An In-Depth Look into Identifying the Differentiation Pathway and Cell Type of Dual-Receptor Expressing Lymphocytes in Type 1 Diabetes

Davis, Hannah (School: Hayti Senior High School)

Type 1 diabetes (T1D) is an auto immune disease driven by auto antibodies that target insulin producing beta cells in the pancreas. Currently there are no prevention methods for T1D. However, in May 2019, a new type of cell that bears lineage markers of both B and T lymphocytes was discovered. Researchers propose that this dual-receptor expressing (DE) lymphocyte binds to the HLA-DQ8 protein during dynamic simulations to mimic insulin and trigger diabetes-specific T Helper cells. These DE cells are hypothesized to be the fundamental factor that causes T1D. Genomic immune cell cDNA from the designated Bio Project was analyzed by use of RNA-seq differential gene expression analysis. Data from bioinformatic analysis supports that the DE lymphocyte is a B lymphocyte mimicking NKT responsible for beta cell islet destruction due to the high levels of perforin and granzyme excretion. The cell function of the DE cell potentially eliminates outside T lymphocyte involvement in islet destruction and defines the DE cell as the primary cause of disease. Furthermore, the unique cell typing of the DE cell implies a closer connection between the development of cells within the innate and adaptive immune systems. Finally, the signaling pathway of the DE cell shows implications in other autoimmune diseases and presents a never before identified cell function. The development, cell typing and function of the DE cell provides insight into T1D disease progression, cellular signaling pathway, and yields potential treatment and prevention opportunities.

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