

Oral presentation

Open Access

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

Paul J Vorster, Alyson Yoder, Dongyang Yu, Yanfang Zheng, Weifeng Wang and Yuntao Wu*

Address: Department of Molecular and Microbiology, George Mason University, Manassas, VA 20110, USA

* Corresponding author

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>

© 2009 Vorster et al; licensee BioMed Central Ltd.

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.

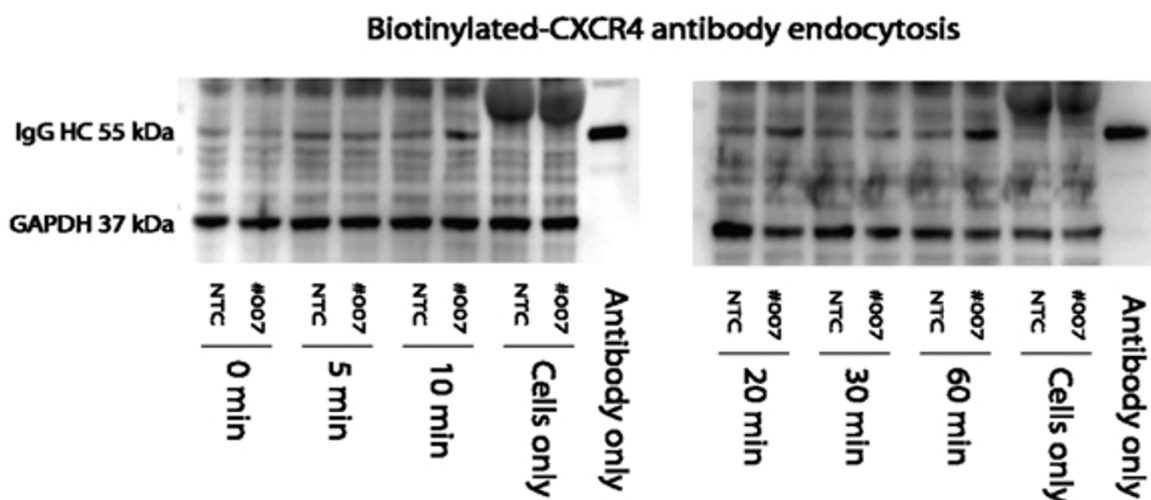


Figure 1
Endocytosis of CXCR4 analyzed by Western blot.

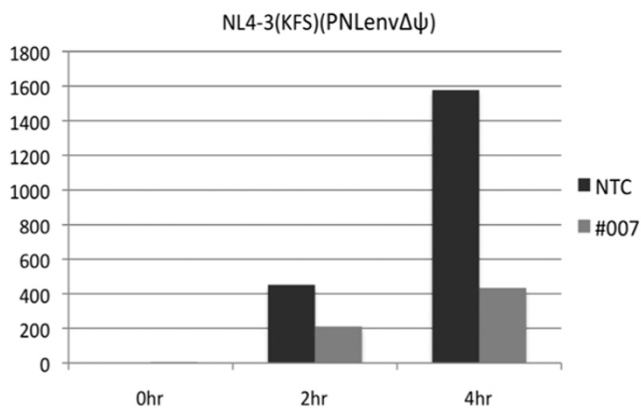


Figure 2
Real-time PCR analysis HIV-1 DNA synthesis in LIMK1 knockdown cells (007-LIMK knockdown cells, NTC-control cells).

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.

Publish with **BioMed Central** and every scientist can read your work free of charge

"BioMed Central will be the most significant development for disseminating the results of biomedical research in our lifetime."

Sir Paul Nurse, Cancer Research UK

Your research papers will be:

- available free of charge to the entire biomedical community
- peer reviewed and published immediately upon acceptance
- cited in PubMed and archived on PubMed Central
- yours — you keep the copyright

Submit your manuscript here:
http://www.biomedcentral.com/info/publishing_adv.asp