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Reconstitution of the lymphoid compartment by naive T cells

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Keywords

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Context

CD8$^+$ or CD4$^+$ T lymphocytes die in the absence of MHC class I or class II molecules. This indicates that the interaction between the T cell receptor (TCR) and self peptide/MHC molecules delivers survival signals in the peripheral T cell compartment. During the recovery from profound lymphopenia *in vivo*, peripheral T cell numbers recover rapidly and acquire a memory T cell phenotype, a process termed homeostatic proliferation. Until now, this process was thought to involve the reconstitution of the T cell pool by memory T cells. Using a TCR transgenic model, the Bevan laboratory explored whether this process might involve the expansion of naive CD8$^+$ T cells.

Significant findings

Following adoptive transfer of naive TCR transgenic CD8$^+$ T cells into syngeneic irradiated recipients, >80% of donor T cells proliferate and express a memory-like phenotype, acquiring CD44, Ly6 and interleukin-2RÎ±, but not CD25, CD49d or CD69. Nor do they downregulate CD62L or the TCR. The proliferation rate stabilises once the lymphoid compartment approaches normal cellularity, with T cells reverting to the naive phenotype. Despite this expansion, T cells do not exhibit CTL effector function in the absence of exogenous antigen; however, transfer into RAG1(-/-) hosts leads to sustained proliferation and CTL activity.

Comments

The authors propose that sustained expansion of naive T cells occurs under circumstances where the lymphoid compartment fails to reconstitute. This process may be beneficial to the host. Since T cell expansion is driven by peripheral self antigens, these findings also have implications for understanding

the mechanisms for persistent autoreactivity of $CD4^+$T cells in autoimmunity and immunodeficiency, diseases where profound T cell lymphopenia may be a feature.

Methods

Adoptive transfer, TCR transgenic mice, RAG-1 deficient mice

References

1. Goldrath W, Bogatzki LY, Bevan MJ: Naive T cells transiently acquire a memory-like phenotype during homeostasis-driven proliferation. J Exp Med. 2000, 192: 557-564.