Inflammatory processes in brain injury

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Inflammatory processes in the brain have been implicated in both acute and chronic neurodegenerative disease. Key inflammatory mediators such as complement, adhesion molecules and cytokines are increased in experimental paradigms of brain injury and many studies report similar changes in the clinical setting. Many of these factors are up-regulated rapidly after an experimental insult, before significant cell death is observed, and intervention studies further support the idea that these compounds contribute directly to the neuronal injury. One of the most studied cytokines in this respect is interleukin-1 (IL-1), which has diverse actions in the central nervous system (CNS) and mediates a wide variety of effects, including the host defense responses to local and systemic disease and injury. IL-1 has also been implicated heavily in acute neurodegenerative diseases such as stroke and head injury as well as more chronic ones, including epilepsy and Alzheimer's. The evidence is derived largely from experimental studies that report increases in IL-1 expression after injury and protective effects of the IL-1 receptor antagonist (IL-1ra). Exactly how IL-1 produces its effects in these conditions is not clear but it appears to involve multiple effects on neuronal, glial and endothelial cells.
Understanding cytokine action in neurodegeneration could lead to novel and effective therapeutic strategies, some of which are already in clinical trials.

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