Public Abstract
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T lymphocytes are critical mediators of the adaptive immune response. T cell receptor (TCR) mediated c-JUN NH2-terminal kinase (JNK) activation is required for mounting proper T cell mediated immune responses. However, little is know as to how JNK activation is coupled to the TCR. This dissertation shows that the scaffold protein Plenty of SH3s (POSH) is required for optimal JNK activation and effector function in both CD4+ and CD8+ T cells. Additionally, this work shows that POSH is dispensable for JNK activation and positive and negative selection in developing thymocytes. Thus, POSH couples the TCR to JNK activation in a cell type and developmental stage dependent manner. This work also revels a novel target for the treatment of autoimmune disorders such as Type I Diabetes and Multiple Sclerosis.