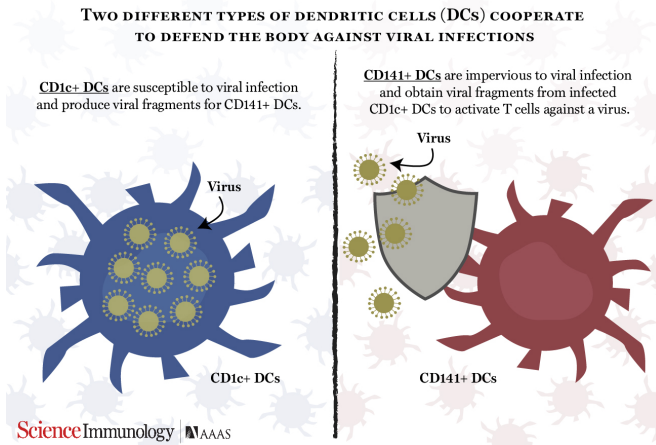


# Dendritic cells 'divide and conquer' to elude viral infection while promoting immunity

7 July 2017



"We show that one DC subset (CD1c+ DCs) is susceptible to viral infection and produces viral fragments," Palucka says. "Another DC subset (CD141+ DCs) uses these viral fragments to activate T cells against the virus. This paradigm may allow a better understanding of the induction of protective immunity against viruses and live-attenuated vaccines against [viral infections](#)."

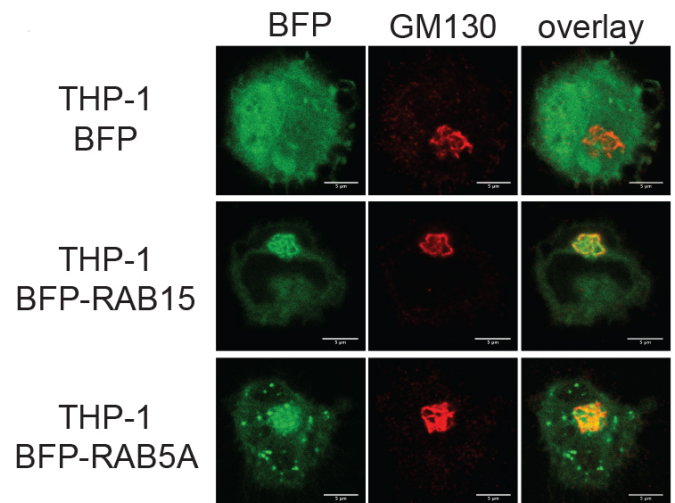
The researchers had isolated these two different DC subsets from blood and lung, and infected them with HIV and influenza viruses. CD1c+ DCs were susceptible to HIV and influenza infection compared with CD141+ DCs.

Infographic depicting a "division of labor" among DC subsets, which protects the body during viral infection. Credit: Carla Schaffer / AAAS

A research team led by Jackson Laboratory (JAX) Professor Karolina Palucka, M.D., Ph.D., in collaboration with a research team at Institut Curie in France led by Dr. Nicolas Manel, have addressed a long-standing puzzle of immunology: How do dendritic cells (DCs) do their job of promoting adaptive immunity to a virus while avoiding getting infected themselves?

DCs are the "beat cops" of the immune system. They round up viral antigens (proteins specific to a given virus), and present them to the receptors on T cells, which in turn promote an [adaptive immune response](#) to that virus. But along the way the DCs are vulnerable to infection by the virus, presumably compromising their protective powers.

The research team reports in *Science Immunology* that two subsets of DCs work together to activate T cells against a virus: one dies and produces the viral antigens that the other then sweeps up and presents to the T cells.



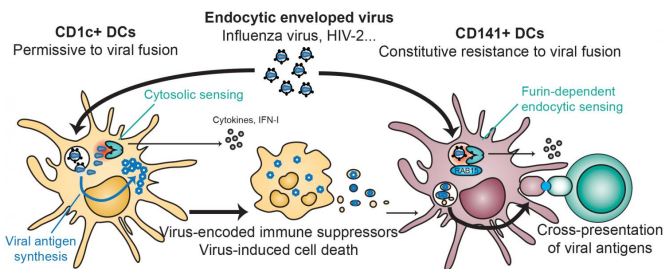
Silvin et al. found that a molecule called RAB15 can help limit viral infection in DCs, and examined its localization in human monocytes growing in culture. Credit: Silvin et al., *Sci. Immunol.* 2, eaai8071

In exploring the reasons for the resistance of CD141+ DCs, the team tested the ability of viruses to fuse with the DCs, and found that CD141+ DCs were resistant to fusion with both HIV and influenza

(both "enveloped" viruses with an outer shell), whereas CD1c+ DCs were not. Further testing showed that CD141+ cells resisted [infection](#) from other endocytic enveloped viruses as well, but not from adenovirus, a non-enveloped [virus](#).

The expression of a specific protein, RAB15, appears to confer the CD141+ DCs' viral resistance, the researchers report.

Their work shows that the DC subsets function together for antiviral response. CD141+ DCs scoop up viral antigens from the dead and dying CD1c+ cells to present to the T cells. This mechanism is in keeping with the known role of CD141+ DCs in presenting antigen from necrotic [cells](#) in general.



CD141+ DCs are dependent on the productive infection of "bystander" CD1c+ DCs, to effectively activate T cells. Credit: Silvin et al., *Sci. Immunol.* 2, eaai8071

**More information:** A. Silvin et al., "Constitutive resistance to viral infection in human CD141+ dendritic cells," *Science Immunology* (2017). [immunology.sciencemag.org/look ... /6/sciimmunol.aai8071](https://immunology.sciencemag.org/look.../6/sciimmunol.aai8071)

Provided by Jackson Laboratory  
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