

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

Varas R.

Ortiz F.C.

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

interleukin 22

interleukin 23

interleukin 4

interleukin 6

monocyte chemotactic protein 1

muscarinic receptor blocking agent

ocrelizumab

reactive oxygen metabolite

reduced nicotinamide adenine dinucleotide phosphate oxidase

reduced nicotinamide adenine dinucleotide phosphate oxidase 2

reduced nicotinamide adenine dinucleotide phosphate oxidase 4

toll like receptor 4

transcription factor Nrf2

transforming growth factor beta

tumor necrosis factor

CD4+ T lymphocyte

demyelinating disease

disease severity

drug targeting

enzyme activity

gene expression

glia cell

human

multiple sclerosis

nervous system inflammation

nonhuman

oligodendrocyte precursor cell

oxidative stress

priority journal

remyelination

Review

Th17 cell

cell communication

cytology

demyelinating disease

glia

inflammation

multiple sclerosis

myelin sheath

oligodendroglia

pathology

pathophysiology

Cell Communication

Demyelinating Diseases

Humans

Inflammation

Multiple Sclerosis

Myelin Sheath

Neuroglia

Oligodendroglia

Oxidative Stress