

Role and mechanism of neurological disorders.

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Introduction

The pros and cons of total lockdown are up for discussion. Children with neurological impairments are a vulnerable population. Compared to the preceding two months the prevalence of symptoms was much lower during lockdown. In of the patients, the underlying neurological disease got worse. Children who frequently left the house when it was in lockdown were more likely to experience symptoms. During lockdown of parents called their paediatrician of parents contacted their child's neuropsychiatric and of parents had a specialist appointment cancelled of patients who typically undergo rehabilitation continued doing so via distance. As a novel type of cell death ferro ptosis differs from conventional cell deaths like autophagy or senescence. Ferro ptosis plays a role in the pathophysiological development of a number of illnesses, such as cancer, cardiovascular disease, nervous system disorders and kidney damage. The significance of ferro ptosis in neurological illnesses has been extensively studied because oxidative stress and iron accumulation are the main pathogenic characteristics of neurological diseases [1].

Changes in iron homeostasis, iron-dependent lipid peroxidation and glutamate toxicity build up are the key characteristics of ferro ptosis, and these changes can all be selectively reversed by substances that either trigger or inhibit ferro ptosis. Iron, lipid, and amino acid metabolism through System voltage-dependent anion channels, p53, p62-Keap1-Nrf2, mevalonate, and other pathways are the primary regulators of ferro ptosis. This review also focuses on Ferro ptosis's regulatory pathways and the advancement of its study in neurological disorders. Ferro ptosis is specifically characterised via way of means of modifications in iron homeostasis, iron-structured lipid peroxidation and glutamate toxicity accumulation of which may be mainly reversed via way of means of ferro ptosis inducers or inhibitors. The ferro ptosis is specifically regulated via way of means of the metabolism of iron, lipids and amino acids thru System voltage-structured anion channels, p53, p62-Keap1-Nrf2, mevalonate and different pathways. This overview additionally attention at the regulatory pathways of ferroptosis and its studies development in neurological diseases [2].

Current research on ferro ptosis in neurological diseases mainly focuses on the key pathways of ferroptosis. At the same time, we found that ferro ptosis has a bidirectional regulatory role in neurological diseases. Therefore, the specific regulatory mechanisms of ferro ptosis in neurological diseases

should be further investigated to open new perspectives on the application of ferro ptosis in the treatment of neurological diseases.

Epilepsy, cerebral palsy and other acquired encephalopathy's, inflammatory and post-infectious diseases, degenerative and metabolic brain diseases, neurodevelopmental disorders, neurogenesis syndromes, brain malformations, etc. Associated impairments were measured in terms of the Cognitive Emotional Motor Sensory Communication Activity [CEMSCA] and participation domains of the International Classification of Functioning, Disabilities and Health (ICFDH) [3].

As one of the most important essential trace elements in the human body, iron is involved in numerous metabolic processes. Ferroptosis was first reported by Dixon et al. Proposed as a unique iron-dependent form of cell death induced by elastin that could not be blocked by inhibitors of apoptosis autophagy and other cell death However, it can be inhibited by certain inhibitors such as ferritin and iron chelators, as well as lipophilic antioxidants . Ferroptosis is primarily caused by an imbalance between the generation and degradation of lipid Reactive Oxygen Species (ROS)

During oxyphosphorylation of iron-containing mitochondria, oxidative stress from excess ROS directly or indirectly damages macromolecular substances. Morphologically, ferroptosis is characterized primarily by cell membrane rupture and blistering, mitochondrial volume reduction, membrane density increase, and apoptotic morphological hallmarks such as cell shrinkage, chromatin condensation, skeletal disruption, and apoptotic formation. Manifests as reduction or disappearance of criste mitochondria body. Ferroptosis is insensitive to inhibitors of apoptosis, thermolysis, and autophagy, but to iron chelators and antioxidants. As a central link in ferroptosis, accumulation of iron and ROS may reduce cystine uptake, deplete glutathione and release arachidonic acid to stimulate the activity of the cysteine glutamate antiporter system and glutathione peroxidase 4 (GPX4). In addition, abnormal expression of various genes was observed especially genes related to iron metabolism such as receptor activator (NCOA4). This aims to focus primarily on neurological disease progression with a view to systematically reviewing the regulation and associated pathways of ferroptosis and providing new insights into ferroptosis in neurological diseases [4,5].

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