

Therapies for Type 1 Diabetes: Current Scenario and Future Perspectives

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ABSTRACT: Type 1 diabetes (T1D) is caused by autoimmune destruction of insulin-producing β cells located in the endocrine pancreas in areas known as islets of Langerhans. The current standard-of-care for T1D is exogenous insulin replacement therapy. Recent developments in this field include the hybrid closed-loop system for regulated insulin delivery and long-acting insulins. Clinical studies on prediction and prevention of diabetes-associated complications have demonstrated the importance of early treatment and glucose control for reducing the risk of developing diabetic complications. Transplantation of primary islets offers an effective approach for treating patients with T1D. However, this strategy is hampered by challenges such as the limited availability of islets, extensive death of islet cells, and poor vascular engraftment of islets post-transplantation. Accordingly, there are considerable efforts currently underway for enhancing islet transplantation efficiency by harnessing the beneficial actions of stem cells. This review will provide an overview of currently available therapeutic options for T1D, and discuss the growing evidence that supports the use of stem cell approaches to enhance therapeutic outcomes.

KEYWORDS: Type 1 diabetes, insulin therapy, islet transplant, immune therapies, mesenchymal stem cells, induced pluripotent stem cells, endothelial colony forming cells.

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Introduction

Type 1 (T1D) and type 2 (T2D) diabetes are the 2 major forms of diabetes, although a recent study proposed that there are actually 5 types of diabetes.¹ While T2D accounts for >85% of global cases, there has been a steady increase (3%–5% annually) in cases of T1D (*IDF Diabetes Atlas 8th Edition*). Tight glucose control during early stages can decrease the susceptibility for progressing towards multi-system complications of microvascular and macrovascular endpoints.² The pathogenesis of T1D is driven by destruction of insulin releasing pancreatic beta cells, accompanied by cellular invasion by both CD4+ and CD8+ T cells, which results in reduction of beta cell mass. Although genetic predisposition is believed to play a role, T1D is mainly a polygenic disease where genetic factors are controversial.³ As such, the rise in global incidences cannot be completely attributed to genetic susceptibility, and exposure to environmental factors also has an important role in progression from islet autoimmunity to clinical T1D. Nevertheless, there are currently more than 50 genetic loci that have been identified using genome-wide association studies and meta-analysis.⁴ Out of many genetic variabilities, polymorphisms within the HLA (human leucocyte antigen) located on chromosome 6 have been implicated in nearly 40% to 50% of cases of T1D.⁵ Other widely reported genetic variations are polymorphisms within the insulin gene (*Ins-FNTR*, *INS/M2*) located at chromosome 2,⁶ CTTLA-4 gene (cytotoxic T lymphocyte antigen-4) located on chromosome 2,⁷ and (Arg620Trp) PTPN22 (protein tyrosine phosphatase non-receptor type 22) located on chromosome 1.⁸ There is a small percentage of T1D

patients (<10%) who display no evidence of autoimmune response and as such are categorised as type 1B diabetic or idiopathic diabetic population.⁹

According to *IDF Diabetes Atlas 8th Edition*, the number of young people (<20 years) living with T1D worldwide is more than 1 million, and if current trends continue, the number of cases is expected to increase by more than 100 000 every year. In the United Kingdom, there are currently 400 000 cases of patients diagnosed with T1D, out of which 29 000 are children (*IDF Diabetes Atlas 8th Edition*). In 1986, a model was proposed by Eisenbarth¹⁰ to understand pathophysiology of T1D. This model described clinical T1D development as a multi-step pathological process. This work also highlighted opportunities for therapeutic interventions at various stages of disease progression, including primary prevention prior to autoimmune activation, secondary treatment after the immune destruction of pancreatic islets, and tertiary therapy after the clinical onset of T1D.¹⁰ As a result, there have been many advancements made in the past decade for the treatment of T1D. This article will focus on recent progress made for the development of novel therapies for T1D.

Insulin Replacement Therapy

Type 1 diabetes is a chronic disease that eventually leads to complete loss of insulin due to destruction of β cells. Also, the lack of appropriate islet cell repair mechanisms which ultimately affects glycaemic control. As a result, insulin replacement therapy is currently the first-line therapeutic option for treating T1D. The use of exogenous insulin as a therapy for T1D was first described by



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