**LESSON 14.  
Introduction to basic virology. Microbiology diagnosis of acute respiratory viral infections (families of *Orthomyxoviridae* and *Paramyxoviridae*)**

**LESSON PLAN:**

• Introduction to specialized virology.

• Collection of examination materials during various viral diseases.

• Microbiological diagnostic methods of viral infections: express, virological and serological.

o Detection of the virus or its components from the examination material taken from the patient (express diagnostics - IFR, IFA, RIM, ZPR, etc.).

o Virological method - cultivation of pathological material in various biological objects (bodies of laboratory animals, chicken embryos and tissue cultures) and subsequent indication and identification

• Virus indication methods (hemagglutination reaction (HAR), hemadsorption phenomenon, cytopathic effect (SPT), intracellular inclusions, "negative colonies", "color test", interference phenomenon, KBR)

• Virus identification methods (BNR, KBR, HALR, hemadsorption retardation reaction, immunodiffusion reactions, PHAR, RIM, IFR, IFA, immunoelectron microscopy)

• Serological method - serodiagnosis of viral infections, taking double sera, establishing serological reactions (KBR, BNR, HALR, IFR, RIM, IFA).

• Viruses that cause acute respiratory infections

• Family Orthomyxoviridae. General characteristics, classification.

• Influenza viruses. Virion structure, cultivation. Influenza virus antigens, antigenic variation, ecology. Pathogenesis of influenza, immunity, microbiological diagnostics. Specific prevention problems.

• Family Paramyxoviridae. General characteristics, classification. Virion structure, cultivation, principles of microbiological diagnostics.

• Parainfluenza viruses, their role in human pathology.

• Epidemic mumps virus, cultivation. Pathogenicity characteristics. Immunity. Specific prevention.

• Respiratory syncytial virus, cultivation. Pathogenicity characteristics. Immunity.

o Genus Morbillivirus. Measles virus. Pathogenicity characteristics. Immunity. Subacute sclerosing panencephalitis. Specific prevention.

**Determinants of Viral Disease**

**Nature of the Disease**

Target tissue

Portal of entry of virus

Access of virus to target tissue

Tissue tropism of virus

Permissiveness of cells for viral replication

Pathogenic activity (strain specific)

**Severity of Disease**

Cytopathic ability of virus

Virus inoculum size

Immune status (naive or immunized)

Competence of the immune system

Immunopathology

Length of time before resolution of infection

General health of the person

Nutrition

Other diseases influencing immune status

Genetic makeup of the person

Age

1 (A) Stages of viral infection. The virus is released from one person, is acquired by another, replicates, and initiates a primary infection at the site of acquisition. Depending on the virus, it may then spread to other body sites and finally to a target tissue characteristic of the disease. (B) The cycle starts with acquisition, as indicated, and proceeds until the release of new virus. The thickness of the arrow denotes the degree to which the original virus inoculum is amplified on replication. The boxes indicate a site or cause of symptoms. (C) Time course of viral infection. The time course of symptoms and the immune response correlate with the stage of viral infection and depend on whether the virus causes symptoms at the primary site or only after dissemination to another (secondary) site. *CMV,* Cytomegalovirus; *HBV,* hepatitis B virus; *HIV,* human immunodeficiency virus.

**Mechanisms of Viral Transmission**

Aerosols

Food, water

Fomites (e.g., tissues, clothes)

Direct contact with secretions (e.g., saliva, semen)

Sexual contact, birth

Blood transfusion or organ transplant

Zoonoses (animals, insects [arboviruses])

Genetic (vertical) (e.g., retroviruses)

**Disease and Viral Factors That Promote Transmission**

Stability of virion in response to environment (e.g., drying, detergents, temperature)

Replication and secretion of virus into transmissible aerosols and secretions (e.g., saliva, semen)

Asymptomatic transmission

Transience or ineffectiveness of immune response to control reinfection or recurrence

**Risk Factors**

Age

Health

Immune status

Occupation: contact with agent or vector

Travel history

Lifestyle

Children in day-care centers

Sexual activity

**Critical Community Size**

Seronegative, susceptible people

**Geography and Season**

Presence of cofactors or vectors in the environment

Habitat and season for arthropod vectors (mosquitoes)

School session: close proximity and crowding

Home-heating season

**Modes of Control**

Quarantine

Elimination of the vector

Immunization/vaccination

Treatment

Education

**Gastrointestinal Viruses**

**Infants**

Rotavirus Aa

Adenovirus 40, 41

Coxsackievirus A24

**Infants, Children, and Adults**

Norwalk virusa

Calicivirus

Astrovirus

Rotavirus A and

**Viral Hemorrhagic Fevers**

Yellow fever virus

Dengue viruses

Hantavirus

Ebola virus

Marburg virus

Lassa fever virus

**Infections of Organs and Tissues**

**Liver**

Hepatitis A,“a”2 B,“a” C,“a” G, D, and E viruses

Yellow fever virus

Epstein-Barr virus

Hepatitis in the neonate or immunocompromised person:

