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Summer Undergraduate Research Celebration
Project: Investigating Myc-Knockout T Cells to Re-engineer Immune Response

Abstract

Upon chronic or long-term stimulation of antigen proteins during infections, T cells (CD8+) are limited in their function as they differentiate into exhaustive states. During activation, T cells reprogram their metabolic pathway to use glycolytic, pentose-phosphate, and glutaminolytic pathways. Myc transcription factor is induced upon T cell activation and highly involved in metabolic pathway linking glutaminolysis to biosynthesize energy for T cells. There is growing evidence that shows Myc plays an essential role in development, differentiation, and activation of immune T cells as it progresses between progenitor-like and exhaustive states. This study investigates the effect of Myc-deleted gene in T cells as it undergoes a chronic antigen response simulated by *in vitro* T cell exhaustion assay. Flow cytometry analysis revealed that more Myc-knockout (Myc-KO) T cells expressed Tim3, which is associated with terminal exhaustion. Furthermore, more control cells expressed CD62L, indicating that Myc is involved in T cell progression towards effector-like state to memory T cells. By understanding the role of Myc in T cell activation, we can manipulate the immune response to avoid overstimulation and exhaustive states and improve immunotherapies for chronic disease.