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# Virology, Spectrum and Structure of Respiratory Syncytial Virus

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Short Communication

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## ABOUT THE STUDY

Human Respiratory Syncytial Virus is a ubiquitous virus and is the leading cause of infant mortality from respiratory infections. By the age of two years nearly all children have been infected and can cause severe bronchiolitis and pneumonia in this age group. Nearly 100% of children in the USA are infected with the virus by 2-3 years of age, several hundred infants may die directly from the infection, while the deaths of an additional several thousand may be attributed to respiratory syncytial virus related complications. The world health organization estimates that respiratory syncytial virus is responsible for 64 million infections world wide and 1,60,000 deaths per annum [1].

Although mostly young infants are affected, it is increasingly recognized as a significant cause of disease in the elderly population and can often be fatal for patients with impaired immune systems. The incubation period of respiratory syncytial virus diseases is estimated to be there to 5 days. The virus can remain viable on hard surfaces for up to 6 hour on rubber gloves for 90 min and on skin for 20 min. this prolonged survival highlights the need for hand washing and contact precautions in limiting the spread of respiratory syncytial virus. Viral shedding is significantly prolonged in immune compromised individuals and can continue for several months.

The spectrum of this disease ranges from mild upper tract illness, infection in middle ear which progresses to acute otitis media, to apnoea in premature infants, pneumonia and bronchitis. At the beginning of the illness the virus replicates in the nasopharynx. Common symptoms of an upper respiratory tract infection include a productive cough and mild to moderate nasal congestion with clear rhinorrhoea. A low grade fever presents in the early stages of the infection and symptoms may persist for 1-3 weeks before complete recovery [2].

Symptoms of lower respiratory tract disease include tachypnea, wheezing that usually appear up to three days following onset of rhinorrhoea. These symptoms are indicative of the virus spreading into the bronchi and bronchioles. A chest radiograph exhibit hyperinflation with flattened diaphragms. If the respiratory syncytial virus further spreads to the alveoli, an interstitial pneumonia may develop with middle and upper lobes affected. In such patients, tachypnea becomes severe respiratory distress, with deep retractions and grunting respirations. The risk for cardiovascular failure secondary to hypoxemia, acidosis and dehydration increases significantly. Immuno compromised and young infants, who have very marrow bronchioles are at particularly high risk for complete bronchiolar obstruction. The risk for vomiting sensation also increases which is often related to respiratory distress and can increase the likelihood and of gastroesophogeal reflux. This may result in decreased oral intake with dehydration. Premature babies born at 30-35 weeks of gestation, HIV infected patients infants with cyanotic congenital heart disease and other immunosuppressive therapy such a bone marrow transplant are at increased risk for morbidity and mortality during Respiratory syncytial infection [3].

Its genome contains 10 mRNA s each coding for an individual protein. The structural proteins are divided into three functional groups, the nucleopsid protein, phosphoprotein and viral polymerase. These proteins together have been demonstrated to function as the RSV replicase. The outer envelope is lined internally with matrix protein and is spiked externally with fusion and attachment glycoprotein projections these are responsible for initiation and propagation of an Respiratory syncytial virus infection [4].

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