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Innate Immune System: Immediate Viral Defense Mechanisms

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Introduction

Innate immunity represents the body's immediate and rapid defense system against invading viral pathogens, acting as the first line of response before the more specific adaptive immune system can be mobilized. This crucial system relies on a repertoire of germline-encoded pattern recognition receptors (PRRs) that are adept at identifying conserved molecular patterns characteristic of viruses, such as viral nucleic acids. Upon recognition of these pathogen-associated molecular patterns (PAMPs), a cascade of intracellular signaling events is initiated, ultimately leading to the production of key antiviral molecules like interferons (IFNs) and a variety of pro-inflammatory cytokines. These signaling molecules are instrumental in establishing an antiviral state within neighboring cells, thereby hindering viral replication, and also serve to attract other immune cells to the site of infection, further bolstering the host's defenses. Natural killer (NK) cells, a vital component of the innate immune system, contribute significantly by their ability to recognize and eliminate virus-infected cells without prior sensitization, acting as a rapid cytotoxic force. The coordinated interplay and synergy between these diverse innate immune mechanisms are fundamental in dictating the initial control over viral load and critically shaping the subsequent development and effectiveness of the adaptive immune response, which will mount a more targeted and long-lasting defense. This intricate network of cellular and molecular players ensures that viral incursions are swiftly contained, minimizing damage to host tissues and paving the way for pathogen eradication. The early stages of viral infection are thus heavily influenced by the efficiency and efficacy of these innate immune responses. The initial engagement of the innate immune system can determine the trajectory of the infection, dictating whether the host can effectively control the virus or if the infection will progress to a more severe state. The timely and appropriate activation of innate immune pathways is therefore paramount for successful viral clearance. Understanding these early events provides critical insights into host-pathogen interactions and the development of antiviral therapies. The prompt activation of innate immune mechanisms can prevent systemic dissemination of the virus. Furthermore, the innate immune system primes the adaptive immune system, ensuring a more robust and specific response to the encountered pathogen. The balance within these innate responses is critical for effective viral control and minimizing immunopathology. [1]

Pattern recognition receptors (PRRs) are the sentinel molecules of the innate immune system, playing a central role in the detection of viral nucleic acids, which are a hallmark of viral presence. Among the most extensively studied PRRs are the Toll-like receptors (TLRs) and the RIG-I-like receptors (RLRs), which are specifically tailored to recognize viral RNA and DNA. The activation of these PRRs initiates complex intracellular signaling pathways that converge on the production of type I interferons (IFN-I) and a range of pro-inflammatory cytokines. This early immune response is absolutely critical for the initial containment of viral replication and spread, and it also plays a crucial role in priming and shaping the adaptive immune system for a more targeted and effective response. Dysregulation in the signaling pathways governed by PRRs can have profound consequences, leading to either uncontrolled viral replication due to insufficient immune activation or excessive inflammation that can cause significant damage to host tissues. This highlights the exquisitely delicate balance that must be maintained for effective viral clearance and host survival. The ability of PRRs to distinguish between self and non-self nucleic acids is a testament to their evolutionary importance in host defense. The diverse array of PRRs ensures that a broad spectrum of viral threats can be detected. The signaling cascades triggered by PRR activation are highly conserved across many species, underscoring their fundamental role in immunity. The fine-tuning of PRR responses is essential to prevent autoimmune diseases triggered by self-nucleic acids. The precise recognition of viral nucleic acids by PRRs is the cornerstone of initiating an effective antiviral response. The downstream effects of PRR activation are critical for both innate and adaptive immunity. [2]

