OPEN ACCESS Freely available online

ISSN: 0974-8369
Biology and Medicine

Commentary

Innate Immunity Against Viral Pathogens

Hans Thulesius*

Department of Health and Caring Sciences, Linnaeus University, Vaxjo, Sweden

DESCRIPTION

Innate immunity is an important front-line defense against viral infection. A well-specialized immune system composed of various physical and chemical barriers. Mucosal surfaces, skin and their secretions counteract viral entry into the host. After being invaded by various pattern recognition receptors, the virus is further recognized and results in activation of innate immune signalling pathways that control the production of interferon, inflammatory cytokines, and chemokines. Type I and type III interferons produced by different cell types stimulate the expression of hundreds of genes collectively known as interferon-stimulating genes (ISGs) that put cells in an antiviral state. In viral infections, the host's innate immune system is designed to act as the first line of defense to prevent viral invasion or replication before the adaptive immune system produces more specific defenses. The congenital immune response uses pattern recognition receptors to recognize specific viral components such as viral RNA, DNA, and viral intermediate infected cells and other immunity to type I interferon (IFN) and inflammatory cytokines. Innate immune receptors and their unique downstream signalling pathways have been identified. The innate immune response to viral infections is due to the distinction between endogenous and viral nucleic acids, and their role in inhibition by virulent factors by coordinating between innate and adaptive immune activation. The innate immune system is essential for the initial recognition of the invading virus and subsequent activation of adaptive immunity. Three classes of receptors, called retinoic acid-inducible genes I -like receptors, Toll-like receptors and NOD-like receptors, sense viral components. Double-stranded RNA, single-stranded RNA and DNA play important roles in cell-type specific production of type I interferon and inflammatory cytokines. While retinoic acid-inducible genes I -like receptors play an important role in recognizing RNA viruses in a variety of cells, plasmacytoid dendritic cells use TLRs to detect virus invasion. On the other hand, NLRs are involved in the production of mature interleukin 1α for dsRNA stimulation. Activation of innate immune cells is important for enhancing the adaptive immune response. This review describes recent advances in understanding the mechanism of viral RNA recognition by these different types of receptors and their relationship to acquired immune responses.

The host cell recognizes the invasion of the virus and initiates a strong antiviral response. The virus first activates the innate immune system, which recognizes viral components *via* pattern recognition receptors Acquired immunity, on the other hand, plays a major role in responding to viral reinfection. The host PRR detects viral components such as genomic DNA, single-stranded RNA, double-stranded (ds) RNA, RNA with 5'-triphosphate ends, and viral proteins. Currently, three classes of PRR, the Toll-like receptor (TLR), the retinoic acid-inducible gene I (RIGI) -like receptor (RLR), and NOD-like receptors are involved in the recognition of virus-specific components of naturally immune cells. It is shown that it is something like a receptor (NOD-like receptors). Among these receptor types, TLRs and RLRs are important for the production of type I interferon (IFN) and various cytokines, whereas NOD-like receptors regulate interleukin 1β (IL1β) maturation through caspase 1 activation. Detection of viral components by RLR and TLR in immune cells activates the intracellular signalling cascade, resulting in the secretion of type I Interferons, inflammatory cytokines and chemokines, and increased expression of co-stimulatory molecules such as CD40, CD80, CD86. Type I Interferon activates intracellular signalling pathways through the type I Interferons receptor and regulates the expression of various genes. IFN-inducible genes such as protein kinase R and 2'5'-oligoadenylate synthase are responsible for removing viral components from infected cells, inducing apoptosis in infected cells, and conferring resistance to viral infections in non-infected cells. Type I Interferon is produced not only by innate immune cells such as dendritic cells (DCs) and macrophages, but also by non-specailised cells such as fibroblasts. Inflammatory cytokines and chemokines are also important in inducing inflammation and eliminating viral infections by mobilizing innate and adaptive immune cells. Co-stimulatory molecules are essential for T cell activation and provide an acquired immune response.

Correspondence to: Thulesius H, Department of Health and Caring Sciences, Linnaeus University, Vaxjo, Sweden, E-mail: johan.skoog@liu.se

Received: 04-Feb-2022, Manuscript No. BLM-22-15982; **Editor assigned:** 07-Feb-2022, Pre QC No. BLM-22-15982(PQ); **Reviewed:** 21-Feb-2022, QC No. BLM-22-15982; **Revised:** 24-Feb-2022, Manuscript No. BLM-22-15982(R); **Published:** 28-Feb-2022, DOI: 10.35248/0974-8369.22.14.472.

Citation: Thulesius H (2022) Innate Immunity Against Viral Pathogens. Biol Med. 14:472.