

Survival protein a potential new target for many cancers

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Researchers from the Walter and Eliza Hall Institute in Melbourne, Australia, have discovered a promising strategy for treating cancers that are caused by one of the most common cancer-causing changes in cells. Credit: Walter and Eliza Hall Institute of Medical Research

Walter and Eliza Hall Institute researchers have discovered a promising strategy for treating cancers that are caused by one of the most common cancer-causing changes in cells.



The discovery offers hope for treating many types of cancer that are driven to grow and spread through the actions of a cancer-causing protein called MYC.

Up to 70 per cent of human cancers, including many leukaemias and lymphomas, have unusually high levels of MYC, which causes <u>cancerous</u> changes in <u>cells</u> by forcing them into abnormally rapid growth.

Dr Gemma Kelly, Dr Marco Herold and Professor Andreas Strasser from the Walter and Eliza Hall Institute in Melbourne, Australia, led a research team investigating how cells with high levels of MYC stay alive and grow. They discovered that lymphomas that have high levels of MYC cannot survive long-term without a protein called MCL-1 which makes cells long-lived. Their research is published this week in the journal *Genes & Development*.

Dr Kelly said the research built on more than three decades of work at the institute into how MYC drives cancer development and how the survival of normal and <u>cancerous cells</u> is regulated. "For many years we have known that proteins from the BCL-2 protein family enhance cell survival and cooperate with MYC to accelerate the development of cancer," she said. "Until now, it was not known which specific BCL-2 family protein was most important for the survival and growth of MYC-driven cancers.

"We discovered that <u>lymphoma cells</u> with high levels of MYC can be killed by disabling a protein called MCL-1. Excitingly, when compared with healthy cells, the lymphoma cells were considerably more sensitive to a reduction in MCL-1 function. This suggests that in the future medicines that block MCL-1 could be effective in treating cancers expressing high levels of MYC with tolerable side-effects on the body's normal cells," she said.



Professor Strasser said the finding was exciting as there was hope that MCL-1 inhibitors may soon be available for clinical use. "MCL-1 is found at high levels in a number of <u>blood cancers</u> and also many solid tumours, so there has been a strong drive to develop potential anti-cancer compounds that target MCL-1," he said.

"Anti-cancer agents that target the protein BCL-2, which is closely related to MCL-1, are already showing promise in clinical trials, including some held in Melbourne. We are hopeful that inhibitors of MCL-1 will soon become available for clinical testing. We will be very interested in determining whether these compounds could be used to treat MYC-driven cancers," Professor Strasser said.

Provided by Walter and Eliza Hall Institute

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