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Guest Editorial

Acute Inflammation in Tissue Repair and Regeneration and Chronic Inflammation in Cell Injury and Tissue Damage

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Inflammation is an injury to the tissue by noxious stimuli release various inflammatory mediators by inflammatory cells. Acute inflammation due to injury, microbial agents, chemical agents, and chronic psychological stress induced release of inflammatory mediators such as cytokines, chemokines, growth factors, and proteolytic enzymes from inflammatory cells such as neutrophils, monocytes, dendritic cells, macrophages. Chemokines, which are produced from immune cells helps in recruitment of inflammatory cells to the site of inflammation. IL-2, IL-12, IFN-δ anti-inflammatory, immunostimulatory cytokines produced by macrophages, dendritic cells, NK cells. Predominant acute inflammatory cell in acute inflammatory microenvironment is neutrophils release ROS, RNS, TGF-β, IL-1, TNF-α involved in antibacterial activity, cell proliferation, cellular development, cellular maturation, deposition of fibroblasts synthesize collagen helps in cell repair and regeneration by activating NF-KB, a key ubiquitous transcription factor involved in transcription of inflammatory mediators.

In chronic inflammation, if the acute inflammation is aggravated chronically persistent, progressive inflammation can cause various cellular and tissue changes. Chronic inflammation induced release of inflammatory mediators such as cytokines, growth factors, and proteolytic enzymes from chronic inflammatory cells such as macrophages, mast cells, T cells and B cells involved in various changes.

Tregulatory cells (Tregs) are formed from Th1 cells producing IL-10 mediated by TGF-β involve in immune modulation. Bregs (Bregulatory cells) are B cells producing IL-10 mediated by TGF-β involved in immune modulation.

Macrophages are the predominant cells in chronic inflammatory microenvironment. Macrophages release various inflammatory mediators such as cytokines (IL-1, TNF-α, IL-6), free radicals (RNS, ROS), growth factors (EGF, FGF, VEGF, PDGF), enzymes (COX-2, Mmp's2, 9, UPA) activate NF-KB, a key ubiquitous transcription factor and STAT-3 transcription factors work together in inflammatory microenvironment. Dysregulated, constitutive activation of NF-KB, a key transcription factor mediated transcription of inflammatory mediators such as IL-3, IL-4, Il-13, IL-17, TGF-β involved in oxidative stress induced tissue damage and immune modulation, cell proliferation (cyclin D, E), cell survival (BCL-2, BCl-XL), fibrosis (TGF-β, FGF), angiogenesis (VEGF, COX-2), genomic instability (ROS, RNS, AID) leads to various diseases such as infectious diseases, heart diseases, cancer, autoimmune diseases, neurodegenerative diseases and metabolic diseases.

Inflammation is a basis of many diseases. Chronic inflammation is considered as a seventh hall mark of cancer. Thorough understanding of inflammatory mediators in pathophysiology of diseases helps in early detection of biomarkers, therapeutic target and prognostic markers for diseases for better futuristic precision management of diseases.

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