

Peter Thompson, Ph.D. Updates

Update on 7-6-18

"I want to thank you and DRC for all of your efforts in funding my research project. You'll be happy to know that because of the DRC grant support and the results we've obtained, I was successful in securing another T1D-based grant from the Larry L. Hillblom Foundation. It's a more substantial grant with funds for up to 3 years, allowing me to continue the work started with the DRC grant!"

Update on 3-1-18

Background:

Type 1 diabetes (T1D) results when immune cells mistakenly destroy beta cells, a specialized cell in the body that produces insulin. Insulin is required for storing energy from the foods we eat, and so is essential for our normal bodily functions. Currently, there is no cure for T1D and since people with T1D don't produce enough insulin they must receive insulin from other sources in order to live. The reasons why beta cells are destroyed in this disease are still a mystery. In this project, we found that a small subset of beta cells turned on a known growth pathway before the disease occurred in a mouse model. This led us to think that this pathway might be a way to compensate for beta cell loss by regrowing or replenishing the pool of beta cells. We hoped to encourage this potential growth of beta cells in such a way that could reverse T1D and restore normal levels of insulin.

Update on 7-3-17

My lab recently found that a beta cell growth pathway, called the mTORC1 pathway, gets switched on in a subset of beta cells during the progression towards and the onset of T1D in mice. Intriguingly, the mTORC1 pathway is normally off in the adult beta cell but is switched on under specific conditions and may help beta cells cope with insults that affect their numbers or function. When the mTORC1 pathway is activated in beta cells, it causes them to grow in size, allowing them to make more insulin and it also can promote their progression towards cell division. The aim of this project is to help beta cells grow (increase in size) and divide (increase in number) using small molecules to stimulate the level of mTORC1 activity in the cells that have already switched it on during the onset of T1D.

In the first stage of this project I will be doing two different experiments. In the first, I will investigate some other important signaling pathways to determine how they dictate the output of mTORC1 signaling in beta cells. In the next part of the project, I'll be setting up two different cell culture model systems that I can use to study this pathway in mouse and human beta cells more effectively than what is currently available.

If we can identify and target the right factors in the mTORC1 pathway, we may be able to reverse the progression towards and onset of diabetes. Ultimately, it would mean that a person with T1D would no longer need insulin injections because they could have sufficient numbers of functioning beta cells to make enough insulin on their own.