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Investigating contact-induced T cell polarisation at virological synapses

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Background

Human Immunodeficiency Virus Type-1 (HIV-1), the main causative agent of Acquired Immunodeficiency Syndrome (AIDS) is a major global health problem. Cell-cell spread of HIV-1 between CD4 T cells confers many advantages including more rapid infection kinetics, evasion of neutralising antibodies and cellular restriction factors, and may pose a barrier to eradicating HIV-1 from the host. HIV-1 cell-cell spread occurs at intercellular contacts called virological synapses (VS) at which HIV-1 preferentially assembles and buds. We have previously shown that the microtubule organising centre (MTOC) and associated organelles are polarised within the HIV-1 infected cell at the VS; however the causes and consequences of this are unknown.

Materials and methods

We have coupled immunofluorescence microscopy with functional virology and mutant T cell lines to investigate recruitment of cellular and viral proteins to the VS.

Results

We show that recruitment of the MTOC to the VS is triggered upon engagement of integrins on the HIV-1 infected cell and that HIV-1 infected T cells appear more prone to polarise. Our data implicate the integrin LFA-1 as an important component of this process. Time course analysis coupled with pharmacological inhibitors reveals that T cell polarisation to the VS is an active process, suggestive of localised synaptic signaling. In support of this, we find evidence for enriched phosphotyrosine staining at the VS.

Conclusions

LFA-1 engagement on HIV-1 infected cells plays a key role in triggering contact-induced T cell polarisation at the VS. This provides further evidence that cell-to-cell spread at the VS is highly regulated.

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