

Poster presentation

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Increased IFN- γ Production by NK and NKT Cells From HIV-1-exposed But Uninfected Individuals

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from 2005 International Meeting of The Institute of Human Virology
Baltimore, USA, 29 August – 2 September 2005

Published: 8 December 2005

Retrovirology 2005, 2(Suppl 1):P85 doi:10.1186/1742-4690-2-S1-P85

Background

Innate immunity is very active at mucosal surfaces, that are the main port of viral entry; it is known that several components of the innate immune response have a direct anti-HIV-1 activity such IFNs and chemokines (1). Interestingly, a report (2) has indicated a high frequency of plasmacytoid dendritic cells and high production of IFN- α in response to Herpes simplex virus infection in long-term non-progressors and long-term survivors, suggesting an important role of these innate mechanisms in the control of HIV-1 infection.

Objective

To establish a relationship between some components of the innate immune system and the phenomenon of natural resistance exhibited by individuals who are continuously exposed sexually to HIV-1 but remain seronegatives (ESNs).

Materials and methods

We evaluated in peripheral mononuclear cells the frequency of plasmacytoid dendritic cells, myeloid dendritic cells, natural killer cells (NK) and NKT cells, and the secretion of IFN- α in unfractionated mononuclear cells stimulated with Herpes simplex virus, as well as the expression of IFN- γ by NK and NKT cells after incubation with PMA/ionomycin, in three groups of individuals: low-risk HIV-1 negative controls (n = 30), sexually ESNs (n = 30), and HIV-1 seropositive individuals (n = 30).

Results

Among the evaluated parameters of the innate response, only the expression of IFN- γ by NK and NKT cells was significantly higher in exposed-seronegative individuals

when compared with controls. As previously reported, HIV-1-infected individuals exhibited a significant decrease in the frequency of myeloid and plasmacytoid dendritic cells, NK cells and invariant NKT cells.

Conclusion

Since it is well known that IFN- γ is effective against HIV-1 *in vitro*, and also activates dendritic cells, NK cells and cytotoxic T lymphocytes (3), this result suggests that the production of IFN- γ might be one of the factors involved in controlling the establishment of HIV-1 infection.