Title:	Inestigating he role of TRIM28 in influenza A virus infection
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Abstract:	Influenza or 'the flu' has plagued mankind for more than 1500 years. Caused by the influenza A virus, flu epidemics and pandemics seriously impact human health, livelihood, and global economy every year. The rapidly evolving ability of the influenza A virus (IAV) is responsible for such recurrent episodes of flu outbreaks. Most of the medications currently available target and inhibit virus-encoded proteins. This strategy fails once the virus acquires resistance to drugs through genetic mutations. An alternative approach to combating viral infection is to identify the host factors that are being manipulated by IAV and design drugs against these components. Through targeting the host, it would be not only be possible to overcome the issue of drug resistance and vaccine ineffectiveness, but also to provide us with broad- spectrum antivirals targeting several viruses that exploit similar pathways in cells for their entry and proliferation. Two genome-wide RNAi screens in 2009 and 2010 commonly identified TRIM28 as a human host factor required for IAV replication 1,2 . TRIM28 is an ubiquitin E3 ligase that functions primarily in the nucleus as a transcriptional co-repressor. This study observed that TRIM28 depletion using different means reduced IAV infection. Infection levels were restored by exogenous supplementation with TRIM28 in lung alveolar epithelial cells. On further investigation, TRIM28 was found to promote nuclear import of vRNPs. Early and late endosome/lysosome expression patterns remained unperturbed, indicating that the effect on IAV infection can be attributed to nuclear functions of TRIM28. In conclusion, this work unraveled the role of TRIM28 as a proviral host factor, and suggests its involvement in the nuclear import of the IAV genome, and possibly in its replication.
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