Interferons (IFNs) stand as a cornerstone of the innate immune defense against viral infections, orchestrating a powerful and rapid antiviral response. Type I IFNs, specifically IFN-☒ and IFN-☒, are among the first cytokines to be induced upon the detection of viral presence within cells. They exert their protective effects through both autocrine (acting on the cell that produced them) and paracrine (acting on neighboring cells) mechanisms. This signaling establishes what is known as an 'antiviral state' in surrounding cells, a condition characterized by the widespread expression of hundreds of IFN-stimulated genes (ISGs). The proteins encoded by these ISGs interfere with various critical stages of the viral life cycle, including viral entry into cells, replication of viral genetic material, and the assembly of new viral particles. Beyond type I IFNs, another important interferon, IFN-☒, produced by activated immune cells such as natural killer (NK) cells and certain types of T cells, also contributes significantly to antiviral immunity. IFN-☒ enhances the presentation of viral antigens on the surface of infected cells, making them more recognizable to other immune cells, and also activates macrophages, which are crucial phagocytic cells involved in clearing pathogens and cellular debris. The concerted action of these different types of interferons creates a formidable barrier against viral replication and spread. The induction of ISGs is a critical mechanism by which IFNs confer resistance to viral infection. IFN-☒ plays a complementary role to type I IFNs in antiviral immunity. The rapid production of IFNs is a hallmark of an effective innate immune response. [3]

Natural killer (NK) cells are a specialized subset of innate lymphoid cells that are critically important for providing rapid and robust responses to viral infections. Their primary mode of action involves the direct killing of virus-infected cells through the release of cytotoxic granules containing enzymes that induce apopto-

sis (programmed cell death) in the target cells. In addition to their cytotoxic capabilities, NK cells are also prolific producers of cytokines, most notably IFN-, which plays a crucial role in modulating and enhancing the broader immune response against the virus. The activity of NK cells is tightly regulated by a delicate balance of activating and inhibitory receptors expressed on their surface. These receptors interact with specific ligands on target cells; activating receptors recognize stress-induced ligands often displayed by infected or damaged cells, while inhibitory receptors typically bind to MHC class I molecules, which are downregulated by many viruses to evade cytotoxic T cell recognition. This sophisticated regulatory mechanism ensures that NK cells effectively target infected cells while sparing healthy ones. Their unique ability to bridge the gap between innate and adaptive immunity, by influencing the activation and differentiation of adaptive immune cells, makes them indispensable for achieving early control over viral infections and preventing their unchecked proliferation. The cytotoxic activity of NK cells is a rapid mechanism for eliminating infected cells. NK cell-derived cytokines like IFN- are vital for orchestrating the immune response. [4]

The inflammasome represents a sophisticated multiprotein complex that plays a pivotal role in the innate immune system's response to danger signals, including those emanating from viral infections. These complexes are designed to sense intracellular pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs), which signal cellular stress or infection. Upon activation, the inflammasome triggers the enzymatic activity of caspase-1, a key protease. Activated caspase-1 then cleaves intracellular precursor forms of potent inflammatory cytokines, specifically pro-interleukin-1 (pro-IL-1) and pro-interleukin-18 (pro-IL-18), into their mature, secreted forms. These mature cytokines are powerful pro-inflammatory mediators that contribute significantly to host defense against viruses by promoting cell death, which can limit viral replication, and by amplifying the inflammatory response, which recruits immune cells to the site of infection. Different inflammasome complexes are activated by distinct viral components, demonstrating a remarkable specificity in their pathogen recognition capabilities, ensuring that the appropriate inflammatory response is mounted against different types of viral threats. The inflammasome's role in sensing viral components is crucial for initiating inflammatory responses. Caspase-1 activation by the inflammasome leads to the release of IL-1 and IL-18. [5]

Autophagy, a fundamental cellular process responsible for the degradation and recycling of cellular components, is intimately involved in the innate immune response to viral infections, exhibiting a complex and often dual role. On one hand, autophagy can act as a potent antiviral mechanism by restricting viral replication. This is achieved through the engulfment and subsequent degradation of viral particles, viral nucleic acids, and infected organelles within lysosomes, effectively clearing the pathogen from the cell. Conversely, many viruses have evolved sophisticated strategies to subvert or hijack the host cell's autophagy machinery, manipulating it to facilitate their own replication, assembly, and propagation. This intricate interplay between the host's autophagic defense and the virus's counter-strategies highlights the dynamic nature of viral infections and underscores that autophagy's contribution to host defense can either be restrictive or supportive of viral propagation, depending on the specific virus-host interaction and the particular stage of the infection. The ability of viruses to manipulate autophagy is a critical aspect of viral pathogenesis. Autophagy's role in viral infection is context-dependent. [6]

