MERS: Pathogenesis and Antivirals

Kwok-Yung Yuen

Since its emergence from Saudi Arabia in 2012, the Middle East Respiratory Syndrome coronavirus (MERS-CoV) has affected over 1000 patients with a crude fatality rate of more than 30% which is three times that of SARS. Besides its broader tissue tropism in cell lines which corroborates with the pathological findings in the common marmoset model, MERS-CoV can cause productive infection in human primary monocyte derived macrophages and dendritic cells associated with cytokine dysregulation. Furthermore MERS-CoV can induce apoptosis in human primary T lymphocytes through the activation of the extrinsic and intrinsic apoptosis pathway. No specific treatment for MERS is available at this stage. Besides convalescent plasma with neutralizing antibody against MERS-CoV, lopinavir-ritonavir or interferon beta-1-b appear to reduce clinical scores, radiological changes, viral load and histopathological changes in the common marmoset model. Despite the potent in-vitro activity of mycophenolate against MERS-CoV, its use in common marmoset may worsen MERS and should not be used alone for treatment.