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Suppression of Rac1 signaling by viral NS1 can facilitate *Influenza A virus* replication

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Influenza A virus (IAV) is a major human pathogen with the potential to become pandemic. IAV contains only eight RNA segments; thus, the virus must fully exploit the host cellular machinery to facilitate its own replication. In an effort to comprehensively characterize the host machinery taken over by IAV in mammalian cells, we generated stable A549 cell lines with over-expression of the viral NS1 protein to investigate potential host factors which might be modulated by NS1 protein. We found that viral NS1 protein directly interacted with cellular Rac1 and facilitates viral replication. Further researches revealed that NS1 can down-regulates the activity of Rac1 via post-translational modifications. Therefore, our results demonstrated that the IAV blocked Rac1-mediated host cell signaling transduction to facilitate its own replication through the NS1 protein. Our findings provide a novel insight into the mechanism of IAV replication and also indicate new avenues for the development of potential therapeutic targets.

Biography

Professor Min Fang got her Ph.D from the Institute of Genetics and Developmental Biology, CAS in 2003. She did her postdoc training in Fox Chase Cancer Center in USA mainly on studying the pathogenesis of viral infection, as well as the mechanisms by which vaccines afford protection. She joined the Institute of Microbiology, CAS in June, 2012 as a professor supported by "Thousand Young Talents Program" of the China's government. Her work was published in esteemed journals such as: Immunity, J Exp Med, PNAS, Plos Pathogen, etc, and multiply works were selected and referred by the "Faculty of 1000".

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