

## Self-antigen recognition causes T-cell activation and systemic inflammation in TGFβ1-deficient mice

Ramireddy Bomireddy<sup>1</sup>, Leena J. Pathak<sup>1</sup>, Jennifer Martin<sup>1</sup>, Gregory P. Boivin<sup>2</sup>, George F. Babcock<sup>3,4</sup>, and Thomas Doetschman<sup>1</sup>

Departments of <sup>1</sup>Molecular Genetics, <sup>2</sup>Comparative pathology and <sup>3</sup>Surgery, University of Cincinnati College of Medicine, Cincinnati, OH, 45267; <sup>4</sup>Shriners Hospital for Children, Cincinnati, OH, 45229.

**INTRODUCTION:** TGFβ1 is an important immunoregulatory molecule as suggested by the lethal autoimmune inflammatory phenotype of *Tgfb1*<sup>-/-</sup> mice. To determine the role of TGFβ1 in checking T-cell activation, we have used *Tgfb1*<sup>-/-</sup> DO11.10 mice (ovalbumin specific transgenic TCR) mice to analyze the activation state of T cells. In these mice a milder inflammation is developed than in *Tgfb1*<sup>-/-</sup> mice. **RESULTS:** Downregulation of CD3, TCR and CD62L, and upregulation of LFA-1, CD49d, CD69 and CD44 on CD4<sup>+</sup> T cells reveals that they are nonetheless activated *in vivo*. Surprisingly, analysis of CD25 and intracellular FOXP3 expression levels suggest that CD4<sup>+</sup>CD25<sup>+</sup>FOXP3<sup>+</sup> T<sub>reg</sub> cells are increased in the TGFβ1-deficient DO11.10 mice. **CONCLUSION:** TGFβ1 deficiency does not reduce the generation of T<sub>reg</sub> cells, but it is required for their regulatory function to inhibit autoreactive T-cell activation.

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