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Guest Editorial

Chronic Psychological Stress-Inflammation: Oxidative Stress: Immunity: Autoimmunity: Cancer

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Abstract

Chronic psychological stress induced release of stress releasing factors activate inflammatory mediators in turn activation of transcription factors mediated inflammatory mediators leads to cellular, vascular, and immunological changes results in various diseases such as autoimmune disease and cancer. This article highlights about the role of psychological stress induced inflammatory mediators results in autoimmune disease and cancer.

Keywords: *Chronic Psychological Stress; NF-KB; IL-1; TNF-α; IL-6; TGF-β*

Chronic psychological stress on inflammation, immunity, oxidative stress, autoimmunity, and cancer

Chronic psychological stress induced release of CRH from hypothalamus activate release of stress releasing factors such as cortisol, ACTH, adrenalin, and noradrenalin, through HPA-axis mediated sympathetic nervous system of ANS activate inflammatory mediators such as IL-1, TNF-α, IL-6, EGF from inflammatory cells such as neutrophils, macrophages, mast cells, which will activate NF-KB, a key transcription factor. If the NF-KB, a key transcription factor constitutively, abnormally activated in chronic smoldering inflammatory microenvironment results in cellular and vascular changes. In chronic inflammatory microenvironment, the abnormally activated ubiquitous NF-KB, key transcription factor results in release of inflammatory mediators involved in cell proliferation (cyclin D, E), cell survival (BCL-2, BCL-XL, survivin), angiogenesis (EGF, FGF, VEGF, PDGF), chronic inflammation (IL-1, TNF-α, IL-6), genomic instability (ROS, RNS, AID), immune modulation (IL-4, IL-10, IL-5, IL-13, IL-14, IL-15, IL-17, TGF-β), invasion and metastasis (UPa, Mmp's) involved in tumor initiation, tumor promotion, and tumor progression.

In chronic inflammatory microenvironment constitutive activation of NF-KB, a key ubiquitous transcription factor results in transcription of inflammatory mediators involved in conversion of TH1 to TH2 lymphocytic type release IL-4, IL-5, IL-13, IL-15, IL-17 pro-inflammatory cytokines mainly involved in cell injury, tissue damage, and immune modulation. Growth factors such as EGF, FGF, VEGF, PDGF and IL-8, HIF-1α involved in angiogenesis, cell proliferation, and cell survival by activating STAT-3 transcription factor. Aitregs (Altered induced T regulatory cells) involved in immune modulation by releasing IL-10 mediated by TGF-β. Proteolytic enzymes (UPa, Mmp's) involved in extracellular matrix degradation induced tissue injury leading to autoimmune diseases. NF-KB, a key ubiquitous transcription factor located in cytoplasm of every cell in an inactive state, where it is activated cytoplasmic NF-KB transcription factor binding to nuclear DNA

results in transcription of inflammatory mediators. NF-KB, a transcription factor involved in immune cells proliferation, immune cells maturation, and immune cells development involved in innate and adaptive immunity.

Oxidative stress induced by inflammatory mediators such as IL-1, TNF-α, IL-8 released by neutrophils, macrophages, and mast cells mediated by NF-KB transcription factor involved in release of free radicals such as ROS, RNS, NO, H_2O_2 involved in cell injury, tissue damage, gene mutation, immunomodulation, cell aging, and cell death.

Conclusion and Future Perspective

Thorough understanding of chronic psychological stress on inflammation, immunity, oxidative stress and in diseases such as autoimmune diseases, cancer helps in preventive and therapeutic effective management of diseases.

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