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DC SIGNR silencing reduces HIV-1 infection in Dendritic cells

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Background
Dendritic cells (DCs) capture HIV-1 from periphery via DC SIGN/R receptors and transfer to T cells. It has been reported that siRNA directed against DC SIGN significantly inhibits HIV infection of DCs. In this study, the expression of DC SIGN/R and its role in HIV-1 infectivity of DCs were assessed.

Methods
DCs were cultured from monocytes from healthy donors and infected with HIV-1 Indian clade C virus. DC SIGN/R expression on DCs was determined by RealTime PCR. The effect of down regulation of this receptor in DCs using DC SIGN/R siRNA on the infection with HIV-1 was assessed. Statistical significance was calculated by Student's t test.

Results
High expression of DC SIGN/R was observed on DCs infected with HIV-1 (p= 0.01). DC sIGNR expression in DCs was maximally downregulated by siRNA at 24 hrs (p=0.002) and was associated with significant reduction in expression of CD40 (p= 0.003), CD80 (p=0.008), CD86 (p=0.007) and P38 MAPK (p=0.005). Transfection of DC SIGNR on DCs at 24 hours followed by infection with clade C HIV-1 demonstrated lower levels of p24 compared to that in untransfected DCs (p=0.0008).

Conclusion
Data demonstrates that down regulation of DC SIGNR expression on DCs decreases activation that in turn may inhibit DC T cell interactions needed for progression of HIV-1 infection. Reduced p24 antigen production may be mediated by P38 MAPK inhibition. Our finding suggests that blocking DC SIGNR expression on DCs may prevent initial binding of HIV-1 and serve as a first step in prevention of HIV-1 infection.

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