

New treatment strategy for leukaemia promises significant reduction in side effects

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A Wright's stained bone marrow aspirate smear of patient with precursor B-cell acute lymphoblastic leukemia. Credit: VashiDonsk/Wikipedia

(Medical Xpress)—Adelaide researchers are zeroing in on a promising new approach to killing off cancer cells in patients with leukaemia.

In a study led by the South Australian Health and Medical Research Institute (SAHMRI) and the University of Adelaide's Centre for



Personalised Cancer Medicine, researchers have found that cancer cells decide whether to live or die after a short period of intense exposure to targeted therapy, opposing the current requirement for continuous treatment.

The researchers say this study presents a new treatment strategy which will translate to a significant reduction in side effects for patients. The results have been published online ahead of print in the journal *Leukemia*.

"This discovery is paradigm shifting," says Professor Deborah White, Director, Cancer Research with SAHMRI and University of Adelaide professor. "Our findings are not just applicable to <u>chronic myeloid</u> <u>leukemia</u> (CML) therapy, but to all targeted cancer treatments.

"In our research, we're looking for methods that will result in the cancer cell killing itself. This would provide an improved treatment and reduce the risk of cancer relapse."

As a consequence of this finding, Professor White and colleagues identified a new target in resistant and persistent disease. They show that by blocking a common protein they can more effectively cause death in leukaemia cells.

Professor White and her research team, including University of Adelaide PhD student Lisa Schafranek, have been investigating the role of a common protein known as STAT5.

"The activity of STAT5 appears to be a critical determinant of the decision for <u>cancer cells</u> to live or die," says Miss Schafranek, a Leukaemia Foundation of Australia PhD scholar.

"Our research has found that by blocking STAT5 in conjunction with



exposure to a regular anti-cancer treatment, we were able to more effectively target the leukaemia cells. We now also better understand the timing required for the combined <u>treatment</u> to be effective."

More information: "Sustained inhibition of STAT5, but not JAK2, is essential for TKI-induced cell death in chronic myeloid leukemia." L Schafranek, et al. *Leukemia* accepted article preview 12 May 2014; <u>DOI: 10.1038/leu.2014.156</u>

Provided by University of Adelaide

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