MULTIPLE SCLEROSIS (MS): balancing the immune system

FX Medicine

MS is a chronic neuroinflammatory disease of the brain and spinal cord[1]

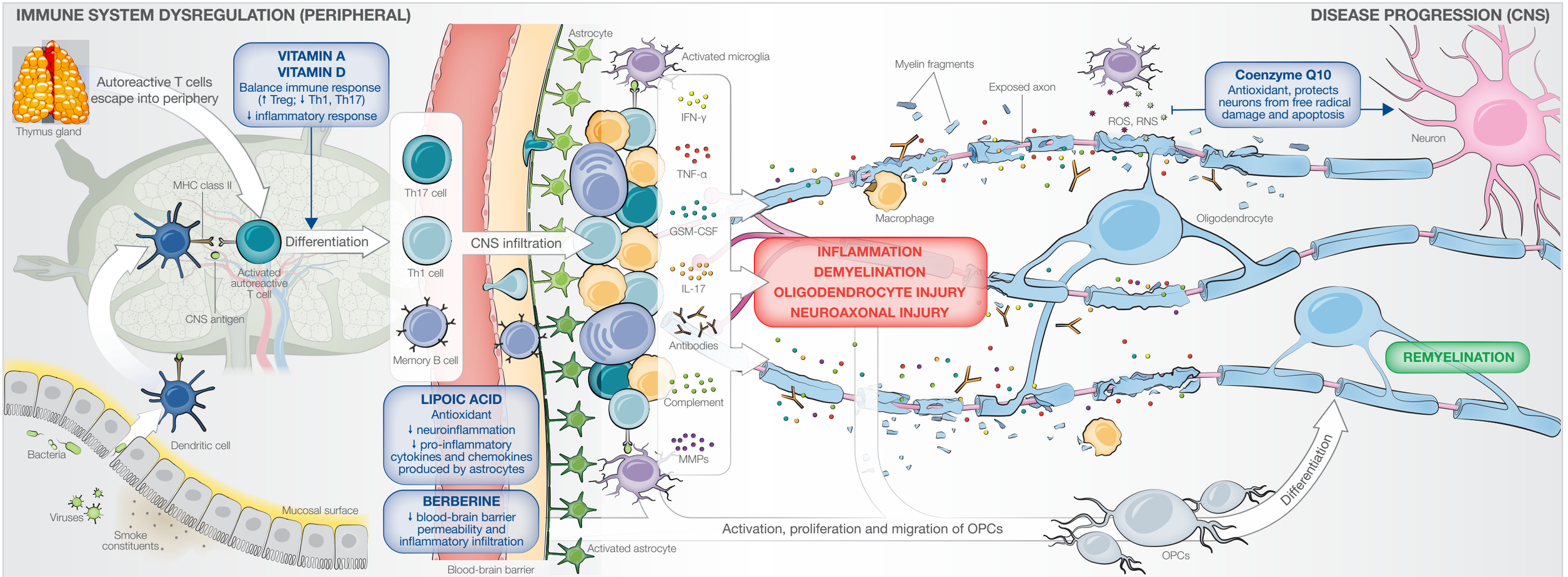
RISK FACTORS FOR MS include:[1,2]

- **Genetic variations**
- **Low vitamin D status**
- **Viruses** (Epstein-Barr virus, human herpesvirus-6)
- **Bacteria** (*H. pylori*, chlamydia, mycoplasmas)
- **Heavy metal exposure** (methylmercury)

3/4 of MS sufferers are **WOMEN**[3]

Up to **90%** of MS patients are **VITAMIN D DEFICIENT**[4]

NATURAL INTERVENTIONS FOR MULTIPLE SCLEROSIS[5-11]



MS is an autoimmune condition which effects the central nervous system (CNS). MS is characterised by destruction of the myelin sheath, which insulates and protects neurons. Without the myelin sheath, neuronal communication is severely disrupted and neurons are susceptible to damage. Symptoms include visual disturbances, motor impairments, fatigue, pain and cognitive deficits. The presentation of symptoms correlates to the location of affected nerves.

IMMUNE SYSTEM DYSREGULATION (PERIPHERAL)

Although the exact cause of MS remains unknown, it is thought that the disease may be triggered in the periphery during the breakdown of central tolerance due to reduced function of regulatory T (Treg) cells and/or resistance of autoreactive cells to suppression. Once activated, differentiated Th1 and Th17 cells, B cells and innate immune cells cross the blood-brain barrier and infiltrate the CNS leading to inflammation and tissue damage. Genetic and environmental factors, such as infectious agents and smoke constituents, contribute to these events.

DISEASE PROGRESSION (CNS)

Immune cells, activated microglia and astrocytes promote inflammation, demyelination, oligodendrocyte and neuroaxonal injury through the production of pro-inflammatory cytokines, chemokines and antibodies, as well as direct cell contact, e.g. macrophages engulf myelin and myelin fragments.

Following demyelination, microglia and astrocytes become activated and in turn activate resident oligodendrocyte progenitor cells (OPCs). Mitogens and promigratory factors released by activated microglia and macrophages promote the proliferation and migration of OPCs to the sites of demyelination. Recruited OPCs differentiate into remyelinating oligodendrocytes and repair damaged myelin.

Over time, chronic neurodegenerative damage overwhelms the neuroprotective and/or neurodegenerative mechanisms. Oligodendrocytes are damaged and remyelination may fail, which may lead to neuron damage, loss of neuron connectivity and apoptosis.

MHC class II: major histocompatibility complex II; CNS: central nervous system; Th: T helper cell; IFN-γ: interferon gamma; TNF-α; tumour necrosis factor-alpha; GM-CSF: granulocyte macrophage-colony stimulating factor; IL-17: interleukin-17; MMPs: matrix metalloproteinases; OPCs: oligodendrocyte progenitor cells, ROS: reactive oxygen species; RNS: reactive nitrogen species