Retrovirology

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# Dichotomous effects of the cofilin kinase LIMK1 on the early steps of HIV-1 infection of CD4 T cells

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*from* Frontiers of Retrovirology: Complex retroviruses, retroelements and their hosts
Montpellier, France. 21-23 September 2009

Published: 24 September 2009

*Retrovirology* 2009, **6**(Suppl 2):O8 doi:10.1186/1742-4690-6-S2-O8

This abstract is available from: http://www.retrovirology.com/content/6/S2/O8



We have previously demonstrated that binding of HIV-1 to the chemokine coreceptor, CXCR4, on resting CD4 T cells activates an actin-depolymerizing factor cofilin to promote the cortical actin dynamic critical for HIV latent infection [1]. The LIM domain kinase 1 (LIMK1) directly phosphorylates cofilin and regulates the actin cytoskeleton. Here, we investigated the role of LIMK1 in HIV-1 infection of resting CD4 T cells and found that HIV-1 infection triggered a rapid, transient activation of LIMK1. We also used siRNA knockdown to inhibit LIMK1 activity and found that knockdown of LIMK1 caused a decrease of F-actin and T cell chemotaxis. LIMK1 also had dichotomous effects on the chemokine coreceptor CXCR4 cycling (Figure 1) and viral DNA synthesis (Figure 2). Our findings are consistent with a model suggesting a multi-functional role of the cortical actin in mediating chemokine coreceptor density, HIV-1 DNA synthesis and intracellular migration.

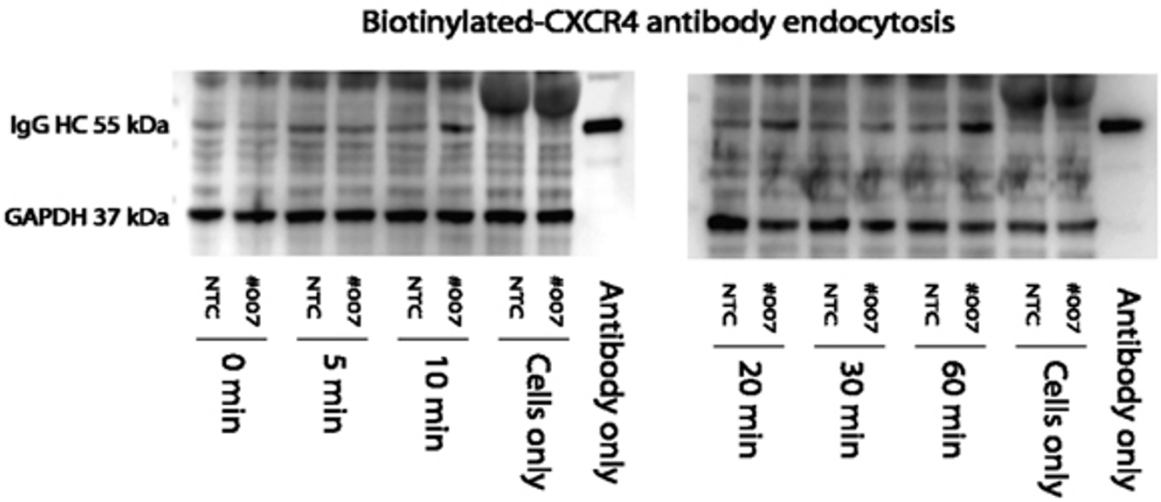


**Figure 1**
**Endocytosis of CXCR4 analyzed by Western blot**.

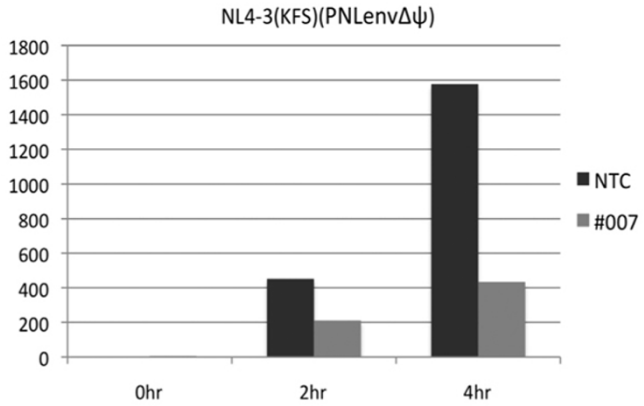


**Figure 2**
**Real-time PCR analysis HIV-1DNA synthesis in LIMK1 knockdown cells (007-LIMK knockdown cells, NTC-control cells)**.

## References

1. Yoder A, Yu D, Dong L, Iyer SR, Xu X, Kelly J, Liu J, Wang W, Vorster PJ, Agulto L, Stephany DA, Cooper JN, Marsh JW, Wu Y: **HIV envelope-CXCR4 signaling activates cofilin to overcome cortical actin restriction in resting CD4 T cells.** *Cell* **134:**782-92.