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ORAL PRESENTATION

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Insect cell endocytosis of chikungunya virus adapted to *Aedes albopictus*, a mosquito recently introduced into southern France

Christian Devaux^{*}, Eric Bernard, Bernard Gay, Nathalie Chazal, Laurence Briant

From 17th International Symposium on HIV and Emerging Infectious Diseases (ISHEID)
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Introduction

Since the first isolation of chikungunya virus (CHIKV) more than 50 years ago in Eastern and Central Africa, CHIKV epidemics have been repeatedly recorded from various countries. The 2005-2006 outbreak in Reunion Island, was characterized by a genome microevolution in the E1 envelope glycoprotein gene (E1-A226V mutation) that enhances CHIKV fitness for *Aedes Albopictus* vector. More recently, CHIKV caused explosive outbreaks in India and propagated to temperate areas in Southern Europe, including France in 2010. Along with *Aedes albopictus* colonization of new geographical areas and climate change facilitating vector proliferation, the epidemic risk for “tropical infectious diseases” represents a real threat for naïve populations. It is therefore important to better understand the replication cycle of CHIKV in cells from *Aedes albopictus*.

Materials and methods

CHIKV strains (the African reference strain of CHIKV 37997; the LR-OPY1 (E1-226V) variant isolated from Reunion Island and the LR-OPY1V226A bearing the reverse E1-V226A mutation) were tested for replication in the C6/36 *Ae. albopictus* cell line. Experiments were performed to assess the role of clathrin and dynamin-dependent endocytic pathways, implication of endosomal pH acidification and requirement for membrane cholesterol in CHIKV infection of mosquito cells.

Result and conclusions

Our data indicate that CHIKV uses a clathrin-dependent, caveolae-independent pathway to infect *Aedes albopictus*

cell cultures and requires membrane cholesterol as well as a low-pH environment for entry. These features, especially membrane cholesterol requirement, are modulated in some extent by the E1-A226V mutation. Altogether, our data provide the first information regarding the pathways used by CHIKV to infect *Aedes albopictus* cells and points the consequences of recent genome microevolution on these entry routes.

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