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Epigenetic and metabolic regulation of leukemia stem cells

Acute myeloid leukemia (AML) remains a significant challenge for oncologists, with a 5-year survival rate of only 27% and a standard treatment that has not changed meaningfully in the past 3 decades. Current treatments are largely ineffective as they do not kill quiescent leukemia stem cells (LSCs) and resistant LSCs will survive to regenerate additional leukemic cells through their self-renewal capacity. Targeting self-renewal pathways that drive LSC development is essential for curative therapy. Oncogenic events including genetic, epigenetic and metabolic abnormalities enable LSCs to hijack normal stem cell of self-renewal mechanisms that allow LSCs to evade therapy and regenerate new leukemia, leading to relapse. Targeted disruption of abnormal stem cell self-renewal represents a novel therapeutic strategy that could significantly reduce the capacity of a tumor to regenerate itself after treatment and has become a new focus for drug development in poor prognosis AML.

Biography

Jenny Wang is the Head of the Cancer and Stem Cell Laboratory at the University of New South Wales, Sydney, Australia. She had worked in Children's Hospital Boston, Harvard Medical School, while doing Postdoctoral Research in Leukemia Stem Cell Biology. Her researches mainly focuses on to develop novel therapeutic strategies specifically targeting leukemia stem cells that are often resistant to commonly used cancer therapies and that are now believed to be the engine driving the growth of a tumor and the major cause for treatment failure and relapse in leukemia.

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