Cytomegalovirus

Herpes simplex virus

Varicella-zoster virus

Rubella virus (congenital rubella syndrome)

**Heart**

Coxsackievirus B

**Kidney**

Cytomegalovirus

BK papillomavirus

**Muscle**

Coxackievirus B (pleurodynia)

**Glands**

Cytomegalovirus

Mumps virus

Coxsackievirus B

**Eye**

Herpes simplex virusa

Adenovirusa

Measles virus

Rubella virus

Enterovirus 70

Coxsackievirus A24

**Central Nervous System Infections**

**Meningitis**

Enteroviruses

Echoviruses

Coxsackievirusa

Poliovirus

Herpes simplex virus 2a

Adenovirus

Mumps virus

Lymphocytic choriomeningitis virus

Arboencephalitis viruses

**Paralysis**

Poliovirus

Enteroviruses D68, 70 and 71

Coxsackievirus A and A16

West Nile virus

**Encephalitis**

Herpes simplex virus 1a

Varicella-zoster virus

Arboencephalitis viruses a

Rabies virus

Coxsackieviruses A and B

Polioviruses

**Postinfectious Encephalitis (Immune Mediated)**

Measles virus

Mumps virus

Rubella virus

Varicella-zoster virus

Influenza viruses

**Other**

JC virus (progressive multifocal leukoencephalopathy [in immunosuppressed

people])

Measles variant (subacute sclerosing panencephalitis)

Prions (spongiform encephalopathy)

Human immunodeficiency virus (AIDS dementia)

Human T-cell lymphotropic virus 1 (tropical spastic paraparesis)

**Viruses Transmitted in Blood**

Hepatitis B, C, G, D

Human immunodeficiency virus

Human T-cell lymphotropic virus 1

Cytomegalovirus

Epstein-Barr virus

West Nile encephalitis virus

**Sexually Transmitted Viruses**

Human papillomavirus 6, 11, 42

Human papillomavirus 16, 18, 31, 45, and others (high risk for human

cervical carcinoma)

Herpes simplex virus (HSV-1 and HSV-2)

Cytomegalovirus

Hepatitis B, C, and D viruses

Human immunodeficiency virus

Human T-cell lymphotropic virus 1

Zika virus

**Screening of the Blood Supply**

HIV-1 and HIV-2

Hepatitis B virus

Hepatitis C virus

Human T-cell lymphotropic virus 1 and 2

West Nile encephalitis virus

*Treponema pallidum* (syphilis)a

a-Other than bacterial growth, *Treponema pallidum* is the only nonviral microbe assayed.

**ORTHOMYXOVIRUSES**

**Trigger Words**

Aerosols, envelope, segmented genome/reassortment, hemagglutinin, neuraminidase, antigenic drift (outbreaks), antigenic shift (pandemics), zoonosis

**Biology, Virulence, and Disease**

ᑏ Large size, enveloped, (−) segmented RNA genome

ᑏᑏ Encodes RNA-dependent RNA polymerase, replicates in nucleus (exception to the rule)

ᑏᑏ Each segment encodes one or two proteins

ᑏᑏ Mixed infection results in genetic mixing of segments: reassortment

ᑏᑏ Binds to sialic acid (HA glycoprotein) and encodes neuraminidase activity (NA glycoprotein)

ᑏᑏ Preexisting antibody can block disease

ᑏᑏ Cell-mediated immune response important for control but causes pathogenesis

ᑏᑏ Influenza A, not influenza B, is a zoonosis

ᑏᑏ Acute flulike symptoms caused by large cytokine release

ᑏᑏ Extensive destruction of ciliated epithelium

ᑏᑏ Pneumonia by influenza or secondary bacterial infection

**Epidemiology**

ᑏᑏ Transmitted by aerosols

ᑏᑏ Annual epidemics caused by mutations, pandemics caused by reassortment of genome segments between human and animal viruses

**Diagnosis**

ᑏᑏ Symptomatology, RT-PCR genome analysis of respiratory secretions, immunology tests (ELISA), hemagglutination and hemagglutination inhibition

**Treatment, Prevention, and Control**

ᑏᑏ Annual vaccine contains two influenza A and one or two influenza B strains: inactivated vaccines contain HA and NA, live attenuated nasal vaccine (for 2 to 49 year olds)

ᑏᑏ Neuraminidase, the M2 channel and the cap-dependent endonuclease are targets for antiviral drugs

**Unique Features of the Influenza A and B Viruses**

The enveloped virion has a genome of eight unique negativesense RNA nucleocapsid segments.

Hemagglutinin glycoprotein is the viral attachment protein and fusion protein; it elicits neutralizing, protective antibody responses.

Influenza transcribes and replicates its genome in the target cell nucleus but assembles and buds from the plasma membrane.

The polymerase uses capped cellular mRNA as primers for mRNA synthesis, and this is a target for baloxavir marboxil.

