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Architecture and assembly of type A influenza virus RNA polymerase

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Influenza A virus is an important pathogen accounting for epidemics and pandemics. The existing vaccines and drugs for influenza are unlikely to protect against some new strains emerging from animal reservoirs which possess the potential to cause high mortality. The influenza A RNA-dependent RNA polymerase (RdRP) complex catalyzes viral RNA replication and transcription activities which make it an attractive target for novel antiviral therapy development. The influenza RdRP consists of PB1, PB2 and PA. PB1 was suggested as the core of the RdRP complex, containing the polymerase active sites. PB2 has a cap-binding motif that snatches the 5'cap of host-cell pre-mRNAs. In our previous reports, we found that the N-terminal of PA carries the endonuclease activity. It cleaves the host pre-mRNAs resulting in primers with a 3′-hydroxyl group for viral transcription. Both structural and functional results implicated that the C-terminal of PA (PA_C) takes part in a diverse range of functions including vRNA/cRNA promoter binding. However, the underlying mechanism of the RdRP complex is elusive due to lacking of whole structure information. Recently, we reconstructed a type A influenza RdRP sub-complex at 4.3 Å resolution using single particle cryo-EM method, compromising PA, PB1 and N-terminal of PB2. The sub-complex folds into a cage-like structure within which PA_C and PB1 creates a partially enclosed central cavity for RNA synthesis. This sub-complex exists as dimer in solution and can assemble into a tetrameric state, regulated by vRNA promoter. Our structural and biochemical results suggest an oligomeric transition model in replication process for influenza RdRP. These results also lead to further understanding of the mechanism of other negative-stranded viral RNA polymerases.

Biography

Yingfang Liu obtained his PhD on Plant Science in Peking University, 1999. After that, he had his postdoctoral studies on structural biology in National Jewish Medical and Research Center, Cold Spring Harbor Laboratory and Duke University Medical Center. In 2005, he built up his own lab in Institute of Biophysics, Chinese Academy of Sciences. From then on, he has been working on some negative stranded RNA viruses and the host innate immune responses upon virus infection. Especially, they are trying to study viral proteins involved in viral genome replication and the host proteins that are induced by interferon upon viral infection. In recent years, his group made great achievement in studies of influenza polymerase.

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