Special Issue

p53 and Ferroptosis

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.

Guest Editor

Prof. Dr. Ji Hoon Jung

Department of Science in Korean Medicine, Graduate School, Kyung Hee University, 26 Kyungheedae-ro, Dongdaemun-gu, Seoul 02447, Republic of Korea

Deadline for manuscript submissions

closed (31 March 2024)

Biomolecules

an Open Access Journal by MDPI

Impact Factor 4.8
CiteScore 9.4
Indexed in PubMed

mdpi.com/si/165712

Biomolecules
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland
Tel: +41 61 683 77 34
biomolecules@mdpi.com

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Biomolecules is a multidisciplinary open-access journal that reports on all aspects of research related to biogenic substances, from small molecules to complex polymers. We invite manuscripts of high scientific quality that pertain to the diverse aspects relevant to organic molecules, irrespective of the biological question or methodology. We aim for a competent, fair peer review and rapid publication. Please look at some of the exciting work that has been published in *Biomolecules* so far. We would be delighted to welcome you as one of our authors.

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Prof. Dr. Peter E. Nielsen
Department of Cellular and Molecular Medicine, Faculty of Health and Medical Sciences, University of Copenhagen, Blegdamsvej 3C, DK-2200 Copenhagen, Denmark

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