

Poster presentation

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PI6-16. Gag and Nef specific T-cell responses in HIV-1 infected long-term non-progressors in Uganda

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Background

HIV-1-specific T-cell responses are preserved in HIV-1 infected individuals with non-progressing HIV-1 disease. "Long Term Non Progressors" (LTNPs) were defined as ART naïve individuals infected with HIV-1 for >8 years, maintaining CD4+ T-cell counts >500, and with minimal CD4+ decline over time. We tested the hypothesis that Gag-specific T-cell responses are inversely correlated to disease progression whereas Nef-specific T-cell responses are not.

Methods

17 ART naïve HIV-1 infected patients from the Entebbe cohort in Uganda were recruited and stratified by CD4+ T-cell count, CD4+ decline slopes, and time of enrolment, into 2 groups – 10 LTNP and 7 Rapid progressors (RP). All patients were women. We measured plasma viral load, current CD4 T-cell count, and IFN- γ , IL-2, IL-4 and perforin ELISpot responses to pools of 22 to 34 peptides (18-mers overlapping by 10 aa) based on consensus sequences of Gag and Nef from HIV-1 clades A1, A2 and D. Medians and inter-quartile ranges were calculated and comparisons between groups were performed using the Mann-Whitney U test. Correlations were presented using Spearman's linear correlation coefficients.

Results

IFN- γ responses to GagA2 pool1 were significantly higher in the LTNP than in RP ($p = 0.02$). IL-4 responses to the

GagA2 pool2 were higher in LTNP ($p = 0.07$), whilst perforin responses to the same pool were higher in the RP ($p = 0.06$). IL-2 responses were low and not significantly different between LTNP and RP. IFN- γ , IL-4 or perforin responses did not correlate with CD4 T-cell counts or viral load. All responses to Nef peptides were not significantly different between the LTNP and RP.

Conclusion

IFN- γ Gag HIV-1 specific responses were higher in LTNPs than in RPs confirming previous results. Higher perforin responses seen in RP perhaps indicate accelerated elimination of infected target cells. Non-specific IL-4 responses were high possibly reflecting TH2 responses to common environmental pathogens in the study population.