Taming the immune system: Advances in targeted therapies for type 1 diabetes.

Julian Chan*

Department of Medicine and Therapeutics, , The Chinese University of Hong Kong, Hong Kong Special Administrative Region, China

Introduction

Type 1 diabetes (T1D) is an autoimmune disease where the body's immune system mistakenly attacks and destroys insulin-producing beta cells in the pancreas. Unlike Type 2 diabetes, which is primarily caused by lifestyle factors, T1D is an autoimmune condition that requires lifelong management through insulin therapy. Recent scientific advances in immunology and diabetes research have opened new doors for targeted therapies aimed at modulating the immune system, offering hope for better management and, potentially, a cure for T1D [1].

In Type 1 diabetes, the immune system targets and destroys the beta cells in the pancreas. These cells are responsible for producing insulin, a hormone that regulates blood glucose levels. Without adequate insulin production, individuals with T1D must rely on external insulin injections or pumps to manage their blood glucose [2].

The underlying cause of this autoimmune attack is not entirely understood, but it is believed to involve a combination of genetic susceptibility and environmental triggers, such as viral infections or dietary factors. Research has shown that specific immune cells, known as T-cells, play a critical role in initiating and sustaining the autoimmune process in T1D. These T-cells mistakenly recognize beta cells as foreign invaders and begin to destroy them. The result is a lack of insulin, leading to hyperglycemia and other complications [3].

The concept of targeted therapies for autoimmune diseases like Type 1 diabetes is rooted in the idea of specifically modulating the immune system to halt the attack on beta cells while preserving the body's ability to fight off infections. Traditional treatments for T1D focus on managing blood glucose levels, but they do not address the underlying immune dysfunction. Targeted therapies aim to stop or slow the progression of the disease by preventing the immune system from attacking the pancreas [4].

Several promising avenues of research have emerged in recent years, focused on immune modulation and reprogramming. These therapies aim to either suppress the autoimmune response or promote immune tolerance to beta cells. Below are some of the most promising targeted therapies currently being explored [6].

Monoclonal antibodies are laboratory-made molecules that can target specific proteins or immune cells. In the context of T1D, monoclonal antibodies are being developed to target immune cells responsible for attacking beta cells. For example, **teplizumab**, a monoclonal antibody that targets the T-cells involved in the autoimmune process, has shown promising results in clinical trials. It has been shown to delay the onset of Type 1 diabetes in high-risk individuals and may help preserve beta cell function in newly diagnosed patients [6].

Co-stimulatory signals are molecules that help activate immune cells, including the T-cells that attack beta cells. By blocking these signals, it may be possible to prevent the activation of the immune cells responsible for the destruction of pancreatic beta cells. **Abatacept**, a drug originally developed for rheumatoid arthritis, is one example of a drug that works by inhibiting these co-stimulatory pathways. Early studies have suggested that abatacept can modulate the immune response in T1D patients and preserve beta cell function [7].

Another promising approach involves promoting immune tolerance, where the immune system learns to recognize beta cells as a natural part of the body rather than as foreign invaders. This strategy focuses on inducing a state of immune unresponsiveness to beta cells, effectively "tolerating" them and preventing further autoimmune attacks [8].

Vaccine-like therapies are being developed to train the immune system to tolerate beta cells. One such therapy is **Diamyd**, a vaccine-based treatment that uses a small portion of the insulin protein to help the immune system recognize and tolerate insulin-producing beta cells. Clinical trials have shown that Diamyd can reduce the immune attack on beta cells and preserve insulin production.Cell-based therapies involve transplanting regulatory T-cells (Tregs), which are immune cells that help suppress autoimmune responses. By increasing the number of Tregs or enhancing their function, researchers hope to restore immune tolerance to beta cells and prevent further autoimmune attacks. Early trials using Treg-based therapies have shown potential, but much more research is needed to refine these treatments and assess their long-term effects [9].

While these targeted therapies for Type 1 diabetes hold great promise, they are still in the early stages of development.

***Correspondence to : **Julian Chan,** Department of Medicine and Therapeutics, , The Chinese University of Hong Kong, Hong Kong Special Administrative Region, China. E-mail: jlan@chn

***Received:** 30-Aug-2024, Manuscript No. AADY-25-158376; **Editor assigned:** 02- Sep -2024, PreQC No. AADY-25-158376 (PQ); **Reviewed:** 11- Sep -2024, QC No. AADY-26-158376;**Revised:** 16-Sep-2024, Manuscript No. AADY-25-158376; **Published:** 25- Sep -2024, DOI: 10.36841/aady-8.5.222*

***Citation:** Chan J: Taming the immune system: Advances in targeted therapies for type 1 diabetes. J Diabetol. 2024;8(5):222*

Much more research is needed to understand their long-term effectiveness and safety. However, the advancements made so far provide hope for a future where the immune system's attack on beta cells can be controlled or reversed, potentially leading to better management of Type 1 diabetes or even a cure [10].

Conclusion

As science continues to unravel the complexities of the immune system and autoimmune diseases, the development of targeted therapies for Type 1 diabetes represents a major step forward in the quest to tame the immune system and ultimately improve the lives of those living with this challenging condition.

References

1. Adams OP. The impact of brief high-intensity exercise on blood glucose levels. Diabetes, metabolic syndrome and obesity: targets and therapy. 2013:113-22.
2. Güemes M, Rahman SA, Hussain K. What is a normal blood glucose?. Archives of disease in childhood. 2016;101(6):569-74.
3. Bruno A, Biller J, Adams Jr HP, Clarke WR, Woolson RF, Williams LS, Hansen MD. Acute blood glucose level and outcome from ischemic stroke. Neurology. 1999;52(2):280-.
4. Gupta S, Nayak MT, Sunitha JD, Dawar G, Sinha N, Rallan NS. Correlation of salivary glucose level with blood glucose level in diabetes mellitus. Journal of Oral and Maxillofacial Pathology. 2017;21(3):334-9.
5. Karter AJ, Ackerson LM, Darbinian JA, D'Agostino Jr RB, Ferrara A, Liu J, Selby JV. Self-monitoring of blood glucose levels and glycemic control: the Northern California Kaiser Permanente Diabetes registry∗. The American journal of medicine. 2001;111(1):1-9.
6. Lösche W, Karapetow F, Pohl A, Pohl C, Kocher T. Plasma lipid and blood glucose levels in patients with destructive periodontal disease. Journal of clinical periodontology. 2000 (8):537-41.
7. Donohoe RT, Benton D. Cognitive functioning is susceptible to the level of blood glucose. Psychopharmacology. 1999;145:378-85.
8. Ripsin CM, Kang H, Urban RJ. Management of blood glucose in type 2 diabetes mellitus. American family physician. 2009;79(1):29-36.
9. Ajabnoor MA. Effect of aloes on blood glucose levels in normal and alloxan diabetic mice. Journal of ethnopharmacology. 1990;28(2):215-20.
10. Hoseth E, Joergensen A, Ebbesen F, Moeller M. Blood glucose levels in a population of healthy, breast fed, term infants of appropriate size for gestational age. Archives of Disease in Childhood-Fetal and Neonatal Edition. 2000;83(2):F117-9.

Citation: *Chan J: Taming the immune system: Advances in targeted therapies for type 1 diabetes. J Diabetol. 2024;8(5):222*