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Inflammation: Biomediators, Therapeutic Implications and Resolution Pathways

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Abstract: Inflammation is a biological reaction of tissues to dangerous stimuli, such as pathogens, injury, and chemical irritants, which are necessary for healing, it may become pathological and lead to the development of chronic diseases. It entails an action of a collaborative response of immune cells, blood vessels and biochemical mediators meant to defend the host and restore tissue homeostasis. Synthetic drugs have many side effects that are associated with therapies and lead to the need to seek more natural therapies. The current review presents the information about the biochemical processes of inflammation, the classification of this phenomenon into acute and chronic inflammation, its clinical presentation, implication, causes, and the conventional and non-conventional methods of its treatment. The combination of conventional knowledge and the current scientific validation results in perspectives of creating effective, safe and accessible anti-inflammatory remedies. These processes are important to understand and to develop specific therapies to reduce the inflammatory diseases at the lowest cost.

Keywords: *Inflammation, Acute Inflammation, Chronic Inflammation, Biochemical Mediators, Immune Response, Therapeutic Targets.*

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I. INTRODUCTION

Inflammation is a multifaceted biological reaction to damaging stimuli of body tissues that is based on the Latin term *inflammatio*. It is an immune response of protective mechanisms against such causes, including immune cells, blood vessels, and molecular agents, and the main task of this mechanism is to remove the initial source of cell injury, remove necrotic cells and injured tissue, and to restore the tissue [1]. Inflammation is one of the most basic biological processes that acts as the protective response of the body to negative logical process, inflammation is the defense mechanism of the body to the invasion of infectious agents, entering of antigens, or cell destruction. It is the most common clinical disease symptom, and it is a local reaction of vascularized tissues to endogenous and exogenous stimuli [2]. This reaction is set out to destroy microbes or irritants and facilitate tissue repair. Nonetheless, overproduction or dysregulated inflammation may cause tissue damage, failure in physiological homeostasis, dysfunction of the organs and fatal outcomes. Inflammation is not a disease as such, but it is a desirable process that follows an injury or disease. It developed in the higher organisms as a defense mechanism to localize and remove harmful stimuli and heal. This complex involves alterations in blood circulation, augmented vascular permeability, and movement of fluid, proteins, and leukocytes out of circulation to the site of injury [3]. Acute inflammation plays a key role in tissue repair and immune response, chronic inflammation is now recognized to be a major cause of many pathological disorders such as cardiovascular disease, diabetes, arthritis and cancer [4].

II. RESEARCH GAP

There is a vast disparity in the validation and application of non-pharmacological interventions despite the identified lack of specificity and long-term risks linked with the existing pharmacological anti-inflammatory medications (e.g., NSAIDs and corticosteroids). Although effective, conventional anti-inflammatory therapy, mostly non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids, has been linked with significant adverse effects such as gastrointestinal toxicity, cardiovascular risks, and immunosuppression [5]. Such restrictions have triggered more finding for more safer and sustainable therapeutic options, especially natural ones.

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These restrictions have led to an increased search of safer, more sustainable modalities of therapy, especially those found in nature. Holistic approaches, including nutritional modulation and exercise hold promise of a safer approach to inflammation in chronic and critical inflammation. Nevertheless, their mechanisms of action, effectiveness in different population groups, and best ways to be incorporated in the usual practice care guidelines have not been extensively studied. This gap needs to be covered by rigorous studies and the creation of comprehensive and personalized management strategies.