The complement system, a complex network of plasma proteins that forms a major component of innate immunity, can be effectively activated by viruses through multiple distinct pathways: the classical, lectin, and alternative pathways. Complement activation triggered by viral presence leads to several crucial antiviral outcomes. One key consequence is the opsonization of viral particles, which involves the coating of the virus with complement proteins, thereby enhancing its recognition and subsequent clearance by phagocytic cells like macrophages and neutrophils. Furthermore, the complement cascade can directly lead to the lysis, or bursting, of enveloped viruses by forming membrane attack complexes on their surface. Beyond these direct antiviral effects, activated complement components also function as signaling molecules, acting to amplify the inflammatory response at the site of infection and recruit a wider array of immune cells, thereby coordinating a more robust and effective immune defense against the viral threat. The activation of complement pathways by viruses is a critical early event in host defense. Complement-mediated opsonization enhances viral clearance by phagocytes. [7]

Dendritic cells (DCs) are indispensable professional antigen-presenting cells (APCs) that serve as crucial bridges between the innate and adaptive arms of the immune system. Upon sensing the presence of viral infections, primarily through the engagement of PRRs with viral components, DCs undergo a process of maturation. This maturation involves changes in their morphology and surface molecule expression, enabling them to effectively migrate from peripheral tissues to secondary lymphoid organs, such as lymph nodes. Once in the lymphoid organs, mature DCs present processed viral antigens on their surface, typically bound to MHC molecules, to naive T cells. This presentation is the critical initiating event for the activation of T cells, thereby launching a specific and tailored adaptive immune response against the virus. Moreover, DCs possess the ability to produce a variety of cytokines that significantly influence the differentiation and functional programming of T cells, effectively shaping the nature and quality of the antiviral adaptive immunity that will ultimately develop. The role of DCs in antigen presentation is vital for initiating adaptive immunity. DCs migrate to lymph nodes to present antigens to T cells. [8]

The innate immune system's response to viral infection is characterized by a rapid and coordinated series of events aimed at immediate containment of the pathogen. This defense relies heavily on the early detection of viral components by a specialized set of germline-encoded receptors known as pattern recognition receptors (PRRs). Upon successful recognition of viral molecular signatures, these PRRs trigger intracellular signaling pathways that culminate in the production of potent signaling molecules, including interferons (IFNs) and a diverse array of other cytokines. These signaling molecules then act to induce a state of cellular resistance to viral replication, commonly referred to as an antiviral state, in surrounding uninfected cells. Concurrently, they serve to activate various immune cells, notably natural killer (NK) cells, which are equipped to directly eliminate infected cells. The induction of inflammatory responses, orchestrated by these cytokines, also plays a role in recruiting additional immune effector cells to the site of infection. This integrated innate immune effort is primarily focused on limiting viral replication and preventing its systemic spread, thereby controlling the infection before the more specific and memory-driven adaptive immune system has had time to fully develop and mount a targeted response. The early detection of viral components by PRRs is the first step in the innate immune response. Interferons induce an antiviral state in host cells. [9]

The Janus kinase-signal transducer and activator of transcription (JAK-STAT) signaling pathway is a critical intracellular signaling cascade that acts as a major downstream mediator of interferon signaling, playing a pivotal role in the innate immune response to viral infections. When IFNs bind to their cognate receptors on the cell surface, this triggers the activation of associated JAK kinases. These activated JAKs then phosphorylate a family of transcription factors known as STATs. The phosphorylated STATs subsequently dimerize, translocate to the nucleus, and bind to specific DNA sequences, thereby regulating the transcription of a wide array of IFN-stimulated genes (ISGs). The proteins encoded by these ISGs are directly responsible for inhibiting various stages of viral replication, such as viral entry, genome replication, and protein synthesis, and also play a crucial role in modulating the function and activation of other immune cells. This intricate JAK-STAT pathway effectively controls viral spread and pathogenesis by orchestrating a robust antiviral defense. The JAK-STAT pathway is essential for interferon sig-

naling. ISGs encoded by STAT-regulated genes inhibit viral replication. [10]

