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RESEARCH ARTICLE

VISUALIZING VIRAL EVASION OF PRRS: A REVIEW OF LIVE CELL IMAGING AND CONFOCAL MICROSCOPY TECHNIQUES

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ABSTRACT

During the initial stage of viral defense, the host maintains vital sentinels through Pattern recognition receptors called RIG-I-like receptors, including RIG-I and MDA5. The successful viral infection and propagation depend on various advanced strategies developed by viruses to bypass these receptors. Biochemical methods combined with immunological approaches have given valuable molecular information about viral immune evasion, yet they fail to detect complex cellular interactions occurring in living cells at the molecular and spatial levels. The combination of live cell imaging techniques with confocal microscopy tools now lets researchers observe virus-host interactions instantly, which provides brand new methods for studying spatial and temporal viral evading patterns. This paper gathers existing information about viral RIG-I and MDA5 evasion pathways while presenting advances in live cell imaging combined with confocal microscopy techniques that researchers use to study the complex viral innate immune sensor blocking strategies. Additionally, we explore the boundaries of existing imaging methods while identifying promising research pathways in this fast-growing field.

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INTRODUCTION

The continuous arms race between pathogens and host immune systems has driven the evolution of sophisticated viral strategies to evade host innate immune responses[1]. Understanding these evasion mechanisms is fundamental to developing effective antiviral therapies and vaccines. Pattern recognition receptors (PRRs) represent a critical first line of defense against viral pathogens, functioning as molecular sentinels to recognize pathogen-associated molecular patterns (PAMPs) and initiate possible protective immune responses[2]. Additionally, as highlighted earlier, despite the sophistication of these innate immune sensors, viruses have evolved numerous strategies to circumvent detection and subsequent antiviral responses. These evasion mechanisms include sequestration of viral RNA in specialized replication compartments, direct inhibition of PRR signaling components, and modification of viral RNA to prevent recognition[3]. However, while considerable progress has been made in identifying viral proteins involved in PRR antagonism, significant gaps remain in our understanding of the spatiotemporal dynamics of these interactions within living cells during infection. Consequently, traditional biochemical and immunological approaches have provided and are providing valuable insights into the molecular mechanisms of viral immune evasion to fill in these gaps. Still, these methods typically provide static snapshots of complex, dynamic processes and often fail to capture the spatial organization and temporal sequence of interactions between viral components and host immune factors. Live cell imaging techniques, particularly confocal microscopy, offer powerful tools to visualize these dynamic interactions in real-time within the native cellular environment[(2,4)]. Live cell imaging and confocal microscopy have emerged as powerful tools for visualizing the real-time dynamics of viral evasion strategies within the native cellular environment. This review aims to provide an overview of how these advanced imaging techniques are being used to study viral evasion of RIG-I and MDA5, summarize key findings, and discuss methodological considerations and prospects.

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II. Background: Viral Evasion of RIG-I and MDA5

A. The Role of RLRs in Antiviral Immunity: RIG-I and MDA5 are cytoplasmic sensors that detect viral RNA and trigger downstream signaling to induce type I interferon (IFN) production and establish an antiviral state. RIG-I recognizes short double-stranded RNA (dsRNA) and single-stranded RNA with 5′-triphosphate or 5′-diphosphate ends, while MDA5 detects longer dsRNA and complex RNA structures[2]. Upon activation, these receptors interact with mitochondrial antiviral signaling protein (MAVS), leading to the activation of transcription factors such as IRF3 and IRF7 and the induction of IFNs and pro-inflammatory cytokines.

B. Mechanisms of Viral Evasion of RIG-I and MDA5: Viruses have evolved diverse strategies to evade detection by RLRs, with three primary mechanisms: (1) sequestration of viral RNA, (2) direct inhibition of PRR signaling, and (3) RNA modification to prevent recognition.

1. Sequestration of Viral RNA: Many viruses establish specialized replication compartments that shield viral RNA from cytoplasmic PRRs. Positive-strand RNA viruses, such as flaviviruses, coronaviruses, and picornaviruses, induce extensive rearrangements of host membranes to create protected replication complexes[5].

