Oxidative stress and antioxidant defense pdf

The survival and death of cells is strictly controlled by various signals and molecules (Freed, 2010). Physiological death of cells is essential for normal function and tissue development. Both physiological cell death can occur under circumstances that causes tissue damage and therefore results in cell death. This first classification of cell death based on necrosis and apoptosis was established by Krammer and colleagues. Necrotic cell death is characterized by the release of intracellular contents into the extracellular space, leading to the loss of cell integrity and the death of the cell. Apoptotic cell death, on the other hand, is characterized by the fragmentation of DNA and the activation of caspases, leading to the formation of apoptotic bodies that are phagocytosed by macrophages.

It has been suggested that ferroptosis is a form of regulated cell death that is characterized by the accumulation of lipid peroxides and the release of reactive oxygen species (ROS) (Stockwell et al., 2008). Ferroptosis is distinct from other forms of cell death, such as apoptosis and necrosis, in that it is regulated by cellular iron metabolism and is dependent on the availability of glutathione (GSH) (Shimada et al., 2016). GSH is a tripeptide that serves as a cellular antioxidant and is involved in lipid metabolism.

The regulation of ferroptosis is complex and involves multiple pathways, including the glutathione peroxidase GPX4, the thioredoxin system, and the NFE2L2 transcription factor. GPX4 is an enzyme that catalyzes the reduction of lipid hydroperoxides to alcohols, thereby preventing the formation of toxic lipid peroxides. Thioredoxin enzymes are involved in the regeneration of GSH, and NFE2L2 is a transcription factor that regulates the expression of genes involved in the metabolism of lipid peroxides.

Ferroptosis is induced by a variety of stimuli, including oxidative stress, nutrient deprivation, and the loss of mitochondrial function. It is also induced by the activation of extracellular signaling pathways, such as the MAPK pathway, which is activated in response to growth factors and cytokines.

Ferroptosis is a potential therapeutic target for the treatment of a variety of diseases, including cancer, neurodegenerative diseases, and inflammatory diseases. The development of selective inhibitors of ferroptosis is an active area of research, and the identification of novel therapeutic targets is essential for the development of new treatments.

In conclusion, ferroptosis is a regulated form of cell death that is characterized by the accumulation of lipid peroxides and the release of ROS. It is regulated by cellular iron metabolism and is dependent on the availability of GSH. The regulation of ferroptosis is complex and involves multiple pathways, including the glutathione peroxidase GPX4, the thioredoxin system, and the NFE2L2 transcription factor. The development of selective inhibitors of ferroptosis is an active area of research, and the identification of novel therapeutic targets is essential for the development of new treatments.
The page contains scientific text discussing ferroptosis, a type of cell death, and its relationship to various conditions and mechanisms. The text references multiple studies and authors, including those on ferroptosis, apoptosis, and other forms of cell death.

For example, one study mentioned is by Zeng, T., Deng, G., Zhong, W., Gao, Z., and others (2015), which discusses ferroptosis caused by erastin in melanoma.

Another study by Yang, W. H., Ding, C.C., Sun, T., and others (2017) explores the role of ferroptosis in the progression of ferroptosis-related cancers.

The text also refers to the work of multiple authors and organizations, such as the National Institutes of Health (NIH), the National Cancer Institute (NCI), and the National Heart, Lung, and Blood Institute (NHLBI).

Overall, the page provides a detailed overview of the latest research findings and developments in the field of ferroptosis and related cell death mechanisms.