Retrovirology

Poster presentation

P04-32. Differential regulation of secondary antibody responses to Gag and Env proteins

G Nabi, V Temchura, C Grossmann, S Kuate, M Tenbusch and K Uberla*

Address: Virology, Ruhr-University Bochum, Bochum, Germany
* Corresponding author

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

Published: 22 October 2009

Retrovirology 2009, **6**(Suppl 3):P60 doi:10.1186/1742-4690-6-S3-P60

This abstract is available from: http://www.retrovirology.com/content/6/S3/P60

Background

Progression to AIDS and loss of CD4+ T cells are associated with a decline in antibody titers to the viral Gag protein, while anti-Env antibodies remain high, suggesting a T cell independent antibody response to Env.

Methods

To test this hypothesis, immunocompetent Balb/c and T-cell deficient nude mice were immunized with non-infectious virus-like particles (VLP) of simian immunodeficiency virus or adenoviral vectors expressing Gag and Env. In addition, B-cells from primed Balb/C mice were transfered into nude mice, to evaluate T-cell dependance of secondary antibody responses.

Results

High levels of antibodies against Gag and Env could only be induced in immunocompetent mice, but not in the immunodeficient mice. Thus, neither cells expressing Env after adenoviral gene transfer nor VLPs induce a T cell independent primary anti-Env antibody response. However, secondary B cell responses to Env, but not to Gag, were observed in immunodeficient mice after transfer of primed B-cells and boosting with VLPs. These T-cell independent secondary antibody levels to Env were lower after stimulation with VLPs modified to contain monomeric membrane bound gp130 and undetectable after injection of soluble gp130.

Conclusion

Membrane-bound trimeric Env seems to be responsible for maintenance of high levels of anti-Env antibodies during progression to AIDS. This T-cell independent secondary antibody response might contribute to viral immune escape by favoring persistence of non-inhibitory antibodies due to competition with T-cell-dependent affinity maturation.