Retrovirology

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Oral presentation

Controlling the Virus Output Via Urokinase Receptor and Integrin Signaling

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We have described that either urokinase-type plasminogen activator (uPA) inhibits HIV expression in monocytic U937 and U1 cell lines (*M. Alfano et al., PNAS, 2002, 99:8862-67*). We have observed that uPA inhibited HIV expression exclusively when both uPAR and CD18/CD11b (Mac-1) were co-expressed at the cell surface. A second interactor of uPAR, FPRL1, was abundantly expressed on the surface of both unstimulated and stimulated U1 cells; however, peptide antagonists of FPRL1 did not interfere with HIV expression from U1 cells. Incubation of U1 cells with Trojan peptides expressing RhoA domains reversed the anti-HIV activity of uPA. In addition to cell line infection, uPA inhibited *in vitro* infection of primary monocyte-derived macrophages and virus replication from monocytes of infected individuals cultivated *ex-vivo*. Thus, RhoA-dependent cytoskeleton rearrangement and intracellular vesicles formation may be related to virion budding and entrapment in intracytoplasmic vacuoles. This is the first report linking integrin activation to a negative control of HIV replication, at least in monocyte/macrophages.