

POSTER PRESENTATION

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IL7 signaling confers polyfunctionality to antitumor CD4+ T cells

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From Society for Immunotherapy of Cancer 29th Annual Meeting
National Harbor, MD, USA. 6-9 November 2014

Mounting evidence indicates that polyfunctional T cells, effector T cells capable of simultaneously producing multiple pro-inflammatory cytokines, are more efficacious in controlling infection and cancer. Our previous study reported that adoptive transfer of tumor-specific CD4+ T cells following chemotherapy gave rise to polyfunctional CD4+ effector cells, characterized by concomitant expression of CD40L, IFN γ , TNF α , IL2 and granzyme B. One unique feature of these highly activated polyfunctional CD4+ effector cells was the high level expression of IL7 receptor, suggesting that IL7 plays a critical role in the generation and maintenance of these cells. In this project, we studied how IL7 signaling confers polyfunctionality to CD4+ T cells. We found that only IL7, but not other IL2 family cytokines can promote the acquisition of polyfunctionality in naïve CD4+ T cells upon antigenic stimulation *in vitro*. IL7 signaling resulted in increased histone acetylation in the promoters of effector molecules including IFN γ and IL2. Mechanistically, PI3K activation was required for polyfunctionality. Administration of rIL7 following chemotherapy and CD4+ T cell adoptive therapy led to enhanced and sustained presence of polyfunctional CD4+ effector cells which mediated durable antitumor effects in mice with advanced B cell lymphomas. Our results provide novel insights into the mechanisms by which IL7 drives the generation of polyfunctional CD4+ effector cells.

Published: 6 November 2014

doi:10.1186/2051-1426-2-S3-P43

Cite this article as: Zhou and Ding: IL7 signaling confers polyfunctionality to antitumor CD4+ T cells. *Journal for ImmunoTherapy of Cancer* 2014 **2**(Suppl 3):P43.

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