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New clues discovered regarding how immune cells operate

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(PhysOrg.com) -- Researchers at the University of Dundee have identified control mechanisms that allow certain white blood cells, which have a vital role in fighting viral infections and dealing with organ transplants, to carry out their work.

Cytotoxic T cells are a subpopulation of white blood cells that work by killing the cells in our bodies that have become infected with viruses. They also play a key part in how the human body rejects organ transplants.

In two recent papers, researchers at the University of Dundee, led by Professor Doreen Cantrell, have examined the control mechanisms that allow these cells to perform their role.

In a paper published in the journal *Nature Immunology*, the researchers reveal the complexity of protein modifications in cytotoxic T cells for the first time. In this paper, the team identified a key molecule that controls the ability of cytotoxic T cells to produce antiviral cytokines.

In a second paper, published in the journal *Immunity*, the Cantrell group turned its focus to the role of protein kinase B (PKB), which controls cell metabolism and energy production in many cell types. The researchers showed that this is not the role of PKB in T cells. Instead, PKB has an essential role in directing the migration of T cells away from lymphoid organs and towards sites of infection in the tissues.

"The most significant finding from these two pieces of work is that they identify two different ways that we can manipulate cytotoxic T-cell function," said Professor Cantrell. "This is important, for instance, for autoimmune diseases, and also following organ transplantation, as it gives us ideas about how to stop the 'killer' function of T cells."

More information: -- Navarro MN et al. Phosphoproteomic analysis identification of an intrinsic pathway for the regulation of histone deacetylase 7 that controls the function of cytotoxic T lymphocytes. *Nat Immunol* 2011 [epub ahead of print]

-- Macintyre AN et al. Protein kinase B controls transcriptional programs that direct cytotoxic T cell fate but is dispensable for T cell metabolism. *Immunity* 2011;34(2):224-36.

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