III. OVERVIEW OF INFLAMMATION

Inflammation is an intricate biological reaction, which consists of various components of cells and molecules, which cooperate to remove the undesirable stimulus and prompt tissue remodeling. Inflammation is a non-specific innate immune reaction, which is different to the adaptive immunity, the one that is directed against specific pathogens [6]. Inflammation is a complicated biological reaction, which consists of various components of cells and molecules, which cooperate to remove the undesirable stimulus and prompt tissue remodeling. The phagocytic cells are recruited locally to eliminate endogenous or foreign matter, whereas the systemic alteration is known to optimize the internal environment to assist the processes. Inflammatory cellular events may be divided based into four phases: (1) a silent phase, where resident tissue cells secrete initial mediators; (2) a vascular phase, when neutrophils raise their levels of vasodilation and increased permeability; (3) a cellular phase, when the neutrophils infiltrate; and (4) phagocytosis, which is a crucial early phase where neutrophils secrete engulf and subsequently destroy invading pathogens [7].

Typically, inflammation is classified as acute or chronic. The immediate and short-term reaction which is characterized by plasma and leucocyte migration into damaged tissues is known as acute inflammation. This response is propagated and matured by biochemical events, which include vascular, immune, and tissue-resident cells [8]. Chronic inflammation refers to a sustained condition; that is, there is a change in the cellular infiltrates to mononuclear cells, and tissue destruction and repair concomitants. Inflammatory biochemical mediators are further divided into pro-inflammatory (e.g., complement components, prostaglandins), inhibitors (e.g., protease inhibitors), and regulators (e.g., tumor necrosis factor, glucocorticosteroids). These molecules mediate the inflammatory cascade caused by various stimuli, including trauma, immune complexes, or bacterial products [9]. One should not confuse inflammation with infection; the first is a process of immunovascular reaction of the body to trauma or stimuli, the second is an indication of microbial pathogens and their activity. Differences are the basis of diagnostic and treatment interventions in clinical practice.

IV. CAUSES OF INFLAMMATION

Inflammation and infection are two different biological term, where the former is immunovascular reaction of the body towards injury-induced or stimuli-induced inflammation and the latter is the presence and activity of microbial pathogens. This difference plays a role in the diagnostic and therapeutic interventions in clinical practice [10]. These triggers may be acute or chronic which leads to inflammation. Chronic inflammation, which can be shown in the case of rheumatoid arthritis, atherosclerosis, and osteoarthritis, may be a consequence of untreated acute inflammation or an unending exposure to irritants.

V. SYMPTOMS OF INFLAMMATION

The typical clinical signs of inflammation also known as the five cardinal signs consist of redness (*rubor*), heat (*calor*), swelling (*tumor*), pain (*dolor*), and loss of function (*functio laesa*). These symptoms are due to a stringent program of vascular, cellular and biochemical events that are used to hold back injury, eliminate pathogens and develop tissue repair [1].

- *Redness (Rubor)*

Vasodilation of small blood vessels in the affected tissues results in redness at the sight of injury. This augmented arterial blood circulation exudes oxygenated blood giving it the characteristic erythematous look. It is mediated by relaxation of smooth muscle cells around arterioles, which is mainly triggered by biochemicals like histamine and prostaglandins [11].

- *Heat (Calor)*

The heat is the result of the increased blood supply (hyperemia) to the inflamed area that is evidence of the elevated activity of metabolism to provide immune responses and cellular restoration. The increase in local temperature can be felt during palpation of the inflamed region and is proportional to the magnitude of a vascular reaction [12].

- *Swelling (Tumor)*

The result of an increase in vascular permeability is swelling as a result of the build-up of fluid in the extravascular extracellular space. This leads to exudation of protein-rich fluid of endothelial barrier which also becomes more permeable to plasma proteins and leukocytes. Mechanical effects of swelling may vary depending on the location, including airway blockage in acute epiglottitis or elevated intracranial pressure in meningitis [13].

- *Pain (Dolor)*

Various processes cause pain which leads to tissue edema, stretching, nociceptors stimulated by inflammatory agents such as bradykinins, prostaglandins, histamine, and serotonin, and direct nerve damage. Pain is a defense mechanism that utilize and restricts additional harm to the tissue in the state of inflammation [14].

