

Understanding the role of CD43 in CD8 T cell activation and trafficking

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Following T cell activation, CD43 expression as measured by the 1B11 mAb increases dramatically on effector T cells, and then decreases on memory T cells. Following viral infection of CD43^{-/-} mice, effector and memory CD8 T cell generation were relatively unaffected. However, we found delayed trafficking of CD8 T cells to the CNS of infected CD43^{-/-} mice. Additionally, we demonstrated delayed contraction of the CD8 T cell response due to decreased apoptosis of effector T cells. To understand the biochemical mechanism(s) of these differences we examined tyrosine phosphorylation of naïve and effector CD8 T cells from ^{-/-} versus ^{+/+} mice. While tyrosine phosphorylation of effector CD8 T cell populations was indistinguishable, surprisingly, we found increased levels of phosphorylated-c-cbl in naïve CD8 T cells from CD43^{-/-} mice compared to ^{+/+} mice. A more detailed analysis of transgenic CD43^{-/-} T cell activation following peptide stimulation revealed that despite increased expression of c-cbl, the TCR specific responses examined remain similar. One defect that was observed revealed that CD43^{-/-} T cells respond suboptimally to chemokine-induced migration. How this migration difference impacts homing to the CNS is currently under investigation.