

**P028** Renal allograft rejection: chemokines enhance the affinity of adhesive CD103<sup>+</sup> T cell interaction with the specific ligand E-cadherin.

**A. Al-hamidi, S. Ali and J. Kirby**

*Applied Immunobiology and Transplantation Research Group, University of Newcastle.*

Acute renal allograft rejection is defined by tubulitis. Many activated T cells that infiltrate to the renal tubules express the  $\alpha E\beta 7$ -integrin (CD103). This integrin plays a crucial role in T cell adhesion to epithelial cells via interaction with specific ligand on epithelial cells called E-cadherin. It was shown that chemo-attractant cytokine can enhance the binding affinity of CD103 to the E-cadherin. This study was designed to assess the role of chemokines in regulation of T cell adhesion to renal epithelium during renal allograft rejection. Recently, we found that MOLT-16 cells which normally express resting CD103 is allowing measurement of the high affinity interaction with immobilised E-cadherin Fc fusion proteins in a static adhesion assay. And 10nM of MCP-3 Chemokine transiently increases this binding affinity in adhesive CD103<sup>+</sup> T cells. However, as pilot data suggests that other CC chemokines produced by kidney cells may play this vital role of increase the affinity binding in addition to its normal function induction of the migration of immune cells to the tubules.