# Special Issue

# Cell Death Signaling of Ferroptosis

## Message from the Guest Editor

Ferroptosis is a unique iron-dependent form of regulated cell death. The accumulation of lipid peroxidation and the depletion of plasma membrane polyunsaturated fatty acids have been well known to result in the lethal event of ferroptosis. Differences in genetic makeup among cancer cells also affect the pharmacodynamic response of ferroptotic agents. High-level RAS-RAF-MEK pathway activity or p53 expression may elevate the generation of ROS through mitochondrial voltage-dependent anion channel 2/3 (VDAC2/3) or inhibit cystine uptake, respectively, and sensitize cancer cells to ferroptosis. Conversely, iron chelators and lipid peroxidation inhibitors are known to suppress ferroptosis and block pathological cell death events in the brain, kidney, and other tissues. Although ferroptosis is a unique iron-dependent form of non-apoptotic regulated cell death, emerging evidence suggests that ferroptosis shares common pathways with different types of cell death. Recent studies reveal that the ferroptotic-agent-induced endoplasmic reticulum stress response plays a pivotal role in the crosstalk between ferroptosis and other types of cell death.

## Guest Editor

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## Deadline for manuscript submissions

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*Cells* has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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