

## Regulation of memory T cell responses to influenza virus infection

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Influenza virus infection elicits memory CD8<sup>+</sup> T cells that persist for the life of the individual and retain the capacity to mediate accelerated clearance of secondary virus infection. The recall of this immunological memory during secondary influenza virus infections in the lungs occurs in two phases. The early phase of the response is mediated by resting memory CD8<sup>+</sup> T cells that are rapidly recruited into the lungs from the circulation and the late phase of the response is mediated by proliferating effector CD8<sup>+</sup> T cells that have been activated by antigen. While it is clear that the accumulation of antigen-specific memory T cells in the lungs is an orchestrated event, relatively little is known about the underlying mechanisms that drive this recruitment process. To address this issue, we have analyzed the role of the CCR5 and CXCR3 chemokine receptors in regulating memory CD8<sup>+</sup> T cell migration to the inflamed lung. We show that different chemokines and their corresponding receptors are involved in each phase of the response. Furthermore, T cells that are recruited into the lungs are induced to express the effector molecules IFN- $\gamma$  and granzyme B. This presentation will discuss the inflammatory mechanisms that regulate chemokine receptor expression and the acquisition of effector functions in memory T cells following influenza virus infection.