Special Issue

Rift Valley Fever Epidemiology, Pathogenesis and Host Response

Message from the Guest Editors

A complete understanding of RVF pathogenesis and host response to infection is also still lacking. Direct tissue injury (i.e., liver, kidneys, adrenal cortex), failure of the adaptive immune response (likely exacerbated by marked loss of lymphocytes), and various innate immune responses (i.e., tissue factor synthesis) have been demonstrated. However, our understanding of the diversity of forms RVF presents across species, age groups, and individuals is woefully incomplete. Usually, human infections present as an acute self-limiting febrile illness, but some patients develop severe or fatal disease that can include hepatic disease, widespread hemorrhages, renal impairment, encephalitis, and ocular lesions. Intriguingly, while encephalitis has been experimentally reproduced in mice, rats, marmosets, and African green monkeys, it has not been described in any natural cases in ruminants.

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