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THE SEVERE ACUTE RESPIRATORY SYNDROME CORONAVIRUS 2 (SARS-COV-2) CAUSES THE NOVEL COVID-19 PANDEMIC: VIRAL TRANSMISSION AND PREVENTION

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ABSTRACT

Severe acute respiratory syndrome Coronavirus 2 (SARS-CoV-2) is the virus that causes COVID-19 and activates the immune system, causing inflammation. SARS-CoV-2 infects lung cells and triggers an immune and inflammatory response, which aim to eliminate the virus.The entry of the virus into the body is handled by the innate immune system, which is the first line of defense mediated by macrophages, dendritic cells, and natural killer (NK) cells. Inflammatory proteins, such as cytokines and chemokines, are produced during the innate response, and in COVID-19, these proteins can cause an excessive inflammatory response called a cytokine storm that damages the lungs and can lead to death. During the adaptive immune response, T cells, including helper T cells and cytotoxic T cells, play a crucial role. Helper T cells produce IL-4 that helps B cells produce antibodies specific to SARS-CoV-2. Vaccines against SARS-CoV-2 are important to effectively fight the virus, reducing the risk of severe disease and transmission. Effective and preventative treatments are needed to mitigate COVID-19 disease.

KEYWORDS: *SARS-CoV-2, COVID-19, inflammation, infection, immune response*

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SARS-COV-2 INFECTION TRIGGERS THE IMMUNE SYSTEM AND CAUSES INFLAMMATION

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ABSTRACT

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection leads to an effective inflammatory response which can also cause severe pathological damage. Inflammation from SARS-CoV-2 involves dysregulated cytokine production, complement activation, cardiovascular damage, and endothelial dysfunction. The SARS-CoV-2 virus infects cells by binding its spike protein to the host cell angiotensin-converting enzyme 2 (ACE2) receptor, which is expressed in cardiac, gastrointestinal, renal, and other tissues. The viral genome is recognized by intracellular pattern recognition receptors (PRRs) such as Toll-like receptors (TLRs). PRRs recognize viral RNA as a pathogen-associated molecular pattern (PAMP), triggering mitochondrial antiviral signaling cascades and activating MyD88. These pathways ultimately activate transcription factors, such as nuclear factor kappa B (NF-κB) and interferon regulatory factors (IRFs) which induce the production of pro-inflammatory cytokines and type I interferons (IFNs). Immune activation involves the complement system, which becomes overactive in COVID-19 patients and leads to the production of C3a and C5a, which are potent inflammatory mediators. In COVID-19, endothelial damage with production of adhesion molecules, inflammation, and endothelial dysfunction may also occur. Inhibitors of pro-inflammatory cytokines may be helpful and reduce mortality in COVID-19.

KEYWORDS: *SARS-CoV-2, COVID-19, infection, immune activation, inflammation*

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COVID-19 DIAGNOSIS AND PATHOGENESIS

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ABSTRACT

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a single-stranded RNA coronavirus that causes COVID-19. The virus enters the body through the respiratory tract and binds to the angiotensin-converting enzyme 2 (ACE2) receptor found on lung epithelial cells and endothelial cells. SARS-CoV-2 replicates and activates the innate immune response, causing a cytokine storm with the release of pro-inflammatory cytokines (primarily IL-6, TNF, and IL-1β). The powerful inflammation causes acute lung injury (ARDS) and multiple organ dysfunction syndrome (MODS). Diagnostic symptoms include dry cough, fever, fatigue, anosmia, ageusia, dyspnea, and gastrointestinal symptoms, amongst others. Clinical presentation may be asymptomatic, mild, moderate, or severe. Laboratory diagnosis includes reverse transcriptase polymerase chain reaction (RT-PCR) and viral RNA detection from nasopharyngeal swabs. Rapid antigen tests detect viral proteins, while serological tests detect IgG and IgM. The virus can infect the endothelium, causing vasculitis, thrombosis, and endothelial dysfunction. From a pathophysiological standpoint, COVID-19 is characterised by a wide spectrum of clinical manifestations, ranging from asymptomatic forms to severe acute respiratory syndromes. The virus enters cells, triggering an inflammatory cascade that can lead to endothelial dysfunction, thrombosis, and cytokine storms, with systemic consequences.

KEYWORDS: *SARS-CoV-2, COVID-19, diagnosis, pathogenesis, symptoms*

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NEUROLOGICAL DISORDERS ASSOCIATED WITH HIV INFECTION: THE IMMUNE AND INFLAMMATORY RESPONSE

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ABSTRACT

Human immunodeficiency virus (HIV) infection can cause neurological disorders and promote activation of the immune system and an inflammatory response in the central nervous system (CNS). HIV-1 crosses the blood-brain barrier (BBB) and directly infiltrates the brain or can infiltrate through infected cells, such as macrophages or CD4 T cells, that have bound the virus. Some viral proteins, such as Tat and gp120, penetrate the BBB through endothelial cells. Microglia and macrophages activated by the virus secrete high levels of pro-inflammatory cytokines such as tumor necrosis factor (TNF), interleukin (IL)-1β, IL-6, and IL-18, reactive oxygen species (ROS), causing neurotoxicity. HIV-1 can also infect astrocytes, which shed viral proteins and other toxic substances that can enhance cytotoxicity. In addition to infecting CD4+ cells with deplition, HIV-1 also provokes immunodeficiency, increasing the risk of opportunistic infections. Neurological complications of HIV include a range of symptoms, including disrupted concentration and memory function, changes in mood, sensory loss, weakness in the body, and increased risk of tumors, such as primary CNS lymphoma.
Common HIV-associated neurological disorders included HIV-Associated Neurocognitive Disorder (HAND), peripheral neuropathy, and HIV-associated vacuolar myelopathy (VM). HIV directly damages nerve cells and can trigger inflammation, leading to a range of cognitive, motor, and behavioral changes.

KEYWORDS: *HIV, infection, neurological disorder, central nervous system, HAND*

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SALMONELLA INFECTION: INNATE AND ADAPTIVE IMMUNITY

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ABSTRACT

The immune response to Salmonella infection involves both innate and adaptive immune mechanisms. Salmonella bacterium causes salmonellosis infection contracted through contaminated products, such as water and food. The bacterial infection transmitted through food is very common and can cause gastroenteritis characterized by abdominal pain, diarrhea, fever and vomiting, and, in severe cases, can cause systemic diseases including typhoid fever. Salmonella enters the stomach and survives the acidic environment with low pH levels and then passes into the small intestine by adhering to the intestinal epithelium. Salmonella injects effector proteins into intestinal epithelial cells, manipulating the host's cellular mechanisms and promoting bacterial entry. The bacterium modifies immune responses which allows it to avoid degradation and to replicate, and leads to cell lysis or release of bacteria into the bloodstream or other tissues in severe infections. In some cases, Salmonella can escape from the intestines and enter the bloodstream, causing a systemic infection with fever. Salmonella utilizes TTSS-1 and TTSS-2 molecules that inject effectors directly into host cells; TTSS-1 is primarily responsible for invasion, while TTSS-2 is used in infection to facilitate intracellular survival. Salmonella also secrete effector proteins such as SopE, SopB, and SipA, which help manipulate host cell signaling, enabling immune evasion. Infection triggers the release of pro-inflammatory cytokines (e.g., IL-1, IL-6, TNF-alpha), which leads to the recruitment of immune cells to the site. Vaccines are currently available for systemic infections such as typhoid fever, but those for non-typhoidal Salmonella infections are also under development.

KEYWORDS: *Salmonella infection, bacteria, innate immunity, adaptive immunity*