

A Short Note on Pluripotent Stem Cells Derived Pancreatic Endoderm Cells

Ekaterina Aksenov*

Department of Intracellular Signaling and Transport, Institute of Cytology, Russian Academy of Sciences, St. Petersburg, Russia

*Corresponding author: Ekaterina Aksenov, Department of Intracellular Signaling and Transport, Institute of Cytology, Russian Academy of Sciences, St. Petersburg, Russia, E-mail: aksenoyek@hotmail.com

Received date: December 08, 2021; Accepted date: December 23, 2021; Published date: December 30, 2021

Citation: Aksenov E (2021) A Short Note on Pluripotent Stem Cells Derived Pancreatic Endoderm Cells. Insights Stem Cells Vol.7 No.3:005.

Description

Pancreas improvement has been the situation of excessive research in quite a few species over the last decades. Elegant comparative researches have proven that early endoderm dedication and pancreatic endocrine specification are conserved amongst species to a positive degree, with versions maximum possibly deriving from evolutionary differences. A distinct and complete expertise of the mechanisms regulating human pancreas improvement is an essential prerequisite to expand a green protocol to obtain *in vitro* managed endocrine differentiation and maturation from human pluripotent stem cells.

The search for alternative β -cell sources has catalysed a series of studies aimed at translating known molecular pathways of pancreatic organogenesis into processes for stem cell differentiation *in vitro*. The prospect of transforming human stem cells, whether embryonic (ES) or induced pluripotent (iPS), into sufficient β -cells to treat many patients, stems primarily from the innovative potential of stem cells, whose populations can be amplified to a clinically significant extent and may be used as transplant material. Second, stem cell pluripotency implies that, under the influence of an appropriate network of inducible growth factors, these cells can give rise to any cell type, including endothelial lines. Fully functional pancreatic secretion has the potential to treat insulin-dependent diabetes.

Diabetes is an incredible health problem that affects more than 300 million people worldwide. By 2030, an estimated 440 million adults will have diabetes. Disease and premature death place an increasing burden on the global health system and on society. Type 1 diabetes (T1D) is defined as insulin deficiency caused by autoimmune destruction of islet β cells. Type 2 diabetes mellitus (T2D) is defined by a progressive decline in the ability of insulin secretion to meet peripheral insulin requirements. The defective innate regenerative capacity of β -cells, either due to β -cell destruction or inadequate reconstitution of β -cells, is increasingly recognized as essential for the pathogenesis of DT1 and DT2.

Despite remarkable advances, the clinical development of cell replacement therapies using human ESCs is still beset by ethical concerns. The use of allogeneic ESC-derived cells has also been associated with immunological mismatches. Although a recent study demonstrated the successful induction of personalized human ESCs by somatic cell nuclear transfer (NTESCs), the

generation of human NTESCs remains challenging. In this regard, nuclear reprogramming technology, which allows generation of pluripotent stem cells from mature somatic cells, has opened a new avenue for generation of pluripotent stem cells specifically for patients. Induced pluripotent stem cells (iPSC) technology is based on the genetic insertion of selected pluripotency-related factors into a source of adult somatic cells, which reprograms the cell fate to allow differentiation into pluripotent stem cell state. Human-derived iPSC lines exhibit characteristics similar to those of human ESCs, including morphology, global gene expression profiles, elongated telomeres, and propensity to differentiate into three germ layers, providing a source self-renewal of new tissues derived from patient cell pool.

Alternative treatment options are crucial in addressing these health challenges, and the field of regenerative medicine is poised to contribute to this. Strategies to induce replication and regeneration of existing β cells can increase the number of β cells available for blood sugar control, and studies of β cell proliferation in many Genetic models have identified candidate pathways. In addition, the discovery that pluripotent embryonic stem cells (ESCs) have the ability to develop into any cell type has inspired a more radical strategy in which defective or deficient tissues are replaced completely.

Patients with type 1 diabetes lack an adequate number of β cells and many patients do not appear to have them. In type 2 patients, β -cell mass is also insufficient to maintain glycemic control. Thus, strategies for generating novel β -cells to replace treatment have generated considerable excitement over the past two decades. A major advance towards this goal has been the identification of human pluripotent ESCs (hESCs) capable of forming tissues from all three developmental germ layers (Thomson et al., 1998). In the decade since this discovery, an additional source of pluripotent stem cells has been identified - induced pluripotent stem cells (iPSCs) reprogrammed from mouse fibroblasts.

iPSCs were also generated from human cells. One of the remarkable features of iPSCs is that, like ESCs, they have the ability to generate all cell types. These cells therefore offer an unprecedented opportunity to generate alternative tissues *in vitro*, including autologous cells from patient-specific cells. For this purpose, autologous mouse iPSCs that have been differentiated into hematopoietic organs have been shown to be

effective in the treatment of sickle cell disease abnormalities in an anemic mouse model.

The exocrine tissue of the pancreas is made up of ductal cells and acinar cells, while the islets provide the endocrine characteristics of the pancreas. After choosing their endocrine

fate, the gonadotropic cells then have to specifically become one of the islet's five mobile endocrine types: insulin-producing β -cells, glucagon-producing α -cells, and δ -cells somatostating, pancreatic polypeptide (PP) cells or ghrelingue-producing ϵ cells.