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IL-10 mediated microvascular and epithelial perturbations in rejecting mouse airway allografts

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Microvascular injuries during inflammation are key cause of transplant malfunctioning and permanent failure, which play a major role in the development of chronic rejection of the transplanted organ. Inflammation induced microvascular loss is a promising area to investigate the decisive roles of regulatory and effector responses. The present study was designed to investigate the impact of IL-10 on immunotolerance, in particular, the microenvironment of the allograft during rejection.

Here, we investigated effects of IL-10 blockade/ reconstitution, and serially monitored regulatory T cells (Tregs), graft microvasculature and airway epithelium in rejecting airway transplants.

We demonstrated that blocking/reconstitution of IL-10 significantly modulate CD4+FOXP3+ Tregs, microvasculature and airway epithelium during rejection. Our findings further highlighted that blockade of IL-10 upregulated proinflammatory cytokines, IL-2, IL-1β, IFN-γ, IL-15 and IL-23, but suppressed IL-5 secretion during rejection, however, reconstitution of IL-10 significantly upregulated CD4+FOXP3+ Tregs, tissue oxygenation/blood flow and airway repair.

Collectively, these findings demonstrate a potential reparative modulation of IL-10 during microvascular and epithelial repair which could provide a vital therapeutic window to rejecting transplants in clinical practice.

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