

A novel role for Inhibitor of DNA binding-2 (Id2) in the regulation of CD8⁺ T cell immunity

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Transcriptional programs that initiate and sustain the proliferation, differentiation and survival of CD8⁺ T cells during the immune response are not fully understood. We found that Id2, an antagonist of E-protein transcription factors, was upregulated by CD8⁺ T cells during infection and maintained in memory cells. Whereas Id2-deficient CD8⁺ T cells recognized antigen and proliferated normally after infection, they failed to accumulate and demonstrated high levels of apoptosis, resulting in the premature contraction of the effector population. Id2-deficiency also diminished effector memory T cell formation and altered expression of genes that influence survival. However, Id2-deficient cells underwent proliferation and accumulated as well as wildtype cells in response to tolerogenic stimuli or in response to a lymphopenic environment. These data highlight a novel function for Id2 in regulating the magnitude of CD8⁺ T cell responses and the formation of memory cells and suggest a mechanism where Id and E proteins regulate mature T cell survival and differentiation. Current studies are underway to identify the signals that regulate the expression of Id2 and to identify the transcriptional targets of Id2 during the immune response.

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