

New way found to boost immunity in fight cancer and infections

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Killer T cells surround a cancer cell. Credit: NIH

An international research team led by Université de Montréal medical professor Christopher Rudd, director of research in immunology and cell therapy at Maisonneuve-Rosemont Hospital Research Centre, has

identified a key new mechanism that regulates the ability of T-cells of the immune system to react against foreign antigens and cancer. T-cells orchestrate the response of the immune system. This work outlines how a receptor termed LFA-1 on the surface of T-cells mediates adhesion to other cells such as cancer cells.

The work, published in *Nature Communications*, shows that LFA-1 mediates adhesion or de-adhesion by engaging a novel intracellular pathway in T-cells. International work over the past decade has underscored the importance of the manipulation of the immune system to combat cancers and infections. Manipulation of the new pathway outlined by Rudd and his co-researchers represents a new targeting strategy to promote immune-cell rejection of cancer.

"With this work," said Rudd, "we have found a new way to alter the overall immune response. We now have new tools to increase immune response against cancer and infections. The discovery could prove to be a major asset in the fight against several pathologies via the targeting of a single immune cell component."

"It is clear that Dr. Rudd's discovery represents a breakthrough in our ability to understand the immune system and to use it in the fight against cancer and infections," added Denis-Claude Roy, director of research at Maisonneuve-Rosemont Hospital. "This new mechanism allows us to identify the weaknesses of our present immunological approaches and to develop new weapons that are even more effective."

More information: Monika Raab, Yuning Lu, Karsten Kohler, Xin Smith, Klaus Strebhardt & Christopher E. Rudd, "LFA-1 activates focal adhesion kinases FAK1 / PYK2 to generate LAT-GRB2-SKAP1 complexes that terminate T-cell conjugate formation", *Nature Communications*, 2017 July 12. [DOI: 10.1038/ncomms16001](https://doi.org/10.1038/ncomms16001)

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