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TNF family members are essential for the regulation of cell survival, differentiation and proliferation. T cells express a wide array of TNF receptors known to both positively and negatively regulate T cell responses including cell division, effector function, survival and memory development. TNF receptor engagement is believed to provide co-stimulatory signals that augment signal transduction pathways initiated by T cell receptor engagement. TRAF6 is an important signaling adaptor downstream from both TNFR family members and IL-1/Toll Like Receptors (TLRs). Although it has been well established that TRAF6 plays a critical role in the innate immune system, a role for TRAF6 in T cells has not been described. To address this, we generated mice with TRAF6 deletion specific to T cells (TT6). Initial analyses reveal that TT6 mice have approximately one half the number of T cells of littermate control mice, with an especially significant decrease in total $CD8^+$ T cell numbers. Upon stimulation *in vitro*, both $CD4^+$ and $CD8^+$ T cells hyperproliferate and $CD4^+$ T cells produce an excess of Th2 cytokines. TT6 mice have an accumulation of $CD44^{hi}$, $CD62L^{lo}$ $CD4^+$ T cells in the periphery, elevated levels of immunoglobulin isotypes in the sera, and a marked expansion of B ells in the lymph nodes. H/E staining of organs from 4 month TT6 mice reveal mononuclear cell infiltrates in liver, lung and kidney. In addition, anti-DNA antibodies are detected in the sera of these mice. These preliminary findings indicate that TRAF6 deletion results in T cell intrinsic defects and may play a role in the maintenance of peripheral tolerance.