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Tissue resident effector memory CD8⁺ T cells can be tolerized in vivo

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Exposure of T cells to antigen under non-inflammatory conditions results in tolerance and is potentially a potent method to prevent or treat autoimmune disease and graft rejection. However, it is not known whether all T cell subsets are equally susceptible to tolerance induction. In this report we have assessed the susceptibility to tolerance induction of tissue resident memory CD8 T cells induced by intranasal infection with influenza virus. A single intravenous injection of a H-2K^d class I binding peptide (NP peptide), derived from the nucleoprotein of the PR8 strain of influenza virus, specifically eliminated the majority of NP-specific memory CD8 cells present in lymphoid tissues as well as extra-lymphoid tissues, including lung and liver. A notable exception was the inability of soluble peptide to reduce the number of memory T cells present in the CNS, presumably due to an inability of peptide to cross the blood-brain barrier. Such tolerance reduced the level of responsiveness to a secondary viral challenge, resulting in a significant reduction in the number of activated memory cells in all tissues, including the CNS. In contrast to the ability of soluble antigen to tolerize both central and tissue-resident memory cells, the introduction of cell bound antigen was effective in tolerizing central memory cells, yet had little impact on tissue-resident memory cells. Therefore, we conclude that peptide antigen is highly effective in tolerizing all central and effector memory CD8 T cells, including tissue-resident CD8 cells.

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