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Absence of Grail promotes CD8$^+$ T cell anti-tumor activity

T-cell tolerance is a major obstacle to successful cancer immunotherapy; thus, it is of high priority to develop strategies to break immune tolerance. Here we report that expression of the E3 ubiquitin ligase Grail is significantly up-regulated in CD8$^+$ T cells infiltrated into transplanted lymphoma tumors and Grail-deficiency confers long-term tumor control. Importantly, therapeutic transfer of Grail-deficient CD8$^+$ T cells was sufficient to repress established tumors. Mechanistically, loss of Grail enhanced anti-tumor reactivity and functionality of CD8$^+$ T cells. In addition, Grail deficient CD8$^+$ T cells exhibited increased IL-21R expression and hyper-responsiveness to IL-21 signaling as Grail promotes IL-21R ubiquitination and degradation. Moreover, CD8$^+$ T cells isolated from lymphoma patients expressed high levels of Grail and lower levels of IL-21R compared with normal donors. Altogether, our data demonstrates that Grail is a crucial factor controlling CD8$^+$ T cell function and is a potential target to improve CTL activity.

Biography

Roza Nurieva has received her PhD in 2000 from the Gabrichevsky Research Institute of Epidemiology and Microbiology, Moscow, Russia. Her Postdoctoral Fellowship, which was supported by the Arthritis Foundation, was with Dr. Chen Dong at the University of Washington, Seattle, USA, focusing on understanding the role of co-stimulatory molecules in regulating T-helper cell activation, differentiation and function. She is currently an Assistant Professor in the Immunology Departments at MD Anderson Cancer Center, USA. Her main research goal is to understand the molecular basis of T-cell mediated immune responses with focus on the regulation of cytokine expression and how abnormal immune regulation leads to autoimmunity, inflammation and cancer.

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