- *Loss of Function (Functio Laesa)*

A dysfunction in a swollen region is often a secondary result of pain and swelling, which limits the movement or the functioning of normal tissues. This was a late addition to the classical four of signs that physicians like Galen and Sydenham used to highlight that there were physiological

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effects that the inflammation had in addition to the initial signs [15]. In combination, these clinical manifestations demonstrate the intricate interaction of cellular and molecular mechanisms coordinated by the immune system to fill the tissue integrity after the damage or a virus attack [16].

VI. TYPES OF INFLAMMATION

The broad categorization of inflammation has been into two types depending on the duration of the inflammation as well as the characteristics of the cellular response: acute and chronic inflammation. This classification indicates that there are quite different physiological and pathological processes, and biochemical and immunological processes are different [17].

- *Acute Inflammation*

Acute inflammation is the first defense response, which is a quick reaction of the immune system to the harmful stimuli which may be infection, tissue damage, or microbial invasion. It is marked by exaggerated plasma and leukocytes (mostly granulocytes) egression out of the circulation into the impacted tissues [18]. This is a self-limiting process which takes place within minutes to days and is mediated by vasoactive amines (e.g., histamine), eicosanoid and several cytokines. Vascular reaction during acute inflammation consists of the brief term vasoconstriction and long last vasodilation, raised vascular permeability and release of protein-rich fluid that results in edema. The cellular responses include leukocyte margination, rolling, adhesion, transmigration, chemotaxis, and phagocytosis, which are directed at the elimination of pathogens and debris [19]. The resolution of acute inflammation normally occurs with the normal tissue structure regained but chronic inflammation can arise due to the inability or length of time of inflammation to resolve normally and inflammation may result in scarring of the tissue, an abscess, or a chronic inflammatory response. Examples of clinical manifestations of acute inflammatory responses include diseases like asthma, glomerulonephritis, as well as septic shock [18].

- *Chronic Inflammation*

Chronic inflammation is sustained and lasts months, years and is characterized by tissue damage, acute inflammation and healing with many times fibrosis [20]. In contrast to acute inflammation, mononuclear cells are the major infiltrators of chronic inflammation, which involves the presence of macrophages, lymphocytes, and plasma cells. Chronic inflammation can emerge as a result of unresolved acute inflammation, chronic infections (e.g., *Mycobacterium tuberculosis*), autoimmune responses, or chronic exposure to irritants, like silica or tobacco smoke [21]. It is the basis of the pathogenesis of many chronic diseases, rheumatoid arthritis, inflammatory bowel disease, atherosclerosis, diabetes and certain cancers. Chronic inflammation is a complex interplay of immune cells with sustained pro-inflammatory cytokine (such as interferon-γ (IFN-γ) and interleukin-17 (IL-17)) generation by T-helper subsets (Th1 and Th17) triggered by complex crosstalk, positive feedback loop amplifying inflammation, and tissue remodeling (fibrosis and angiogenesis). The occurrence of epigenetic modifications also leads to persistent inflammatory phenotypes [22].

VII. BIOMEDIATORS OF INFLAMMATION

The most important mediators of inflammatory response are the biomediators, which include cellular mediators as well as biochemical mediators of the initiation, progression, and resolution of inflammatory responses. The role of these biomediators in inflammation is important in understanding the pathogenesis of disease and creating therapeutic and non-therapeutic interventions [23]. The inflammatory reaction is a complex process of interactions of different cell types and soluble factors that may be divided into cell-derived and plasma-derived. These biomediators have redundant actions and they comprise vasoactive amines, lipid mediators, cytokines, and complement components that act in combination to organize the inflammatory [24] response.