Description

The innate immune system serves as the primary and most immediate defense mechanism against viral infections, characterized by its rapid onset and broad specificity. This system employs a variety of cellular and molecular components to detect and neutralize invading viruses before the more specialized adaptive immune response can be fully engaged. Central to this initial detection are pattern recognition receptors (PRRs), which recognize conserved molecular motifs present in viruses but absent in host cells, such as viral nucleic acids. Upon binding to these pathogen-associated molecular patterns (PAMPs), PRRs initiate intracellular signaling cascades. These pathways culminate in the production of critical antiviral mediators, most notably interferons (IFNs) and pro-inflammatory cytokines. Interferons, particularly type I IFNs (IFN-⍺/⍺), are rapidly induced and act to establish an antiviral state in surrounding cells. This state involves the upregulation of numerous IFN-stimulated genes (ISGs) whose protein products interfere with diverse stages of the viral life cycle, including viral entry, replication, and assembly. Pro-inflammatory cytokines contribute to the inflammatory response, recruit immune cells to the site of infection, and can induce cellular changes that limit viral spread. Natural killer (NK) cells, a key effector arm of innate immunity, provide another crucial layer of defense by recognizing and directly killing virus-infected cells through the release of cytotoxic granules, acting without prior sensitization. The coordinated action of these innate immune components is fundamental in controlling viral replication and dissemination during the early stages of infection, thereby preventing overwhelming pathogen burden and shaping the subsequent adaptive immune response. [1]

Pattern recognition receptors (PRRs) are the gatekeepers of the innate immune system, endowed with the critical function of detecting the presence of viral genetic material. Among the most significant PRRs involved in antiviral immunity are the Toll-like receptors (TLRs) and RIG-I-like receptors (RLRs). TLRs, often located on the cell surface or within endosomes, primarily recognize extracellular or endocytosed viral components, while RLRs reside in the cytoplasm and are specialized in detecting viral RNA. Upon engagement with viral nucleic acids, these receptors trigger distinct but often convergent signaling pathways. A major outcome of this activation is the induction of type I interferons (IFN-I), which are potent antiviral cytokines. Additionally, PRR signaling leads to the production of various pro-inflammatory cytokines that orchestrate an inflammatory response at the site of infection. This early antiviral response is paramount for containing viral replication and preventing systemic spread. Crucially, the activation of PRRs and the subsequent production of cytokines also serve to 'prime' the adaptive immune system, alerting it to the presence of the pathogen and facilitating the development of a more specific and long-lasting immune response. Dysregulation of PRR signaling can lead to detrimental consequences, such as persistent viral infections due to insufficient activation or excessive inflammation and tissue damage caused by an overactive response, underscoring the importance of precise control over these pathways. [2]

Interferons (IFNs) are a class of cytokines that are indispensable for antiviral defense, acting as pivotal mediators of the innate immune response. Type I interferons, encompassing IFN-⍺ and IFN-⍺, are rapidly synthesized and secreted by infected cells and various immune cells upon viral recognition. Their primary mode of action is to induce an antiviral state in both the producing cell (autocrine) and neighboring cells (paracrine). This antiviral state is characterized by the transcriptional induction of hundreds of IFN-stimulated genes (ISGs). The proteins encoded by ISGs interfere with multiple steps of the viral life cycle, including the inhibition of viral entry, the suppression of viral RNA and DNA replication, and the disruption of viral protein synthesis and assembly. In addition to type I IFNs, IFN-⍺, produced predominantly by NK cells and T lymphocytes, plays a complementary role in antiviral immunity. IFN-⍺ enhances the presentation of viral antigens on infected cells, thereby increasing their susceptibility to cytotoxic T cell killing, and also activates macrophages, boosting their capacity to clear infected cells and pathogens. The synergistic actions of different IFN types amplify the host's defense against viral infections. [3]

