Neurodegenerative diseases such as Alzheimer’s disease (AD) and Amyotrophic Lateral Sclerosis (ALS) are chronic progressive disorders characterized by loss of neurological functions. Although the enormous progress has been made in the understanding of diseases mechanism, no effective treatments to stop or slow the progression of neurodegeneration are currently available [1].

**Introduction**

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**Ferroptosis**

Ferroptosis is an iron-dependent and peroxidation-driven form of regulated cell death that is distinct from other types of cell death [2]. The main hallmark of ferroptosis is an excessive accumulation of lipid hydroperoxides leading to iron-dependent oxidative stress [3]. GPX4 as the only enzyme that can directly reduce phospholipid hydroperoxide in membranes is the master regulator of ferroptosis [4]. Ferroptotic hallmarks are well-demonstrated in AD and ALS [5, 6].

**Results**

1. Conditional ablation of Gpx4 in neurons (brain and spinal cord) of adult mice resulted in rapid onset and progression of paralysis and death after tamoxifen (TAM) treatment [7].

2. Paralyzed mice had a dramatic degeneration of motor neurons in the spinal cord [7].

3. Gpx4 forebrain neuron specific neuron specific knockout mouse models exhibited significant deficits in spatial learning and memory after TAM treatment compared with control mice [8].

**Conclusion**

The primary focus of our laboratory’s research is to investigate the importance of ferroptosis in neuron death in AD and ALS using different mouse models of these diseases. We are interested in determining whether inhibition of Ferroptosis is effective in retarding neurodegeneration and increasing neurological functions in those models. By understanding the importance of ferroptosis in AD and ALS, we hope to gain important insights into whether ferroptosis inhibition is a novel therapeutic approach for AD and ALS.

**References**