Conclusion: There is sufficient evidence to indicate an autochtonous transmission occurs in Reunion Island. Specifics studies have to be conducted to assess the impact on public health, and to describe the epidemiology of the disease (vector, reservoir, human cases). Health professionals should be aware for early diagnosis and early administration of effective treatments.

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Phylogeny of Rift Valley fever virus isolates recovered from humans during 2008-2011 disease outbreaks in South Africa

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Background: Last major outbreaks of Rift Valley fever (RVF) in South Africa occurred 39 years ago, in 1974-76. Re-emergence of RVF in South Africa in 2008-2011 affected large populations of both livestock and humans. Full genome sequencing of 100 human isolates from 302 laboratory confirmed cases was done to study their genetic diversity.

Methods & Materials: Viral RNA was extracted from infected Vero cell culture supernatant fluids and the entire S, M and L RNA segments amplified. RT-PCR products were purified and sequenced using the Ion Torrent sequencing platform. MEGA software using a neighbor-joining distance method was used to perform phylogenetic analysis of sequence data of isolates from recent outbreaks in South Africa including sequences obtained from Genbank of isolates collected throughout Africa, Madagascar and the Arabian Peninsula.

Results: Isolates from outbreaks in South Africa during 2008 and 2009 belong to lineage C, containing isolates from major outbreaks in Zimbabwe, Madagascar, Kenya and Saudi Arabia. All the 2010-2011 isolates cluster into lineage H baring one isolate from a patient potentially exposed to wild virus and live attenuated Smithburn vaccine strain used for animal immunization. This isolate sorted with the parent vaccine strain in lineage K in the M segment tree, but sorted in lineage H in the S and L segment trees indicating segment reassortment.

Conclusion: Recent re-emergence of RVF in South Africa, was first associated with outbreaks of the disease in adjacent parts of north-eastern parts of the country in 2008 and outbreaks in KwaZulu Natal and North Cape Provinces in 2009, all caused by lineage C genotype. RVF virus had progressively re-infiltrated much of the interior plateau of South Africa in 2010 with last human cases reported in May of 2011. Most of the confirmed cases diagnosed in 2010-2011 were caused by the novel lineage H genotype. All fatal cases (n = 25) resulted from infection with lineage H genotype. Reassortment between vaccine virus and wild virus has implications for the safety of the live-attenuated veterinary vaccine. The massive use of the vaccine during outbreaks might contribute to the evolution of RVF virus, including the possibility of acquiring new tissue tropism and pathogenic properties.

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Dengue virus infection induces endothelial cells senescence

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Background: Dengue is a zoonotic mosquito-borne disease. The virus that causes dengue, DENV infects the endothelium and trigger intravascular leakage. The mechanism contributing to the leakage, however is still unknown. Here, we examined if DENV infection induces endothelial cells senescence, a mechanism previously shown to affect endothelial cells permeability.

Methods & Materials: The well-established endothelial cells model culture system, human umbilical vein endothelial cells (HUVECs) were continuously passaged until they reached senescence. Senescence-associated (SA)-β-gal in situ staining and flow cytometry cell cycle analysis were used to stage cells at the different cell passage number. The staged cells were infected with DENV type-2 (DENV-2). Foci-forming assay and quantitative RT-PCR (qRT-PCR) were used to quantitate virus infectivity and replication. (SA)-β-gal staining and flow cytometry analyses were used to stage post-infection cells. Impedance-based real-time growth kinetics analysis was performed to monitor cell morphological changes in real time.

Results: Staging of of HUVECs using SA-β-gal staining and cell cycle distribution method showed a significant increment in the SA-β-gal positive cells and cells arrested at the G2/M phase of the cell cycle with concomitant reduction of cells in the G0/G1 and S phase with the increasing cell passage number. Significant reduction in DENV-2 infectivity and replication, determined by foci-forming assay and qRT-PCR, was obtained for infection of intermediate young and early senescent HUVECs in comparison to infection of young HUVECs. DENV infection increases the percentage of HUVECs expressing senescence-associated (SA)-β-gal, cells arrested at the G2/M phase and cells with enlarged morphology, indicative of senescing cells.

Conclusion: This study highlights that DENV infection induces HUVECs senescence suggesting possible role of induction of senescence in dengue.

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