Neuroinflammation in demyelinating diseases: Oxidative stress as a modulator of glial cross-talk

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Myelin is a specialized membrane allowing for saltatory conduction of action potentials in neurons, an essential process to achieve the normal communication across the nervous system. Accordingly, in diseases characterized by the loss of myelin and myelin forming cells-oligodendrocytes in the CNS-, patients show severe neurological disabilities. After a demyelinated insult, microglia, astrocytes and oligodendrocyte precursor cells invade the lesioned area initiating a spontaneous process of myelin repair (i.e. remyelination). A preserved hallmark of this neuroinflammatory scenario is a local increase of oxidative stress, where several cytokines and chemokines are released by glial and other cells. This generates an environment that determines cell interaction resulting in oligodendrocyte maturity and the ability to synthesize new myelin. Herein we review the main features of the regulatory aspect of these molecules based on recent findings and propose new putative signal molecules involved in the remyelination process, focused in the etiology of Multiple Sclerosis, one of the main demyelinating diseases causing disabilities in the population. © 2019 Bentham Science Publishers.

Glial cross-talk

Microglia

Multiple Sclerosis

Neuroinflammation

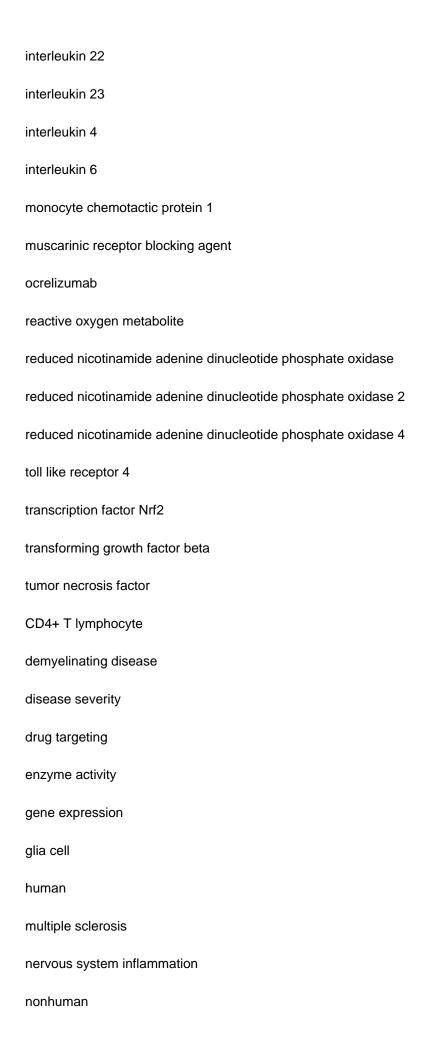
Oxidative stress

Remyelination

immunoglobulin enhancer binding protein

interleukin 17

interleukin 1beta



oligodendrocyte precursor cell
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