J Clin Cell Immunol 2018, Volume 9
DOI: 10.4172/2155-9899-C6-064

3rd International Conference on Autoimmunity

November 26-27, 2018 | Dublin, Ireland

The role of programmed death-1 (PD-1) receptors and programmed death receptor ligand-1 (PD-L1) co-inhibitory pathway in the regulation of autoimmune diabetes mellitus (type 1 diabetes mellitus)

Abdelhakim Ezzat Ali Elyamani
Medical Research Institute (MRI), Alexandria University, Egypt

Diabetes type 1(T1D) is an autoimmune disease characterized by disability of the body to make insulin as a result of pancreatic beta cells (β cells) destruction. The onset most often occurs in childhood, but the disease can also develop in adults in their late 30s and early 40s. T1DM management remains a daunting clinical challenge. International Diabetes Federation (IDF) estimates that there are 34.6 million people with diabetes in the Middle East and North Africa, a number that will almost double to 67.9 million by 2035. Despite on-going technological advances, the majority of affected patients are not able to achieve recommended glycemic targets. Daily insulin injections are the standard of care, but they are not a cure. Due to artificial blood glucose regulation, T1D patients remain at an increased risk of heart and kidney disease, blindness and peripheral neuropathy. T1DM occurs in individuals with underlying genetic risk, coupled with synergy from one or more heretofore poorly characterized environmental triggers. Clinical evidence demonstrated that CD4+, CD8+ T cells and B cell-mediated autoantibody are critical for beta cell destruction. In fact, B cell-mediated autoantibody production against islet antigens (specifically, insulin autoantibody) precedes T1D onset and is currently the only immunological biomarker of disease progression. Programmed death-1 (PD-1) is a T cell inhibitory receptor, and it is highly expressed on recently activated effector T cells as well as chronically stimulated (anergic) CD4+ and (exhausted) CD8+ T cells, thus limiting their antiviral and antitumor activity. Blocking PD-1 signaling has the potential to reinvigorate anergic or exhausted cells. This spurred the development of PD-1 pathway inhibitors (checkpoint blockade) for the treatment of advanced malignancies. Deficiency in, or blocking PD-1 from interacting with its ligand programmed death ligand-1 (PD-L1), accelerates T1D onset in NOD mice.

hakimyamani@yahoo.com