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Editorial

Diabetic Mice by Reducing Inflammation and Inducting Beta Cell Regeneration

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INTRODUCTION

Diabetes is a complex metabolic disorder resulting from abnormally high blood glucose levels (Hyperglycemia) mainly caused by insulin insufficiency, insulin dysfunction and/or loss of insulin-producing pancreatic beta cells . Maintenance of a healthy blood glucose level can be achieved by regular injections of exogenous insulin in patients suffering from T1D, reducing the risk of various diabetic complications, including nephropathy and neuropathy. However, achieving long-term glucose levels within a narrow physiological range by regular insulin injections is still impossible. Thus, beta cell pool recovery is still one of the primary goals in treatment of diabetes. Addressing this, pancreas and pancreatic islets transplantation were considered as potential therapeutic options . However, a chronic shortage of donor organs and the need for concomitant immunosuppressive therapy restrict the widespread use of pancreatic cell transplantations. As an alternative, in vitro generation of insulin-secreting cells and their subsequent transplantation into patients with T1D has been proposed. Insulinsecreting cells can be generated by differentiating Stem Cells (SCs), particularly pluripotent SCs and pancreatic progenitor cells . Here, adult pancreatic alpha- cells and precursors of acinar cells have been considered as suitable candidates for cell replacement therapy . Despite the potential benefits of SC-derived cells, the insulin response to glucose in the differentiated beta cell is less intense Macrophage-Colony Stimulating Factor (GM-CSF) and Interleukin (IL)-7, the proinflammatory cytokine IL-1β (but not IL-6 or Tumour Necrosis Factor (TNF)-α), Th17 cytokines, and the regulatory cytokines IL-10 and IL27 in the blood of T1D patients. Moreover, Burke, et al. reported that IL-1β, a major mediator of inflammatory responses associated with diabetes development, is co-ordinately and reciprocally regulating chemokine and insulin secretion. In addition, IL-1β is known to have potent cytotoxic effects leading to progressive beta cell death . In particular, inflammation negatively affects GLP-1 binding to G ProteinCoupled Receptors (GPCRs) located on pancreatic beta cells. GLP-1 is a neuropeptide and an incretin derived from the transcription product of the proglucagon gene, exerting insulinotropic actions that include the stimulation of insulin gene transcription, insulin biosynthesis, and insulin secretion. Diminished GLP-1 signalling mediated by inflammatory triggers lead to a considerable deterioration of insulindependent homeostasis of glucose. During the study of the reserpine effect on mice maintained under optimal conditions we used additional eighteen C57BL/6 male mice. Mice were randomized into 3 equal groups (n=6). Group 1 mice (intact controls) were kept under standard vivarium conditions; groups 2 and 3 mice received sympatholytic reserpine. Reserpine was injected intraperitoneally in single dose 0.1 mgkg-1 in 0.1 ml of 0.9% NaCl. Control group was consisted of

compared to native beta cells. Degeneration of insulin- producing beta cells in Langerhans islets is a hallmark of insulin-dependent T1D. Recent research showed that the loss of beta cells is at least partly caused by an on-going inflammation . In this regard, Alnek, et al. observed an increased abundance of growth factors, including Granulocyte intact mice. Control animals were administered a single intratracheal 0.1 ml 0.9% NaCl. We evaluated the effect of reserpine on the amount of Lin- - cells, HSCs and HPCs in bone marrow, pancreata and blood of mice in 7 min (group 2) and 2 hrs (group 3) after injection

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