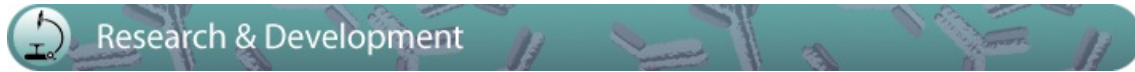





Slit2N Inhibits Transmission of HIV-1 from Dendritic Cells to T-cells by Modulating Novel Cytoskeletal Elements

By *mora*
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Slit2N Inhibits Transmission of HIV-1 from Dendritic Cells to T-cells by Modulating Novel Cytoskeletal Elements ^[1]



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Dendritic cells are among the first cells to encounter sexually acquired human immunodeficiency virus ([HIV-1](#) ^[3]), in the mucosa, and they can transmit HIV-1 to [CD4+](#) ^[4] T-cells via an infectious synapse. Recent studies reveal that actin-rich membrane extensions establish direct contact between cells at this synapse and facilitate virus transmission. Genesis of these contacts involves signaling through c-Src and [Cdc42](#) ^[5], which modulate actin polymerization and filopodia formation via the Arp2/3 complex and Diaphanous 2 (Diaph2). We found that Slit2N, a ligand for the Roundabout (Robo) receptors, blocked HIV-1-induced signaling through Arp2/3 and Diaph2, decreased filopodial extensions on dendritic cells, and inhibited cell-to-cell transmission of HIV-1 in a Robo1-dependent manner. Employing proteomic analysis, we identified Flightless-1 as a novel, Robo1-interacting protein. Treatment with shRNAs reduced levels of Flightless-1 and demonstrated its role in efficient cell-to-cell transfer of HIV-1. These results suggest a novel strategy to limit viral infection in the host by targeting the Slit/Robo pathway with modulation of cytoskeletal elements previously unrecognized in HIV-1 transmission.

To read more, please click [here](#) ^[6].

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