- *Cellular Biomediators of Inflammation.*

The initial controllers of inflammation, either generating or down-regulating it, are cellular biomediators. Mast cells are strategic because they are found at tissue-environment interfaces, and when activated, they release histamine, leukotrienes, and cytokines to orchestrate vascular permeability and recruit immune cells. The neutrophils are rapid response inflammatory cells that phagocyte pathogens and release toxic oxygen products prior to apoptosis, whereas the eosinophils attack parasites and play a role in allergic pathology. The central location is held by the monocytes and macrophages which generate cytokines like TNF-α and IL-1, reactive oxygen and nitrogen species, proteolytic enzymes and thus mediates between the innate and adaptive immunity. The vascular endothelial cells mediate leukocyte adhesion and plasma exudation with the help of nitric oxide and expression of adhesion molecules, whereas platelets mediate vasoactive compounds and growth factors that mediate clotting and tissue healing. Other roles of sensory neurons that mediate inflammation include the release of neuropeptides like substance P and Gene-Related Peptide (CGRP) which connects the sensory neuron and inflammatory pathways [25].

- *Cell-Derived Biochemical Mediators*

The main categories of cell derived biochemical mediators are lipid derivatives and signaling molecules which enhance or eliminate inflammation. The metabolism of arachidonic acids produces eicosanoids, such as prostaglandins, thromboxanes, and leukotrienes, which control the vascular tone, chemotaxis, and smooth muscle contraction. The enzymes cyclooxygenase isoforms (COX 1 and COX 2) mediate the production of prostanoids (PGE2) which is central to fever and inflammatory cell activation, whilst the lipoxygenase pathways mediate the production of leukotrienes (which have strong chemotactic and bronchoconstrictive effects). Platelet activating factor (PAF) has powerful effects in low concentrations, which cause vasodilation, permeability and leukocyte recruitment. In contrast, Lipoxins are called pro resolution mediators and they reduce neutrophil migration and increase the uptake of

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apoptotic cells. Collectively, these lipid mediators create an ascending-descending balance to amplify pro inflammatory and resolution processes, which condition the direction of the inflammatory response [26].

➢ *Plasma-Derived Mediators*

Plasma Derived Mediators Inflammatory cascade is prolonged by plasma derived mediators via soluble proteins and peptides. The cytokines and chemokines, IL-1, IL-6, TNF-α, IFN-γ and histamine and serotonin as vasoactive amines control leukocyte trafficking, acute phase protein synthesis, and fever responses, and regulate the vascular permeability and smooth muscle tone, respectively. Bradykinin plays a role in vasodilation, pain and spasmogenicity usually enhanced by prostaglandins. NF-κB is a master transcriptional regulator which stimulates cytokine, adhesion molecule, and enzyme (COX 2 and iNOS) genes. Markers of systemic inflammation, like C reactive protein (CRP), are acute phase proteins, whereas reactive oxygen species (ROS) are antimicrobial agents as well as damaging signals in case of excessive production. Opsonization, chemotaxis, and cell lysis, which are mediated by the complement system, activated either through classical, lectin, or other pathways, are accompanied by regulatory factors that avoid collateral tissue damage (Petrucci *et al.*, 2025). The combination of these plasma mediators with cellular and lipid signals has a role in sustaining, amplifying, and finally resolving inflammation.

VIII. TREATMENT OF INFLAMMATION

The therapeutic process in the management of inflammation is to control the inflammatory reaction to reduce the symptoms, avoid tissue destruction, and induce healing. They involve medications to modulate major biochemical pathways, especially those participating in generating and acting on pro-inflammatory mediators [28]. With its extraordinary biodiversity comprising of various ecological regions, Nigeria has a rich pool of medicinal plants that have been in use in centuries and centuries in the management of inflammatory diseases. The ethnobotanical background of the country covers the presence of more than 7,000 plant species, most of which have strong anti-inflammatory effects [29]. Emerging trends in phytochemical studies as well as molecular biology have started to unravel the scientific foundation of these traditional applications and have shown advanced mechanisms of action that compete with conventional pharmaceuticals. In this way, considering the negative side of synthetic conventionally accessible steroidal or non-steroidal medications [30], the discovery of fresh anti-inflammatory agents through a herbal source is becoming trendy with the aim of achieving a higher level of safety, higher efficacy, and a more cost-effective means of curing inflammation.

