Diabetes is a metabolic disease that affects millions of people worldwide. Most common types of diabetes are called type 1 and type 2 (T2D), which are exemplified by loss of specific pancreatic cells, named as β-cells. The level of glucose in the blood is regulated by coordinated action of various pancreatic hormones, including insulin which orchestrates glucose uptake by other cells in the periphery. Insufficient number of β-cells solely producing insulin leads to a chronic condition of hyperglycemia, with elevated levels of glucose and finally to severe diabetes. The current pharmaceutical approaches aim to keep the levels of glucose close to the norm, however no cure for severe condition is yet offered. One of the critical questions that relates to the function of β-cells and insulin insufficiency in diabetes is how these dependencies rise and whether/how the observed robust changes in cellular metabolism and signaling mechanisms that govern the maturation of β-cells influence each other is not well understood. Similarly, are all these to be observed at the very early stage, do the diabetes related alterations follow the same developmental pattern and how? The current project utilizes the combination of high-end sequencing and mass spectrometry-based techniques to measure the changes occurring the level of mRNA in single cells, mitochondrial proteomes, lipidomes (assessment of cellular fats) and metabolomes (targeted assessment of cellular metabolites related to mitochondria) to visualize the specific changes in rare diabetes type 2 human induced pluripotent stem cells (**Aim 1**), verify them using various microscopy and experimental fluorescence sorting techniques (**Aim 2**), and contest them with a panel of known small compounds to facilitate the routes for possible future treatments (**Aim 3**). We will employ here the well-established human induced pluripotent stem cells model, to recapitulate human pancreatic β-cells development in the dish (*in vitro*). We chose the group of very well-characterized patients with rare forms of T2D. This project will be performed at the Adam Mickiewicz University in Poznań, Poland in collaboration with our colleagues in Finland and the USA. We expect to reveal in detail the cellular mechanisms underlying type 2 diabetes with particular emphasis on changed processes occurring in pancreatic β-cells at the early stages of maturation at the level of mitochondria, as well as changes in fat metabolism.