## Retrovirology



Poster presentation

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# P16-18. Regulatory T cell frequencies correlate with T cell activation in chronic HIV-1 infection

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from AIDS Vaccine 2009 Paris, France. 19–22 October 2009

Published: 22 October 2009

Retrovirology 2009, 6(Suppl 3):P247 doi:10.1186/1742-4690-6-S3-P247

This abstract is available from: http://www.retrovirology.com/content/6/S3/P247 © 2009 Angin et al; licensee BioMed Central Ltd.

### **Background**

Regulatory T cells (Tregs) are potent modulators of immunity and may have an impact on T cell regulation and activation in the setting of HIV-1 infection. However, studies on Tregs in HIV-1 infection have shown divergent results and the exact role of Tregs in HIV-1 infection remains poorly understood. We here investigated Treg frequencies, immune activation and HIV-1 specific immunity in elite controllers (EC) and chronic progressors.

#### Methods

We studied the frequency of CD4+CD25+FoxP3+Tregs in 16 HIV-1 elite controllers (HIV-RNA <50 copies/ml), 17 individuals with untreated chronic progressive HIV-1 infection and 9 HIV negative individuals by flowcytometric analysis. We also evaluated T cell activation by measurement of CD38 and HLA-DR expression on CD8 and CD4 T lymphocytes. HIV-1 specific CD8 T cell responses were available for a subset of individuals.

#### **Results**

In concordance with previous data, EC had significantly lower immune activation than chronic progressors (p < 0.005). The frequency of CD4+CD25+FoxP3+ regulatory T cells was positively correlated with T cell activation as measured by frequency of CD8+CD38+T cells (R² 0.42, P = 0.006). There was a strong trend to decreased Treg frequencies in elite controllers and HIV negative individuals compared to chronic progressors. In the elite controller subset HLA-B57 positive EC had lower Treg frequencies

than non-HLA-B57 EC, but this trend did not reach statistical significance.

#### Conclusion

Our data suggest that Treg frequencies positively correlate with immune activation in this cohort of individuals with chronic HIV-1 infection and that elite control is associated with lower T cell activation as previously described. Treg frequencies may be lower in HIV-1 elite controllers compared to chronic progressors but further studies are needed to investigate this finding in more detail and to address the impact of Tregs on HIV-1 specific T cell responses.