Dengue virus (DENV) and other flaviviruses reshape the endoplasmic reticulum (ER) to form characteristic vesicle packets where viral RNA synthesis occurs. These structures effectively sequester viral RNA, limiting its exposure to cytoplasmic RIG-I and MDA5. Similarly, hepatitis C virus (HCV) creates a "membranous web" derived from ER membranes that harbors viral replication complexes[7]. Futhermore, coronaviruses, including SARS-CoV and SARS-CoV-2, induce double-membrane vesicles (DMVs) that encapsulate viral RNA during replication[2]. These DMVs contain pores that allow the selective export of newly synthesized viral RNA while protecting replication intermediates from detection[6]. Consequently, poliovirus for instance, generates vesicles derived from the secretory pathway that enclose viral replication complexes. Picornaviruses on the other hand reorganize host membranes to form replication organelles with distinct morphologies. While enteroviruses have been shown to sequester viral RNA in autophagosome-like structures that shield replication intermediates from RLRs[7,8]. Despite these insights, the precise spatiotemporal dynamics of viral RNA sequestration and the mechanisms by which some viral RNA inevitably escapes these compartments to trigger immune responses remain poorly understood. This represents a significant gap in our knowledge of viral evasion strategies.

2. Direct Inhibition of PRR Signaling: Viruses can directly target components of PRR signaling pathways to prevent the initiation or propagation of antiviral responses. These strategies involve viral proteins that interact with and inhibit key molecules in the RLR signaling cascade.

At the level of RNA sensing, viral proteins can bind directly to RIG-I or MDA5 to impede their activation. The NS3 protein of dengue virus interacts with the CARD domains of RIG-I, preventing its interaction with MAVS[9]. Similarly, the V proteins of paramyxoviruses specifically bind to MDA5, preventing its oligomerization and downstream signaling[9]. MAVS represents another critical target for viral antagonism. The NS3-4A protease of HCV cleaves MAVS from the mitochondrial membrane, disrupting its ability to aggregate and propagate signaling, which is a direct inhibition. Moreso, the NS1 protein of influenza A virus (IAV) inhibits MAVS-mediated signaling through multiple mechanisms, including the suppression of TRIM25-mediated RIG-I ubiquitination. Downstream of MAVS, viruses target TBK1 and IKKε to prevent IRF3 phosphorylation. The $\gamma_1$34.5 protein of herpes simplex virus 1 (HSV-1) recruits protein phosphatase 1α (PP1α) to dephosphorylate TBK1. Multiple viral proteins, including the leader proteinase (Lpro) of foot-and-mouth disease virus and the papain-like protease (PLpro) of coronaviruses, deubiquitinate signaling components to disrupt pathway activation.

At the terminal stages of the pathway, viruses can prevent IRF3 activation or nuclear translocation. The VP35 protein of Ebola virus binds to and masks the phosphorylation sites on IRF3, while the C6 protein of vaccinia virus inhibits the translocation of phosphorylated IRF3 to the nucleus. While these individual mechanisms have been characterized using traditional biochemical approaches, the spatiotemporal dynamics of these interactions in living cells remain largely unexplored. Additionally, how viruses coordinate multiple evasion strategies during infection is not well understood, highlighting the need for techniques that can visualize these processes in real-time.

3. RNA Modification to Prevent Recognition: Viruses can modify their RNA genomes and replication intermediates to avoid detection by PRRs. These modifications include alterations to the RNA structure, chemical modifications of nucleotides, and the incorporation of host-derived RNA sequences.

Many viruses conceal their 5′-triphosphate moieties, which are potent RIG-I activators, through various mechanisms. Influenza virus for example employs a "cap-snatching" mechanism to cleave 5′ caps from host mRNAs and use them to prime viral transcription[10]. Arenaviruses, including Lassa virus, possess a 5′-3′ exoribonuclease that processes the 5′-triphosphate to a 5′-monophosphate, rendering the viral RNA less stimulatory to RIG-I. RNA secondary structures can also influence PRR recognition. Hepatitis C virus contains structured RNA elements in its 3′ untranslated regions that can mask PAMPs and reduce recognition by RIG-I. Similarly, the structured nature of picornavirus internal ribosome entry sites

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(IRES) may help evade detection. The chemical modifications of viral RNA represent another evasion strategy. N6-methyladenosine (m6A) modifications in viral RNA have been shown to reduce recognition by RIG-I and MDA5[2]. Similarly, 2′-O-methylation of viral RNA can prevent activation of PRRs and subsequent interferon production[11]. Some viruses however, integrate host RNA sequences into their genomes, creating chimeric RNA molecules that are less likely to trigger immune responses. For instance, certain flaviviruses incorporate host RNA sequences within their 3′ untranslated regions to evade immune detection. Despite growing appreciation for these RNA modification strategies, much remains unknown about how viruses coordinate these changes during infection and how they interact with other evasion mechanisms. The dynamic nature of viral RNA synthesis, modification, and packaging is difficult to capture using traditional techniques, highlighting the need for advanced imaging approaches. Having realized this lacuna along with others earlier highlighted, there is a need to investigate the current limitations of studies, in order to explore pathways for future experiments.