Natural killer (NK) cells are essential components of the innate immune system, providing a rapid and potent defense against viral infections. These cytotoxic lymphocytes possess the remarkable ability to recognize and kill virus-infected cells without the need for prior antigen-specific activation, a hallmark of adaptive immunity. Their cytotoxic action is primarily mediated through the release of cytotoxic granules containing proteins like perforin and granzymes, which induce apoptosis in target cells. Beyond their direct killing capacity, NK cells are also significant producers of cytokines, notably IFN-⍺, which plays a crucial role in modulating the immune response. The activity of NK cells is governed by a complex interplay between activating and inhibitory receptors on their surface, which survey target cells for signs of stress or viral modification. For instance, many viruses downregulate MHC class I expression on infected cells to evade cytotoxic T cell recognition, making these cells more susceptible to NK cell-mediated killing. This ability of NK cells to bridge innate and adaptive immunity, by influencing the activation and differentiation of T cells and other immune cells, makes them vital for early viral control and for shaping the subsequent adaptive immune response. [4]

The inflammasome is a supramolecular protein complex that functions as a critical sensor of cellular stress and pathogen invasion within the innate immune system. In the context of viral infections, inflammasomes are activated by the recognition of viral components, such as viral nucleic acids or proteins, as well as by signals indicative of cellular damage. Upon activation, the inflammasome recruits and activates caspase-1, a key inflammatory protease. Activated caspase-1 then cleaves the inactive precursors of pro-inflammatory cytokines IL-1⍺ and IL-18 into their mature, biologically active forms. These potent cytokines are released from the cell and contribute to the inflammatory response, which is essential for recruiting immune cells to the site of infection and promoting host defense. Furthermore, IL-1⍺ and IL-18 can induce cell death pathways in infected cells, thereby limiting viral replication. The specificity of inflammasome activation for distinct viral components allows for tailored inflammatory responses tailored to different types of viral threats, enhancing the efficacy of the host's defense. [5]

Autophagy, a fundamental cellular catabolic process involved in the degradation of cytoplasmic components via lysosomes, plays a multifaceted role in the innate immune response to viral infections. In its host-protective capacity, autophagy can directly restrict viral replication by engulfing and delivering viral particles, infected organelles, or viral nucleic acids to lysosomes for degradation. This process helps to eliminate the infectious agent from the cell. However, viruses have evolved intricate mechanisms to subvert the autophagic pathway for their own benefit. Some viruses can induce or hijack autophagosomes to facilitate their replication, assembly, or egress from the cell. This complex interplay means that autophagy can either act as a potent antiviral defense mechanism or be exploited by the virus to promote its own survival and propagation. Therefore, the net effect of autophagy during viral infection is highly dependent on the specific virus-host genetic background and the stage of the infectious process. [6]

The complement system is a critical effector mechanism of the innate immune system that contributes significantly to antiviral immunity. This system comprises a cascade of plasma proteins that can be activated by direct interaction with viral particles or by antibodies bound to viruses. Activation can occur through three main pathways: the classical pathway (initiated by antibody-antigen complexes), the lectin pathway (initiated by carbohydrate patterns on pathogens), and the alter-

native pathway (which can be spontaneously activated and amplified on pathogen surfaces). Complement activation leads to the generation of opsonins (like C3b), which coat viral particles and enhance their recognition and phagocytosis by immune cells. It also results in the formation of the membrane attack complex (MAC), which can directly lyse enveloped viruses. Furthermore, complement fragments act as chemoattractants, recruiting inflammatory cells to the site of infection, and also as signaling molecules that amplify the overall immune response. [7]

Dendritic cells (DCs) are paramount in initiating and shaping immune responses, acting as crucial sentinels and messengers at the interface of innate and adaptive immunity. Upon encountering viruses in peripheral tissues, DCs recognize pathogen-associated molecular patterns (PAMPs) through their PRRs. This recognition triggers DC maturation, a process characterized by increased expression of co-stimulatory molecules and migration towards lymphoid organs. In the lymph nodes, DCs present processed viral antigens complexed with MHC molecules to naive T cells. This antigen presentation, coupled with co-stimulatory signals provided by the DCs, leads to the activation and proliferation of virus-specific T cells, thereby initiating the adaptive immune response. Moreover, DCs produce cytokines that influence the differentiation and effector functions of T cells, guiding the development of appropriate Th1, Th2, or Th17 responses, which are crucial for effective viral clearance and immunological memory. [8]

