

## **Project 1.10 Role of metabolic stress in differentiation of pancreatic progenitor cells**

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**Laboratory:** Laboratory of Cell Signaling and Metabolic Disorders

### **Background:**

Type 2 diabetes (T2D) is associated with impaired and insufficient insulin secretion from pancreatic beta cells in response to glucose, greater glucagon secretion from pancreatic alpha cells, dysregulation of lipid homeostasis, and chronic inflammation. Recent studies suggest a new mechanism for profound loss of beta cell function in T2D, that comprises metabolic stress-induced loss of the beta cell identity, involving its reversal to a fetal state (dedifferentiation) and reprogramming to express hormones of other islet cell types including glucagon and somatostatin. The maintenance of pancreatic islet cells identity and function depends on dynamic control of transcription factors expression, although the extent of the phenomenon remains unclear. Based on our preliminary studies we hypothesize that metabolic pathway networks controlled by SCD are likely to represent critical steps in differentiation balance of pancreatic mature beta cells and specification of pancreatic progenitors into the endocrine lineage.

### **Aim:**

The main objective of the project is to determine a possible role of SCD1 in regulation of pancreatic progenitor-cells differentiation and metabolic stress-driven reprogramming of beta cells.

### **Requirements:**

- MSc in the field of biochemistry, molecular biology or biotechnology.
- laboratory experience with working knowledge of molecular biology and/or biochemistry techniques.
- knowledge of histological/morphological techniques.
- candidate should be highly motivated (as demonstrated via joint former education, additional courses completed, list of publications and conferences attended, previous scientific experience, references of the candidate's thesis tutor, letter-of-intent).
- ability to work as a team member along with written and spoken presentation skills required.