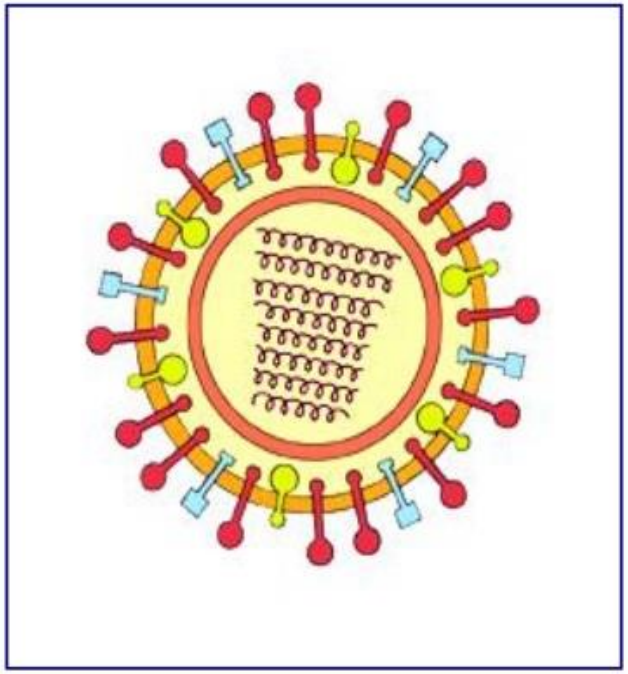
LECTURE XIII

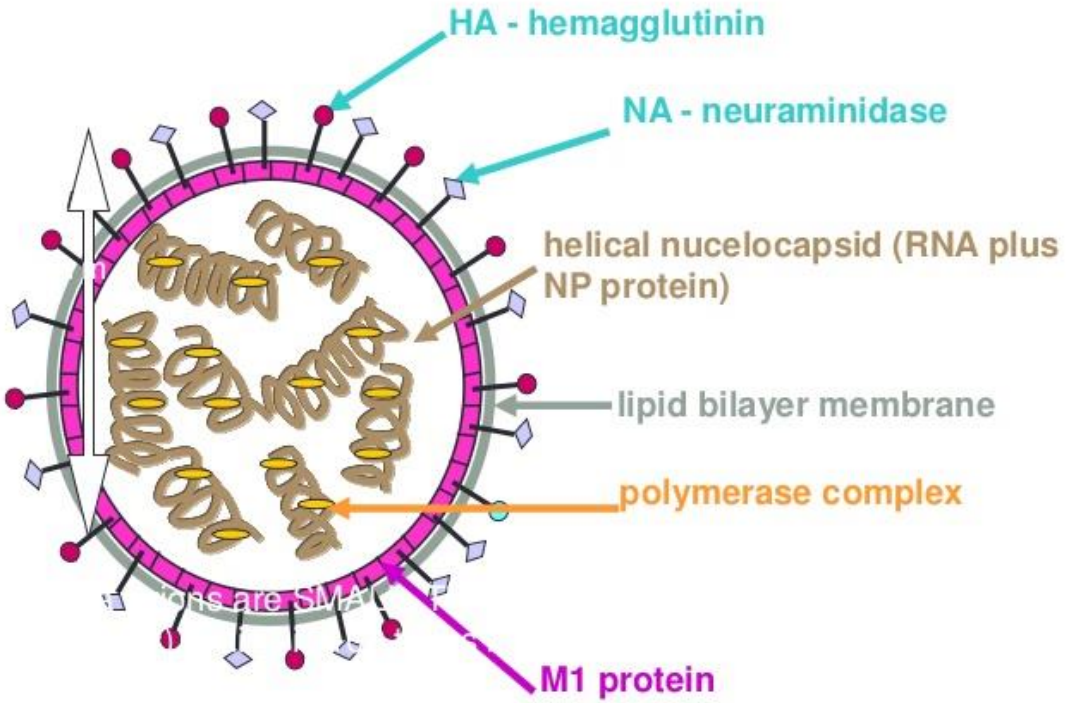
Introduction to special virology. Causative agents of respiratory viral infections (*Orthomyxoviridae*, *Paramyxoviridae*,*Adenoviridae*, *Coronaviridae* family and genus *Rhinovirus*).*Poxviridae* family, the role in human pathology.

