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HIV-1 p24 derived epitopes modulate KIR2DL2-binding to HLA-Cw03

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Background

Recent studies have suggested that HIV-1 can evade Natural Killer (NK)-cell-mediated immunity by mutating viral epitopes to enhance engagement of inhibitory Killer Ig-like receptors (KIRs) expressed on NK cells. However, the precise mechanisms modulating the interaction of inhibitory KIRs and their HLA class I ligands, and the role that HIV-1 epitopes might play in this interaction are not well understood. In this study we investigated whether HLA-Cw3-presented epitopes within HIV-1 p24 Gag can modulate binding of KIR2DL2, an inhibitory KIR, to HLA-Cw03.

Methods

Using tapasin-deficient 721.220 cell line expressing HLA-Cw*0304 we initially screened for HIV-1 peptides that stabilized HLA-Cw*0304 expression using 222 10-mer peptides overlapping by 9 amino acids spanning the entire HIV-1 p24 Gag sequence. Peptides stabalizing HLA-Cw*0304 expression were thereafter investigated for their ability to facilitate binding of a KIR2DL2-IgG fusion construct.

Results

We identified several HIV-1 p24 epitopes that were able to stabilize HLA–Cw*0304 expression. A subset of these epitopes also allowed for binding of KIR2DL2. Currently we are investigating the consequences of KIR2DL2-binding to HLA class I presented HIV-1 epitopes for the antiviral function of primary NK cells from KIR2DL2+ individuals.

Conclusion

Taken together, these studies have identified epitopes within HIV-1 that enhance the binding of the inhibitory NK cell receptor KIR2DL2 to its ligand HLA-Cw3, and are in line with recent data suggesting that the sequence of the HLA class I presented epitope has an important impact on the interaction between KIR and HLA class I (Boyington et al. Nature 2000, Vivian et al. Nature 2011).

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