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Nipah Virus (Niv) and Hendra Virus (Hev): 2018

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Letter to the Editor,

Hendra virus (HeV) and Nipah virus (NiV) are emerging zoonotic viruses that cause severe and often lethal respiratory illness andencephalitis in humans. Henipaviruses can infect a wide range of species and human-to-human transmission has been observed for NiV. While the exact route of transmission in humans is not known, experimental infection in different animal species suggests that infection can be efficiently initiated after respiratory challenge. The limited data on histopathological changes in fatal human cases of HeV and NiV suggest that endothelial cells are an important target during the terminal stage of infection; however, it is unknown where these viruses initially establish infection and how the virus disseminates from the respiratory tract to the central nervous system and other organs. Here we review the current concepts in henipavirus pathogenesis in humans. Nipah Virus (NiV) infection is a recently promising zoonosis that causes chronic disease in both animals and humans. The accepted host of the virus is fruit bats of the Pteropodidae Family, Pteropus genus.

NiV was first identified during an epidemic of disease that took place in Kampung Sungai Nipah, Malaysia in 1998. On this time, pigs were the transitional hosts. Though, in subsequent NiV outbreaks, there were no middle hosts. In Bangladesh in 2004, humans became infected with NiV as a result of overwhelming date palm sap that had been infected by infected fruit bats. Human-to-human transmission has also been recognized, counting in a hospital setting in India. NiV infection in humans has a variety of clinical presentations, from asymptomatic infection to the severe acute respiratory syndrome (SARS) and fatal encephalitis. NiV is also competent of causing disease in pigs and other household animals.

There is no vaccine for either humans or animals. The primary action for human cases is exhaustive sympathetic care. There are at present no drugs or vaccines specific for NiV infection although this is a main concern disease on the WHO, R&D Blueprint. Intensive helpful care is suggested to treat chronic respiratory and neurologic complications. Currently, there is no vaccine or drug available for humans or animals. The primary treatment is intensive supportive care for people suffering from severe respiratory and neurologic complications. From contracting the disease to the onset of the symptoms, the incubation period ranges between 4 and 14 days. In some case, an incubation period of 45 days has also been reported. People are expected to make full revival after existing acute encephalitis. Though, survivors have shown long-term neurological circumstances like convulsion disorder and character changes. After revival, a small number of people are seen to have relapsed or developed delayed onset encephalitis.

The molecular mechanisms of HNV pathogenesis remain largely unknown. Several animal models are available, such as hamsters, ferrets and African green monkeys which closely mimic the disease progression seen in human cases; however, this research is hampered by lack of reagents and sufficient animal numbers. Biologically relevant in vitro models of primary human cell cultures are also available to study the different components of HNV infection in the respiratory epithelium, endothelium and neurons and will allow for an in-depth analysis of the host-pathogen interactions. These studies have already shown differences in host responses depending on the cell types and virus strains. The latter is of particular interest since strain variations have been observed between the NiV outbreaks in Malaysia and Bangladesh, correlating with differences in outcome of disease. Similarly, the more recent equine cases of HeV have been associated with a higher prevalence of neurological disease and were associated with strain variations. It is hypothesized that these genetic changes may result in differences in cell and/or tissue tropism. The recent development of reverse genetics systems for NiV and HeV will allow direct testing of this hypothesis. Finally, a better understanding of the molecular mechanisms of HNV pathogenesis will be important for developing effective counter measures to prevent and treat infection with these often lethal viruses.

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