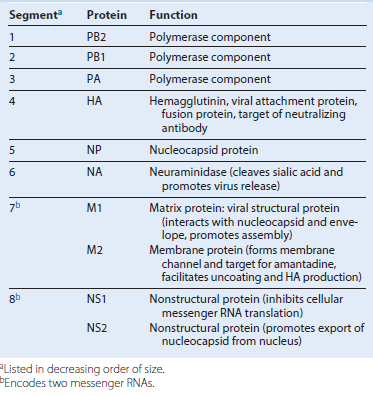
The antiviral drugs amantadine and rimantadine target the M2 (membrane) protein for *influenza A only* to inhibit the uncoating step.

The antiviral drugs zanamivir, oseltamivir, and peramivir inhibit the neuraminidase protein of influenza A and B.

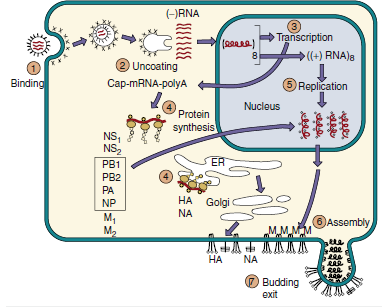
The segmented genome promotes genetic diversity caused by mutation and reassortment of segments on infection with two different strains.

Influenza A infects humans, other mammals, and birds (zoonosis).

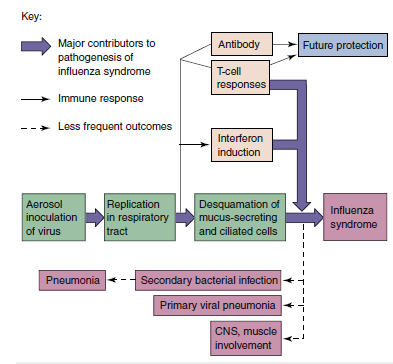
**Products of Influenza Gene Segments**

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Replication of influenza A virus. After binding *(1)* to sialic acid–containing receptors, influenza is endocytosed and fuses *(2)* with the vesicle membrane. Unlike for most other ribonucleic acid *(RNA)* viruses, transcription *(3)* and replication *(5)* of the genome occur in the nucleus. Viral proteins are synthesized *(4)*, helical ribonucleoprotein complex nucleocapsid segments form and associate *(6)* with the M1 protein–lined membranes containing M2 and the hemagglutinin *(HA)* and neuraminidase *(NA)* glycoproteins. The virus buds *(7)* from the plasma membrane and eventually kills the cell. *(−),* Negative sense; *(+),* positive sense; *ER,* endoplasmic reticulum; *NP,* nucleocapsid protein; *NS1, NS2,* nonstructural proteins 1 and 2; *PA, PB1, PB2,* polymerase components; *polyA,* polyadenylate.

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Pathogenesis of influenza A virus. The symptoms of influenza are caused by viral pathologic and immunopathologic effects, but the infection may promote secondary bacterial infection. *CNS,* Central nervous system.

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**Disease Mechanisms of Influenza A and B Viruses**

Virus infects the upper and lower respiratory tract.

Systemic symptoms are caused by the interferon and cytokine response to the virus. Local symptoms result from epithelial cell damage, including ciliated and mucus-secreting cells.

Interferon and cell-mediated immune responses (natural killer and T cells) are important for immune resolution and immunopathogenesis.

Infected people are predisposed to bacterial superinfection because of the loss of natural barriers and exposure of binding sites on epithelial cells.

Antibody is important for future protection against infection and is specific for defined epitopes on HA and NA proteins.

The HA and NA of influenza A virus can undergo **major (reassortment: shift)** and **minor (mutation: drift)** antigenic changes to ensure the presence of immunologically naive susceptible people.

Influenza B virus undergoes only minor antigenic changes.

