Plasmacytoid DCs control HIV latency in resting T-cells by type I IFNα
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Background

We previously demonstrated that myeloid DC (mDC), but not plasmacytoid DC (pDC), induce HIV latency in T-cells in vitro1,2. Since pDC produce high levels of type-I interferon (IFN), we asked whether different IFNs have an effect on the establishment, maintenance and reversal of HIV latency in CD4+ T-cells.

Aims

1. Determine the effect of increasing concentrations of IFNα on productive infection
2. Determine the effect of increasing concentrations of IFN on latency establishment
3. Determine the effect of IFN on latency reversal

Methods

In vitro cell culture set-up

Ex vivo cell culture set-up

Results

Type I IFNα, IFNβ and IFNγ inhibit establishment of latent infection

IFNα activates latent HIV in vitro

IFNα activates latent HIV ex vivo

Conclusions

1. Using an in vitro latency model we have shown that IFNα can inhibit productive infection and can inhibit the establishment of HIV latency. Similar results were obtained with IFNβ and IFNγ.

Implications

IFNα effects on control of latency are complex. It can act as an latency reversing agent but can also block productive infection in activated cells and the virus production induced by TCR stimulation. Understanding these mechanisms may allow targeted use of IFNα.

References