



ADENOVIRUSES INFECT HUMANS THROUGH THE RESPIRATORY AND GASTROINTESTINAL TRACTS

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ABSTRACT

Adenovirus infections primarily affect the respiratory and gastrointestinal tracts. Adenoviruses consist of a linear, double-stranded DNA genome of approximately 36 Kb and an icosahedral, non-enveloped capsid. The main proteins of the virus are fibers which bind to the target cell, pentons which serve for cell penetration, and hexons which comprise the main structure. The fiber protein binds to specific host receptors in the respiratory tract. In the gastrointestinal tract, integrins $\alpha\beta 3$ and $\alpha\beta 5$ may also be involved. This is followed by penetration, transport, and release of the genome. The viral DNA is released into the nucleus through nuclear pores. Viral replication involves the transcription of the early genes E1A, E1B, E2, E3, and E4, and late expression involves the L1-L5 structural genes for capsid assembly. Clinical effects include pneumonia, nasopharyngitis, tonsillitis, and bronchitis, with gastrointestinal symptoms that may include diarrhea, nausea, and vomiting, especially in children. No specific antiviral therapy is routinely effective.

KEYWORDS: *Adenovirus, infection, respiratory tract, gastrointestinal tract, viral protein*



MODULATION OF THE IMMUNE SYSTEM BY BACTERIAL, PARASITIC, AND VIRAL INFECTIONS

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ABSTRACT

The immune system defends the body from pathogenic microorganisms such as viruses, bacteria, and parasites. The innate response against pathogenic viruses is implemented through the recognition of viral RNA or DNA by Toll-like receptors (TLRs)-3, -7, and -9 and the cytosolic receptors retinoic acid-inducible gene I (RIG-I) and melanoma differentiation-associated gene 5 (MDA5). Viruses typically induce the production of type I interferon (IFN)- α and - β , which activate natural killer (NK) cells to kill infected cells. In bacterial infections, the innate immune response occurs through pattern recognition receptors (PRRs) with recognition of TLRs that identify bacterial components such as lipopolysaccharide (LPS) (using TLR4), peptidoglycans (TLR2), and flagellin (TLR5). The production of inflammatory cytokines can induce local and systemic inflammation with fever. The immune system responds specifically to the type of pathogen, and each pathogen develops mechanisms to evade these responses.

KEYWORDS: *Infection, pathogenic microorganism, immune modulation, immune response, innate immunity*



INNATE IMMUNITY AND THE LOSS OF CD4+ LYMPHOCYTES IN HIV INFECTION

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ABSTRACT

Human immunodeficiency virus (HIV) primarily targets CD4+ T cells, which are important lymphocytes of the adaptive immune system. During HIV infection, the progressive loss of CD4+ lymphocytes plays a key role in the immune response, and the loss of these cells leads to acquired immunodeficiency syndrome (AIDS). Immune deficiency begins with the activation of macrophages, natural killer (NK) cells, and dendritic cells. These cells are activated by HIV via Toll-like receptors (TLRs) responding to viral RNA, resulting in the induction of NF- κ B and the production of cytokines and interferons (IFNs). Macrophages act as reservoirs for the virus and produce inflammatory cytokines that mediate chronic inflammation, activated dendritic cells produce IFN α that contributes to immune activation and CD4+ T cell death, and NK cells kill HIV-infected cells by releasing viruses into the surrounding environment. HIV infects CD4+ T cells activated via the coreceptors CCR5 or CXCR4, and therefore, infected cells undergo apoptosis. Uninfected cells also die due to the lytic action of inflammatory cytokines and exposure to the envelope protein gp120. Quiescent CD4+ lymphocytes die through caspase-1-mediated pyroptosis, with viral RNA being transformed into incomplete DNA that is recognized by the innate immune sensor IFI16, which activates the caspase-1 inflammasome.

KEYWORDS: *HIV infection, AIDS, CD4+ cell, lymphocyte, innate immunity*



NLRP3 MEDIATES THE INFLAMMATORY RESPONSE IN INFLUENZA VIRUS INFECTION

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ABSTRACT

In influenza virus infection, NLR family pyrin domain containing 3 (NLRP3) is a key mediator that plays both protective and pathogenic roles. The influenza A virus induces cellular damage due to reaction oxygen species (ROS) formation, ionic changes, damage-associated molecular pattern (DAMP) release, and M2 ion channel activity. These signals lead to activation of the NLRP3 sensor, which activates caspase-1, which processes pro-IL-1 β and pro-IL-18 into mature IL-1 β and IL-18. The protective role of NLRP3 involves the activation of IL-1 β and IL-18, which stimulate the recruitment and antiviral activation of immune cells such as neutrophils, natural killer (NK) cells, and T cells. The pathogenic role of NLRP3 occurs as a result of its excessive activation, which leads to the excessive production of inflammatory cytokines.

KEYWORDS: *NLRP3, NLR, infection, inflammasome, inflammatory response, influenza virus*



HEPATITIS B AND C VIRUSES ACTIVATE INNATE IMMUNE RECEPTORS AND PATHOGEN- OR DAMAGE-ASSOCIATED MOLECULAR PATTERNS

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ABSTRACT

Hepatitis B virus (HBV) and hepatitis C virus (HCV) activate the innate immune response and subsequently, the adaptive immune response. HBV and HCV are viruses that primarily infect the human liver, which can lead to chronic hepatitis, cirrhosis, and often, liver cancer. These two viruses replicate differently and both recruit innate immune cells that rush to defend the body. Activation of innate immunity occurs through pattern recognition receptors (PRRs) that are detected by innate immune receptors such as Toll-like receptors (TLRs), which detect viral RNA or DNA. RIG-I-like receptors (RLRs) detect viral RNA in the cytoplasm. NOD-like receptors (NLRs) can respond to viral infection and cellular stress. These receptors act as pathogen-associated molecular patterns (PAMPs) and activate antiviral immunity. Infection can cause cellular damage mediated by damage-associated molecular patterns (DAMPs).

KEYWORDS: *Hepatitis B, hepatitis C, virus, pattern recognition receptor, Toll-like receptor, pathogen-associated molecular pattern, damage-associated molecular pattern*