**Influenza Pandemics Resulting from Antigenic Shift**

**Year of Pandemic Influenza A Subtype**

1918 - H1N1

1947 - H1N1

1957 - H2N2; Asian flu strain

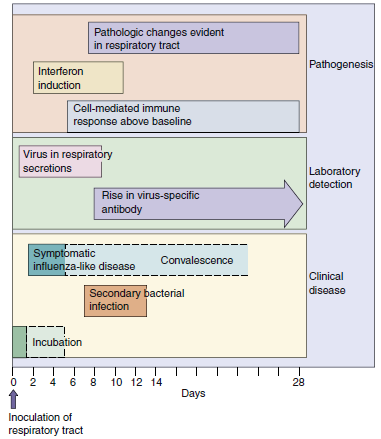
1968 - H3N2; Hong Kong flu strain

1977 - H1N1; Russian

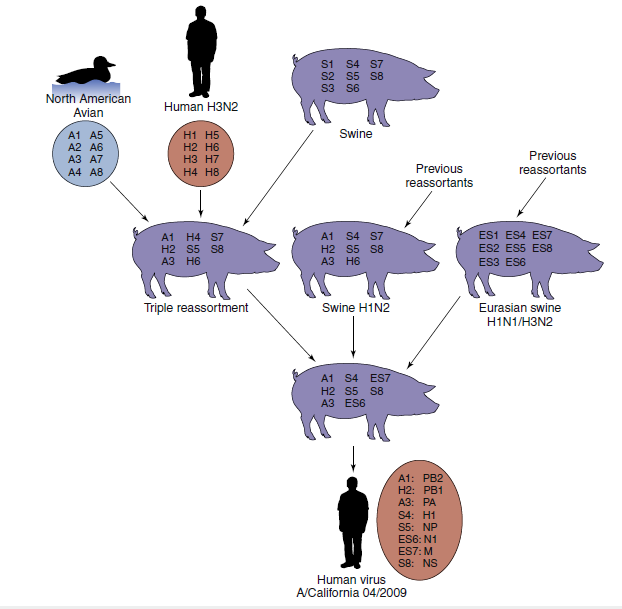
1997, 2003 - H5N1: China, avian

2009 - H1N1, swine flu

Time course of influenza A virus infection. The classic “flu syndrome” occurs early. Later, pneumonia may result from bacterial pathogenesis, viral pathogenesis, or immunopathogenesis.



Generation of A/California/04/2009 (H1N1) pandemic swine flu by multiple reassortments of genomic segments of influenza A virus. The pandemic H1N1 virus arose from the mixing of a triple reassortment of avian, human, and swine viruses with two other swine viruses, each of which was also generated by reassortment between swine, human, and other influenza viruses. This new virus emerged in the spring of 2009 (out of season) in Mexico but was first identified in California.



**Epidemiology of Influenza A and B Viruses**

**Disease/Viral Factors**

Virus has a large, enveloped virion that is easily inactivated by dryness, acid, and detergents.

Segmented genome facilitates major genetic changes, especially on hemagglutinin and neuraminidase proteins.

Influenza A infects many vertebrate species, including other mammals and birds.

Coinfection with animal and human strains of influenza A can generate very different virus strains by genetic reassortment.

Transmission of virus often precedes symptoms.ᑏ

**Transmission**

Virus is spread by inhalation of small aerosol droplets expelled during talking, breathing, and coughing.

Virus likes a cool, less humid atmosphere (e.g., winter heating season).

Virus is extensively spread by schoolchildren.ᑏ

**Who Is at Risk?**

Seronegative people

Adults: classic flu syndrome.

Children: asymptomatic to severe respiratory tract infections.

High-risk groups: elderly and immunocompromised people, people in nursing homes or with underlying cardiac or respiratory problems (including asthma sufferers and smokers).ᑏ

**Geography/Season**

Worldwide occurrence. Epidemics are local; pandemics are worldwide.

Disease is more common in winter.ᑏ

**Modes of Control**

Antiviral drugs have been approved for prophylaxis or early treatment. Killed and live vaccines contain predicted yearly strains of influenza A and B viruses.

**Clinical Summary**

**Influenza A:** A 70-year-old woman has rapid onset of fever with headache, myalgia, sore throat, and nonproductive cough. The disease progresses to pneumonia with bacterial involvement. There is no history of recent immunization with influenza A vaccine. Her husband is treated with amantadine or a neuraminidase inhibitor.

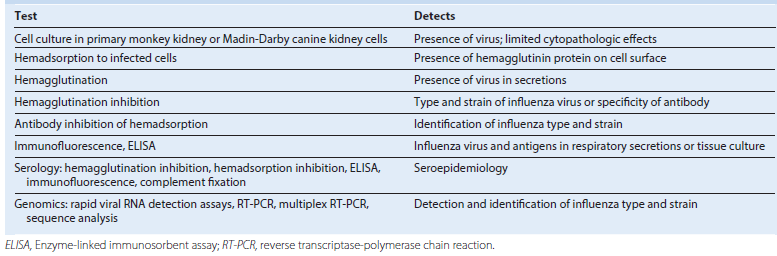
**Diseases Associated With Influenza Virus Infection**

Acute influenza infection in adults - Rapid onset of fever, malaise, myalgia, sore throat, and nonproductive cough

Acute influenza infection in children - Acute disease similar to that in adults but with higher fever, gastrointestinal tract symptoms (abdominal pain, vomiting), otitis media, myositis, and more frequent croup

Complications of influenza virus infection - Primary viral pneumonia, Secondary bacterial pneumonia, Myositis and cardiac involvement, Neurologic syndromes: Guillain-Barré syndrome, Encephalopathy, Encephalitis, Reye syndrome

**Laboratory Diagnosis of Influenza Virus Infection**

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**PARAMYXOVIRUSES**

**Trigger Words**

ᑏ Fusion, syncytia, aerosols, envelope

ᑏᑏ Measles: cough, conjunctivitis, coryza, photophobia, Koplik spots, rash, fever, SSPE, postmeasles encephalitis

