

A Mechanistic Insight on Pathophysiological Mechanisms of Inflammatory Diseases and Potential Therapeutic Targets

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Abstract

Inflammation is a response initiated by a damaged or living tissue in defense of the infection and injury. Moreover, it localizes and eliminates the injurious agent for proper healing. Inflammatory response initiates transfer of fluid, proteins, vascular changes, white blood cells, blood flow, and an enhancement in permeability of blood vessels at the site of damage

Keywords: cognitive functioning: traumatic brain injury (TBI)

Introduction

Inflammation is a response initiated by a damaged or living tissue in defense of the infection and injury. Moreover, it localizes and eliminates the injurious agent for proper healing. Inflammatory response initiates transfer of fluid, proteins, vascular changes, white blood cells, blood flow, and an enhancement in permeability of blood vessels at the site of

damage. [1] An improper inflammatory response initiates tissue destruction, and further the process of clearance of damaged tissue and foreign substances. [2] In these circumstances, the inflammatory responses impair the beginning (Figure. 1). Environmental agents, (pollen) include allergic, hypersensitivity, reactions, stimulate inflammation, and autoimmune reactions. [2] Later on, the body's immune response transforms initial signs into chronic inflammation in contrast to its tissues.

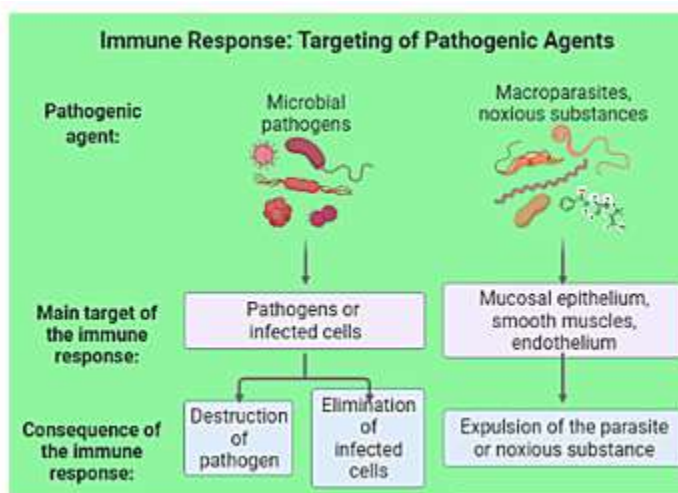


Figure 1: An illustration of immune response_ targeting of pathogenic agents.

The physical and chemical mediators, acids, alkalis, oxidizing agents, infectious agents, microorganisms, unfitting immunological responses, and tissue death are identified as key factors as the stimuli of inflammation. [4] The physiology of inflammation has two key processes, one is vascular changes that consist of various steps, including exudate, clotting factors, antibodies, axial stream. Next is cellular change covering most of the vital constituents, including phagocytes, leukocytes having granules of cell-destroying enzymes and proteins, and mechanism of connected routes and paths? Defused chemical elements create a concentration gradient and neutrophils follow it. [5] These gradients are

defined as chemotactic factors and when it moves only in one direction are renamed as chemotaxis. Cell-eating macrophages are existent at the site of injury and are reflected as a cellular hallmark of chronic inflammation. [6,7] White blood cells (basophils, macrophages, neutrophils, and monocytes), endothelial cells, platelets, and mast cells, created primarily from blood plasma, lining the blood vessels, and finally, damaged tissue cells (Figure. 2). Histamine is known as a chemical mediator of inflammation and is considered as a key element of inflammation that triggers vasodilation and enhances vascular permeability as well.

