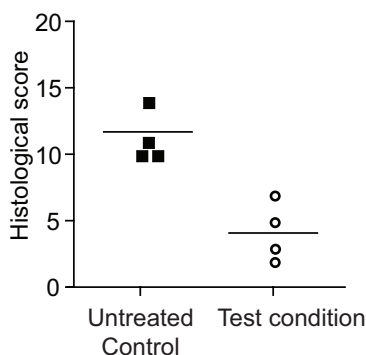
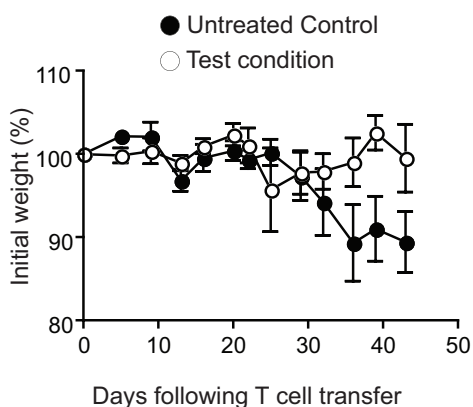


T cell-induced colitis

The transfer of naïve CD4⁺CD45RB^{high} T cells into RAG deficient mice is known to result in intestinal inflammation, which is dependent on the presence of intestinal bacteria. Mucosal inflammation involves the activation of transferred T cells within the draining lymphoid organs followed by their recruitment to the large intestine resulting in a local inflammatory response at this site. Intestinal inflammation involves an influx of inflammatory cells, epithelial cell hyperplasia, mucin depletion from goblet cells, and in severe cases crypt abscess formation and ulceration. Intestinal inflammation is associated with the production of pro-inflammatory cytokines by intestinal APC (IL-12, IL-23, IL-18) and donor T cells (IFN γ , IL-17, TNF α and IL-2). Attenuation of IL-23, IFN γ or TNF α production protects mice against weight loss and intestinal inflammation, and thus disease in this mouse model appears to most closely reflect those patients in which Infliximab treatment is effective. Increased IL-10 production in transgenic mice, or the presence of IL-10 producing CD25⁺ regulatory T cells is also protective against the murine disease.



Experimental readouts:

- Weight loss
- Histological analysis
- Disease pathology scoring
- Quantitative PCR analysis of tissue cytokines and chemokines

Duration:

40-60 days dependent upon experimental readouts

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Service Package I

- Administration of test compounds
- Induction of colitis model
- Measurement of weight loss and colon length

Service Package II

- Histological analysis

Service Package III

- Quantitative PCR analysis of tissue cytokines and chemokines