Influenza Virus

- Virus are spherical in shape
- Size is 80 -120 nm
- Pleomorphism is common with variant forms

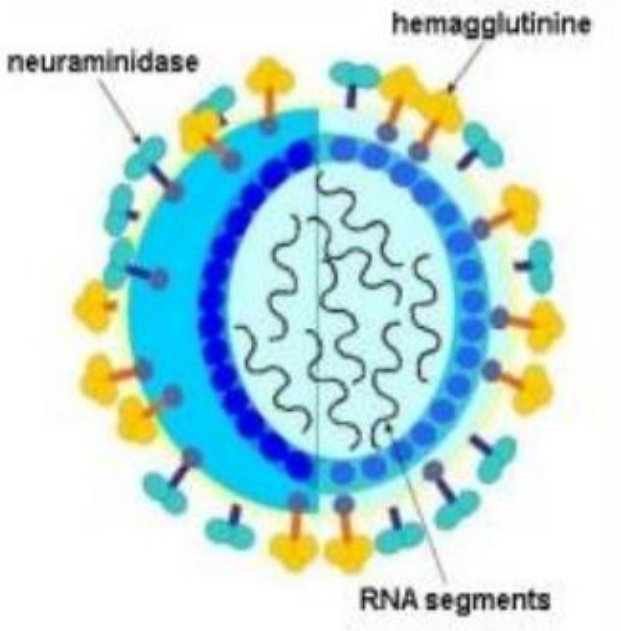


Structure of Virion



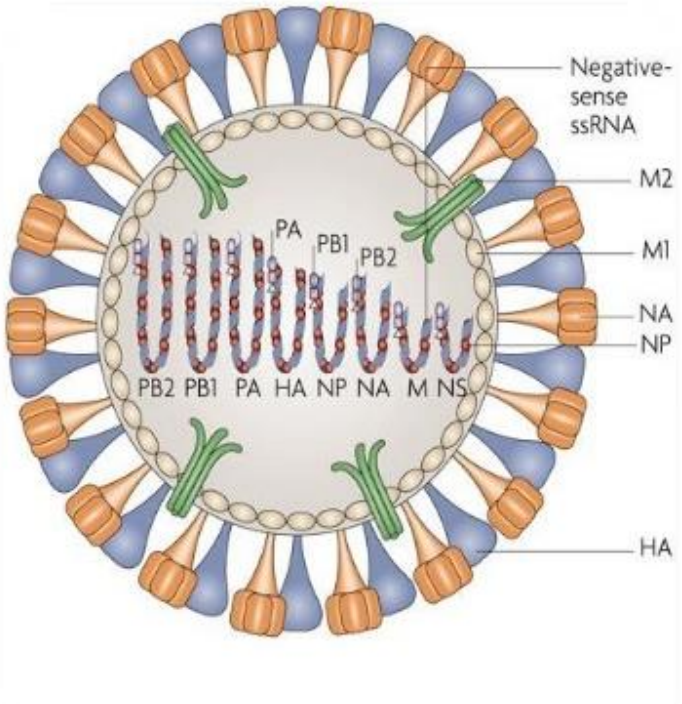
Viral structure

- The nucelocapsid is surrounded by an envelope with inner membrane protein layer and outer lipid
- From the envelop there are projections of two types
- 1 **Hem agglutinins**
- **2 Neuraminidase**



