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SHORT COMMUNICATION

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Host Defense Mechanisms and Tissue Tropism in Emerging Infectious Diseases

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About the Study

Emerging Infectious Diseases (EIDs) are infections that are either completely new to a community or that have always existed but are spreading quickly both geographically and in terms of frequency. EIDs can be caused by a variety of pathogens, including viruses, bacteria, fungi, and parasites. Each type of pathogen has unique biological characteristics that influence its pathogenicity and interaction with the host. Viruses are obligatory intracellular pathogens that reproduce by taking over the host's biological processes. They can cause direct cytopathic effects, such as cell lysis, or cause apoptosis. Some viruses integrate into the host genome, potentially causing oncogenesis or persistent infections [1]. Viral mutations, especially in RNA viruses, can lead to increased virulence, immune evasion, and resistance to antiviral treatments.

Bacteria may be extracellular or intracellular. Pathogenic bacteria often produce toxins (endotoxins or exotoxins) that cause tissue damage and inflammation. They can also avoid the host immune system through mechanisms like antigenic variation, secretion systems that interfere with host cell functions which protect them from immune responses and antibiotics [2]. Pathogenic fungi can be adaptive or primary pathogens. They can cause direct tissue invasion and destruction, and produce toxins. Granulomatous inflammation is a common symptom of fungal infections, which can progress to chronicity because the pathogen is hard to remove entirely. Parasitic infections can cause a wide range of pathological effects, from direct tissue damage to chronic inflammation. They often have complex life cycles and can modify host immune responses to ensure their survival [3].

The interaction between a pathogen and the host is a dynamic process that determines the severity and outcome of the infection. This interaction involves the pathogen's virulence factors and the host's defense mechanisms [4]. Virulence factors are molecules produced by pathogens that enhance their ability to cause disease. They include adhesins, enzymes that degrade host tissues, and toxins. The ability of a pathogen to avoid or reduce the host immune response is also an important virulence factor. For instance, some pathogens produce proteins that inhibit complement activation or phagocytosis [5].

The initial line of protection is the innate immune system, which consists of soluble components, cellular defenses and physical barriers. Immune cells Pattern Recognition Receptors (PRRs) identify pathogens, which causes the activation of inflammatory pathways [6]. While severe or uncontrolled inflammation can cause tissue damage, it is necessary for the treatment of infections. T and B cell activation is one of the antigen-specific mechanisms involved in adaptive immune response. T cells can directly kill infected cells or assist in coordinating the immunological response, while B cells produce antibodies that neutralize pathogens. Immunopathology can occur if the adaptive response is too vigorous, leading to autoimmune reactions, or if it is insufficient, allowing persistent infection [7].

Acute inflammation is a common sign of many infections and is characterized by redness, swelling, heat, and pain. It results from the influx of immune cells and the release of pro-inflammatory cytokines and chemokines. While inflammation is necessary to control infection, it can also lead to collateral damage to host tissues. Some infections result in chronic inflammation, which can cause tissue damage and fibrosis [8]. This is often seen in infections that are not fully cleared by the immune system, leading to a state of persistent immune activation. Direct damage

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to host cells by the pathogen can result in cell death and tissue necrosis. Infections caused by viruses frequently result in lysis or death of the affected cells [9].

In some cases, the immune response itself can cause significant tissue damage. Chronic infections, especially those caused by intracellular pathogens like certain bacteria and fungi, can lead to the formation of granulomas. These are organized collections of immune cells that try to secure the infection, but they can also impair normal tissue function [10]. Severe infections can lead to sepsis, a life-threatening systemic inflammatory response. Septic shock, a subset of sepsis, involves severe hypotension and multiple organ dysfunctions. The pathogenesis of sepsis includes widespread endothelial dysfunction, dysregulated immune responses, and coagulopathy. Some pathogens can establish latent or persistent infections, evading the immune system and causing long-term pathology.

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