➢ *Pharmacological Interventions*

- *Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)*

Drugs that inhibit platelet production or the synthesis of other inflammatory mediators [31] are known as Nonsteroidal Anti-Inflammatory Drugs (NSAIDs). Aspirin and ibuprofen, diclofenac and celecoxib are examples of NSAIDs, which is an inhibitor of the cyclooxygenase (COX) enzymes that catalyze the production of prostaglandins out of arachidonic acid. NSAIDs suppress the production of prostaglandins to reduce the symptoms of inflammation, pain, fever. Nevertheless, they do not, as a rule, stop the progress of underlying chronic inflammatory diseases. Taking NSAIDs over a long period of time is linked to several side effects osteoporosis, inhibition of response to infection or injury, euphoria, cataracts, glaucoma, peptic ulcers and bronchospasm through the inhibition of the physiological and inflammatory prostaglandin and simultaneous release of leukotrienes [32]. The drug is steroidal are known leads to adrenal atrophy [33].

- *Corticosteroids*

Prednisone, dexamethasone and prednisolone are steroidal anti-inflammatory agents, which mimic the endogenous glucocorticoids, with a broad anti-inflammatory and immunosuppressive effect. They suppress several inflammatory processes such as cytokines formation and the migration of leukocytes. In spite of its effectiveness, chronic use has such risks as adrenal atrophy, osteoporosis, an increased risk of infections, and metabolic imbalance [34].

- *Biological Agents and Immunosuppressants.*

Biological therapies interfere with certain immune elements as tumor necrosis factor-alpha (TNF-α), interleukins, and cell surface receptors to suppress the inflammatory pathway. Infliximab, adalimumab, etanercept and rituximab are used to treat autoimmune and chronic inflammatory conditions. Immunosuppressed drugs such as methotrexate and cyclophosphamide suppress the immune system but can be toxic with their dose [35].

➢ *Non-Pharmcological Treatments*

The use of herbal supplements and dietary interventions high in anti-inflammatory phytochemicals to offer safer and adjunctive treatment options with less side effects is a topic of growing interest [36]. Lifestyle modifications (weight control and quitting smoking) and nutritional modulation are also very critical in controlling chronic inflammation. Medical plants are less risky than the traditional NSAIDs and corticosteroids that have the disadvantage of being gastrointestinal toxic, cardiovascular, and immunosuppressive.

➢ *Sources of Natural Anti-inflammatory Agents*

- *Animal Sources*

Several animal-based products have high anti-inflammatory effects. Fish oil is a good source of omega-3 fatty acids (EPA and DHA) which has been shown to have strong anti-inflammatory properties by altering the production of prostaglandins and cytokines. Melittin is a peptide that is an anti-arthritic and anti-inflammatory agent and is found in bee venom [37].

- *Marine Sources*

Marine organisms represent a new source of new anti-inflammatory compounds that is more relevant. Astaxanthin

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is a marine algae derivative, which has outstanding antioxidant and anti-inflammatory effects. Marine sponges generate several bioactive compounds, which have strong anti-inflammatory effects due to phospholipase A2 inhibition, such as monalides and variabilins [38].

- *Plant Sources*

Plants continue to be the most prolific in terms of bioactive compounds which have anti-inflammatory properties. These are known as phytochemicals and they are flavonoids, alkaloids, terpenoids, phenolic compounds and saponins. Plant-based anti-inflammatory agents have several benefits over their synthetic counterparts, including more than one mechanism of action, synergy, and, as a rule, have a positive safety profile [39].

