

The requirement of ST3Gal-I sialyltransferase for lymphocyte homeostasis and stimulation

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Changes in glycosylation accompany T cell development and activation, and these are thought to influence T cell trafficking and antigen sensitivity. During thymic development T cells change in their sialylation state, and previous data from our lab indicate this influences the ability of CD8 to interact directly with MHC Class I molecules (termed non-cognate binding). Deficiency in a developmentally regulated sialyltransferase called ST3Gal-I leads to a profound defect in peripheral CD8 T cells, and it has been proposed that ST3Gal-I is key in regulating non-cognate CD8 binding. Here we studied non-cognate CD8 binding and T cell homeostasis in ST3Gal-I deficient mice. Surprisingly, we find that ST3Gal-I deficiency does not appear to influence CD8 non-cognate binding during T cell development. However, the ST3Gal-I deficient T cells are intrinsically impaired in their expansion when responding to cognate antigen, but survival in the presence IL-7 is not affected in vitro. In addition, our data shows ST3Gal-I deficiency dramatically impairs lymphocyte recovery from lymphoid organs after adoptive transfer in vivo, and this defect is not corrected by antigenic stimulation.