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HTR1F Antagonists for Treatment of Diabetes and Improvement of Peri-Transplant Beta Cell Survival and Function for Type 1 Diabetes

Tech ID: 32759 / UC Case 2020-217-0

TECHNOLOGY DESCRIPTION

The invention is a novel target to improve peri-transplant human beta cell (HBC) survival and function during pancreatic islet transplant. HTR1F agonists have been shown to inhibit HBC function (insulin secretion) while causing HBC death.

Approximately 1.6 million people in the United States are living with T1D, of which 200,000 are under the age of twenty. It is anticipated that 5 million people in the U.S. will have T1D by 2050. While islet transplantation can cure T1D, peri-transplant HBC death limits the procedure to those who have low insulin requirements. Currently, there are no approved treatments to improve HBC survival and function during transplant.

ADVANTAGES

- ▶ Potential to improve HBC survival to make islet transplantation a possibility for more T1D patients, including those with higher insulin requirements.
- ▶ Increasing HBC proliferation has the potential to serve as a complementary approach to reducing HBC death.
- ▶ Potential to increase insulin secretion in a type 2 diabetic patient while potentially modifying disease progression by preventing HBC death/increasing HBC proliferation.

APPLICATIONS

- ▶ Improve HBC survival and function during islet transplant
- ▶ Method of treating Type 1/Type 2 diabetes without transplant (i.e., administration of HTR1F chemical antagonists to improve diabetes)

LOOKING FOR PARTNERS

To commercialize the technology

CONTACT

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OTHER INFORMATION

CATEGORIZED AS

- ▶ **Medical**
- ▶ Disease:
[Metabolic/Endocrinology](#)
- ▶ [Therapeutics](#)

RELATED CASES

2020-217-0

STAGE OF DEVELOPMENT

Proof-of-concept: demonstration that blocking HTR1F with a specific antagonist increases insulin secretion in human islets and prevents human islet cell death due to cytokines.

DATA AVAILABILITY

Under CDA

PATENT STATUS

Patent Pending

OTHER INFORMATION

Published patent application: WO2022155152A1

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