



# **Autophagy: Chapter 7. Induction of Autophagy in HIV-1-Uninfected Cells: Role of Fusogenic Activity of gp41**

*Lucile Espert, Martine Biard-Piechaczyk*

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Autophagy is recognized as an innate mechanism by which intracellular pathogens are degraded into autolysosomes. In consequence, to avoid their destruction, many pathogens have evolved to block at least one step of the autophagy process or to exploit autophagic membranes for their own self-serving purposes. Autophagy is triggered by molecules that sense a danger, such as pathogen-recognition receptors (PRRs), damage-associated molecular pattern molecules (DAMPs), pathogen receptors, and cytokines. Interestingly, several enveloped viruses can induce autophagy through membrane-fusion events that occur at the entry step of their life cycle. For human immunodeficiency virus type 1 (HIV-1), the envelope glycoprotein gp41 is responsible for the fusion between the membrane of the virus, or the infected cell, and the membrane of the uninfected target cell. This fusion event triggers autophagy in CD4 T lymphocytes, leading to their apoptosis – the mechanism responsible for development of AIDS. Thus, autophagy plays an important role in the pathogenesis of HIV-1 infection by killing specifically the uninfected CD4 T lymphocytes through membrane-fusion events.

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