**Ferroptosis, an iron-dependent regulated cell death**

**: An emerging target in cancer and cardiovascular diseases**

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Ferroptosis is a type of cell death, which is morphologically and mechanistically distinct from other type of cell death pathways such as apoptosis and necroptosis. Increasing interest in ferroptosis is emerging due to its critical roles in several pathological conditions, such as ischemia-reperfusion (I/R) injury, neurodegeneration, and cancer. Lipid peroxidation is a hallmark of ferroptosis and directly destroys cellular membranes, thereby causing ferroptosis. Among the various lipids, polyunsaturated fatty acids (PUFAs) associated with several phospholipids, such as phosphatidylethanolamine (PE) and phosphatidylcholine (PC), are responsible for ferroptosis-inducing lipid peroxidation. Since the *de novo* synthesis of PUFAs is strongly restricted in mammals, cells take up essential fatty acids from the blood and lymph to produce a variety of PUFAs via PUFA biosynthesis pathways. Free PUFAs can be incorporated into the cellular membrane by several enzymes, such as ACLS4 and LPCAT3, and undergo lipid peroxidation through enzymatic and non-enzymatic mechanisms. In this talk, I will discuss how various lipid metabolism and signaling pathways are associated with lipid peroxidation and ferroptosis, and will provide insights into treatment strategies for ferroptosis-related diseases.