



RESPIRATORY SYNCYTIAL VIRUS (RSV): PATHOPHYSIOLOGY, CURRENT TREATMENT ADVANCES & PREVENTION STRATEGIES

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Abstract

Respiratory syncytial virus (RSV) is a viral pathogen affecting the lungs mainly lower lobe causing infections. It is an enveloped RNA virus having a single stranded, negative sense genome. It belongs to the family Pneumoviridae and the genus Orthopneu Amovirus having two genomes A and B. RSV is one of the most important viral causes of bronchiolitis and pneumonia in infants and young children, and it also significantly affects elderly adults, patients that have gone under transplants and immuno-compromised patients. Worldwide RSV continues to be a major cause of hospital admissions. According to World Health Organization, an estimated value of 3.6 million hospitalizations and approximately 100 000 deaths each year among children younger than five years old are related to RSV. The RSV epidemic, unlike other respiratory viruses such as influenza happens each year, the RSV infections can always be seen in chilly weather such as late fall and winter. The Respiratory Syncytial Virus is highly contagious with an incubation period of 4 to 5 days and can spread through respiratory droplet or close contact with contaminated surface. Since RSV has heavy droplets, it cannot be aerosolized for long distances thus contact is the primary route of transmission.



Modern American Journal of Medical and Health Sciences

ISSN (E): 3067-803X

Volume 01, Issue 09, December, 2025

Website: usajournals.org

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PATHOGENESIS

The pathogenesis of the respiratory syncytial virus begins when it enters the host body through the nasopharynx route. It then attaches itself to the host cell which the respiratory epithelium through a special protein called (G) Glycoprotein and the (F) fusion protein helps the virus to fuse the viral envelope to the host cell. This allows the virus to enter the host cell and begin the process of viral replication. One of the prominent features of the RSV is that the fusion protein can also fuse and bind the infected cells leading to formation of a giant multinucleated cells or a syncytium thus getting its name the syncytial virus. This is also a distinct feature under the microscope. The Respiratory syncytial virus initially affects the upper nasopharyngeal epithelium before traveling through to the lower respiratory tract usually after 1-3 days via aspiration or the interstitial infection as mentioned prior. When the RSV spreads from the upper tract of the respiratory system to the lower tract towards the bronchioles it causes desquamation and necrosis of the ciliated epithelium. The damage of the airway lining causes edema, infiltration of the inflammatory cells and markers and excessive mucous production which leads to the narrowing and obstruction of the airway. This mechanism also explains the difficulty breathing and wheezing signs which are typical of bronchiolitis, it can also cause prolonged breathing or hypoxia. The innate immunity acts first by releasing interferons, interleukins, chemokines attracting macrophages, neutrophils, and T-cells to the infected area. However, this inflammatory response increases the edema and mucous production and in children with narrow airways or with developing immunity can cause severe respiratory distress. Adaptive immunity encompasses both humoral and cellular immunity, but their defenses are insufficient since mucosal IgA responses are short lived and have limited response, they allow re-infections throughout their lives. RSV also has the ability to evade immunity by interfering with the interferons and suppressing the antiviral pathways facilitating its continuous spread in the population.



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SYMPTOMS

The clinical manifestation of Respiratory Syncytial Virus differs based on the patient's age, immune status, and the infection's severity. RSV can cause any respiratory infection varying from a common cold to pneumonia.

Initial Symptoms

- . headache
- . low grade fever
- . fatigue
- . sore throat
- . runny nose (rhinorrhea)

low respiratory tract infection symptoms

As the RSV progresses to the lower respiratory tract it can cause severe symptoms

- . Tachypnea
- . Dyspnea
- . Wheezing
- . Tachycardia
- . Hypoxia which can manifest as cyanosis or low oxygen saturation
- . Loss of appetite (especially in infants)
- . Lethargy
- . Irritability

SEVERE SYMPTOMS

- . Apnea (difficulty breathing)
- . Respiratory failure

RSV is also associated with an increased risk of otitis media. Reinfection may present itself as exacerbated asthma or common cold. The recovery time for RSV ranges from one to two weeks but in some cases, it can cause prolonged wheezing and coughing. RSV may be fatal in premature infants, immunocompromised patients, and patients with underlying lung conditions.



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LABORATORY DIAGNOSTICS

RSV infection is verified by the identification of viral antigen, RNA, or through virus isolation. Nasopharyngeal aspirates or swabs are the specimens of choice. Rapid antigen detection tests (RADTs) are frequently employed in pediatric populations because of their prompt results. RT-PCR is the most sensitive and specific technique, extensively utilized in clinical environments. Viral culture is infrequently utilized due to protracted turnaround times. Chest X-ray may reveal hyperinflation or peri bronchial thickening in severe instances; however, the findings are non-specific.

TREATMENT

The respiratory syncytial virus has no specific targeted therapy, so the main course of action is supportive care this includes supplemental oxygen, antipyretics, and hydration . In infants with severe bronchiolitis to relief nasal congestion saline drops in a suction bulb maybe used along humidified oxygen and mechanical ventilation. Ribavirin, an antiviral, is rarely used and reserved for high-risk patients, such as those with congenital heart disease, prematurity, or immunodeficiency. Palivizumab, a monoclonal antibody, is used prophylactically rather than for treatment, given monthly during RSV season in high-risk infants to reduce hospitalization rates.

PREVENTION

Maternal immunization, monoclonal antibody prophylaxis, and infection-control measures are the main ways to prevent RSV infection. Transplacental transfer of protective antibodies is made possible by maternal immunization during the third trimester (after 28 weeks gestation), giving newborns passive immunity for the first six months of life. A long-acting monoclonal antibody given intramuscularly can also prevent severe lower respiratory tract infections in infants starting their first RSV season. High-risk children up to 24 months old who are starting their second RSV season, such as those with immunodeficiency, congenital heart disease, or chronic lung disease, may also be eligible for it. Other prevention techniques include good hygiene; be sure to wash hands, avoid exposure by



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Volume 01, Issue 09, December, 2025

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reducing your contact with individuals with a respiratory infection, wear protective gear such as gloves, masks and gowns.

CONCLUSION

Respiratory Syncytial Virus (RSV) is still a major cause of respiratory illness, especially in high-risk groups, infants, and young children. Reducing disease severity and hospitalization requires early detection, supportive care, and adherence to preventive measures. Long-acting monoclonal antibodies and improvements in maternal immunization provide promising ways to safeguard infants at risk. To reduce RSV's worldwide impact, research, surveillance, and public health initiatives must continue.

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***Modern American Journal of Medical and
Health Sciences***

ISSN (E): 3067-803X

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Website: usajournals.org

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