Molecular Characterization of Rift Valley Fever Virus during the East

African Outbreak, 2006 – 2007

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ABSTRACT

Rift Valley Fever (RVF) is a disease caused by Rift Valley Fever Virus (RVFV). It belongs to the phlebovirus family. It causes a febrile illness in livestock and humans. RVF outbreaks follow heavy rainfall and flooding. The virus is transmitted to humans through mosquito bites or contact with infected animal products. RVF has periodically caused major outbreaks in Sub-Saharan Africa, and elsewhere in Africa and recently also in the Arabian Peninsula. From December 2006, RVF outbreak occurred sequentially in East Africa (Kenya, Somalia, and Tanzania); starting from Northeastern province (NEP) of Kenya, then in Coast province of Kenya and also in mid and Southern regions of Somalia in early January, 2007. In February 2007, the outbreak was reported in Rift Valley province of Kenya and in Tanga and Dodoma regions of Tanzania. Following these sequential outbreaks, the question was whether the outbreaks were caused by a viral strain that was spreading from region to region or viral strains that were dormant within the regions but were activated by the heavy rains and flooding. To address this question, full genome sequencing of RVF virus isolates from each of the four regions was done and compared with historic strains. The full genome of the 2006-2007 strains displayed minimal changes from the historic strains. The variation in all the segments was highest in the M segment (1.9%), followed by S segment (1.7%), and finally L segment (1.4%). An analysis done by region indicated the highest average divergence to be in the Tanzanian isolates (0.8%). The Garissa isolates (Northeastern province, Kenya) had the next highest diversity (0.7%). Isolates from Baringo, (Rift Valley province, Kenya) and those from Kilifi and Malindi (Coast province, Kenya) were the least divergent (0.6%). These results indicated that strains were highly conserved in relation to historic outbreaks. However; detailed analysis of nucleotide and predicted amino acid sequences revealed that unique differences occurred between isolates of various regions; this was more evident in the Tanzania and Garissa strains. These results clearly indicate that virus strains from each region were uniquely different, representing de novo activation of dormant RVFV strains in these regions. A single formulation of vaccine could be used to protect the population at risk from different geographical regions and this should be taken into consideration. Continuous surveillance and characterization of the virus should also be carried out as the virus has been shown to be endemic to different regions and could cause outbreaks following heavy rains and flooding.