Chapter 13
Hostile Communication of Measles Virus
with Host Innate Immunity and Dendritic Cells

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Abstract Following measles virus (MV) infection, host innate immune responses promptly operate to purge the virus. Detection of alerting measles viral components or replication intermediates by pattern-recognizing host machinery of Toll-like receptors and RNA helicases triggers signaling to synthesize array of anti-viral and immunoregulatory molecules, including type I interferon (IFN). Diverse subtypes of dendritic cells (DCs) play pivotal roles in both host innate immunity on the primary MV-infected site and initiating adaptive immune responses on secondary lymphoid tissues. Responding to the predictable host immune responses, MV appears to have devised multiple strategies to evade, suppress, or even utilize host innate immunity and DC responses. This review focuses on versatile actions of MV-induced type I IFNs causing beneficial or deleterious influence on host immunity and the interplay between MV and heterogeneous DCs at distinct locations.

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**Introduction**

Host immunity has evolved along with continuous pathogenic invasions. During the last decade, great scientific interests have arisen in the field of host innate immunity, especially of type I interferon (IFN) synthesis and toll-like receptor (TLR) signaling, as well as diverse dendritic cell (DC) responses. DCs not only regulate host innate immune response, but also control initiation of host adaptive immunity by stimulating virus-specific naïve T lymphocytes. Upon measles virus (MV) infection, the host immune system is activated to eventually clear the virus. However, MV in return eludes or counterattacks the first line of host defense, the critical innate immune system of the type I IFN network and T cell stimulatory capacity of DCs as well. Consequently, MV could induce profound suppression of host immunity, predisposing MV-infected individuals to other microbial pathogens. Recent studies on MV immunobiology yielded interesting and astonishing results and helped us better understand how host immunity has evolved to combat pathogenic MV and more importantly how immunosuppressive MV contends with protective host innate immune response and DCs.

**Host Innate Immunity Following Measles Virus Infection**

MV is transmitted via aerosol and thus thought to initially infect cells located in respiratory tissues, including epithelial cells and mucosal DCs. On the primary sites infected by MV, multiple host innate immune responses are swiftly operated by