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Poster presentation

P07-02. Cytotoxic T lymphocyte-mediated immune responses in HIV-1 clade C infected mother-child pairs

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Background

Mother to child HIV transmission represents a significant proportion of HIV/AIDS burden in resource-poor settings. The aim of this study is to characterize specific HIV-specific CTL responses in mother-child transmission pairs and characterize pathways of immune escape in children who acquire HIV-1 from their mothers.

Methods

Sixty mother-child pairs were recruited from clinics in Durban, South Africa. The ages of the mothers ranged from 17–42 years while children ranged from 0.3–10 years. Viral load measurement and CD4 counts were performed on all subjects at 3–6 month intervals. High resolution HLA typing was performed. Comprehensive screening of HIV-1 specific CTL responses by an interferon-gamma ELISpot assay using pools of overlapping peptides (18 mers) spanning all expressed viral proteins was performed.

Results

As predicated, the viral load results showed children to exhibit a significantly higher viral load than their mothers ($P < 0.0001$). Children aged between 3–6 months exhibited a CD4% between 10–42% (median 25%) and children aged between 6–12 months between 8–57% (median 23%). Despite an overall stable CD4 only 40% of all children responded targeting between 1–6 (median 1) overlapping peptide pools. As expected almost 94% of mothers responded showing a broader repertoire of responses targeting 1–20 (median 4) overlapping peptide pools ($P < 0.0038$ However, the viral loads between ELISpot responders and non-responders in both mother and child (0.1179) groups were not significantly different. The viral regions most targeted by responder children were Nef (49%), Gag (17%) and Env (14%). Protective HLA alleles (B57/B81/B27/B13/B3910/B5801) were not associated with lower viral loads among mothers or children in this cohort.

Conclusion

Adults have higher frequency of CTL responses than children. HLA and CTL data alone is not sufficient in determining what enables relative viral control in this cohort. Additional studies are required to better define the immune mechanisms of control in this cohort.