## Poster presentation

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## **HIV-I infection in dendritic cells in HAART suppressed patients** Irvin M Maldonado<sup>\*1</sup>, Sharilyn Almodóvar<sup>1</sup>, Giselle Marrero<sup>1</sup>, María del C Colón<sup>1</sup>, Juan C López<sup>1</sup>, Jesús Rosado<sup>1</sup>, Carlos Domínguez<sup>5</sup>, René Herrera<sup>4</sup>, Martin Hill<sup>2</sup>, Alberto S Cornier<sup>3</sup> and Eric Lorenzo<sup>1</sup>

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The different pathways of HIV transmission from dendritic cells to T cells and their possible capacity to harbor the virus, indicates the potential of this group of cells to be a viral reservoir, allowing the virus to evade immune responses. Therefore, we isolated in vivo blood dendritic cells (DCs) from HIV-1 infected patients undergoing HAART. Proviral DNA amplification from DCs was obtained from all infected patients except in patients with non-detectable viral loads. A significant finding was that DCs proviral DNA amplification were obtained from three patients with extremely low viral loads, which resembles the normal viral blips in HAART suppression. This DCs viral infection may be responsible for the replenishment of important reservoirs. DCs' capacity to harbor archaic viral quasispecies was also revealed in two of six patients and those quasispecies were at least as archaic as resting CD4+ T cells viral quasispecies. In addition, the DCs proviral DNA of these two patients presented multiple changes in glycosylation patterns equal to resting CD4+ T cells reservoir. Other important analyses of nucleotide distance and phylogeny revealed a close relation between the viral populations of DCs and resting CD4+ T cells in four HAART failure patients. These data suggest that DCs can act as a short-term reservoir and could be partially responsible for the replenishment of other important reservoirs. Due to the well-documented capacity of DCs to pass the HIV infection to T cells and phylogenetic evidence, we suggest that the infection of DCs in HAART suppression may be partially responsible for the replenishment of the resting CD4+ T cell reservoir.