Respiratory Syncytial Virus (RSV)

By Lauren Childers

Etiological agent: Respiratory Syncytial Virus (RSV)

Transmission: RSV is an airborne disease and transmitted indirectly when droplets containing the virus are released into the air by an infected person and then breathed in by another individual or come into contact with another persons’ nose, eyes, or mouth (1). RSV can also be transmitted directly if individuals come into contact with the oral or nasal secretions of an infected individual (1).

Reservoirs: Humans are the only known reservoirs of RSV. However, the virus can survive on soft tissues, such as the hands, and fomites like countertops, toys, or other hard surfaces, for many hours (2).

General characteristics of RSV: RSV is a single-stranded, enveloped, non-segmented, membrane-bound, negative-sense RNA virus (2, 5, and 6). The nucleocapsid genome encodes for eight structural proteins and two non-structural proteins (6). RSV is medium sized virus (85-350nm in diameter) that is closely related to other viruses such as measles, mumps and parainfluenza strains 1, 2 and 3 (6). RSV is a member of the Paramyxoviridae family (5) and the subfamily Pneumovirinae (6). RSV has two subtypes: A and B. Both subtypes A and B are simultaneously present in most cases, and strain A is thought to be more virulent (5). Several distinct genotypes are present within each subtype and the dominant strains shift yearly, which may account for the high frequency of reinfection (5). The incubation period of RSV is typically 4-6 days and a full recovery is expected within 1-2 weeks (4). Even after recovery, very young infants may be able to spread the virus for 1-3 weeks (1).

Tests for identification: Rapid antigen detection tests (RADTs) are the most commonly used test for RSV detection and are available in ready-made commercialized kits that can be performed at the point of care without a trained lab professional (3). A sample of secretions from the nasopharyngeal cavity is obtained and a positive or negative result can be read in 10-30 minutes. There are three kinds of tests: immunochromatographic (ICR) tests, enzyme immunoassays (EIA), and optical immunoassays (OIA) (3). The principle of all three kits is the same; however, they differ in apparatus, sequence of binding reaction, and macroscopic signaling (3). The basics of all three tests are that if RSV is present, a macroscopic color change will occur – the tests do this by targeting the RSV fusion surface glycoprotein (3). The macroscopic color change that is observed is mediated by the binding of viral protein from the nasopharyngeal sample to the test’s RSV-specific antibodies (3). Testing for RSV is a common in young children, as virtually all children are infected by three years of age (2).

Signs and Symptoms: Signs and symptoms include persistent coughing and/or wheezing, fast or troubled breathing, fever (>100.4°F), coryza, rhinorrhea, conjunctivitis, and decreased energy (2, 4). RSV is “also likely to cause sinus and ear involvement” (4). Young infants may become irritable (1).

Historical information: RSV was discovered in 1956 while scientists were observing a respiratory virus in chimpanzees (chimpanzee coryza agent). Conversely, it was later discovered that the chimpanzees were...
contracting the virus from their human care takers (6). In 1963 Robert Chanock and associates were able to isolate, characterize, and name the virus (6). RSV is a descriptive term related to the pathological effects the virus has on the epithelium cell of the respiratory tract; the virus merges epithelial cell walls to create a syncytia (6). Work in the 1960s with vaccines “failed to impart protection” and even worse, it was seen that infants who received the vaccine had more severe RSV infections in the following years (6).

**Control, treatment and vaccination:** Presently there is no cure or vaccine for RSV, and rehydration therapy remains the best treatment (5). Bronchodilators, which are medicines that help reduce airway resistance, may be prescribe to make breathing easier; while antibiotics may be prescribed to help with secondary infections contracted while the immune system is compromised due to RSV (6). Researchers are currently working to develop a vaccine for RSV, but none have been approved by the FDA for human trials (1). There is a drug called Palivizumab that is available to prevent severe RSV in high risk infants and children, but it is not a cure and cannot help children already suffering from the virus (1). It should also be noted that Palivizumab may interfere with rapid immunoassay testing, which could lead to a falsely negative result (1).

**Prevention:** Good hand hygiene and wiping off of hard surfaces with soap and water or disinfectants remains the best way of preventing the spread of RSV (1). Infected individuals should not share cups or eating utensils with others, and should cover their nose and mouth when coughing or sneezing (1). In addition, parents should refrain from kissing their children’s’ face and hands if the child is infected (1).

**Risk factors:** Risk factors include infants <6 months of age, infants, children and elderly with lung or congenital heart diseases, children who are born premature, immunocompromised patients, any age group with severe asthma, institutionalized elderly, and residence living at an altitude above 2500m (2). Scientists have also discovered a genetic predisposition to severe RSV disease associated with polymorphisms in cytokine- and chemokine-related genes (2).

**Virulence factors:** As noted above, the single-stranded genome of RSV codes for eight structural proteins and two non-structural proteins. Three of the eight structural proteins code for transmembrane surface glycoproteins (6). The two transmembrane surface glycoproteins are that are key in virulence for RSV are the fusion proteins (F) and attachment proteins (G); these two proteins are responsible for “the initiation and propagation of RSV infection” (6). The G protein attaches the virus to epithelial cell walls where the F protein is then cleaved by proteolytic enzymes of the infected cell (6). The virus then fuses with the epithelial cell membrane and enters the cytoplasm where is can then take control of the host cell and replicate (6). The virus lyses the host cell and releases complete viral particles to infect other cells. The F protein, once in the cytoplasm, can also cause fusion of adjacent host cell membranes by degrading individual membranes, which creates a large multinucleated epithelial cell, or syncytia (6). Viral RNA can then spread without the formation of complete viral particles (6).

**Global and local outbreaks:** RSV causes seasonal outbreaks throughout the world. In the northern hemisphere the season runs from November – April, with peaks in January or February (2). The southern hemisphere experiences outbreaks in May – September with peaks in May, June or July (2). Tropical and semitropical regions see outbreaks associated with the rainy season, and temperate regions do not
typically see sharp a endemic peak and RSV may be seen eight months out of the year (2). RSV is the most common cause of lower respiratory tract infections (LRTI) in children four years of age or younger. It is estimated that globally, RSV causes 34 million episodes of LRTI infections in children five years or younger, and results in approximately 3.4 million hospitalizations per year (2). It is estimated that when exposed for the first time, 25 – 40% of children will show signs or symptoms of bronchiolitis or pneumonia and 0.5 – 2% of these children will require hospitalization (1).

In the United States, it is likely that 132,000 – 172,000 pediatric hospitalizations among children five or younger are associated with RSV, and doctors are seeing that number increasing in frequency (2). The rate of hospitalization is greater in children younger than 3 years of age, approximately 40% (2). In adults, particularly the elderly and immunosuppressed, it is “estimated that RSV may be responsible for as much as 25% of excess wintertime mortality,” although RSV is regularly unrecognized as a cause of LRTI infections in adults, and often misdiagnosed as influenza (2).

References: