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Opinion Article

Development of Transplantable Beta Cells for Cell Therapy using Patient-Specific Cells

Divya Verma*

Department of Cell Science, Banaras Hindu University, Varanasi, Uttar Pradesh, India

DESCRIPTION

One of the most prevalent chronic diseases in children and adolescents, type 1 diabetes is brought on by the autoimmune destruction of the insulin-producing beta cells in the islets of Langerhans. Patients are thus totally dependent on insulin injections. Most young children require ongoing insulin injection therapy to manage their hyperglycemia. Exogenous insulin, however, frequently causes serious hypoglycemia-related problems. Thus, insulin therapy prolongs life but does not provide a cure. On the other hand, beta-cell replacement therapy through transplantation may provide a cure since it can restore insulin secretion that is responsive to glucose levels and can offer the best level of control to prevent hypoglycemia when insulin is secreted. Whole-pancreas transplantation can reinstate endogenous insulin production, although it is uncommonly performed on young patients with diabetes due to the possibility of perioperative morbidity from digesting enzyme damage caused by the exocrine pancreas during surgery. Contrarily, islet-cell transplantation offers beta cells that produce insulin in a relatively non-invasive manner. For recipients who are younger, it becomes a more practical choice. In fact, a great deal of progress has been made in islet-cell transplantation as a result of the Edmonton protocol's success, which emphasizes both an adequate number of donor islets and immunosuppressive regimens free of steroids. But there aren't nearly enough transplantable islets because it takes two to four donors to reverse diabetes. This scarcity is made worse by immunosuppressive medications' cytotoxicity, which kills transplanted In this aspect, using a different source of beta cells is essential to bridging the supply and demand gap for cells. Therefore, encouraging the growth of new beta cells is a key objective of diabetes therapy. Autologous cells could provide a safer option if immunosuppressant are eliminated. A patient-specific strategy would ideally improve the efficacy and security of islet transplantation. The viability of changing one type of cell into another has been established by recent developments in stem cell biology. By promoting the transdifferentiation of widely available cells, such fibroblasts, into therapeutically useful cells, like blood, neuron, cardiomyocyte, and islet-like cells, this innovation promotes autologous cell therapy. The engineering of new beta cells from patients' own cells, the abolition of lifelong immunosuppressant use, bio incompatibility, and disease transmission associated with donor cells are important uses of such patient-specific therapy. In this phase, transcription factors for beta cell differentiation and pancreatic stem cell formation are essential. Because of their high flexibility, stem cells can develop into any form of cell. Adult multipotent stem cells are highly uncommon and challenging to separate, nevertheless. Embryonic stem cells and Induced Pluripotent Stem Cells (iPSs) are both inducible *ex vivo*. iPSs can be reprogrammed from somatic cells by certain stimuli. IPSs can develop into certain cell types under particular circumstances, such as the presence of growth factors and many transcription factors. This patient-specific conversion would be significant since it would reduce the possibility of immunological rejection. There are two main techniques to reprogramme numerous somatic cells into the desired cell types, either directly or indirectly. In the former, somatic cells like fibroblasts serve as the direct source of beta cells. They can also be produced using the so-called indirect pathway or an iPS step. New research demonstrates the possibility of employing patient-specific iPSs as a source of transplantable beta cells for cell replacement therapies in diabetes by revealing that islet-like insulin-producing cells can be transformed from iPSs. This method is made viable by the fact that specific factors can produce iPSs from somatic cells. Directed reprogramming is the process of directly changing somatic cells into beta cells without the use of a multipotent or pluripotent intermediary. This process is typically triggered by the presence of specific growth factors and the overexpression of important transcription factors. Fibroblasts can be reprogrammed directly into neurons, cardiomyocytes, insulin-producing cells, and blood-cell progenitors, proving that it is possible to change one specific cell type into another. Future patient-specific treatments will be greatly influenced by both direct and indirect pathways for the regeneration of new beta cells. Our understanding of the crucial steps in the differentiation of beta cells during the embryonic development of the pancreas is crucial to the success of developing islet-like insulin-producing cells. The various transcription factors have

Correspondence to: Divya Verma, Department of Cell science, Banaras Hindu University, Varanasi, Uttar Pradesh, India, E-mail: VermaDv@gmail.com

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been used to force available cells to differentiate into desired types in a distinct delineation pathway, including across lineages, like from fibroblasts into iPSs, or from one fully functional lineage to another, like from fibroblasts into insulin-positive cells. However, serious safety concerns persist.

CONCLUSION

Despite the encouraging advancements in the hunt for novel sources of insulin-producing beta cells, the functionality of new cell types that replace the same type *in vivo* needs to be closely scrutinized. If long-term normoglycemia is to be accomplished, the function of these new transplantable beta cells must extremely nearly resemble that of the normal beta cells. The vast intricacy of the beta cell makes this particularly difficult for the development of real insulin-producing cells. In order to achieve the ultimate aim of discovering a treatment for diabetes, it is crucial that we fully comprehend the mechanisms that regulate the formation and destiny of beta cells.