Plants, such as *Zingiber officinale* (ginger), *gingerols* in ginger inhibit COX-2, LOX-5, NF-κB, and MAPK [40], *Curcuma longa* (turmeric), *curcumin* dual COX/LOX inhibits, RANKL, and epigenetic modulation [41], *Azadirachta indica* (neem), azadirachtin and nimbin inhibit T-cell proliferation, IL-6, TNF-α, IL-1β [42], *Vernonia amygdalina* (bitter leaf), vernodalin and caffeoylquinic acids offer antioxidant effects and cytokine modulation [43], and *Garcinia kola* (bitter kola), kolaviron targets respiratory inflammation through multiple mechanisms in reducing inflammatory markers [42]. Others, such as *Allium cepa* (quercetin), *Cymbopogon citratus* (citral), *Syzygium aromaticum* (eugenol), *Nauclea latifolia* (caffeic acid), and *Ocimum gratissimum*, have antirheumatoid arthritis potential through inhibition of intracellular pathways and soluble factor, and more recent research validates antioxidant, analgesic, and antipyretic actions of the *S. occidentalis/V. amygdalina*. These phytocompounds act via different mechanisms: the selective inhibition of COX-2 with reduced gastrointestnal risks, the inhibition of nuclear translocation of NF-κB, the JAK-STAT regulation, the inhibition of ROS (reduction of TNF-α/IL-6 40-60%), and an increase in IL-10, which is safer than NSAIDs (adverse events <5) and has improved multi-target action (pooled SMD -0.75, I2=45) [44].

IX. CONCLUSION

Treatment of inflammation involves a combined effort against the biochemical mediators and immune pathways involved in the pathogenesis of inflammation and must balance between therapeutic and adverse outcomes. Further investigation of new agents and precision medicine will be able to improve the results in inflammatory diseases. Nigerian plants are considered on comparative profile characteristics as low cost, high rural accessibility, and as adjunctive corticosteroid-sparing agents. The knowledge of such mechanisms is necessary to design specific therapeutic interventions that have the potential to control the effects of inflammatory processes without affecting the positive effects of the inflammatory response required to repair tissues and provide defense against host diseases. To incorporate them into healthcare across the globe, future focus will be on the use of RCTs, pharmacokinetic research, drug interactions as well as personalized regimens to utilize the Nigeria biodiversity to develop new phytotherapies.

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REFERENCES

[1]. Y. Geng, Z. Wang, J. Zhou, M. Zhu, J. Liu, and T. D. James, "Recent progress in the development of fluorescent probes for imaging pathological oxidative stress," Chemical Society Reviews, vol. 52, no. 11, pp. 3873-3926, 2023.
[2]. G. Harvanová and S. Duranková, "Inflammatory process: Factors inducing inflammation, forms and manifestations of inflammation, immunological significance of the inflammatory reaction," Alergologia Polska-Polish Journal of Allergology, vol. 12, no. 1, pp. 54-61, 2025.
[3]. M. Lekander, The Inflamed Feeling: The Brain's Role in Immune Defence, Oxford University Press, 2022.
[4]. M. E. Kim and J. S. Lee, "Molecular foundations of inflammatory diseases: insights into inflammation and inflammasomes," Current Issues in Molecular Biology, vol. 46, no. 1, pp. 469-484, 2024.
[5]. S. Akhter et al., "Role of Th17 and IL-17 cytokines on inflammatory and auto-immune diseases," Current Pharmaceutical Design, vol. 29, no. 26, pp. 2078-2090, 2023.
[6]. M. A. Chowdhury, N. Hossain, M. A. Kashem, M. A. Shahid, and A. Alam, "Immune response in COVID-19: A review," Journal of Infection and Public Health, vol. 13, no. 11, pp. 1619-1629, 2020.C. Malainou, S. M. Abdin, N. Lachmann, U. Matt, and S. Herold, "Alveolar macrophages in tissue homeostasis, inflammation, and infection: evolving concepts of therapeutic targeting," The Journal of Clinical Investigation, vol. 133, no. 19, 2023.
[7]. D. Chopra et al., "Overview of inflammation," in Inflammation Resolution and Chronic Diseases, Singapore: Springer Nature Singapore, 2024, pp. 1-18.
[8]. V. J. Clemente-Suárez et al., "New insights and potential therapeutic interventions in metabolic diseases," International Journal of Molecular Sciences, vol. 24, no. 13, p. 10672, 2023.
[9]. L. L. Alex and J. P. Michael, "Endothelium and smooth muscle interactions in hypertension," in Blood Vessel Changes in Hypertension Structure and Function, Volume II, CRC Press, 2024, pp. 43-70.
[10]. D. S. Goldstein, "The extended autonomic system, dyshomeostasis, and COVID-19," Clinical Autonomic Research, vol. 30, no. 4, pp. 299-315, 2020.
[11]. T. Iba, J. Helms, M. Levi, and J. H. Levy, "Inflammation, coagulation, and cellular injury in heat-induced shock," Inflammation Research, vol. 72, no. 3, pp. 463-473, 2023.
[12]. B. Saravi et al., "Capillary leak and endothelial permeability in critically ill patients: a current

