Chikungunya virus-host interplay at the keratinocyte level

Laurence Briant*[1]

[1]Centre d'études d'agents Pathogènes et Biotechnologies pour la Santé (CPBS) CNRS, UMR 5236, CNRS, CPBS, 1919 route de Mende F-34293 Montpellier, France. – Université Montpellier 1, université montpellier 2, CNRS : UMR5236 – France

Résumé

Transmission of chikungunya virus (CHIKV) to humans is initiated by puncture of the skin by a blood-feeding Aedes mosquito. Despite the growing knowledge accumulated on CHIKV, the interplay between skin cells and CHIKV following inoculation still remains unclear. In this study we questioned the behavior of human keratinocytes, the predominant cell population in the skin, following viral challenge. We report that CHIKV rapidly elicits an innate immune in these cells leading to the enhanced transcription of type I/II and type III interferon genes. Concomitantly, we show that despite viral particles internalization into Rab5-positive endosomes and efficient fusion of virus and cell membranes, keratinocytes poorly replicate CHIKV as attested by absence of nonstructural proteins and genomic RNA synthesis. Accordingly, human keratinocytes behave as an antiviral defense against CHIKV infection rather than as a primary targets for initial replication. This picture significantly differs from that reported for Dengue and West Nile mosquito-borne viruses.

Mots-Clés: Chikungunya virus, host, pathogen interaction, skin

*Intervenant