



p53 and Ferroptosis

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Message from the Guest Editor

The tumor suppressor p53 is frequently mutated in various human cancers. p53 is activated to induce or repress the transcription of numerous target genes important for multiple biological functions through various stressors, such as DNA damage, metabolic stress, oxidative stress, etc.

Ferroptosis is caused by the accumulation of iron-dependent lipid reactive oxygen species. Ferroptosis is caused when glutathione, an anti-oxidant, is depleted and the activity of glutathione peroxidase 4 (GPX4), a lipid repair enzyme, is lost. This process relies on intracellular iron, which acts as a catalyst to form free radicals from peroxides.

Many of the processes of p53 in ferroptosis have not yet been identified.

We invite all scientists working on p53 and ferroptosis to participate in this Special Issue. Original research articles or reviews on all aspects of their molecular mechanisms are welcome.

We look forward to your contributions.





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