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Amount of Non-Self RNA Is Matter to Regulate Nucleic Acid Sensor Retinoic Acid-Inducible Gene I (RIG-I) Activation

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The differentiation of cytosolic self vs non-self RNA is mainly dependent on detection of structural differences by nucleic acid sensors. They are crucial to detect RNA viruses and eliminate it through activation of interferon/ -stimulated genes (ISGs). In our previous studies, the infectivity rate of extracellular vesicles containing multiple virus particles is higher than infection with equivalent numbers of free viruses. Besides, innate immunity related genes decreased in vesicular form of infection. We evaluated whether this effect is due to viral nucleic acid amount presented in cytosol at the very beginning of the infection. We used several different viruses to infect different cell lines at low and high MOIs. We checked both viral RNA load and ifn-β expression via qPCR. The common trend was that while infection increases with higher MOIs, innate response was decreasing. We repeated our experiments using a mRNA analog, low molecular weight poly(I:C). When multiple non-self-viral nucleic acids enter the cytosol en masse, it results in suppression of RIG-I activation and decrease in interferon expression. Finally we demonstrated that this suppression of innate immune activation is in part due to the triggering of RIG-I protein degradation. Our study reveals a heretofore unknown cytosolic non-self RNA threshold above which viruses can suppress the activation of the innate immune system and replicate.

Keywords: Innate Immunity, RIG-I, Virus infection