

Immunomodulation by Interleukin-10 Therapy in Mouse Airway Transplantation

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Abstract : Microvascular injuries during inflammation are key causes 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 the 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 the blocking/reconstitution of IL-10 significantly modulates CD4+FOXP3+ Tregs, microvasculature, and airway epithelium during rejection. Our findings further highlighted that blockade of IL-10 upregulated proinflammatory cytokines, IL-2, IL-1 β , 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.

Keywords : interleukin -10, regulatory T cells, allograft rejection, immunotolerance

Conference Title : ICOTS 2020 : International Conference on Organ Transplantation Surgery

Conference Location : Dubai, United Arab Emirates

Conference Dates : March 19-20, 2020