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Defining the role of CD69 in the formation of resident memory CD8⁺ T cells

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Abstract

Resident memory CD8⁺ T cells (T_{RM}) reside in nonlymphoid tissues. There, they play a key role in preventing reinfection by exerting cytotoxic and inflammatory functions upon exposure to previously encountered pathogens. CD69 is often used as a definitive marker of T_{RM} cells. CD69's interaction with the G-protein-coupled receptor S1PR1 has been identified as one mechanism by which CD69 can regulate tissue residency. However, the functional requirement for CD69 in promoting the generation and maintenance of CD8⁺ T_{RM} under a wide variety of circumstances remains unclear. We explored the role of CD69 in tissue residency using co-transfer of antigen specific CD69 sufficient and deficient CD8⁺ T cells in the context of acute LCMV, Influenza, and VSV infections. Strikingly, we found that CD69 was not necessary for T_{RM} establishment in most tissues, although it can promote T_{RM} localization under some circumstances. This seems to be influenced by the focal point of infection. Interestingly, the kidney appears to rely on CD69 for tissue residency with every model pathogen examined. We propose that the requirement for CD69 is context dependent rather than absolute, and that a combination of factors, including tissue microenvironment and infectious agent, dictate CD69's influence on development of CD8⁺ resident memory.

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