Trama et al. Retrovirology 2012, 9(Suppl 2):P201
http://www.retrovirology.com/content/9/S2/P201

RETROVIROLOGY

POSTER PRESENTATION Open Access

Lack of IgA envelope-reactive antibody producing cells in terminal ileum in early and chronic HIV-1 infection

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From AIDS Vaccine 2012
Boston, MA, USA. 9-12 September 2012

Background

HIV-1 vaccines must induce protective antibodies at mucosal surfaces; the role of IgA in protection remains unknown. The HIV-1 Env antibody response begins ~day 17 after transmission, and derives from a polyreactive memory B cell pool of gut flora-reactive IgG1 and IgA B cells. Whereas the IgG Env antibody response persists years after acute HIV-1 infection, the initial IgA response decreases over the first month. There is also selective destruction of terminal ileum germinal centers in early HIV-1 infection (EHI). To determine HIV-1 IgA responses in gut, we isolated Env-reactive antibodies from ileum from patients in EHI and chronic HIV-1 infection (CHI).

Methods

Single plasma cells (PCs) and IgD- memory B cells were sorted from the ileum and/or blood of 7 EHI and 3 CHI. Antibodies were isolated by PCR amplification of Ig heavy chain V(D)J and light chain VJ genes and characterized by ELISA and Luminex.

Results

Whereas CHI blood memory IgA+ B cells reactive with HIV-1 envelope ranged from 0.20-0.79%, only 0-0.07% of ileum IgA+ B cells were Env-reactive. Of 254 mAbs isolated from EHI ileum, only 3 (1.2%) were HIV-1-reactive. In CHI, 9 (5.7%) of 158 mAb were HIV-1 reactive. None of the HIV-1 reactive ileum antibodies were of the IgA isotype.

Conclusion

HIV-1 envelope reactive IgA+ memory B cells and PCs can be found in the blood, but there is a dearth of HIV-1 reactive memory IgA+ B cells and PCs in ileum in EHI and CHI. Loss of IgA in plasma after acute HIV-1 infection is paralleled by the loss of IgA+ B cells in ileum, and is likely a consequence of HIV-1-induced ileum germinal center apoptosis. For vaccine design, it will be important to determine if mucosal IgA+ B cell loss is due to replicating virus or is triggered by soluble HIV-1 envelope.

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Published: 13 September 2012

doi:10.1186/1742-4690-9-S2-P201
Cite this article as: Trama *et al.*: Lack of IgA envelope-reactive antibody producing cells in terminal ileum in early and chronic HIV-1 infection. *Retrovirology* 2012 **9**(Suppl 2):P201.

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