C. Current Limitations in Studying Viral Evasion of PRRs: Traditional biochemical approaches, such as co-immunoprecipitation and Western blotting, have been invaluable for identifying protein-protein interactions but provide only static snapshots of complex, dynamic processes. These methods often disrupt the native cellular environment, potentially altering the interactions being studied. Furthermore, they typically require cell population averaging, obscuring cell-to-cell variability that may be critical for understanding viral infections. Conventional immunofluorescence microscopy offers improved spatial resolution but lacks temporal information and often requires cell fixation, which brings the risk of artificial results along with preventing analyses of dynamic cellular events[4]. Additionally, the resolution of standard confocal microscopy (200-300 nm) is insufficient to resolve many of the fine subcellular structures involved in viral replication and PRR signaling.

RNA detection methods like fluorescence in situ hybridization (FISH) can visualize viral RNA but typically require fixed samples and have limited sensitivity[12]. Furthermore, distinguishing between different RNA species (genomic, replicative intermediates, mRNA) remains challenging. Most importantly, the kinetics of viral evasion strategies in relation to PRR activation are poorly understood. The temporal window during which viruses must implement these evasion mechanisms to establish why productive infection remains largely uncharacterized. Questions persist regarding the sequence of events: Do viral antagonists act preemptively, preventing PRR activation entirely, or do they function to suppress already initiated signaling cascades?. The subcellular localization of these interactions is another area requiring further investigation. While certain viral components are known to target specific organelles (e.g., mitochondria for MAVS inhibition), the precise spatial organization of viral evasion mechanisms and their coordination within the infected cell remain unclear. Finally, addressing these limitations requires innovative approaches that can visualize the dynamics of virus-host interactions in living cells with high spatial and temporal resolution. Live cell imaging and advanced confocal microscopy techniques offer promising solutions to these challenges, enabling the study of viral evasion strategies in their native cellular context and in real time.

III. Live Cell Imaging and Confocal Microscopy: Methodological Advances: The study of dynamic processes in living cells has been revolutionized by advances in live cell imaging and confocal microscopy, offering unprecedented opportunities to visualize virus-host interactions in real-time and at high resolution. Confocal microscopy, in particular, overcomes the limitations of traditional widefield microscopy by using pinholes to eliminate out-of-focus light, thereby allowing for optical sectioning and three-dimensional reconstruction of cellular structures[13].

A. Principles and Advantages: Live cell imaging enables direct observation of cellular events as they unfold, offering several key advantages for the study of viral evasion of PRRs. Real-time observation allows researchers to capture transient interactions between viral components and host proteins that might be missed in fixed samples. For example, live cell imaging has been used to track the rapid relocalization of viral proteins to mitochondria to inhibit MAVS signaling, revealing the speed and efficiency with which viruses can disrupt innate immune responses[14]. Single-cell resolution provides insights into the heterogeneity of viral infections, as neighboring cells can exhibit markedly different stages of infection and immune activation. This is particularly relevant for understanding how viral evasion strategies impact cell-to-cell variability in interferon responses[15].

Finally as regards live imaging, spatiotemporal mapping allows for the correlation of specific cellular compartments with viral replication events and immune signaling. By tracking the formation of viral replication compartments and their interactions with PRRs, researchers can elucidate how viruses create specialized microenvironments to shield their genomes from immune detection[16].

B. Fluorescent Tagging Strategies: The ability to specifically label viral and host proteins with fluorescent tags is central to live cell imaging. Genetically encoded fluorescent proteins (FPs), such as GFP, mCherry, and their derivatives, have become indispensable tools for visualizing protein localization, interactions, and movement within living cells. These FPs can be fused to proteins of interest through molecular cloning techniques, creating chimeric proteins that retain their biological function while becoming fluorescently visible[4]. Key considerations include the choice of tag position (N-terminal vs. C-terminal) to minimize interference with protein function, the use of flexible linker sequences to improve protein folding

[11]Unterholzner, L., Sumner, R. P., Baran, M., Ren, H., Mansur, D. S., Bourke, N. M., ... & Bowie, A. G. (2011). Vaccinia virus protein C6 is a virulence factor that binds TBK-1 adaptor proteins and inhibits activation of IRF3 and IRF7. PLoS pathogens, 7(9), e1002247.