ᑏᑏ Mumps: parotitis, orchitis, aseptic meningitis

ᑏᑏ Parainfluenza: croup, barking seal, pneumonia

ᑏᑏ RSV: infant, pneumonia

**Biology, Virulence, and Disease**

ᑏᑏ Large size, enveloped, (−) RNA genome, fusion protein

ᑏᑏ Encodes RNA-dependent RNA polymerase, replicates in cytoplasm

ᑏᑏ Parainfluenza and mumps bind to sialic acid and encode neuraminidase activity (HN glycoprotein); measles and RSV glycoprotein bind to proteins

ᑏᑏ Fusion protein promotes entry and cell-cell fusion (syncytia)

ᑏᑏ Cell-mediated immune response essential for control but causes pathogenesis

ᑏᑏ Measles: maculopapular rash, high fever with cough, conjunctivitis, coryza, Koplik spots (small gray lesions in mouth); more severe if vitamin A deficient, giant cell pneumonia if T-cell deficient, postmeasles encephalitis, SSPE 5–7 years later caused by measles variant

ᑏᑏ Mumps: parotitis, orchitis, aseptic meningitis

ᑏᑏ Parainfluenza: common cold, croup, bronchitis

ᑏᑏ RSV: common cold, pneumonia, bronchiolitis, life-threatening for premature

infants

**Epidemiology**

ᑏᑏ Transmitted by aerosols

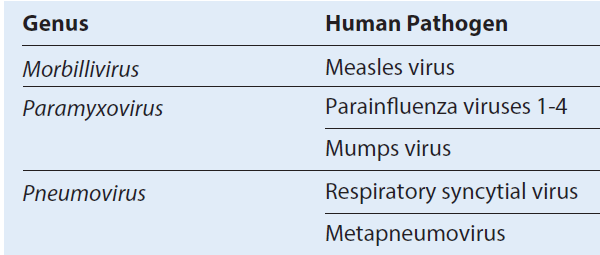
**Diagnosis**

ᑏᑏ Symptomatology, RT-PCR genome analysis of respiratory secretions

**Treatment, Prevention, and Control**

ᑏᑏ Live attenuated vaccine for measles and mumps; RSV: passive immunization for premature infants at high risk; aerosolized ribavirin *RSV,* Respiratory syncytial virus; *RT-PCR,* reverse transcriptase-polymerase chain reaction; *SSPE,* subacute sclerosing panencephalitis.

**Paramyxoviridae**

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**Unique Features of the Paramyxoviridae**

Large virion consists of a negative-sense RNA genome in a helical nucleocapsid surrounded by an envelope.

The three genera can be distinguished by the activities of the viral attachment protein: **HN** of parainfluenza virus and mumps virus binds to sialic acid and red blood cells (hemagglutinin and neuraminidase activity), neuraminidase facilitates release from cell;

**H** of measles virus binds protein receptors and is also a hemagglutinin;

**G** of RSV binds to cells but is not a hemagglutinin.

Virus replicates in the cytoplasm.

Virions penetrate the cell by fusion with the plasma membrane and exit by budding from the plasma membrane without killing the cell.

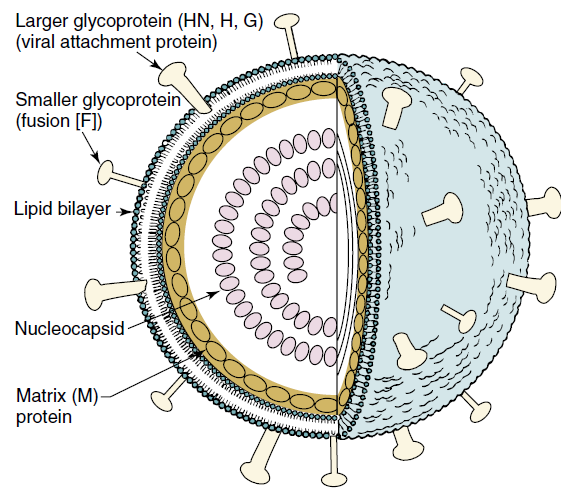
Viruses induce cell-to-cell fusion, causing multinucleated giant cells **(syncytia).**