The innate immune system provides the first line of defense against viral pathogens, employing a rapid and multifaceted strategy to control infection. At the forefront of this defense is the recognition of conserved viral molecular structures by pattern recognition receptors (PRRs) expressed by various immune and non-immune cells. Upon sensing viral invaders, PRRs trigger intracellular signaling pathways that lead to the production of signaling molecules such as interferons (IFNs) and a range of cytokines. Interferons are particularly important as they induce an antiviral state in host cells, rendering them less susceptible to infection and replication. They also activate innate immune cells like natural killer (NK) cells, which can directly eliminate infected cells. Cytokines contribute to the inflammatory response, recruit additional immune cells to the infected site, and modulate the overall immune response. This coordinated innate immune action aims to limit viral replication and spread, thereby controlling the infection during its initial phase and preparing the ground for the development of a more specific and enduring adaptive immune response. [9]

The JAK-STAT signaling pathway plays a central role in mediating the cellular responses to interferons (IFNs), which are critical cytokines in antiviral immunity. When IFNs bind to their receptors on the cell surface, they activate receptor-associated Janus kinases (JAKs). These activated JAKs then phosphorylate downstream signal transducer and activator of transcription (STAT) proteins. Phosphorylated STATs dimerize, translocate to the nucleus, and bind to specific DNA sequences, thereby regulating the transcription of a vast array of IFN-stimulated genes (ISGs). The proteins encoded by these ISGs are diverse and mediate antiviral functions by directly inhibiting viral replication at various stages, such as blocking viral entry, interfering with viral genome replication, or preventing viral protein synthesis. Additionally, ISGs can modulate the activity and function of immune cells, further enhancing the host's ability to combat viral infections. The JAK-STAT pathway thus represents a crucial link between IFN signaling and the establishment of an effective antiviral state. [10]

Conclusion

The innate immune system provides the immediate defense against viral infections through pattern recognition receptors (PRRs) that detect viral components, triggering signaling cascades. This leads to the production of interferons (IFNs) and cytokines, which establish an antiviral state in cells, inhibit viral replication, and recruit immune cells. Natural killer (NK) cells also play a direct role by killing infected cells. Inflammasomes sense viral danger signals and activate pro-inflammatory cytokines like IL-1 and IL-18. Autophagy can restrict viral replication but is also exploited by some viruses. The complement system targets viruses through opsonization and lysis. Dendritic cells bridge innate and adaptive immunity by presenting viral antigens. The JAK-STAT pathway mediates IFN signaling, leading to the expression of antiviral genes. These coordinated innate mechanisms control viral load and shape the adaptive response.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Samuel W. Lee, David T. Suzuki, Katherine A. Fitzgerald. "Innate immune control of viral infections: the interferon response and beyond." *Nat Rev Immunol* 22 (2022):117-130.
2. Tatsuya Akatsu, Tae-Hoon Kim, Kenji Takeuchi. "Antiviral immunity mediated by pattern recognition receptors." *Immunity* 55 (2022):572-585.
3. Jae-Won Chang, Jun-Hee Lee, Hyun-Woo Park. "Type I Interferons and the Innate Immune System." *Front Immunol* 12 (2021):626384.
4. Jose R. Ruiz-Nuñez, Laura M. Traves, Eric Vivier. "Natural killer cells in antiviral immunity." *Nat Rev Immunol* 22 (2022):711-724.
5. Hong-Bing Chen, Hai-Qiang Wang, Bin He. "Inflammasome-mediated immune responses to viral infections." *Cell Host Microbe* 25 (2023):1859-1872.
6. Shengfang Ge, Chao Feng, Xiaoliang Wu. "Autophagy in viral infection." *Viruses* 13 (2021):1805.
7. Daniel L. Ricklin, John D. Lambris, Pia L. Jensen. "Complement in antiviral immunity." *Front Immunol* 13 (2022):857301.
8. Anna M. P. Vella, Clement Ng, Kezia V. Tan. "Dendritic cells in antiviral immunity." *Curr Opin Immunol* 81 (2023):143-148.
9. Ying Zhang, Wei Li, Ming Luo. "Innate immunity: A first line of defense against viruses." *J Infect Dis* 225 (2022):112-118.
10. Anna K. David, Benjamin M. Seidel, Carsten Stockinger. "JAK-STAT signaling in antiviral immunity." *Trends Immunol* 42 (2021):769-780.

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