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Poster Presentation

Key Role of Inflammation in Central Nervous System Damage and Disease; TNFα, IL-1

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Abstract

Inflammation is portion of the body's immune response and it is basically a host protective response to tissue ischemia, injury, autoimmune responses or infectious agents. Although the information presented so far points to a detrimental role for inflammation in central nervous system (CNS) disease, it may also be useful. CNS demonstrates characteristic of inflammation, and in response to damage, disease or infection, resident CNS cells generate inflammatory mediators, including prostaglandins (PGs), pro-inflammatory cytokines, free radicals and complement, which in turn induce chemokines and adhesion molecules, recruit immune cells, and activate glial cells. In response to a brain injury, as trocytes become activated, increasing expression of glial fibrillary acidic protein, and producing cytokines. Cytokines including both tumor necrosis factor-α (TNF-α) and IL-1 are strongly implicated in neuronal loss during acute and chronic neurodegenerative disease, but also participate in repair and recovery. Although TNF-α is found associated with active MS lesions, induces death of oligodendrocytes. TNF-α appears not to be needed for mast cell–dependent pelvic pain. TNF-α, is released from Schwann cells immediately after nerve damage. IL-1 can also attach nerve terminals and influence substance P release and migration of polymorphonuclear White blood cells (WBCs). IL-1β is also selectively upregulated in astrocytes in the spinal trigeminal nucleus, spinal cord and rostral ventromedial medulla in models of inflammation, cancer pain and nerve damage. IL-1β is an important messenger between neurons and glia.

Keywords: CNS Damage, Inflammation, Cytokine, Interleukin

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