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EUROPEAN ENDOCRINOLOGY AND DIABETES CONGRESS

August 22-23, 2022 | Webinar

A novel approach for creating beta cells toward Type 1 Diabetes

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The reprogramming of somatic and stem cells into pancreatic β cells is a promising approach toward type 1 diabetes as a cell replenishment therapy. Pancreatic lineage cells including acinar, duct, delta and alpha cells can Trans differentiate into β cells in vivo. Instead of limited these cells in vivo, however, a large number of somatic cells such as fibroblasts or hepatocytes is required for the clinical therapy as a resource, and cell fate converter into direct β cell reprogramming remains to be uncovered in vitro in the context of potential future therapy for all T1D patients. We found, however, that no induction of insulin in murine embryonic fibroblasts (MEFs) by above 3 factors. Here we propose a hypothesis in which another cell fate key driver might be involved in MEFs-derived cell reprogramming. To address this, we have established an unique cell line, Tec-3p which is very likely immature endocrine progenitors. We have also developed the lineage-tracing model from endocrine progenitors to beta cells in dual-labelled transgenic mice expressing GFP and DsRed2 under the control of Ngn3 and insulin promoters, respectively. Integrative screening analyses with our unique models has performed to identify potential key factors such as growth factors facilitating beta cell differentiation from progenitors mediated by Notch signalling. Moreover, we found that these factors accelerated the differentiation through the down regulation of Neurog3 gene expression through epigenetic control, indicating that chromatin remodelling mediated epigenetically by coordinating the integrative Notch and Ebb signalling contributes to progenitor maintenance and pancreatic endocrine cell fate determination. Taken together, we hypothesize a key driver of cell fate determination contribute to direct conversion from somatic cells into functional insulin producing cells in concert with these signals.

Biography

Masa Matsumoto has his expertise in regenerative medicine toward type 1 diabetes, and received his PhD in immunology from Tokyo University. He worked with Wylie Vale at Salk institute on functionalities of urocortin family on pancreatic beta cells. He is currently a full project professor of regenerative medicine using direct cell reprogramming at Juntendo University in Tokyo. He was awarded Ochai memorial foundation in 2004, Kowa life science foundation in 2002, an excellent poster of JBMR in 2003, RCGM frontier international symposium in 2012 and 2014, Terumo life science and art research foundation and a prize of innovation idea contest of TMDU in 2020.