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Islet Cell Transplantation for Treating Type 1 Diabetes: The Bright Future of Drug Designing and Development

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Short Communication

The revolutionary process of islet transplantation for treating type 1 diabetes has been limited by the high level of graft failure. The process of islet isolation alone has been shown to negatively impact islet survival and function *in vivo*. In addition, insults mediated by the instant blood-mediated inflammatory reaction, hypoxia, ischemia and immune response significantly impact the islet allograft post transplantation [1,2]. Moreover, the immune response to alloantigens and recurrence of autoimmunity contribute to pancreatic islet transplant dysfunction [3]. Several approaches are being developed and tried to overcome this serious problem and novel drug designing is heavily involved with promising results and bright rewards. Some of these approaches are being discussed briefly in this commentary.

Regenerating islet-derived protein 3 alpha; a pancreatic secretory protein, which functions as an antimicrobial peptide in control of inflammation and cell proliferation has been shown to enhance islet engraftments through its cytocprotective effect and advance the therapeutic efficacy of islet transplantation in mice [1].

Further, human mesenchymal stromal cells were shown to have therapeutic potential in allogeneic islet transplantation for type 1 diabetes patients and to promote islet survival and function in vivo. Some of the mechanisms which promote islet regeneration/survival include cyto protective and immune modulatory properties with expression up-regulation of Annexin A1, Elastin microfibril interface 1 and integrin-linked protein kinase [2].

Moreover, the adoptive transfer of T regulatory cells which play a central role in maintaining immune homeostasis and peripheral tolerance to foreign antigens in humans has the potential to significantly improve islet graft survival. The combination of islet and T regulatory cells co-transplantation was shown to be feasible and to have great potential to improve islet graft survival with the possibility to wean off or withdraw traditional immunosuppressive agents and improve patient quality of life [3].

Additionally, red ginseng has been reported to enhance insulin secretion-stimulating and anti-apoptotic activities in pancreatic beta-cells improving islet cell and graft function after isolation and transplantation, respectively. Islet pretreatment with red ginseng showed 1.4-fold higher glucose-induced insulin secretion than did control islets. Red ginseng pretreatment up regulated B-cell lymphoma 2 (Bcl-2) expression and down regulated Bcl-associated X protein (BAX), caspase-3, and inducible nitric oxide synthase (iNOS) expression. On the other hand, glucose-induced insulin release, NO, and apoptosis were significantly improved in red ginseng-pretreated islets compared with cytokine-treated islets in mice [4].

Similarly, islet PEGylation was shown to protect against nonspecific islet destruction in the early post-transplant period of intra portal islet transplantation in rats leading to better islet mass preservation and post-transplant outcomes [5].

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