Retrovirology

Poster presentation

P01-04. Investigation of the sensitivity of acute-phase HIV-1 isolates to type I interferons

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Background

Type I interferons (IFNs) stimulate the establishment of an antiviral state in infected and susceptible cells and play a key role in the initial containment of many virus infections. Most viruses have evolved at least one strategy for the evasion of this antiviral response. The acute burst of viral replication in primary HIV-1 infection is associated with a transient elevation in systemic IFN-alpha levels. This study addressed the relationship between acute-phase viral sensitivity to type I IFNs *in vitro* and the prognostically-important setpoint persisting viral load established in subjects with primary HIV-1 infection.

Methods

Primary HIV-1 isolates were derived from a cohort of acutely-infected patients who subsequently went on to establish setpoint persisting viral loads of between 3,000 and 900,000 viral RNA copies/ml. An *in vitro* system using stimulated peripheral blood mononuclear cells was devised to assess the sensitivity of these virus isolates to the antiviral effects of type I IFNs.

Results

Both IFN-alpha and IFN-beta were shown to inhibit the replication of the lab-adapted HIV-1 isolate W6BC *in vitro*. IFN-beta was found to require a significantly shorter exposure time than IFN-alpha for exertion of its full antiviral effect. *In vitro* IFN-alpha and IFN-beta IC50 values were successfully calculated for an initial set of acute-phase primary HIV-1 isolates. The IFN sensitivity of different HIV-1 isolates was shown to vary, and preliminary results suggested a correlation between IFN sensitivity and setpoint viral load. A more extensive group of virus isolates are currently being studied to confirm or deny this hypothesis.

Conclusion

Primary HIV-1 isolates differ in sensitivity to control by type 1 IFNs. Preliminary results suggest that viral IFN sensitivity could be among the factors that impact on the setpoint persisting viral load established *in vivo*.