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p53 protects tumor cell survival via a long non-coding RNA under glucose starvation

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p53 is one of the most important tumor suppressors that have been found thus far. Approximately 50% of tumors harbor p53 mutations and which is believed to contribute to tumor's malignancy. However, the remaining 50% of tumors possess wild type p53, and the exact role it plays in tumor cells is not clear. Cancer cells prefer to metabolize glucose through aerobic glycolysis, known as the Warburg effect. Due to its low efficiency of ATP production, it is therefore reasonable to believe that tumor cell rely more heavily on glucose compared to normal cells. Yet under glucose starvation conditions, we found wild type p53 can up-regulates a long non-coding RNA, which interferes with a novel necrotic signaling pathway, and protects tumor cells against necrosis under glucose starvation. Our data suggest that wt-p53 can act as an oncogenic factor to promote tumor cell survival by metabolic reprogramming.

Biography

Mian Wu was graduated from Nanjing Normal University in 1981 (BS) and obtained his PhD degree from Columbia University, USA in 1988. He has conducted his Post-doctoral research at Harvard University during 1988-1991. Thereafter, he moved to Singapore as an Assistant Professor at School of Biological Sciences of National University of Singapore. From 2000, he is working as a full Professor at University of Science and Technology of China in Hefei, Anhui. His research interests focus on molecular mechanisms for p53-regulated tumor development and regulation of non-coding RNA in tumor metabolism. He has published more than 60 research papers on international peer-reviewed journals with more than 2900 citations.

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