

Researcher steps closer to understand autoimmune diseases

March 6 2012

Understanding why immune cells, called T-cells, attack the body is vital in the war against autoimmune diseases like diabetes. University of Alberta researcher Troy Baldwin is a step closer to understanding why the body's T-cells sometimes attack healthy cells causing autoimmune diseases.

Baldwin and graduate student Alex Suen looked at a specific molecule, Bim, which is vital in regulating T-cell death. When they removed Bim they found that the autoreactive T-cells, which are normally eliminated from the blood, were able to survive but were left inactive.

The other part of this study, which is published in the <u>Proceedings of the</u> <u>National Academy of Sciences</u>, is that there is another mechanism controlling these T-cells above and beyond Bim.

"When the autoreactive [T-cells] survived because Bim was absent, they weren't able to kill the insulin producing cells in the pancreas," said Baldwin, who is looking specifically at the T-cells that target the pancreas, which causes diabetes.

"Our future work is going to look at what those mechanisms are that are controlling these cells," said Baldwin. "Even though they [T-cells] are present in the body and should be able to target, in this case, the pancreas and cause diabetes, they don't."

This work has big potential. Understanding why these cells didn't target



the <u>pancreas</u> and why they are inactive could lead to new therapies to control auto-reactive T-cells and help people with autoimmune diseases.

"Something is telling them not to become active," said Baldwin. "If we can figure out what that something is, we could then potentially use that information to try and either suppress cells that would normally become activated and cause <u>autoimmunity</u> or vice versa. We could now take T-cells that are not active and make them more active.

"Cancer therapies, for example, are one place where we want to boost the T-cell response. So if we turn that suppressive mechanism off, then perhaps we can make a cell that wouldn't normally respond, responsive."

The lab, in the Department of Medical Microbiology and Immunology, continues its work in this area. The next step will be to find other mechanisms that regulate T-cells.

"First, we want to try and understand if there are other cells in the laboratory model that are controlling this auto-reactive population or if it is something in the cells themselves that is preventing them from being active," said Baldwin.

"If we can break those control mechanisms, then we can understand how <u>autoimmune diseases</u> can progress," said Suen. "That will give us an idea of what to target to generate therapies to either try to prevent breaking that control or enforcing that control more strictly."

Provided by University of Alberta

Citation: Researcher steps closer to understand autoimmune diseases (2012, March 6) retrieved 2 May 2024 from <u>https://medicalxpress.com/news/2012-03-closer-autoimmune-diseases.html</u>



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