

Proposing biomarkers to evaluate the alterations in the brain iron homeostasis and their relation with the physiopathology of Alzheimer's disease Proponiendo biomarcadores para evaluar las alteraciones en la homeostasis cerebral de hierro y su relación co

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Multiple lines of evidence have implicated oxidative stress and free radical damage to the pathogenesis and etiology of Alzheimer's disease (AD). Amyloid-beta peptide contributes to oxidative damage in AD by inducing lipid peroxidation. In addition, iron might contribute to the increased susceptibility of the brain to iron-induced oxidative damage, due to its ability to catalyze the generation of free radicals in biological systems. There are several points in the iron regulation pathway in which alterations may occur, affecting iron metabolism. Altered expression and altered cellular distribution of melanotransferrin, lactotransferrin, and neuromelanin have been reported in the brain tissue of patients suffering AD. In addition, disruptions in lactotransferrin, ceruloplasmin, neuromelanin, and hemo oxygenase may result in oxidative stress. In conclusion, in AD it appears to be an excessive accumulation of iron in the brain and oxidative damage, suggesting a loss of the homeostatic mec