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Iron Deposition in the Central Nervous System of the Human Brain

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Iron (Fe) In the central nervous system is linked to proteins, involved in many important processes such as oxygen transportation, oxidative phosphorylation and synthesis and metabolism of neurotransmitters. Capillary endothelial cells are connected to the astrocytes. The latter are ideally positioned to transport Fe to other brain cells [1].

Transverse relaxation R2 and R2* magnetic resonance imaging (MRI) is the best technique to demonstrate the distribution and the concentrations of Fe in the different deep brain structures during life. However, it cannot be used for the Fe evaluation in the cerebral cortex. In descending amount, the highest concentrations of Fe during normal adult age are observed in the substantia nigra, the red nucleus, the globus pallidus, the dentate nucleus, the putamen, the corpus geniculatum, the subthalamic nucleus and the caudate nucleus. The incidence is low in the thalamus and in the cerebral white matter. The Fe content is lower in infant brains compared to adult ones. It increases progressively during the brain maturation process, but most strongly in the basal ganglia and brainstem nuclei, mainly reflecting the accumulation of dopamine and neuromelanin. The Fe content decreases moderately after the age of 80 years, probably related to the normal age-related cognitive decline [2].

Ferroptosis is a unique form of programmed cell death, characterized by cytosolic accumulation of Fe, lipid hyperperoxides and metabolites, and affected by the fatal peroxidation of polyunsaturated fatty acids in the plasma membrane [3].

Brain accumulation of Fe is often detected in patients suffering from neurodegenerative diseases. Fe increase, mainly in the caudate nucleus, is related to working memory disturbances. The Fe accumulation is the most prominent in frontotemporal lobar degeneration, mainly in those of the FUS and TDP types, compared to the Tau form. Moderate Fe collection is found in Alzheimer and Parkinson brains but also in less frequent diseases such as Huntington's disease, Hallervorden-Spatz syndrome, neuroferrinopathy and aceruloplasminemia [4].

Hemorrhagic as well as non-hemorrhagic infarcts contain an increased iron content in the acute as well as in the residual stages. In non-hemorrhagic infarcts the Fe concentration further increases during their progressive resolution. In lobar hematomas the Fe accumulation moves from the core of the bleeding in the acute stage to the boundaries in the residual phases. Superficial siderosis, frequently linked to cerebral amyloid angiopathy, is easily to detect with MRI [5].

Post-mortem evaluation of Fe content in different neurodegenerative and cerebrovascular diseases can be demonstrated by 7.0-tesla MRI but also by particle induced X-ray examination [6].

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