Review of: "Human rhinovirus promotes STING trafficking to replication organelles to promote viral replication"

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This study focused on the non-canonical function of stimulator of interferon genes (STING) in the human rhinovirus (HRV) life cycle. Previously, McKnight et al. identified STING as a necessary host factor for HRV propagation, and they proposed that non-canonical function of STING is involved in it (Proc Natl Acad Sci U S A. 2020 Nov 3; 117(44): 27598–27607). As the inhibitory effect after the depletion of STING is quite dramatic, it was assumed that STING has essential roles in HRV life cycle. This study by Triantafilou M et al. tried to clarify the involvement of STING in HRV propagation. They found that STING recruitment to the viral replication organelle was triggered by the dissociation from the STIM1, which is induced by 2B viroporin. They also showed the presence of STING in autophagosomes and proposed a model suggesting that this process promotes non-lytic viral particle release from the cells.

The role of autophagy in HRV replication is controversial. The modulation of autophagy function dramatically alters cellular environment, including innate immune responses, lipid and protein homeostasis, damaged organelle accumulation, and so on. Importantly, several previous reports indicated that the inhibition of autophagy leads the activation of innate immune responses (i.e., Tal MC PNAS, 2009 Feb 24;106(8):2770–5). It seems more likely that autophagy induced by STING may negatively regulate RLR signaling pathway. We expect further critical analysis regarding precise function of STING in HRV replication.