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and function, and the selection of FPs with appropriate brightness, photostability, and spectral properties. For example, researchers have successfully tagged the viral NS3 protein of dengue virus with GFP to track its localization to the endoplasmic reticulum and its interaction with the PRR signaling pathway[3].

Furthermore, recent advances in genome editing, such as CRISPR/Cas9-mediated knock-in, have further expanded the possibilities for fluorescent tagging. This technology allows for the endogenous tagging of proteins at their native loci, preserving native expression levels and regulatory control. A good example is with CRISPR/Cas9, which has been used to tag endogenous MAVS with GFP. This process now enables the visualization of MAVS aggregation and signaling dynamics in response to viral infection.

C. Imaging Modalities: Confocal microscopy is a workhorse technique for live cell imaging, providing optical sectioning and improved resolution compared to traditional widefield microscopy[17]. By using a pinhole aperture to eliminate out-of-focus light, confocal microscopy generates high-resolution images of specific planes within a cell, which can then be compiled into three-dimensional reconstructions.

Several variants of confocal microscopy offer different trade-offs between speed, sensitivity, and phototoxicity[18]. Point-scanning confocal microscopy provides excellent optical sectioning but is relatively slow, making it less suitable for capturing rapid dynamic events[17,18]. Spinning disk confocal microscopy, on the other hand, offers faster acquisition speeds with reduced phototoxicity, making it ideal for long-term live imaging[19]. To visualize finer details of virus-host interactions, super-resolution microscopy techniques along with confocal microscopy are a necessity. These techniques include stimulated emission depletion (STED) microscopy, structured illumination microscopy (SIM), and photoactivated localization microscopy (PALM/STORM), which can surpass the diffraction limit of light and achieve resolutions down to 20-30 nm[20]. These techniques have been used to resolve the nanoscale architecture of viral replication compartments and to visualize the organization of PRR signaling complexes at the plasma membrane[20].

D. Image Analysis and Quantification: The extraction of meaningful data from live cell imaging experiments requires sophisticated image analysis and quantification techniques. Hence, deconvolution algorithms can be used to remove out-of-focus blur and enhance the contrast and spatial resolution of images[21]. For specifics, object segmentation algorithms can identify and delineate structures of interest within complex images, such as viral replication compartments or PRR signaling complexes. Particle tracking algorithms can follow this up by observing the movement of labeled molecules over time, providing insights into their diffusion rates and interaction kinetics.

Additionally, Machine learning-based segmentation tools, such as Ilastik and Weka, have emerged as powerful solutions for automating complex image analysis tasks. These tools act a softwares to train algorithms to recognize patterns and features in images, enabling the rapid and accurate quantification of large datasets[22]. Colocalization analysis is another important technique for quantifying the spatial overlap between different fluorescent signals. By measuring the degree of colocalization between viral proteins and host PRRs, similar studies on viral imaging can assess the efficiency with which viruses target and inhibit immune signaling pathways.

VI CONCLUSION

This review has synthesized current advances in the use of live cell imaging and confocal microscopy to elucidate how viruses evade detection by pattern recognition receptors (PRRs) such as RIG-I and MDA5. The literature demonstrates that these advanced imaging techniques have transformed our understanding of the dynamic and spatially complex strategies employed by viruses to subvert innate immune defenses. Live cell imaging has revealed that viral antagonism of PRR signaling is a highly dynamic process, involving rapid relocalization of viral and host proteins, the formation of specialized replication compartments, and the precise temporal deployment of viral antagonists. These studies underscore the importance of both spatial compartmentalization and timing in viral immune evasion. Notably, the sequestration of viral RNA within replication organelles emerges as a common and effective strategy to shield viral genomes from cytosolic sensors. Super-resolution and correlative imaging approaches have further uncovered nanoscale features of these interactions, such as the selective targeting of activated PRR complexes and the structural remodeling of replication compartments. To expand the frontiers of this field, future research should prioritize the integration of complementary methods, such as intravital microscopy, confocal microscopy, and label-free imaging, to validate and extend current findings in more physiologically relevant contexts.

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