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Abstract

Influenza A Virus Disturbs the Host Cell Protein Homeostasis by Inducing the Accumulation of Insoluble Proteins †

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Abstract: Influenza A virus (IAV) is the causative agent for most of the annual respiratory epidemics in humans and the major influenza pandemics in the last century, and is associated with high morbidity and mortality, especially in the elderly. In order to efficiently replicate, this virus hijacks the host cellular machinery and requires precise interactions with host components. However, cells have evolved specific defense mechanisms to counteract the effects induced by the virus. In fact, upon IAV infection, several processes within the cytosol and the endoplasmic reticulum, related to protein synthesis and processing, have shown to contribute either as part of an effective replication cycle or as part of an effective cellular antiviral response. Recent reports show contradictory findings regarding the control of the cellular proteostasis mechanisms by both the virus and the host cell. With this study, we aimed to further unravel the interplay between IAV and the host cell proteostasis-related mechanisms at early time-points post-infection. Our results suggest that the virus disturbs host cell protein homeostasis by inducing the accumulation of insoluble proteins in a process that seems to be related to viral RNA processing. We further analyzed the interplay between IAV infection and the endoplasmic reticulum unfolded protein response. Our results may lead to a better understanding of the interplay between IAV and the host cell and, furthermore, contribute to the development of novel antiviral strategies.

Keywords: influenza A virus (IAV); virus–host interaction; proteostasis; protein quality control; protein aggregation; unfolded protein response