

2nd International Conference on

PANCREATIC DISORDERS & TREATMENT

September 13-14, 2017 Dallas, USA



Nicholas Zavazava

University of Iowa, USA

Human iPS cells are an alternative source of pancreatic beta cells

Type 1 diabetes (T1D) is caused by autoimmunity whereby pancreatic beta cells are destroyed by autoimmune disease. Standard therapy is replacement of insulin by regular injections of recombinant insulin. However, T1D can be cured by the transplantation of pancreatic islets or that of the whole pancreas organ. Our approach is to generate iPS from the patient and derive pancreatic beta cells from those cells and transplant them back to the patient. The advantage of this approach is that we could potentially transplant patients early after diagnosis with no waiting times for the availability of organs and we would not require the use of immunosuppressant since the cells are self-derived. Fibroblasts from a healthy individual were transduced with the Yamanaka factors in a retroviral vector. After confirming that the cells were pluripotent, the cells were differentiated into the endoderm which co-expresses CXCR4 and Sox 17. Next the cells were exposed to growth factors that converted them into the foregut endoderm. The cells were further treated to form the pancreatic precursor cells which expressed Pdx1. After further treatment with GLP-1 and nicotinamide, the cells formed pancreatic beta cells. These cells highly express GLP-1R and are responsive to high glucose by secretion of insulin. When transplanted in diabetic mice, they correct hyperglycemia within 3-4 weeks. Thus human iPS cells are an alternative source of pancreatic beta cells that can be harnessed for the treatment of diabetes.

Biography

Dr. Nicholas Zavazava is currently working as a Professor and Director of Transplantation Research at University of Iowa, USA. His work primarily focusses on stem cells. His lab is interested in studying stem cell derived hematopoietic cell and insulin producing cells.

nicholas-zavazava@uiowa.edu

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