Types of Haemagglutinnins

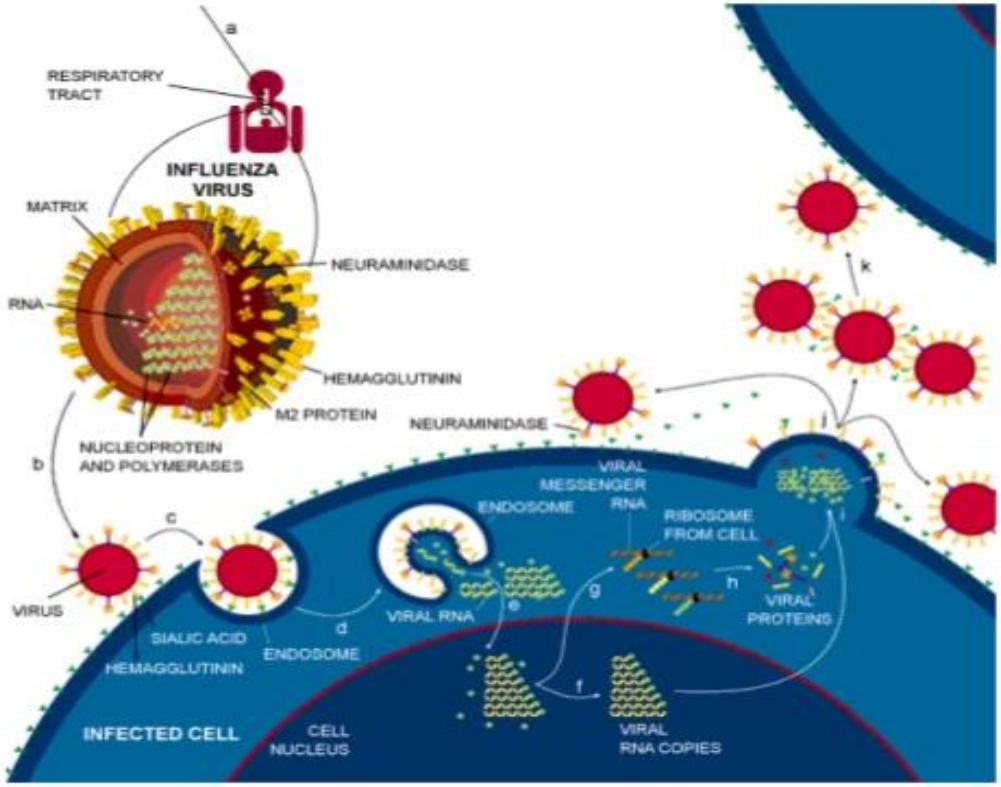
- Haemagglutination is strain specific
- Great variation
- H A there are 15 subtypes H 1 to H15 in avian influenza
- But only 4 variants in humans



Neuraminidases

- Neuraminidase are glycoprotein's
- Destroys cell receptors by hydrolysis cleavage
- Anti neuraminidase antibodies are produced following infection and immunization
- Not protective as Antihemagglutinin antibodies
- Helps to inhibit the release and spread of progeny
- Strain specific exhibit variation, There are nine different subtypes N 1 – N9.

Life cycle of the ‘flu virus



Antigenic Variation

- Unique feature of this virus lies with antigenic variation.
- High in type A virus
- Less in type B virus
- Not in type C virus
- RNP and Matrix proteins are stable
- Haemagglutination and Neuraminidase are independent of the variations.

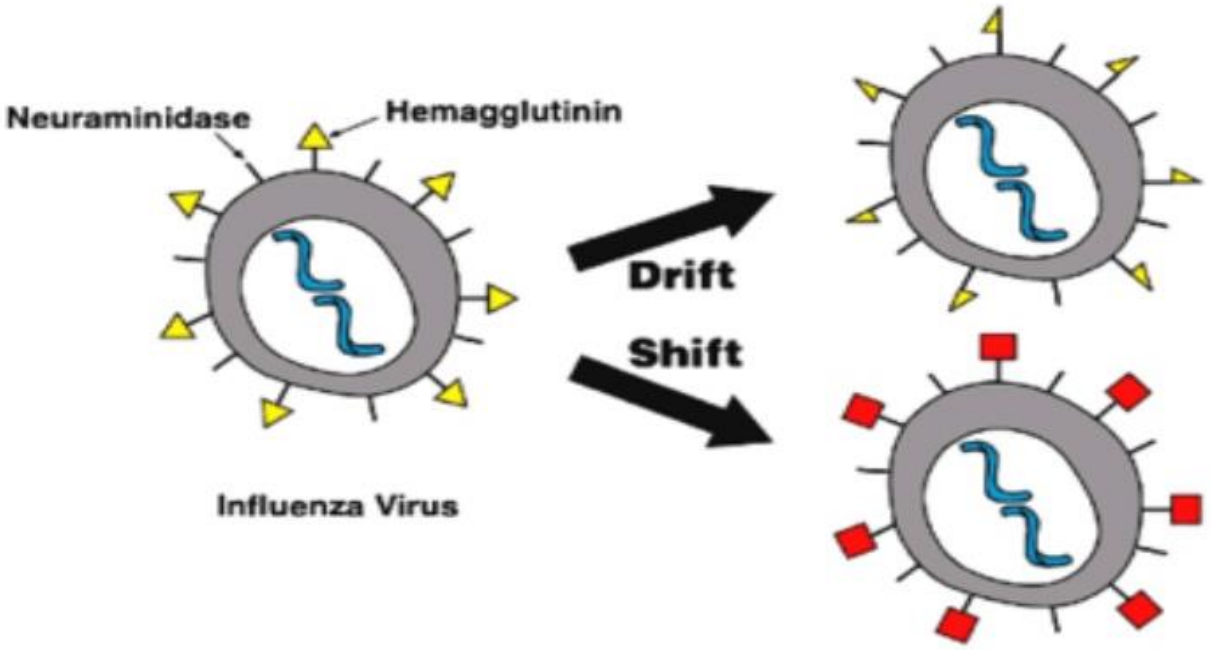
Influenza prominent Antigenic Changes

- Antigenic Shift
  - major change, new subtype
  - caused by exchange of gene segments
  - may result in pandemic
- Example of antigenic shift
  - H2N2 virus circulated in 1957-1967
  - H3N2 virus appeared in 1968 and completely replaced H2N2 virus

