

CONFÉRENCE

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HIV Persistence in the Setting of Antiretroviral Therapy: When, Where and How HIV hides?

Advances in the treatment of HIV infection have dramatically reduced the death rate from AIDS and improved the quality of life of many HIV-infected individuals. Although lifelong suppression of HIV replication with ART seems possible, side effects, resistance, stigma and cost all contribute to the necessity of finding a cure. It is now clear that ART alone does not eradicate HIV: Even after more than 15 years of intensive and continuous therapy, the spread of the virus resumes within a few weeks upon cessation of ART in all but exceptional cases. The failure to cure HIV infection is believed to result from low-level viral production/replication, the presence of latent replication-competent provirus in resting CD4⁺ T cells, and T-cell dysfunction stemming from persistent immune activation. Current ART does not target the reservoir of long-lived latently infected cells and does not fully restore immune functions. Insights into cellular mechanisms that control HIV gene expression and chronic immune activation suggest that the modulation of immune functions may accomplish both of these goals. Several immunologic strategies aimed at curing HIV infection are currently being investigated. They include the blockade of negative regulators of T cell activation, the administration of the gamma-c cytokine IL-7, HIV immunization and many other potential therapies. Determining both the immunological and the virological impacts of such strategies would be essential to develop successful therapies aimed at reducing the size of the latent HIV reservoir.

Jeudi 23 janvier 2014 à 11h30

Pavillon Claire McNicoll, salle Z-209

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