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Editorial

Human stem cell pancreatic development and disease

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INTRODUCTION

The pancreas encompasses both endocrine and exocrine compartments with important functions for nutrient metabolism. The endocrine pancreas is comprised of five cell types clustered in the pancreatic islets that regulate blood glucose homeostasis: glucagon-producing alpha cells, insulin-producing beta cells, somatostatin-producing delta cells, pancreatic polypeptide-producing gamma cells, and ghrelin-producing epsilon cells. The exocrine pancreas consists of acinar and ductal cells, which secrete the digestive enzyme-containing pancreatic juice and transport it into the duodenum, respectively. Several chronic diseases are associated with dysfunction of pancreatic cells, the most notable being diabetes mellitus, in which beta cells are destroyed by the immune system or functionally impaired. Our current understanding of disease mechanisms has mostly been gained from the analysis of mouse models. While the molecular pathways that govern pancreas development are largely conserved between rodents and humans, there are also important differences .For example, the first endocrine cells to arise in human development are beta cells, whereas alpha cells are the first to develop in mice. As discussed in detail in this review, a number of mutations that are associated with human diabetes do not cause diabetes in mice. Therefore, human cell models are needed to understand disease mechanisms and to develop novel therapies. Over the past 15 years, protocols have been developed to generate the different cell types of the pancreas from human pluripotent stem cells .In this review, we discuss how pancreatic cells differentiated from have advanced our understanding of human pancreas development and disease and how technological advances are now beginning to enable large-scale disease modeling and screening for new therapeutics. Recapitulating human pancreas development in vitro Compared to the detailed knowledge that exists in mice, the molecular mechanisms of human pancreas development are still largely unknown. This is especially true for early developmental stages that include induction of the endoderm germ layer, formation and patterning of the gut tube, and pancreas organ formation. The limiting factors for studying human pancreas development are the limited availability of embryonic and fetal tissue samples and, until the advent of single cell technologies, the cellular heterogeneity found within these tissues. The development of protocols for the differentiation of hPSCs towards the pancreatic lineage has provided a readily available and scalable human model in which to explore the basis of human development The current framework for our understanding of pancreas development has been mostly derived from studies in vertebrate model organisms. This knowledge has served as the guide to devise protocols for the stepwise differentiation of hPSCs into pancreatic progenitor cells. In vitro differentiation of human pluripotent stem cells into different cell types of the pancreas. Overview of the stages of in vitro differentiation into the different pancreatic cell types, namely alpha, beta, acinar, and ductal cells. Beta cell differentiation protocols produce some monohormonal insulin+ cells that are glucose-responsive (beta-like cells) as well as nonfunctional insulin+ cells coexpressing other pancreatic hormones (polyhormonal cells). Upon implantation into mice, hPSCderived pancreatic progenitors differentiate into glucose-responsive, insulin-secreting beta cells, as well as other endocrine cell types. For example, the use of agonists in combination with the TGFβ receptor ligand active A to induce definitive endoderm from hPSCs is based on the finding in vertebrate embryos that both Wnt and Tgfβ signaling are required for definitive endoderm formation.Likewise, observations in mice that pancreas induction requires inhibition of sonic hedgehog and that bone morphogenic protein signaling favors the hepatic at the expense of the pancreatic fate

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