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## Introduction

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia due to absolute and relative insulin deficiency. Type 1 diabetes mellitus also known as insulin dependent diabetes mellitus accounts for 5-10% of all causes of the syndrome, is a T-cell-mediated autoimmune disease that begins, in many cases, three to five years before the onset of clinical symptoms, continues after diagnosis, and can recur even after islet transplantation. The effector mechanisms which is responsible for the destruction of beta cells involves the action of cytotoxic T cells as well as soluble T-cell products [1,2].

## Immunosuppressive agents and Diabetes Mellitus

These are the agents which decrease the destruction of pancreatic beta cells. One of the examples for the immunosuppressive agent is Cyclosporin A which blocks cytokine production by all T cells thus limiting production of the T-cell, it also prevents the secretion of cytokines, which are important direct mediators of beta cell destruction, these include Interferon C (IFN-c) and Tumor Necrosis Factor alpha (TNF-a) [3,4]. Cyclosporin A, has been reported to decrease destruction of beta cells [5]. Although Cyclosporin A targeted cytokine production, other broad-spectrum immunosuppressive regimens also may be effective in preventing the loss of insulin production [6]. These immunosuppressive agents are nephrotoxic and have other side effects making it highly inappropriate for long-term uses [6].

## Antigen Specific Immunization

These strategies are based on the fact that a response to antigen is affected by many factors which include the antigenic signal strength, co-stimulation, and the cytokine environment. Therefore, by modulating these parameters, it is possible to divert pathogenic responses of the antigens into a protective, nonpathogenic response [4,6]. In addition to modifying the strength of the T-cell receptor signal with altered ligands or adjuvants, the prevention of antigen can be altered [7,8,9]. By this way type 1 Diabetes Mellitus can be prevented by inducing immune regulation to the administered antigen.

## Anti-CD3 Antibodies

Anti CD 3 molecules contain FcR-binding portion which is responsible for T-Cell activation signals and other effects of T-Cells, thus by eliminating FcR binding portion of Anti CD3 molecule type 1 diabetes mellitus can be prevented. It has been reported that non-FcR binding antibody induces previously activated T-Cells but naïve cells were unaffected. Also, the other inhibitory effects were limited

to the previously activated T-helper cells- which are involved and are present in pancreas of subjects with type 1 diabetes mellitus. Anti CD 3 antibody molecule may induce tolerance to autoimmune destruction of pancreatic beta cells preventing diabetes mellitus type 1. The non-FcR binding antibodies activate signal to T cells resulting in release of Interleukin 10 (IL 10). The conventional Anti CD3 Antibodies release IFN-c [10-13].

## Anti-CD20 Antibodies

These molecules inhibit B cells, it has been reported that anti CD 20 Antibodies also provoke C-peptide responses. Through their action prevents type 1 diabetes but long-term action has not been studied in detail [4].

## References

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