Retrovirology

Poster presentation

Impact of the cellular protein Dlg 1 on HIV infectivity and cell to cell transfer

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HIV-1 spread in an infected organism is highly dependent on proper virus assembly and budding for the efficient formation of infectious particles. To achieve this goal, the virus hijacks numerous cellular proteins such as members of the ESCRT pathway, Tip47 or AP complexes. These are generally partners for Gag, Env or both, mediating their intracellular traffic to the site of release, or even favouring their interaction with one another. We have previously identified Dlg1, the human homologue of Drosophila Discs Large protein as a new partner for HIV-1 Gag [1]. Dlg1 underlies the plasma membrane of different cell types (epithelial, neural cells, T lymphocytes) and acts as a scaffold for the formation of multiprotein complexes. We reported that depleting endogenous Dlg1 in T cells does not affect Gag production, maturation or release. Nevertheless, Dlg1 knockdown enhances the infectivity of released virions, which correlates with an increase of the amount of Env in producer cells as well as in viral particles. Depletion of Dlg1 also induces a modification of Gag and Env cellular localisation from the plasma membrane to an apparent internal punctate structure where they colocalize. Thus Dlg1 can be considered as a new negative regulator of HIV-1 virions infectivity.

Dlg1 regulates the immunological synapse, playing a role in lymphocyte polarisation, signal transduction and effector function. Besides, Dlg1 is a partner of Zap70, a kinase involved in both the immunological and the virological synapse formation, also known to facilitate HIV-1 cell to cell transmission. Overall these data led us to question the impact of Dlg1 in HIV-1 cell to cell transmission. To this end we used a cell culture system in which virus release from donor cells is unaffected while virus transfer from donor to recipient cells is severely impaired. The impact of Dlg 1 under these culture conditions will be presented.

References

1. Perugi F, Muriaux D, Ramirez BC, Chabani S, Decroly E, Darlix JL, Blot V, Pique C: **Human Discs Large is a new negative regulator of human immunodeficiency virus-1 infectivity.** *Mol Biol Cell* 2009, **20:**498-508.