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Short Communication

Chronic Psychological Stress Mediated Immune Modulation Induced Autoimmune Diseases

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Abstract

chronic psychological Stress is a main etiological factor for autoimmune diseases by releasing neuropeptides activates inflammatory mediators such as IL-1,TNF-α,IL-6, and COX-2 , proinflammatory cytokines induce activation of NF-KB a key transcription factor, which activate inflammatory mediators involved in chronic inflammation , immune modulation, cell proliferation, cell survival, angiogenesis involved in autoimmune diseases . This article briefs about the role of chronic psychological stress mediated immune modulation induced autoimmunity diseases.

Keywords: *HPA-axis; Autoimmunity; Neuropeptides; Cortisol; NF-KB; Tregs*

Chronic psychological stress induced immunomodulation leads to autoimmune diseases

Chronic psychological stress, anger, hatred, depression, frustration induced release of CRH (Corticotropin releasing hormone) from hypothalamus activate HPA-axis through ANS release stress releasing neurohormones such as cortisol, noradrenaline, and ACTH. These neurohormones activates inflammatory mediators such as IL-1β,TNF-α, IL-6 and COX-2 ,which activates NF-KB and STAT-3 key transcription factors, which further activate inflammatory mediators involved in chronic inflammation (IL-1β,TNF-α), immune modulation (IL-10,TGF-β, iNOS), tissue damage (MMP's2,9,ROS,RNS), cell proliferation (Cyclin D,E), angiogenesis (IL-8,COX-2,VEGF), cell survival (BCL-XL,BCL-2) [1-10].

NF-KB a key transcription factor induced expression of inflammatory mediators such as chemokines, cytokines, growth factors, and proteolytic enzymes involved in conversion of TH1 lymphocytic type to TH2 lymphocytic type mediated by IL-4,STAT6 transcription factor release IL-4,IL-13,IL-5 proinflammatory cytokines along with TH17 cells involved in chronic inflammation, immune modulation, and tissue damage. IL-1, COX-2, and TNF-α pro-inflammatory cytokines activate NF-KB a key transcription factor, IL-6, IL-10,EGF, FGF activate STAT-3 transcription factor, both transcription factors work together involved in cell proliferation by expression of cyclin D,E cell cycle regulatory proteins and cell survival by BCL-2, BCL-XL anti-apoptotic proteins.

Growth factors such as EGF, FGF, VEGF involved in cell proliferation, cell survival, and angiogenesis by activation of STAT-3 transcription factor. Altered induced regulatory T cells (iTregs) formed from TH1 cells mediated by TGF-β inflammatory mediator release IL-4,IL-2,IL-10IL-17,IL-13,IL-5, pro-inflammatory cytokines involved in immune modulation by inhibiting innate and adaptive immune cells (involved in decreased mitogenic response in lymphocytes, natural killer cell cytotoxicity is decreased , IgA secretion) ,otherwise normal regulatory T cells (nTregs) involved in self tolerance and immune homeostasis. Proteolytic enzymes such as matrix metallo proteinases 2,9 (Mmp's2,9), Urokinase plasminogen activator (UPA) involved in tissue damage, all these changes leads to autoimmune diseases [10-19].

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