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/26jan1249

overview," Intensive Care Medicine Experimental, vol. 11, no. 1, p. 96, 2023.
[13]. G. I. Lee and M. W. Neumeister, "Pain: pathways and physiology," Clinics in Plastic Surgery, vol. 47, no. 2, pp. 173-180, 2020.
[14]. T. Pascart and E. Drouin, "Gout: the deeply rooted image of the self-inflicted disease of kings comes from misinterpreting history," The Lancet Rheumatology, vol. 7, no. 6, pp. e388-e390, 2025.
[15]. M. S. Merakeb, N. Bribi, R. Ferhat, and B. Yanat, "Inhibitory effects of linum usitatissimum alkaloid on inflammatory and nociceptive responses in mice," Current Bioactive Compounds, vol. 19, no. 2, pp. 56-62, 2023.
[16]. N. Zotova, Y. Zhuravleva, V. Chereshnev, and E. Gusev, "Acute and chronic systemic inflammation: features and differences in the pathogenesis, and integral criteria for verification and differentiation," International Journal of Molecular Sciences, vol. 24, no. 2, p. 1144, 2023
[17]. B. Katt and R. Coyle, "Thermal burns, chemical burns, and frostbite," in Review of Hand Surgery, E-Book, 2021, pp. 76.
[18]. S. Hannoodee and D. N. Nasuruddin, "Acute inflammatory response," in StatPearls [Internet], StatPearls Publishing, 2024.
[19]. K. S. Alharbi, S. K. Alenezi, and G. Gupta, "Pathophysiology and pathogenesis of inflammation," in Recent Developments in Anti-Inflammatory Therapy, Academic Press, 2023, pp. 1-9.
[20]. M. Pahwa, A. Cavanagh, and M. Vanstone, "Key informants in applied qualitative health research," Qualitative Health Research, vol. 33, no. 14, pp. 1251-1261, 2023.
[21]. C. L. R. Soares et al., "Biochemical aspects of the inflammatory process: A narrative review," Biomedicine & Pharmacotherapy, vol. 168, p. 115764, 2023.
[22]. C. Puricelli et al., "Platelets, protean cells with all-around functions and multifaceted pharmacological applications," International Journal of Molecular Sciences, vol. 24, no. 5, p. 4565, 2023.
[23]. C. Luo et al., "A review of the anti-inflammatory effects of rosmarinic acid on inflammatory diseases," Frontiers in Pharmacology, vol. 11, p. 153, 2020.
[24]. J. L. Harwood, "Polyunsaturated fatty acids: conversion to lipid mediators, roles in inflammatory diseases and dietary sources," International Journal of Molecular Sciences, vol. 24, no. 10, p. 8838, 2023.
[25]. G. Petrucci, M. A. Farhoud, and A. Habib, "Platelet thromboxane A2 and prostaglandin receptors," in Platelet Physiology I: Volume 1, Cham: Springer Nature Switzerland, 2025, pp. 291-302.
[26]. H. Zhao et al., "Inflammation and tumor progression: signaling pathways and targeted intervention," Signal Transduction and Targeted Therapy, vol. 6, no. 1, p. 263, 2021.
[27]. V. L. Petricevich and R. Abarca-Vargas, "Allamanda cathartica: a review of the phytochemistry, pharmacology, toxicology, and biotechnology," Molecules, vol. 24, no. 7, p. 1238, 2019.
