Title:	Role of FUS and autophagy in regulation of influenza A virus life cycle
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Abstract:	Influenza is a global disease caused by influenza A virus (IAV), responsible for millions of infections and deaths worldwide each year. Devoid of any locomotion and metabolic activities, IAV, like other viruses, rely on host cells at each stage of its life cycle. Several RNAi screens have identified various host factors that IAV needs at different stages of its life cycle. An RNAi screen in Drosophila cells revealed FUS as a potential factor for IAV infection [48]. FUS is an RNA and DNA binding protein that functions in multiple gene expression processes, including transcription, splicing, mRNA transport, stress granules formation, and translation. Some studies also showed that it interacts with the IAV nucleoprotein (NP) and polymerase (PA) [49, 50]. The aim of this research was to investigate the role of the FUS in the life cycle of the IAV. Two siRNAs targeting different regions of FUS were used to deplete the proteins in human lung epithelial cells (A549). The results indicated that although FUS was not involved in the entry of IAV, it promoted viral transcription/replication. Additionally, FUS was observed to accumulate around the nucleoli during the late stage (24 h) of IAV infection. IAV relies on the host's cellular processes, including autophagy. Studies suggest that the matrix protein 2 (M2) of IAV inhibits the fusion of autophagosomes with lysosomes, and M2 has an LC3 interacting motif, which is responsible for overturning the autophagy process and maintaining virion stability [62, 63]. We further explored the significance of autophagy in the life cycle of IAV using microtubule-associated protein 1A/1B-light chain 3 (LC3) as an autophagy marker. Our findings revealed the presence of ‘contour-like structures’ of the viral hemagglutinin (HA) during the late stages of infection, which colocalized with LC3, vimentin, tubulin, actin, and partially with the ER proteins. We term the structures ‘contourulums’ as they appeared in the cell-like contour maps. We also explored the effect of the nuclear export block on the distribution of LC3 and HA during IAV infection.
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