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### **► To cite this version:**

Maryse Peressin, Vincent Holl, Sylvie Schmidt, Thomas Decoville, J Penichon, et al.. P11-16. Transfer of HIV-1 from Langerhans and interstitial dendritic cells to T lymphocytes: protection mediated by antibodies?. *AIDS Vaccine* 2009, Oct 2009, Paris, France. pp.P161, 10.1186/1742-4690-6-S3-P161 . inserm-00663924

**HAL Id: inserm-00663924**

**<https://inserm.hal.science/inserm-00663924>**

Submitted on 27 Jan 2012

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## **PII-16. Transfer of HIV-1 from Langerhans and interstitial dendritic cells to T lymphocytes: protection mediated by antibodies?**

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from AIDS Vaccine 2009  
Paris, France. 19–22 October 2009

Published: 22 October 2009

*Retrovirology* 2009, **6**(Suppl 3):P161 doi:10.1186/1742-4690-6-S3-P161

This abstract is available from: <http://www.retrovirology.com/content/6/S3/P161>

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### **Background**

Langerhans (LC) and interstitial dendritic cells (IDC) present in mucosal tissues are among the first HIV-1 target cells. Infected DC have been shown to transfer the virus to nearby CD4 T lymphocytes. We have demonstrated that neutralizing antibodies (NAb) were able to inhibit the transfer of HIV-1 from monocyte-derived DC to CD4-T lymphocytes. In the context of HIV-1 transmission at mucosal site, we analysed the transfer of HIV-1 from LC and IDC to CD4-T lymphocytes, and determined the capacity of NAb to inhibit this transfer.

### **Methods**

LC and IDC were obtained by differentiation of CD34+ cord blood cells. Immature LC/IDC were infected during 2 hours with HIV-1 and extensively washed prior to the coculture with primary lymphocytes or CD4-T cell lines, in presence or absence of NAb. HIV-1 infection was determined by the detection of infected cells by intracellular p24-staining and flow cytometry analysis, or by the quantification of HIV-1 released in the supernatant by p24-ELISA dosage.

### **Results**

We demonstrated that LC and IDC transfer HIV-1 to T lymphocytes. Moreover, neutralizing antibodies efficiently inhibit HIV-1 replication CD4-T lymphocytes when cocultured with infected LC/IDC. These results show that HIV-1 transfer to CD4-T cells is not resistant to neutralization. Surprisingly, when primary CD4-T or non-

permissive B lymphocytes were cocultured with HIV-1 exposed DC, a strong stimulation of HIV-1 production was detected in LC and IDC. This augmentation was not observed in the coculture of LC/IDC with CD4-T cell lines.

### **Conclusion**

Altogether, these data demonstrate that, in the mucosal context where DC and T or B lymphocytes are in close contact, infected DC may become highly HIV replicating cells. As Ab were able to efficiently inhibit HIV-1 transfer from LC/IDC to T lymphocytes, they should be locally induced at the mucosal site by vaccination to prevent HIV-1 dissemination.