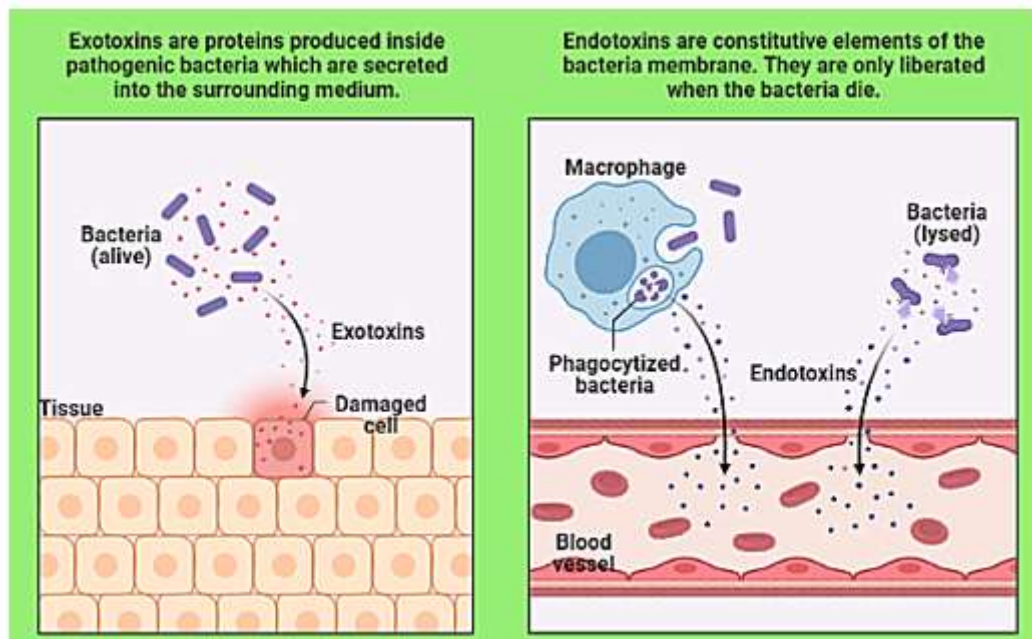


Figure 2: A comparative illustration of the routes of Exotoxins vs Endotoxins.

Even lysosomal promotes vascular permeability. Inflammatory molecules like cytokines also display vasoactive and chemotactic properties. The association of the immune system and inflammatory processes join in the initiation and propagation of several disorders, even failure of these systems can initiate mental and physical health problems. [8] These inflammatory edges are considered the most significant cause for enhancing morbidity and mortality worldwide, more than 50% of all deaths being attributable to the diseases that originated because of inflammation. [9] Moreover, inflammation is one of the key players of healing, but its chronic form promotes the initiation and propagation of the diseases, even though it can induce complications in physiology that can transform into a fatal disease. [10] Therefore, inflammation generate the causes that initiate various diseases and various inflammatory induced physiological complications, including metabolic syndrome, neurodegenerative, multiple autoimmune syndromes, ischemic heart disease, stroke, allergic, asthma, Alzheimer disease, chronic kidney disease, inflammatory bowel disease, Crohn's Diseases, and associated infections, along with a specific emphasis on the corona infections are elaborated. [11] The infection turns into a more complicated situation that persists around the location and associated pathology of the diseases that are induced by inflammation. Therefore, infection and infectious disease are some of the main causes or vice versa of mortality, and morbidity worldwide. [12] New pathogens are continuously emerging. Besides, the identified infectious diseases are re-emerging, and its most complicated aspects are their increase of microbial resistance toward antimicrobial agents. Environmental factors induce new causes that are generating immunosuppressed generations. Thus, the recognition of the type of

infection or infectious diseases at the location of the inflammatory induced diseases is crucial in exposing the pathology of these diseases. For example, inflammatory diseases, including fibromyalgia, sarcoidosis, lupus, rheumatoid, and arthritis can involve the lungs. These conditions trigger inflammation and initially upset joints and muscles. Lung infection and inflammation induce disease of the lung, causing infection. It is a tough task to differentiate their pathology and therefore, in these situations, especially immunosuppressant's, which enhance the chances of infections are the best marked therapeutic target for treating such types of inflammatory surroundings. [13] The key features of the mechanistic Insight on Inflammation, pathophysiological mechanisms, and pathology of the above-mentioned diseases, as well as potential therapeutic targets, are presented. [14] Author pointed out the importance of the mechanistic insight on pathophysiological mechanisms of inflammatory diseases for search out the potential therapeutic targets. These strategies can be fruitful in developing novel remedies for such kind of diseases and disorders.

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Availability of data and materials

Wherever necessary, relevant citations are included in the reference section.

Competing interests

The author has declared that no competing interest exists.

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