

## Press release

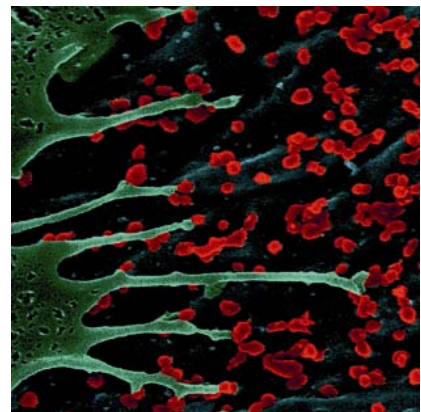
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# Chikungunya : discovery of a human-specific factor involved in the virus replication

Scientists from the Institut Pasteur, Inserm and CNRS, have identified a human-specific factor involved in the replication of Chikungunya virus which accounts for the species specificity of this virus. Chikungunya virus is an emerging virus that in 2005 caused, for the first time, an outbreak in La Réunion island, a French overseas district where more than 30% of the population was infected, and has recently emerged in temperate regions of Europe. The identification of this new host factor enriches our understanding of the molecular bases of Chikungunya virus infection, which were characterized so far. This work also paves the way for the development of a more relevant humanized animal model to better understand the pathophysiology of infection. This research has been published online on April 26, at the *European Molecular Biology Organization reports* (EMBO reports).

Chikungunya virus was first identified in Tanzania in the 1950s and emerged in the islands of the Indian Ocean in 2005 where it caused a large outbreak, most notably in La Réunion island. In 2007, this virus was imported to Europe, causing an outbreak in Italy. In late summer 2010, the two first autochthonous cases were reported in the French Riviera.

The molecular mechanisms of Chikungunya virus infection in human cells have been poorly characterized. Scientists at the Institut Pasteur, Inserm and CNRS, under the leadership of Marc Lecuit, head of the Biology of Infection Unit at the Institut Pasteur in Paris, have made a significant advance: they have identified a human-specific intracellular molecular factor called NDP52 that is involved in the replication of Chikungunya virus. The scientists have demonstrated that in human cells, NDP52 is able to bind to a protein of Chikungunya virus, nsP2, and that this interaction promotes the replication of the virus inside its target cells, thereby contributing to the development of human Chikungunya virus infection. In contrast, mouse NDP52 does not interact with nsP2, and NDP52 therefore does not promote infection in murine cells.



In 2008, the team headed by Marc Lecuit had developed the first animal model to experimentally reproduce the human disease caused by Chikungunya virus. This animal model has since allowed a better understanding of the physiopathology of infection, and has enabled the identification of Chikungunya virus target cells and tissues to test the efficacy of preventive and curative approaches. The discovery of the role of the human factor NDP52 in Chikungunya virus infection opens new avenues for the development of a humanized animal

model, which would be instrumental for deeper understanding of the pathophysiology of Chikungunya virus infection.

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Caption – Chikungunya viruses at the surface of an infected human fibroblast.

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## Source

**Species-specific impact of the autophagy machinery on Chikungunya virus infection, *European Molecular Biology Organization reports*, April 26, 2013**

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