Poster

## [P25-8] P25-8: Immunosuppressive drugs (3): Biomarkers and

pharmacokinetics

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# [P25-8-5] Regulation of cellular and soluble Tim-3 in renal transplant recipients treated with sirolimus- or tacrolimus-based

### immunosuppressive regimens

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#### Background

T-cell immunoglobulin domain and mucin domain-3 (Tim-3) is an activation induced negative regulator of adaptive immune response and is reported to participate in the induction of allograft tolerance. However, whether Tim-3 is affected by different immunosuppressive regimens has not been well documented. Therefore, our aim was to explore whether the tacrolimus (TAC) and sirolimus (SRL) would affect the cellular and soluble Tim-3 in renal transplant recipients (RTs) differently.

#### Methods

We collected periphery whole blood and serum samples from 15 RTs treated with TAC-based regimen, 16 RTs treated with SRL-based regimen and 16 healthy controls. The expressions of Tim-3 on CD4+ and CD8+ T cells were determined by flow cytometry. The concentration of soluble Tim-3 (sTim-3) was determined by enzyme-linked immunosorbent assays (ELISA).

#### Results

Surface expressions of Tim-3 on CD4+ and CD8+ T cells were significantly higher in SRL group than those in HC group (Fig. A and B), while no difference was observed between SRL and TAC groups. In the two RTs groups (SRL and TAC), sTim-3 were significantly increased when compared with HC group, but no difference was observed between SRL and TAC groups (Fig. C). Correlation analysis revealed that sTim-3 was significantly and positively correlated to cellular Tim-3 (Fig. D and E). In addition, serum creatinine (Scr) levels were positively correlated with sTim-3 (Fig. F), but not with cTim-3.

#### Conclusions

Our results indicated that there was a comparable up-regulation of cellular and soluble Tim-3 in RTs under different immunosuppressive treatment. Moreover, sTim-3 is positively associated with Scr, suggesting that sTim-3 may be a promising noninvasive biomarker of renal function in RTs.

#### Zoom image

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