Influenza Antigenic Changes

- Antigenic Drift
  - minor change, same subtype
  - caused by point mutations in gene
  - may result in epidemic
- Example of antigenic drift
  - in 2002-2003, A/Panama/2007/99 (H3N2) virus was dominant
  - A/Fujian/411/2002 (H3N2) appeared in late 2003 and caused widespread illness in 2003-2004

Antigenic Variations



Resistance of Virus

- Inactivated by heating at 50^0c for 30 mt
- Survive for 1 week at 0 – 4^0c for 1 week
- Virus preserved at – 70^0c
- **Survive in the blankets for 2 weeks**
- Ether, formaldehyde, Phenol destroy the virus

INFLUENZA

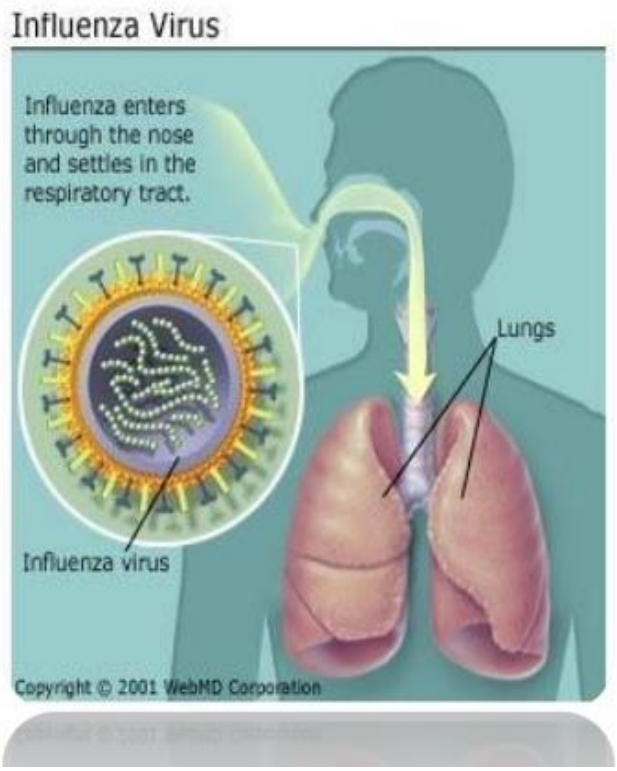
- Cause of the infection of the Respiratory tract.
- Occurs as

  Sporadic

  Epidemic

  Pandemic

Major pandemic in 1918 – 1919



Epidemiology

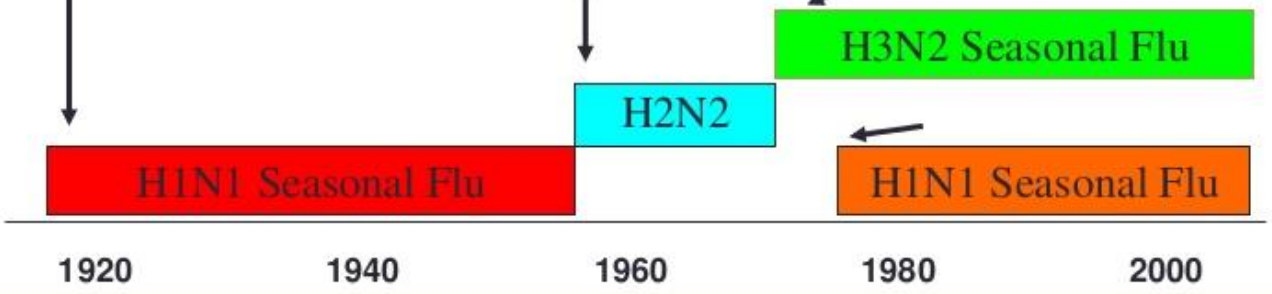
- Virus enters through respiratory route
- In 3 – 4 days majority manifest
- Many are subclinical infections
- Type A produce pandemics
- Type B sporadic cases, epidemics
- Dangerous in the Temperate regions
- Higher mortality in aged and patients with existing cardiopulmonary involvement

