Host Jump: Flu, MERS-CoV and Ebola

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The emergence of viral infectious diseases in recent years has posed great public health concern worldwide. For example, flu continues to be a major cause of mortality worldwide and highly pathogenic MERS-CoV infection occurred in 2012 in Middle East. Recently, a novel Ebola virus (EBOV) first identified in March 2014 has caused more than 25,000 human infections in West Africa, resulting in more than 10,000 deaths. Most of the infectious viruses are zoonotic pathogens that crossed the species barriers to infect humans. The mechanism of viral interspecies transmission is an important scientific question to be addressed, leading to the control and prevention of infectious disease. Here, we discuss the functional and structural progress in understanding the interspecies transmission of influenza viruses and MERS-CoV. The receptor binding property of virus is a major determinant for the host tropism, which enables interspecies transmission. First, the receptor binding specificity of influenza virus is determined by the viral hemagglutinin (HA) and we summarize recent crystallographic studies that provide molecular insights into HA-host receptor interactions that have enabled several influenza A virus subtypes to 'jump' from avian to human hosts. Second, the surface-located spike (S) protein of coronavirus initiates infection by mediating receptor-recognition and membrane fusion and is therefore a key factor in host specificity. We summarize the progress made in the past decade in understanding the cross-species transmission of SARS-CoV and MERS-CoV by focusing on S features, their receptor-binding characteristics, and the priming cleavage process.

It has been reported that the EBOV genome variation might have potential impact on the efficacy of sequence-based candidate therapeutics. However, only limited viral information is available since July, when the outbreak entered a rapid growth phase. Here, we describe 175 full-length EBOV genome sequences from five severely stricken districts in Sierra Leone from September 28 to November 11, 2014. We found that the 2014 EBOV has become more phylogenetically and genetically diverse from July to November, 2014, characterized by the emergence of multiple novel lineages. Because genetic diversity of the 2014 EBOV has increased sharply, extensive EBOV surveillance in Sierra Leone, Guinea and Liberia, is highly appreciated to better understand the viral evolution and transmission dynamics of the ongoing outbreak. These data should be able to facilitate international efforts in respects of development of vaccines and therapeutics.