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Morphology and morphogenesis of arboviruses circulating in Brazil: Dengue, Yellow Fever, Zika and Chikungunya

Debora Ferreira Barreto-Vieira

1Laboratory of Morphology and Viral Morphogenesis, Brazil.

Abstract

The impact of the cocirculation of different arbovirus in Brazil is still little known. As in the case of reinfection by different serotypes of dengue virus (DENV), the interaction of arboviruses (DENV serotypes 1-4, chikungunya [CHIKV] and vírus zika [ZIKV]) could theoretically result in more intense viremias or other immunological alterations. The epidemic of ZIKV in 2015 in the Americas it provided evidence for features of pathogenicity that had not been observed previously in infections by flaviviruses. ZIKV was shown to be able to cross the placental barrier to cause congenital infections and to be transmitted sexually among humans. Studies in vitro by transmission electron microscopy shows that part of the ZIKV morphogenesis may occur within viroplasm-like structures, never seen in other flaviviruses. Current studies suggest that flaviviruses produce an ensemble of structurally different virions circulating in an organism, collectively contributing to tissue tropism and virus dissemination. In this work, Aedes albopictus mosquito lineage cell (C6/36 cells) and Kidney epithelial cells of African green monkey (Vero cells) were infected with samples of the main circulating arboviruses in Brazil (DENV-1, DENV-2, DENV-3, DENV-4, ZIKV, yellow fever [YF] and CHIKV) and ultrastructural studies by transmission electron microscopy were performed. In all cells cultures inoculated, virus particles and replication was observed. The particle morphology was similar between the four serotypes of DENV, YF and ZIKV, with differences only in diameter. Cells infected with DENV, YF presenting tubular structures and, after the appearance of these structures, particles in the assembly process and particles already presenting envelope structure were observed in cisterns of the rough endoplasmic reticulum and in cytoplasmic vesicles. In cells infected with large viroplasm-like compartments, localized in the perinuclear area together with peripheric rough endoplasmic reticulum, mitochondria and microtubules, were verified. ZIKV particles were observed inside lysosomes and rough endoplasmic reticulum. The CHIKV particles showed spherical format with an approximate diameter of 50-60nm and with the envelope structure enough evident. In cells infected with CHKV it was observed that the particles are internalized mainly by clathrin-mediated endocytosis. Virus particles was observed in endocytic vesicles and in compartments formed by unitary membranes where nucleocapsids were observed. The release of CHKV particles was by budding. In this study we demonstrated that morphology of DENV of four different serotypes is similar with differences only the diameter and that there is no difference between the replicative cycles; the ZIKV had a different replicative cycle than the Flavivirus pattern. Regarding CHKV, the replicative cycle occurred as described in the literature.

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