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B AND T CELL RESPONSE TO SARS-COV-2 INFECTION

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ABSTRACT

The immune system is activated during infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus that causes coronavirus disease (COVID-19). Adequate inflammation helps eliminate the infection, but excessive inflammation leads to serious complications for the patient infected with SARS-CoV-2. The strong immune response can lead to uncontrolled inflammation that can result in a cytokine storm. T cells are a type of lymphocyte that participate in the adaptive immune response and play a key role in fighting viral infections. Cytotoxic T cells (CD8+) directly attack and kill cells infected by viruses, and in SARS-CoV-2 infection, cytotoxic T cells recognize infected cells and destroy them, reducing viral replication. Helper T cells (CD4+) are so called because they help other immune cells by releasing cytokines that activate and guide cytotoxic T cells and B cells. In COVID-19, CD4+ cells help orchestrate an immune response against the virus and are responsible for producing antibodies by B cells, which can neutralize the viruses. Both T and B cells coordinate to fight SARS-CoV-2 infection by recognizing infected cells (T cells) and producing neutralizing antibodies (B cells).

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

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SARS-COV-2 INDUCES COVID-19 AND CAUSES DEADLY LUNG INFLAMMATION

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ABSTRACT

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the virus responsible for coronavirus disease 2019 (COVID-19) which can cause severe lung inflammation, a hallmark symptom of the disease. The virus enters cells in the respiratory tract by binding to the angiotensin-converting enzyme 2 (ACE2) receptor via its spike protein and infects type II pneumocytes through endocytosis. Transmembrane serine protease 2 helps activate the spike protein, facilitating viral entry into the host cell. Inside the cell, the virus releases RNA, needed to produce viral proteins, into the cytoplasm that helps the virus replicate.The infection activates the immune system by engaging pattern recognition receptors (PRRs) and Toll-like receptors (TLRs) that recognize viral RNA and induce NF-kB transcription with production of inflammatory cytokines. In COVID-19, the immune response can become dysregulated and may lead to a "cytokine storm", an event characterized by an abundant release of pro-inflammatory cytokines which can cause severe lung inflammation. With lung inflammation, various biochemical pathways are activated, such as NF-κB, JAK-STAT, and the NLRP3 inflammasome, leading to the production of pro-inflammatory cytokines that contributes to the worsening of lung inflammation. Lung inflammation leads to tissue damage, edema formation, and fibrosis, that can lead to multi-organ failure and patient death in severe cases.

KEYWORDS: *Lung inflammation, SARS-CoV-2, COVID-19, virus, inflammatory cytokine, cytokine storm*

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ROLE OF ACE2 IN SARS-COV-2 INFECTION

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ABSTRACT

The angiotensin-converting enzyme 2 (ACE2) receptor is used by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) to cause coronavirus disease 2019 (COVID-19). ACE2 is a transmembrane protein found in several human cell membranes, including blood vessels, lungs, heart, kidneys, and intestines. ACE2 regulates the renin-angiotensin-aldosterone system (RAAS) by converting angiotensin II to angiotensin 1–7. These angiotensins can have both vasoconstrictor and vasodilator effects, respectively, and are involved in the regulation of blood pressure and inflammation. Angiotensin-converting enzyme (ACE) and ACE2 regulate the renin-angiotensin system and thus, cardiovascular vasoactive function. SARS-CoV-2 binds to this receptor, causing COVID-19 that contributes to systemic inflammation. Models using SARS-CoV-2 and ACE2 have enabled novel therapies and a better understanding of COVID-19.

KEYWORDS: *ACE2 receptor, SARS-CoV-2, COVID-19, angiotensin, renin-angiotensin system*

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COVID-19 VACCINE STRATEGIES ARE NECESSARY TO FIGHT SARS-COV-2 INFECTION

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ABSTRACT

The immune system is involved in the biochemical mechanisms that occur during severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and the development of vaccines to combat coronavirus disease 2019 (COVID-19). SARS-CoV-2 infects human cells by activating immunity that should provide protection. The spike protein S is used by the SARS-CoV-2 virus to bind to the angiotensin-converting enzyme 2 (ACE2) receptor on host cells, especially in the respiratory system. The ACE2 receptor present on lung cells, cardiac tissue, kidneys and the intestines allows the virus to enter the cell. ACE2 is widely expressed in the lungs, heart, kidneys and intestine. The S protein of SARS-CoV-2 has a strong affinity for ACE2, facilitating viral entry. Viral particles are assembled in the Golgi apparatus and released by exocytosis. The innate immune system detects viral components, such as RNA, using pattern recognition receptors (PRRs) such as Toll-like receptor (TLR)7 and TLR8, which lead to the production of cytokines (IL-6, TNF e IL-1β), initiating an inflammatory response. In adaptive immunity, B cells recognize viral antigens and, once activated, differentiate into plasma cells to produce antibodies, particularly IgG and IgA. Helper T cells (CD4+) support B cell function, while cytotoxic T cells (CD8+) target and kill infected cells. COVID-19 mRNA vaccines use a variety of approaches to stimulate an immune response without causing disease. These vaccines deliver synthetic mRNA encoding the SARS-CoV-2 spike protein. Upon entry into host cells, this mRNA is translated by ribosomes into a spike protein that is processed and presented on major histocompatibility complex (MHC) molecules, activating both CD4+ and CD8+ cells. Studies focusing on the mechanisms of viral entry and the immune response are crucial to understanding COVID-19 infection, immunity, and vaccine development.

KEYWORDS: *COVID-19, vaccine, SARS-CoV-2, mRNA, viral entry, infection*

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THE INTERFERON RESPONSE IN SARS-COV-2 INFECTION

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ABSTRACT

In 2020, interferon gamma (IFNγ) was found to be involved in critical cases of Coronavirus Disease 2019 (COVID-19) pneumonia. Interferons (IFNs) are signaling proteins in the immune system that play a central role in the response to viral infections, including that of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus responsible for COVID-19. When cells detect the presence of the virus, they release IFNs that signal other cells to increase their antiviral defenses. However, SARS-CoV-2 is capable of evading the immune system and developing mechanisms to disrupt IFN responses. During SARS-CoV-2 viral infection, type I IFN-α and -β are generated to counteract the pathogenic effect of the virus. Starting from the beginning of the infection, SARS-CoV-2 replicates by implementing mechanisms to suppress IFNs and therefore immunity. The evasion of IFNs by SARS-CoV-2 can lead to uncontrolled viral spread and the eventual immune response can become excessive and result in a "cytokine storm" with severe lung inflammation. Therapeutic approaches aim to administer exogenous IFNs early in the infection, which could offset the virus's immune evasion tactics.

KEYWORDS: *Interferon, COVID-19, signaling protein, viral infection, SARS-CoV-2*