Circulating Seasonal Influenza A Sub-Types from Pandemics of the 20th Century

1918/19
40-100 million deaths

1957/58
~2 million deaths

1968/69
~1 million deaths



4 pandemics since 1889, with 11 to 39 years (average ~30 years) between each = ~3.3% annual risk of pandemic onset (but likely higher now)

TRANSMISSION

- AEROSOL
  - 100,000 TO 1,000,000 VIRIONS PER DROPLET
- 18-72 HR INCUBATION
- SHEDDING

Spread of Influenza

- The virus is transmitted easily from person to person via droplets and small particles produced when infected people cough or sneeze.

Pathogenesis

- Infects the respiratory tract
- Even 3 or few viral particles can infect
- Neuraminidase facilitates infection reducing the viscosity of Mucous
- Ciliated cells are infected in the Respiratory tract - site of viral infection
- When superficial layers are damaged exposes the basal layers
- And exposure of the basal layer causes the bacterial infections.

Pathogenesis – Viral Pneumonia

- Thickening of the Alveolar cells
- Intestinal infiltration with leucocytes with capillary thrombosis of Leucocytic exudates
- Hyaline membrane is formed occupying alveolar ducts and alveoli
- In late stages infiltration with Macrophages

Clinical features

- Incubation 1 to 3 days
- Present with mild cold lead to fulminating rapidly fatal Pneumonia
- Can abruptly present with head ache malign
- Can also present with abdominal pain with type B in children
- Bacteria superinfect

SYMPTOMS

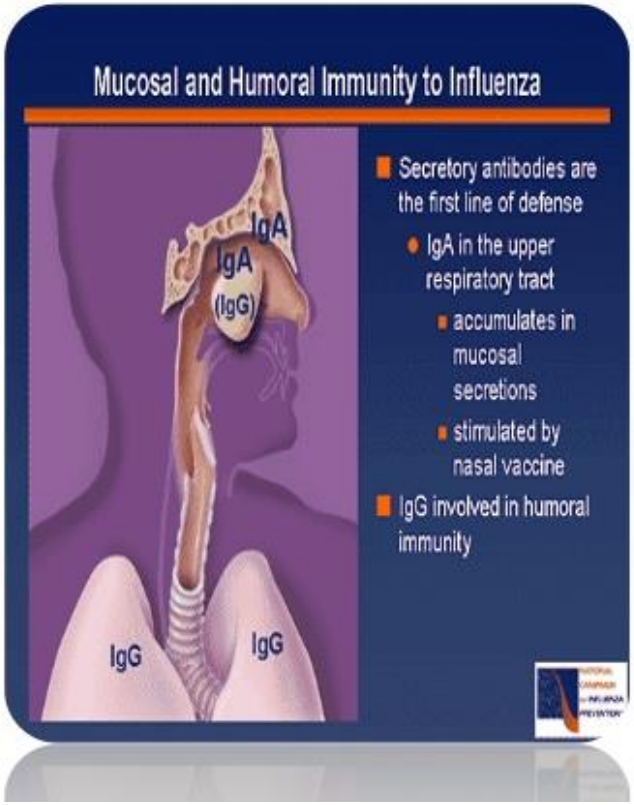
- FEVER
- HEADACHE
- MYALGIA
- COUGH
- RHINITIS
- OCULAR SYMPTOMS

Complications of Influenza

- Bacterial super infections
- Cardiac complications
- Congestive heart failure
- Myocarditis
- Neurological involvement
- Encephalitis
- Type B virus can produce Reye's syndrome
- Degenerative changes in the Brain and Liver
- Gastric flu with type B virus

Immunity in Influenza

- After infection immunity lasts 1 to 2 years
- Immunity lasts short duration due antigenic variants infecting at intervals.
- Antibodies produced locally are effective IgA immunoglobulin.
- Anti Hemagglutinins and Antinerumanidase are effective in prevention of infection.



