

THE MIDWINTER CONFERENCE OF IMMUNOLOGISTS POSTER ABSTRACT - 2005

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A20 is an NF- κ B responsive gene that negatively regulates TNF- and TLR-induced NF- κ B and JNK signaling via its ubiquitin-modifying function. While A20 is inducible in most cell lines and tissue types, its lymphoid expression is unique, characterized by high mRNA levels in primary and secondary lymphoid tissue and constitutive protein expression in thymocytes and naïve T cells. No function role for A20 in these cells has thus far been defined. In order to determine if A20 regulates TCR-induced NF- κ B, we studied T cells from A20-deficient mice. A20-deficient T cells have prolonged I κ B α degradation following TCR stimulation. This can be explained by exaggerated IKK kinase activity in these cells, indicating that A20 likely acts at or above the level of the IKK complex. Phosphorylation of JNK is also prolonged in A20-deficient T cells; however, early events such as tyrosine phosphorylation and ERK MAPK activation are normal in T cells lacking A20. A20-deficient T cells also exhibit higher expression of NF- κ B-regulated genes, such as CD25, CD69, and IL-2, following TCR stimulation. These results suggest that A20 plays a novel role in regulating TCR signaling and T cell function.