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Defective viral genomes as primary triggers of the antiviral immune response

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The innate immune response to viruses is initiated when specialized cellular sensors recognize viral danger signals. In infections with viruses well adapted to the host, virus-encoded proteins that delay the cellular response allow the virus to replicate to high titers prior to host intervention. The mechanisms overcoming viral evasion of the immune system and leading to the production of the primary antiviral cytokine IFN are not well established. Our data indicate that the appearance of defective forms of the viral genome may provide the answer. Defective viral genomes, which were until recently considered an epiphenomenon of in vitro virus replication, accumulate in infected cells at a faster rate than standard viral genomes and potently trigger the sustained activation of the transcription factors IRF3 and NF-B and the production type I IFNs through a mechanism independent of IFN signaling. Remarkably, our most recent work demonstrates that defective viral genomes are generated naturally during respiratory infections in vivo even in mice lacking the type I IFN receptor, and their appearance coincides with the production of cytokines during infections with Sendai virus or influenza virus. Notably, the hallmark antiviral cytokine IFN is only expressed in lung epithelial cells containing defective viral genomes, while cells within the lung that contain standard viral genomes alone do not express this cytokine. Together, our data indicate that defective viral genomes generated naturally during viral replication are a primary source of danger signals for the initiation of the host immune response to infection in vivo.

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