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Changes in gd T cell function and gut homing receptors following SIV infection of rhesus macaques

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Background

During simian immunodeficiency virus (SIV) infection changes occur in the gd TCR T cell population. The major subset in mucosal tissue, Vd1, becomes prevalent in peripheral blood relative to the Vd2 subset. gd T cells have the ability to expand in vitro and in vivo making them attractive as cytotoxic effectors against infections like SIV and HIV.

Methods

We studied gut homing receptors and functionality of Vd1 and Vd2 T cells in blood and jejunum of naïve (8 and 3 respectively) and SIV infected (13 and 9 respectively) animals. Vd1+ and Vd2+ T cells were identified by flow cytometry using pan gd TCR-PE and Vd2-FITC mAbs. Intracellular staining for IFN-g and perforin was performed after Phorbol-Myristate-Acetate/Ionomycin stimulation.

Results

Here, the Vd1 subset in SIV infected macaques was not significantly increased in blood, but in jejunum, Vd1 T cells became predominant compared to Vd2 T cells (p=0.0044) and to the level in blood (p=0.0014). Expansion of the Vd1 subset in jejunum was linked to increased expression of the a4b7 gut homing marker on peripheral blood Vd1 T cells (p=0.033). After stimulation, Vd1 and Vd2 T cells in blood produced both IFN-g and perforin. Higher frequencies of Vd1 T cells produced perforin in SIV+ animals compared to naïve (p=0.0186), while IFN-g producing cell frequencies decreased (p=0.0066). Most Vd1 T cells were CD4-CD8- (DN). Those producing perforin were also mainly DN compared to CD4+ and CD8+ Vd1 T cells (p=0.0156).

Conclusion

Expansion of Vd1 T cells in the jejunum is associated with increased trafficking from peripheral blood to the mucosal site. This expanded population with cytotoxic potential could contribute to viremia control.

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