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Therapeutics for Ferroptosis in CNS Disease

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Message from the Guest Editor

Iron (Fe) is a vital element in oxygen transport, enzyme activity, and oxide-reduction reactions for almost all organisms. However, iron is responsible for a recently described programmed cell death mechanism. This process is known as ferroptosis, where antioxidant defenses such as glutathione peroxidase 4 (GPX4) are inhibited or overwhelmed. For proper intracellular use, iron must cross biological membranes, which requires conversion from its oxidized to reduced state and vice versa, depending on the metabolic step involved. The complex molecular mechanisms involved in ferroptosis can be activated in all cell types, including those of the CNS, developing degenerative (NDG) processes also known as neurodegeneration. The progressive loss of neural functions and neuronal death with iron accumulation is closely associated with intellectual and/or motor impairment. In this Special Issue of Pharmaceuticals, we intend to update the most relevant information on the potential therapeutic action of ferroptosis in the CNS, with a focus on its implication in different types of neurological pathologies and its most representative biomarkers.

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Message from the Editor-in-Chief

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