**Cell-mediated immunity** causes many of the symptoms but is essential for control of the infection.

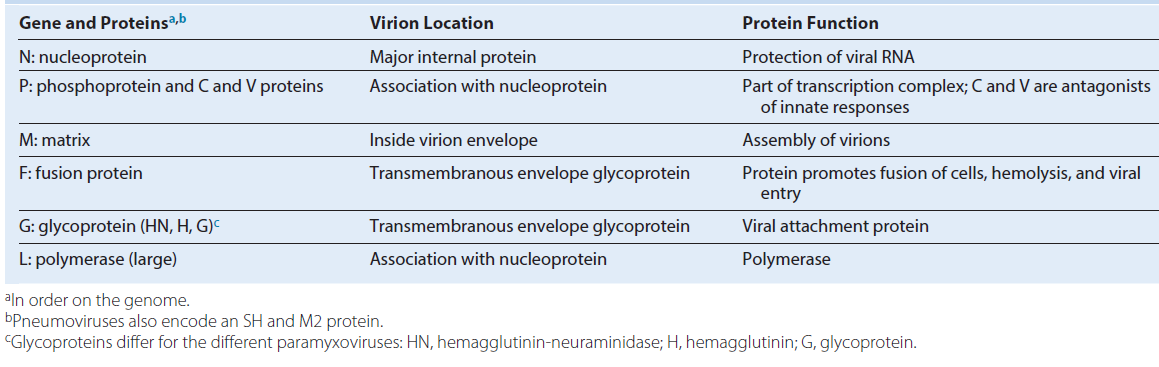
Paramyxoviridae are transmitted in **respiratory droplets** and initiate infection in the respiratory tract.

Measles and mumps establish viremia and spread to other body sites.

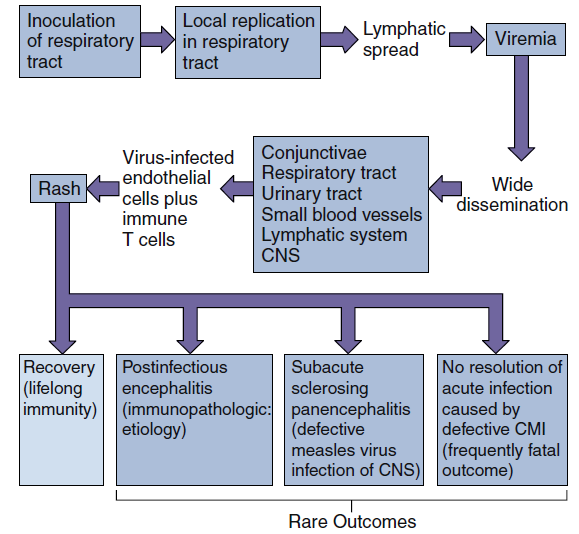
Model of paramyxovirus. The helical nucleocapsid—consisting of negative-sense, single-stranded RNA and the polymerase *(P),* nucleoprotein *(N),* and large protein *(L)*—associates with the matrix *(M)* protein at the envelope membrane surface. The nucleocapsid contains RNA transcriptase activity. The envelope contains the viral attachment glycoprotein (hemagglutinin-neuraminidase *[HN],* hemagglutinin *[H],* or G-protein *[G],* depending on the virus) and the fusion *(F)* protein.

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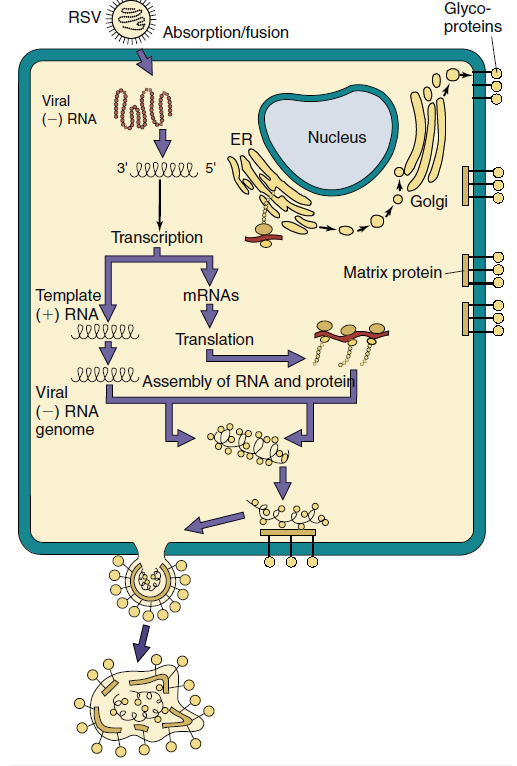
**Major Viral-Encoded Proteins of Paramyxoviruses**

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Mechanisms of spread of the measles virus within the body and the pathogenesis of measles. *CMI,* Cell-mediated immunity; *CNS,* central nervous system.

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Replication of paramyxoviruses. The virus binds to glycolipids or proteins and fuses with the cell surface. Individual messenger RNAs *(mRNAs)* for each protein and a full-length template are transcribed from the genome. Replication occurs in the cytoplasm. Proteins associate with the new genome, and the nucleocapsid associates with matrix and glycoprotein-modified plasma membranes. The virus leaves the cell by budding. *(−),* Negative sense; *(+),* positive sense; *ER,* endoplasmic reticulum; *RSV,* respiratory syncytial virus. (Modified from Balows, A. et al., 1988. Laboratory Diagnosis of Infectious Diseases: Principles and Practice. Springer-Verlag, New York, NY.)

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**Disease Mechanisms of Measles Virus**

Virus infects epithelial cells of respiratory tract.

Virus spreads systemically in lymphocytes by **viremia.**

Virus replicates in cells of conjunctivae, respiratory tract, urinary tract, lymphatic system, blood vessels, and CNS.

