



PAR-2 Deficient CD4+ T Cells Exhibit Downregulation of IL-4 and Upregulation of IFN- γ after Antigen Challenge in Mice

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Background: To investigate the functional role of protease activated receptor (PAR) -2 in T lymphocytes, we analyzed TCR-mediated inflammatory cytokine production using PAR-2 deficient (KO) and wild type (WT) mice.

Methods: Production of serum IgE and cytokines by spleen CD4+ T cells was determined in OVA-sensitized and OVA-challenged mice of PAR-2 KO in contrast to WT mice. Phosphorylation of JNK1 and 2 was determined by Western blotting.

Results: A reduction in serum levels of IgE and IL-4 production by splenic CD4+ T cells from OVA-sensitized and OVA-challenged KO mice compared to WT mice was observed. By contrast, IFN- γ production was upregulated after antigen stimulation in KO mice. Anti-CD3-mediated phosphorylation of JNK1 was upregulated in splenic CD4+ T cells from KO mice compared to WT mice.

Conclusions: PAR-2 participates in the regulation of T cell cytokine production that may be caused by modulation of JNK1 phosphorylation.

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