



# THE SEVERE ACUTE RESPIRATORY SYNDROME CORONAVIRUS-2 IS A HIGHLY CONTAGIOUS RESPIRATORY PATHOGEN

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## ABSTRACT

Severe acute respiratory syndrome Coronavirus 2 (SARS-CoV-2) is a highly contagious virus that causes coronavirus disease 2019 (COVID-19). The virus spreads mainly through respiratory droplets when an infected person coughs, sneezes, talks or breathes, making it easily transmissible. SARS-CoV-2 reproduces with a high  $R_0$  value that is between 2 and 3, indicating that one person could potentially infect more than one other. Symptoms of the infection can appear after 2-14 days and patients may also show no symptoms. Several biological pathways in humans are exploited by SARS-CoV-2 including modulation of the renin-angiotensin system, the cytokine storm pathway, and hypoxia and hypoxic pathways. Preventive measures, such as hand hygiene, ventilation, and wearing a mask, reduce the transmission of the virus and COVID-19 disease.

**KEYWORDS:** *SARS-CoV-2, virus, COVID-19, infection, transmission*



# ROLE OF PATTERN RECOGNITION RECEPTORS IN IMMUNE AND INFLAMMATORY RESPONSES

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## ABSTRACT

Pattern recognition receptors (PRRs) are germline-encoded receptors (encoded in DNA from birth) that present on various cell types such as macrophages, dendritic cells, neutrophils, epithelial cells, and endothelial cells. PRRs detect two types of signals: Pathogen-associated molecular patterns (PAMPs) which are conserved microbial molecules such as lipopolysacchired (LPS), peptidoglycan, and viral RNA, and damage-associated molecular patterns (DAMPs), which are molecules such as ATP, urate, and cytosolic DNA/RNA released by damaged cells. The PRR family includes Toll-like receptors (TLRs), NOD-like receptors (NLRs), RIG-I-like receptors (RLRs), and C-type lectin receptors (CLRs). TLRs are required for cytokine activation through MyD88/TRIF and NF- $\kappa$ B; NLRs are required for inflammasome formation, caspase-1 formation, and IL-1 $\beta$ /IL-18; RLRs are required for the production of anti-viral type I interferons (IFNs); and CLRs are required for opsonization and phagocytosis. PRRs on antigen presenting cells (APCs) induce expression of costimulatory factors (CD80/86, CD40) and modulate Th1/Th2/Th17 polarization. In conclusion, PRRs recognize conserved signals from pathogens and host cell damage, trigger inflammation, and initiate adaptive immunity. If activated uncontrollably, they can cause systemic damage or chronic disease.

**KEYWORDS:** *Pattern recognition receptor, pathogen-associated molecular pattern, damage-associated molecular pattern, immune response, inflammation*



## COVID-19 RESPIRATORY DISEASE

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### ABSTRACT

Coronavirus disease 2019 (COVID-19) is a respiratory disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). From a molecular and cellular perspective, the infection involves a complex series of events that lead to respiratory compromise and other systemic complications. SARS-CoV-2 is a positive-sense RNA virus enveloped in a lipid envelope containing four main structural proteins: Spike (S), which is important for cell entry; Envelope (E), Membrane (M), and Nucleocapsid (N). Infection begins with the binding of the S protein to the angiotensin-converting enzyme 2 (ACE2) receptor on target cells. Activation of intracellular receptors including Toll-like receptors (TLRs), RIG-I-like receptors (RLRs), and MDA5 receptors, that recognize viral RNA leads to the production of type I interferons (IFN- $\alpha$ , IFN- $\beta$ ) and pro-inflammatory cytokines (IL-6, TNF, IL-1 $\beta$ ). In COVID-19, the adaptive response activates T and B lymphocytes, particularly CD8<sup>+</sup> lymphocytes, which eliminate infected cells, and CD4<sup>+</sup> lymphocytes, which support B lymphocytes. COVID-19 causes alveolar collapse due to infected alveoli, pulmonary edema with interstitial pneumonia, and acute respiratory distress syndrome (ARDS) due to inflammatory cytokines and hypoxemia. In conclusion, COVID-19, caused by the SARS-CoV-2 virus, is a respiratory disease with profound implications at both the molecular and cellular levels. SARS-CoV-2 triggers an immune response that, if unregulated, can lead to severe lung inflammation and syndromes such as ARDS.

**KEYWORDS:** *COVID-19, SARS-CoV-2, ARDS, infection, respiratory disease, lung inflammation*



# THE LONG-TERM EFFECTS FOLLOWING COVID-19 INFECTION

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## ABSTRACT

COVID due to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection can produce long-term effects which persist for weeks or months after recovery from the acute phase of the disease. Long-term symptoms can be seen even in patients who have had a mild form of COVID, including young people. Persistent symptoms can include chronic fatigue, dyspnea, brain fog, muscle and joint pain, sleep disturbances, anxiety and depression, persistent changes in taste and smell, and palpitations. The disease can also affect organs such as the heart, brain, immune system, and gastrointestinal tract, possibly resulting in hospitalization. Vaccination reduces the risk of severe COVID and eventual persistent symptoms.

**KEYWORDS:** *SARS-CoV-2, COVID, symptom, long-term, respiratory, pandemic*



# BACTERIAL AND VIRAL INFECTIONS IN ATOPIC DERMATITIS, THE MOST COMMON ALLERGIC SKIN DISEASE

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## ABSTRACT

Atopic dermatitis (AD) is a chronic inflammatory skin disease occurring primarily in children that is one of the most common allergic diseases. AD is characterized by intense itching, dry skin, and eczematous lesions, and is often associated with other atopic conditions such as asthma and allergic rhinitis. One of the clinically relevant aspects of AD is its increased susceptibility to both bacterial and viral infections due to a compromised skin barrier and a disrupted immune system. Skin barrier dysfunction results in reduced production of filaggrin and epidermal lipids, favoring the penetration of microorganisms. Immune imbalances are characterized by an increased Th2 response with inflammation and impaired antimicrobial defenses. *Staphylococcus aureus* is the most prevalent bacteria responsible for infection in patients with AD, while *Streptococcus pyogenes* are less common for infections. Additionally, patients may experience viral infections due to the *Herpes simplex virus* (HSV), which causes the potentially serious infection eczema herpeticum. Management of AD requires attention to associated skin infections and early recognition and prompt treatment of bacterial and viral infections can prevent complications and improve quality of life.

**KEYWORDS:** *Atopic dermatitis, infection, bacteria, virus, skin, allergic disease*