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Infection and Tissue Damage: Reactive Oxygen Species

Stephen Jones*

Department of Cancer, Sidney Kimmel Comprehensive Cancer Center, Baltimore, Maryland

Editorial

Inflammation is a protective immunological response that the host produces in response to invading infections. This survival strategy trait has developed in animals' immune systems to aid tissue healing. Invertebrates have two immune systems that identify and eradicate pathogens: the innate immune system and the adaptive immune system. When pathogens are encountered, the innate immune system produces an initial inflammatory response that includes systemic vasodilation, vascular leakage, and leukocyte emigration.

While this is beneficial to the organism, it may also lead to inflammation and illness if left unchecked. The four cardinal indications of localised acute inflammation, as recorded by the Roman physician Celsus about 2000 years ago, are as follows: Calor heat, Rubor redness, tumour swelling, and Dolor pain all contribute to Functiolaesa function loss (or impairment). Pattern-recognition receptors, which are germline-encoded, let the innate immune system detect a wide range of pathogens, including viruses, bacteria, and fungus.

Membrane-bound receptors such as Toll-like receptors and c-type lectin receptors, as well as cytoplasmic nod-like receptors, are all members of the PRR family. These receptors identify pathogen-associated chemical patterns such as flagellin, carbohydrates, and different microbe cell wall components such as peptidoglycan and lipopolysaccharide, as well as danger-associated molecular patterns such as mammalian dsDNA and uric acid crystals generated by wounded cells.

PRRs are expressed by a wide range of immune cells, including macrophages, monocytes, dendritic cells, and neutrophils, allowing for early pathogen identification. Immune cells initiate an acute inflammatory response shortly after the innate immune system is activated, allowing the production of different cytokines and chemokines to attract immune cells to the infection site. Neutrophils are the first cells that bind to endothelial cells and move through the vascular wall at the site of infection to ingest invading pathogens and emit vasoactive and pro-inflammatory mediators.

Inflammatory mediators generated by inflammatory cells at the site of damage are responsible for the majority of the early vascular alterations seen in acute inflammation. Histamine, platelet-activating factors, bradykinin, and thrombin are among the mediators that enhance vascular permeability, resulting in fluid build-up and leukocyte extravasation. Bacterial or viral infection, tissue necrosis, trauma, radiation, burns, or any foreign body present in tissue can all produce acute inflammation. When the innate immune system's capacity is exceeded or its protective function is compromised, it activates the adaptive immune system, which activates specialised T and B cells to eliminate pathogens.

If this process is protracted or ineffective, it leads to a chronic state of inflammation, which is linked to a variety of ailments, including heart disease and rheumatoid arthritis. Chronic inflammation has also been linked to TB, ARDS, autoimmune illnesses, inflammatory bowel disease, atherosclerosis, and neurodegenerative and metabolic hormonal problems.

Many inflammatory disorders proceed because of the production of reactive oxygen species. ROS are created by cells engaged in the host defence response, such as polymorphonuclear neutrophils, and they cause endothelial dysfunction by oxidising important cellular signalling proteins like tyrosine phosphatases. ROS function as both a signalling molecule and an inflammatory mediator. Superoxide and other reactive oxygen species (ROS) may interact with NO at a diffusion-limited pace to create reactive nitrogen species like peroxynitrite, which is three to four times quicker than superoxide dismutation by superoxide dismutase.

***Corresponding author:** Stephen Jones, Department of Cancer, Sidney Kimmel Comprehensive Cancer Center, Baltimore, Maryland, E-mail: jones_s@ccnt.usc.edu

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