Perspective

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Single RNA Viruses in Translation and Replication Dynamics

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Abstract

Among the most common infections and a significant burden on society are RNA viruses. Although RNA viruses have been extensively investigated, due to a lack of sensitive assays, little is known about the processes that take place within the initial few hours after infection. Here, we create the virus infection real-time imaging (VIRIM) single-molecule imaging technique to examine the translation and replication of individual RNA viruses in living cells. Virim revealed substantial coordination between the translation and replication of individual viral RNAs as well as a startling variability in replication kinetics between cells. Additionally, we use VIRIM to pinpoint the host genes that prevent early viral replication and identify the replication stage of the incoming viral RNA as a significant bottleneck of effective infection. Single-molecule photography the study of virus replication and virus-host interactions is a potent technique that can be broadly applied to RNA viruses.

Keywords: +RNA virus; Viral translation; Viral replication; Virus-host competition; Single-molecule imaging; Suntag; Virim

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Introduction

Numerous virus families, such as the Coronaviridae (including the Middle East respiratory syndrome coronavirus [MERS-CoV] and severe acute respiratory syndrome coronavirus, Caliciviridae, and Picornaviridae, which are significant human and animal pathogens, make up the group of positive-strand RNA viruses. The majority of +RNA viral infections currently have relatively limited treatment options, placing a significant financial and health burden on society [1]. When released into the cytoplasm of a host cell, the single-strand positive-sense RNA genome of the majority of +RNA viruses can be immediately translated into viral proteins [2]. Viral proteins perform a variety of tasks after being synthesised, including viral RNA replication, altering host cell activities to aid in virus proliferation, and suppressing antiviral signalling in the host cell [3]. After the incoming text has been translated Negative-sense RNA is produced by the newly formed vRNA-dependent RNA polymerase and is then used as a template for the synthesis of further +RNAs [4]. These fresh +RNAs may start a fresh cycle of translation and replication or they may be encapsulated to create fresh infectious virus particles [5]. Due to the many functions that vRNA molecules can perform, closely regulated switching between these dynamic processes is probably crucial for virus reproduction [6]. A translation-to-replication switch is required to start virus replication in freshly infected cells, even for incoming vRNA [7].

Discussion

There are currently few mechanistic insights into this switch, despite the fact that some elements that may influence it have been found [8]. Cells have many defences against viral infection, such as protein sensors that can recognise lengthy double-stranded When +RNA viruses replicate, RNA (dsRNA) is created [9]. When host cell signalling detects viral dsRNA, innate antiviral mechanisms including the interferon induction pathway are quickly activated [10]. IFN-induced genes are upregulated as a result of subsequent IFN signalling, which is essential for limiting viral proliferation. In response, viruses actively block antiviral signalling pathways. To avoid detection of the virus and

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subsequent synthesis of antiviral signalling molecules, many picornaviruses, for instance, create proteases that specifically target host dsRNA sensors or components of the IFN signalling cascade Furthermore, many RNA viruses prevent host translation and transcription, which could impair the immune system's ability to fight off the virus. Early virus discovery may thereby increase the likelihood of building a successful antiviral defence. Inside a diseased cell thus, competition between viral translation/replication kinetics and host-cell antiviral signalling kinetics is likely what determines the outcome of a viral infection. Interestingly, antiviral signalling has been found to exhibit significant cell-to-cell variation even in homogeneous populations of cells in culture. Indicating that virus-host conflict may involve cellular and/or viral heterogeneity. The assays that are now available are not ideal for studying viral translation and replication dynamics or virus-host rivalry for a variety of reasons. First off, the majority of current assays are not sensitive enough to identify the virus in the early stages of infection, when viral translation, replication, and antiviral defences are starting. Because the arriving virus particle only includes one vRNA molecule and can readily avoid detection, it is difficult to examine it. Second, because the majority of tests call for cell lysis or fixation, they cannot measure infected cells in real time. As a result, it is challenging to link the early stages of an infection's molecular events to how the illness will ultimately turn out. Third, for analysis of dynamic processes that are not synchronised in time, it is particularly challenging when numerous cells are needed for a single measurement.

Conclusion

An ensemble method is insufficient to analyse temporally defined events, such as replication of the incoming vRNA or the start of antiviral signalling, if several cells in a population are infected at various periods. A further issue for ensemble approaches is the highly diverse response to viral infection. Fourth, the majority of assays only evaluate one aspect of viral infection. However, due to the interdependence of viral translation and replication, a translation error leads to decreased polymerase output, which could slow down the pace of replication. Thus, the potential of single-parameter experiments to explicitly elucidate mechanistic insights into regulation of translation or replication is restricted.

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None

Conflict of Interest

No conflict of interest

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