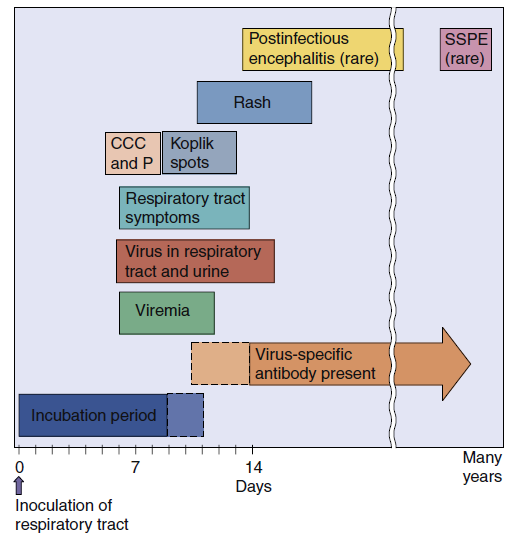
Rash is caused by T-cell response to virus-infected epithelial cells.

Virus causes immunosuppression.

**Cell-mediated immunity** is essential to control infection.

Sequelae in the CNS may result from immunopathogenesis (postinfectious measles encephalitis) or development of defective mutants (subacute sclerosing panencephalitis).

Time course of measles virus infection. Characteristic prodrome symptoms are cough, conjunctivitis, coryza, and photophobia *(CCC and P),* followed by the appearance of Koplik spots and rash. *SSPE,* Subacute sclerosing panencephalitis.

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**Epidemiology of Measles**

**Disease/Viral Factors**

Virus has large enveloped virion that is easily inactivated by dryness and acid.

Contagion period precedes symptoms.

Very contagious with 95% infectivity rate.

Host range is limited to humans.

Only one serotype exists.

Immunity is lifelong.

**Transmission**

Inhalation of large-droplet aerosols.

**Who Is at Risk?**

Unvaccinated people, especially infants <1 year old.

Malnourished people, especially vitamin A deficient, who have more serious outcomes.

Immunocompromised people, who have more serious outcomes.

**Geography/Season**

Virus found worldwide.

Virus endemic from autumn to spring, possibly because of crowding indoors.

**Modes of Control**

Live attenuated vaccine (Schwartz or Moraten variants of Edmonston B strain) can be administered.

Immune serum globulin can be administered after exposure.

**Clinical Consequences of Measles Virus Infection**

Measles - Characteristic maculopapular rash, cough, conjunctivitis, coryza, photophobia, Koplik spots *Complications*: otitis media, croup, pneumonia, blindness, encephalitis

Atypical measles - More intense rash (most prominent in distal areas); possible vesicles, petechiae, purpura, or urticaria

Post measles encephalitis - Acute onset of headache, confusion, vomiting, possible coma after rash dissipates

Subacute sclerosing panencephalitis - Central nervous system manifestations (e.g., personality, behavior, and memory changes; myoclonic jerks; spasticity; blindness)

**Measles-Mumps-Rubella Vaccine**

Composition: live attenuated viruses

Measles: Schwartz or Moraten substrains of Edmonston B strain

Mumps: Jeryl Lynn strain

Rubella: RA/27-3 strain

Vaccination schedule: after 12 months of age and at age 4 to 6 years or before junior high school (12 years of age)

Efficiency: 95% lifelong immunization with a single dose

**Disease Mechanisms of Parainfluenza Viruses**

There are four serotypes of parainfluenza viruses.

Infection is **limited to the respiratory tract;** upper respiratory tract disease is most common, but significant disease can occur with lower respiratory tract infection.

Parainfluenza viruses do *not* cause viremia or become systemic.

Diseases include **coldlike** symptoms, **bronchitis** (inflammation of bronchial tubes), and **croup** (laryngotracheobronchitis).

Infection induces protective immunity of short duration.

**Epidemiology of Parainfluenza Virus Infections**

**Disease/Viral Factors**

Virus has a large enveloped virion that is easily inactivated by dryness and acid.

Contagion period precedes symptoms and may occur in absence of symptoms.

Host range is limited to humans.

Reinfection can occur later in life.

**Transmission**

Inhalation of large-droplet aerosols.

**Who Is at Risk?**

Children: at risk for mild disease or croup.

Adults: at risk for reinfection with milder symptoms.

**Geography/Season**

Virus is ubiquitous and worldwide.

Incidence is seasonal.

**Modes of Control**

There are no modes of control.

**Disease Mechanisms of Mumps Virus**

Virus infects epithelial cells of respiratory tract.

Virus spreads systemically by viremia.

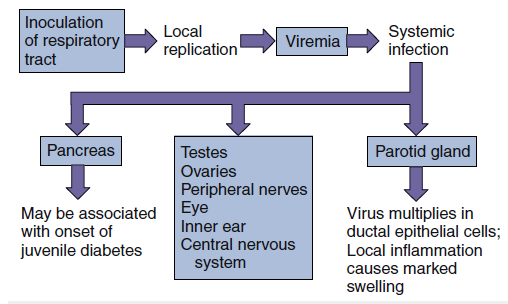
Infection of parotid gland, testes, and central nervous system.