[28]. I. Attaf and A. E. Cherbal, "The mechanistic aspects of the anti-inflammatory activity of Lavandula stoechas," Ph.D. dissertation, Université de jijel, Algeria, 2021.
[29]. Y. Y. Zhong, H. Wang, and Y. Y. Wang, "Nature's pain relievers: Unveiling the power of plant extracts against non-steroidal antiinflammatory drugs," Next Research, p. 100685, 2025.
[30]. S. Fareed, "Exploring the role of local glucocorticoid metabolism in osteoblasts during chronic inflammation," Ph.D. dissertation, University of Birmingham, UK, 2023.
[31]. J. H. Stone et al., "Inebilizumab for treatment of IgG4-related disease," New England Journal of Medicine, vol. 392, no. 12, pp. 1168-1177, 2025.
[32]. A. Nisar et al., "Phytochemicals in the treatment of inflammation-associated diseases: the journey from preclinical trials to clinical practice," Frontiers in Pharmacology, vol. 14, p. 1177050, 2023.
[33]. M. Mengistu, M. Kebebew, and V. B. Meyer-Rochow, "Ethnozoological study of medicinal animals used by the inhabitants of the Kucha District, Gamo Zone, Southern Ethiopia," Journal of Ethnobiology and Ethnomedicine, vol. 20, no. 1, p. 72, 2024.
[34]. D. Placha and J. Jampilek, "Chronic inflammatory diseases, anti-inflammatory agents and their delivery nanosystems," Pharmaceutics, vol. 13, no. 1, p. 64, 2021.
[35]. N. C. Uche-Okereafor, "Studies on Solanum Mauritianum Scopoli bacterial endophytes and the effects of their crude secondary metabolites on common pathogenic bacteria and resistant human cancer cells," Ph.D. dissertation, University of Johannesburg, South Africa, 2020.
[36]. U. O. Arunsi, O. E. Chioma, P. E. Etusim, and S. E. Owumi, "Indigenous Nigeria medicinal herbal remedies: A potential source for therapeutic against rheumatoid arthritis," Experimental Biology and Medicine, vol. 247, no. 13, pp. 1148-1178, 2022.
[37]. D. Jain et al., "Exploring synergistic benefits and clinical efficacy of turmeric in management of inflammatory and chronic diseases: A traditional Chinese medicine based review," Pharmacological Research-Modern Chinese Medicine, p. 100572, 2025.
[38]. M. F. Olaniyan, O. D. Olaniyi, F. Odegbemi, T. B. Olaniyan, and O. B. Odegbemi, "Isolation and purification techniques for bioactive compounds from Nigerian medicinal plants and their therapeutic applications," Discover Chemistry, vol. 2, no. 1, p. 13, 2025.
[39]. E. C. Pham, V. Van Doan, T. V. Le Thi, C. Van Ngo, and L. V. Van, "In vivo and in silico antihypertensive, anti-inflammatory, and analgesic activities of Vernonia amygdalina Del. leaf extracts," Heliyon, vol. 10, no. 19, 2024.
[40]. S. Fakhri et al., "Modulation of TLR/NF-κB/NLRP signaling by bioactive phytocompounds: A promising strategy to augment cancer chemotherapy and immunotherapy," Frontiers in Oncology, vol. 12, p. 834072, 2022.