



## Neurodegeneration - Neurodegenerative Diseases

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### Editorial

The role of biometals during a growing list of brain disorders is supported by evidence from a good range of sources including genetics, biochemical studies and biometal imaging. These studies have spurred a growing interest in understanding the role of biometals in brain function and disease also because the development of therapeutic approaches which will be ready to restore the altered biometal chemistry of the brain. We hope that these exciting insights will provide a robust platform to develop advances within the rapapeutics which will allow us to “restore the balance” in metal homeostasis in the brain.

The review describes how abnormalities during this complex process can cause loss of Fe, which is related to changes in neurotransmission, energy production and myelination, and is related to diseases like AD. Conversely, abnormal Fe handling also can cause Fe accumulation, which is related to AD and PD and may be a major target of therapeutic developments supported Fe chelation. The article provides a timely insight into the consequences of abnormal Fe metabolism through loss of ferritin function, a key Fe-regulatory protein and the way these changes can cause ferritin accumulation, reactive oxygen species formation and oxidative stress.

Despite a little cohort and rare alleles the studies provide important insights on altered Fe in neurodegeneration and illustrate that the role of metals in neurodegeneration must be examined in association with genetics, and disease sub-populations to realize a transparent insight into the contributory role of metals in these diseases. They supply important insights into the cell fate when abnormal Cu trafficking occurs, as is evidenced by the genetic diseases, Menkes and Wilson disease, and more broadly in AD and prion diseases. The recent interest in concussion in sport has resulted in significant media focus about chronic traumatic encephalopathy (CTE), although an immediate causative link(s) between concussion and CTE isn't established.

Typically, sport-related CTE occurs during a retired athlete with or without a history of concussion(s) who presents with a constellation of cognitive, mood, and/or behavioral symptoms and who has postmortem findings of tau deposition within the brain.

There has been significant interest within the presence of neurodegenerative diseases in athletes during the past decade, especially those involved in touch sports who may be exposed to concussive and subconcussive impacts during their sporting careers. The traditional histological examination demonstrated normal cerebral mantle, substantia alba, and brainstem, with mild-to-moderate neuronal dropout of the nigral pars compacta and pars reticularis, without Lewy bodies. Neurodegenerative diseases target large-scale neural networks. Four competing mechanistic hypotheses are proposed to elucidate network-based disease patterning: nodal stress, transneuronal spread, trophic failure, and shared vulnerability. These data enabled us to research how intrinsic connectivity in health predicts region-by-region vulnerability to disease.

A key molecular pathway implicated in diverse neurodegenerative diseases is that the misfolding, aggregation, and accumulation of proteins within the brain. Compelling evidence strongly supports the hypothesis that accumulation of misfolded proteins results in synaptic dysfunction, neuronal apoptosis, brain damage, and disease. However, the mechanism by which protein misfolding and aggregation trigger neurodegeneration and therefore the identity of the neurotoxic structure remains unclear.

Let's learn to cope with our new reality of being in this virtual life that includes virtual work, virtual consultations, virtual exercise lesson and virtual family and friend meets. It is normal to feel sad, stressed, scared during such a crisis. Remember, it is a physical lockdown for everyone, not a lockdown of your mind and soul, it is not a lockdown of help, hope and kindness.

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