Principal symptom is swelling of parotid and other glands caused by inflammation.

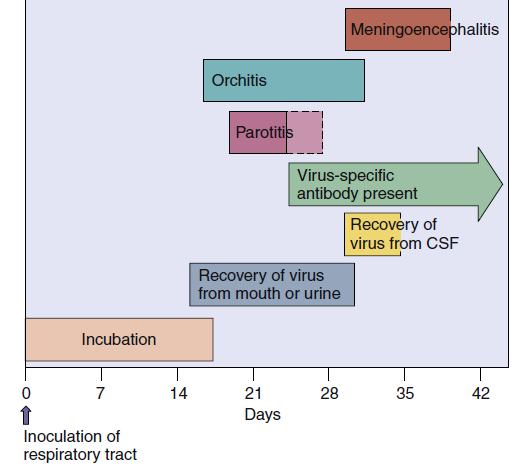
Cell-mediated immunity is essential for control of infection and responsible for causing some of the symptoms.

Antibody is not sufficient because of the virus’ ability to spread cell to cell.

**Mechanism of spread of mumps virus within the body.**

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**Time course of mumps virus infection. *CSF,* Cerebrospinal fluid.**

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**Epidemiology of Mumps Virus**

**Disease/Viral Factors**

Virus has large enveloped virion that is easily inactivated by dryness and acid.

Contagion period precedes symptoms.

Virus may cause asymptomatic shedding.

Host range is limited to humans.

Only one serotype exists.

Immunity is lifelong.

**Transmission**

Inhalation of large-droplet aerosols.

**Who Is at Risk?**

Unvaccinated people, especially infants <1 year old.

Immunocompromised people, who have more serious outcomes.

**Geography/Season**

Virus is found worldwide.

Virus is endemic in late winter and early spring.

**Modes of Control**

Live attenuated vaccine (Jeryl Lynn strain) is part of measles mumps-rubella vaccine.

**Disease Mechanisms of Respiratory Syncytial Virus**

Virus causes localized infection of respiratory tract.

Virus does not cause viremia or systemic spread.

Pneumonia results from cytopathologic spread of virus (including syncytia).

Bronchiolitis is most likely mediated by the host’s immune response.

Narrow airways of young infants are readily obstructed by virus induced pathologic effects.

Maternal antibody is insufficient to protect infant from infection.

Natural infection does not prevent reinfection.

**Epidemiology of Respiratory Syncytial Virus**

**Disease/Viral Factors**

Virus has a large enveloped virion that is easily inactivated by dryness and acid.

Contagion period precedes symptoms and may occur in the absence of symptoms.

Host range is limited to humans.

**Transmission**

Inhalation of large-droplet aerosols.

**Who Is at Risk?**

Infants: lower respiratory tract infection (bronchiolitis and pneumonia).

Premature neonates: serious disease.

Children: spectrum of disease from mild to pneumonia.

Adults: reinfection with milder symptoms.

Immunocompromised, chronic heart and lung problems: serious disease.

**Geography/Season**

Virus is ubiquitous and found worldwide.

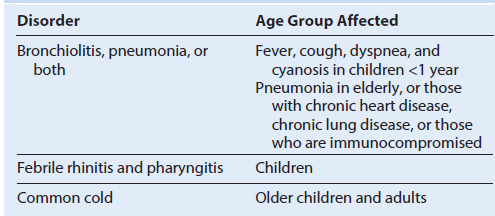
Incidence is seasonal.

**Modes of Control**

Immunoglobulin is available for infants at high risk.

Aerosol ribavirin is available for infants with serious disease.

**Clinical Consequences of Respiratory Syncytial Virus Infection**

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**Clinical Summaries**

**Measles:** An 18-year-old woman had been home for 10 days after a trip to Haiti when she developed a fever, cough, runny nose, and mild redness of her eyes. She now has a red, slightly raised rash over her face, trunk, and extremities. There are several 1-mm white lesions inside her mouth. She was never immunized for measles because of misinformation that an “egg allergy” would be a problem. The vaccine is not produced in eggs.

**Mumps:** A 30-year-old man returning from a trip to Russia experienced a 1- to 2-day period of headache and decreased appetite, followed by swelling over both sides of his jaw. The swelling extended from the bottom of the jaw to in front of the ear. Five days after the jaw swelling appeared, the patient began complaining of nausea and lower abdominal and testicular pain. He never received a booster immunization with the MMR vaccine.

**Croup:** An irritable 2-year-old toddler with little appetite has a sore throat, fever, and hoarse voice and coughs with the sound of a barking seal. A high-pitched noise (stridor) is heard on inhalation. Flaring of the nostrils indicates difficulty breathing.