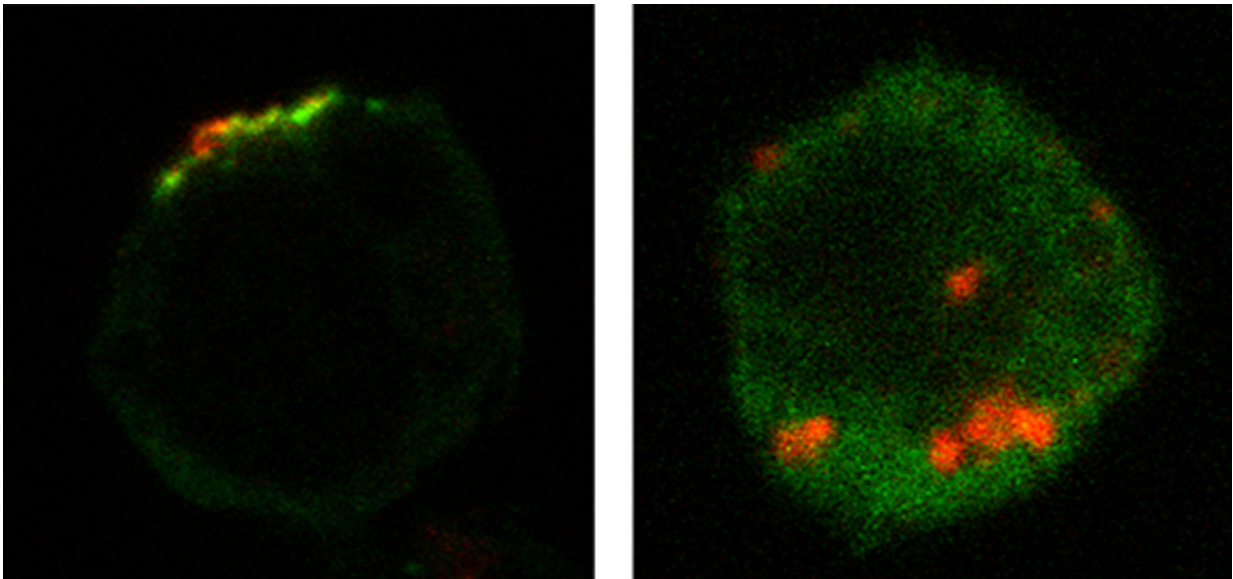


Study reveals how a Rab protein controls HIV-1 replication

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Compared with a control T cell (left), loss of Rab27a (right) blocks the delivery of late endosomes (red) to the plasma membrane, inhibiting the recruitment of Gag (green) to HIV-1 assembly sites. Credit: Pereyra Gerber et al., 2015

HIV-1 replication requires the coordinated movement of the virus's components toward the plasma membrane of an immune cell, where the virions are assembled and ultimately released. A study in *The Journal of Cell Biology* reveals how a Rab protein that controls intracellular trafficking supports HIV-1 assembly by promoting high levels of an important membrane lipid.

New HIV-1 particles assemble at specialized sites in the plasma membrane that are enriched in PIP₂, a phospholipid component of the membrane that recruits a [viral protein](#) called Pr55^{Gag} (Gag) that directs HIV-1 assembly. Because certain cell secretion pathways have been suggested to be required for this process, University of Buenos Aires researcher Matías Ostrowski and colleagues investigated whether a role might be played by Rab27a, a protein that guides delivery of membrane-bound compartments called endosomes to the plasma membrane.

Ostrowski and colleagues found that [viral replication](#) was impaired in [immune cells](#) lacking Rab27a. These cells showed reduced levels of PIP₂ at the plasma membrane and thus failed to recruit Gag to form viral assembly sites. The researchers determined that Rab27a boosted PIP₂ production at the [plasma membrane](#) by controlling the endosomal delivery of an enzyme that is necessary for production of the phospholipid to the cell periphery.

Ostrowski believes that these results open a path to investigate whether manipulating endosomal traffic could be a new target for anti-HIV-1 therapies.

More information: Pereyra Gerber, P., et al. 2015. *J. Cell Biol.* [DOI: 10.1083/jcb.201409082](https://doi.org/10.1083/jcb.201409082)

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