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Harnessing Ferroptosis to Combat Alzheimer's Disease

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Abstract

Millions of individuals all over the world suffer with Alzheimer's disease (AD), a devastating neurological condition. There is currently no cure for AD, and the medications that are available only offer short-term symptom alleviation at best. So, it is crucial to find novel treatment targets for AD. In addition to apoptosis and necrosis, ferroptosis has emerged as a potential therapeutic target. Lipid peroxide buildup is a hallmark of ferroptosis, which is controlled by a web of interconnected signalling pathways. Targeting ferroptosis may offer therapeutic promise, as it has recently been implicated in the pathophysiology of AD.

Ferroptosis Linked GPX4: A Key to Unlocking Alzheimer's Treatment

There is mounting proof that ferroptosis plays a role in the development of Alzheimer's disease. For instance, oxidative stress and lipid peroxidation have been linked to AD pathogenesis due to studies showing higher amounts of lipid peroxidation products in the brains of AD patients. In addition, ApoE4 and TREM2, two known genetic risk factors for AD, have been related to lipid metabolism and ferroptosis control [1]. The glutathione peroxidase 4 (GPX4) enzyme plays an important role in ferroptosis control by converting lipid hydroperoxides into alcohols. Decreased GPX4 activity in the AD brain has been linked to lipid peroxide buildup and ferroptotic cell death [2]. Alterations in iron metabolism, mitochondrial malfunction, and inflammation are all possible contributors to the dysregulation of ferroptosis in AD. Excess iron can cause lipid peroxidation and ferroptosis, and it has been linked to Alzheimer's disease pathogenesis. Impaired mitochondrial activity has been linked to the formation of reactive oxygen species and lipid peroxidation, both of which have been implicated with Alzheimer's disease. Lastly, inflammatory stimuli can induce ferroptosis by increasing lipid peroxidation and decreasing GPX4 function; persistent inflammation is a characteristic of AD. Considering that ferroptosis may play a part in the development of AD, inhibiting it may be a useful treatment approach. Various strategies have been developed for addressing ferroptosis in Alzheimer's disease. One strategy is shooting for GPX4 specifically.

Protecting against ferroptotic cell death in vitro and in vivo, small molecule GPX4 activators have been created. An amyloid beta-reducing and cognition-enhancing GPX4 activator was also shown in a recent study in a mouse model of Alzheimer's disease. Targeting iron metabolism is another option. Lipid peroxidation and ferroptotic cell death are two processes that iron chelators have been demonstrated to mitigate. Furthermore, a recent study shown that iron chelation can ameliorate cognitive impairment and decrease amyloid beta deposition in an AD animal model. Lastly, addressing inflammation might be an effective strategy. In vitro and in vivo studies have revealed that anti-inflammatory medications can mitigate lipid peroxidation and ferroptotic cell death. In addition, new research has shown that an anti-inflammatory medication can lessen amyloid beta buildup and boost cognition in an Alzheimer's disease mice model. Many strategies have been proposed to target ferroptosis as a potential treatment for Alzheimer's disease. Although these methods are still in the research and development

stage, they provide a new hope for the future development of AD treatment [1,2].

References

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