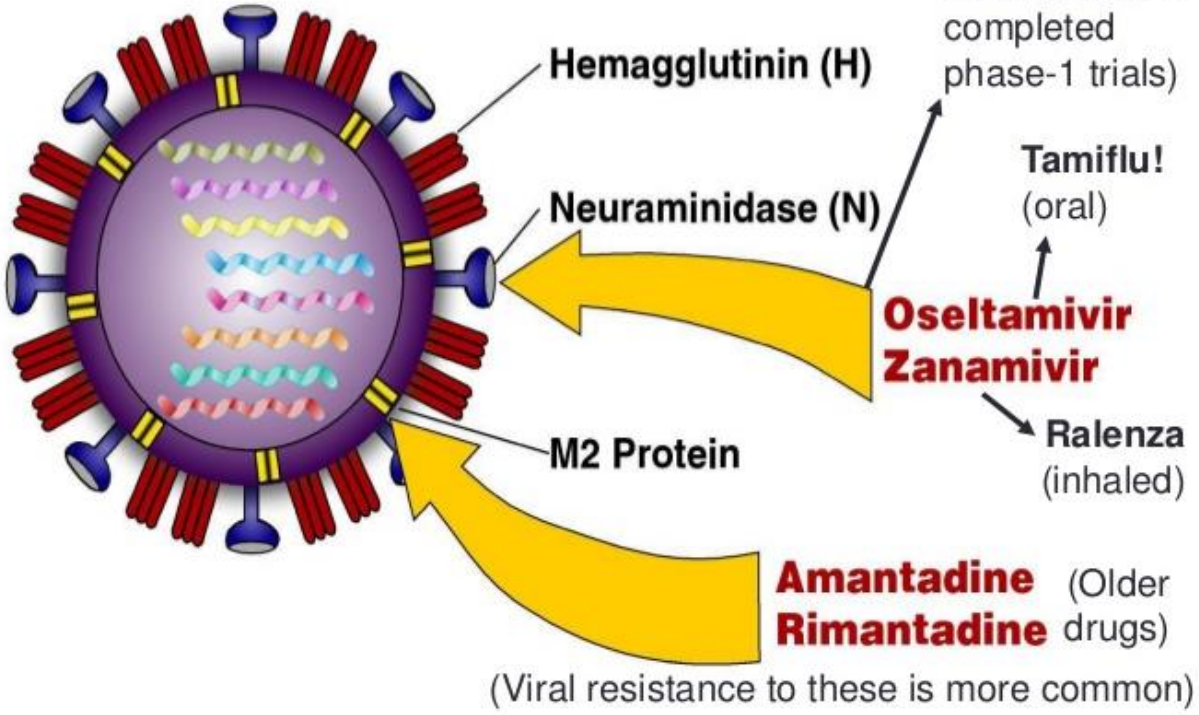
Influenza Diagnosis

- Clinical and epidemiological characteristics
- Isolation of influenza virus from clinical specimen (e.g., nasopharynx, throat, sputum)
- Significant rise in influenza IgG by serologic assay
- Direct antigen testing for type A virus

Serology

- Compliment fixation test
- Haemagglutination Inhibition testing
- *Testing on paired sera*
- Detection of Haemagglutination Inhibition testing
- Radial Immunodiffusion

Antiviral Therapies for Influenza



New strain Hon Kong

H5 N1 strain

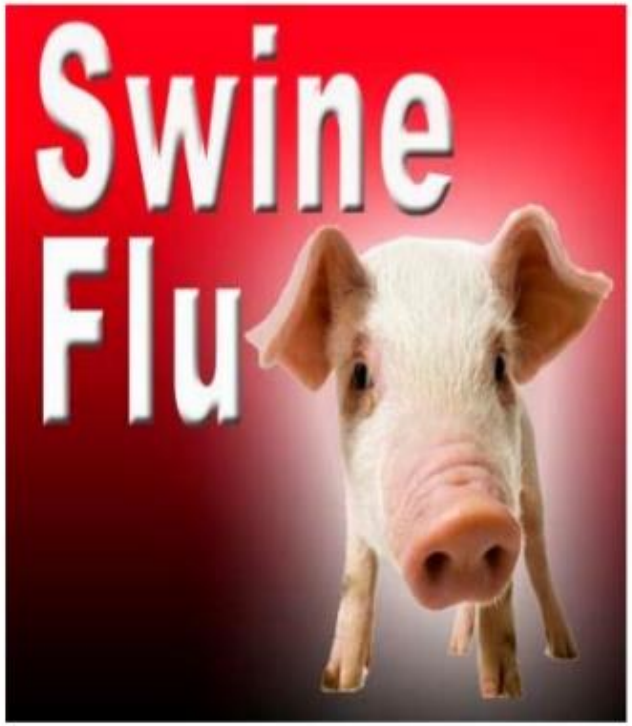
- Originated in Hong Kong
- 18 confirmed 6 dead
- Can spread from Chicken to Humans
- Wild aquatic birds spread.

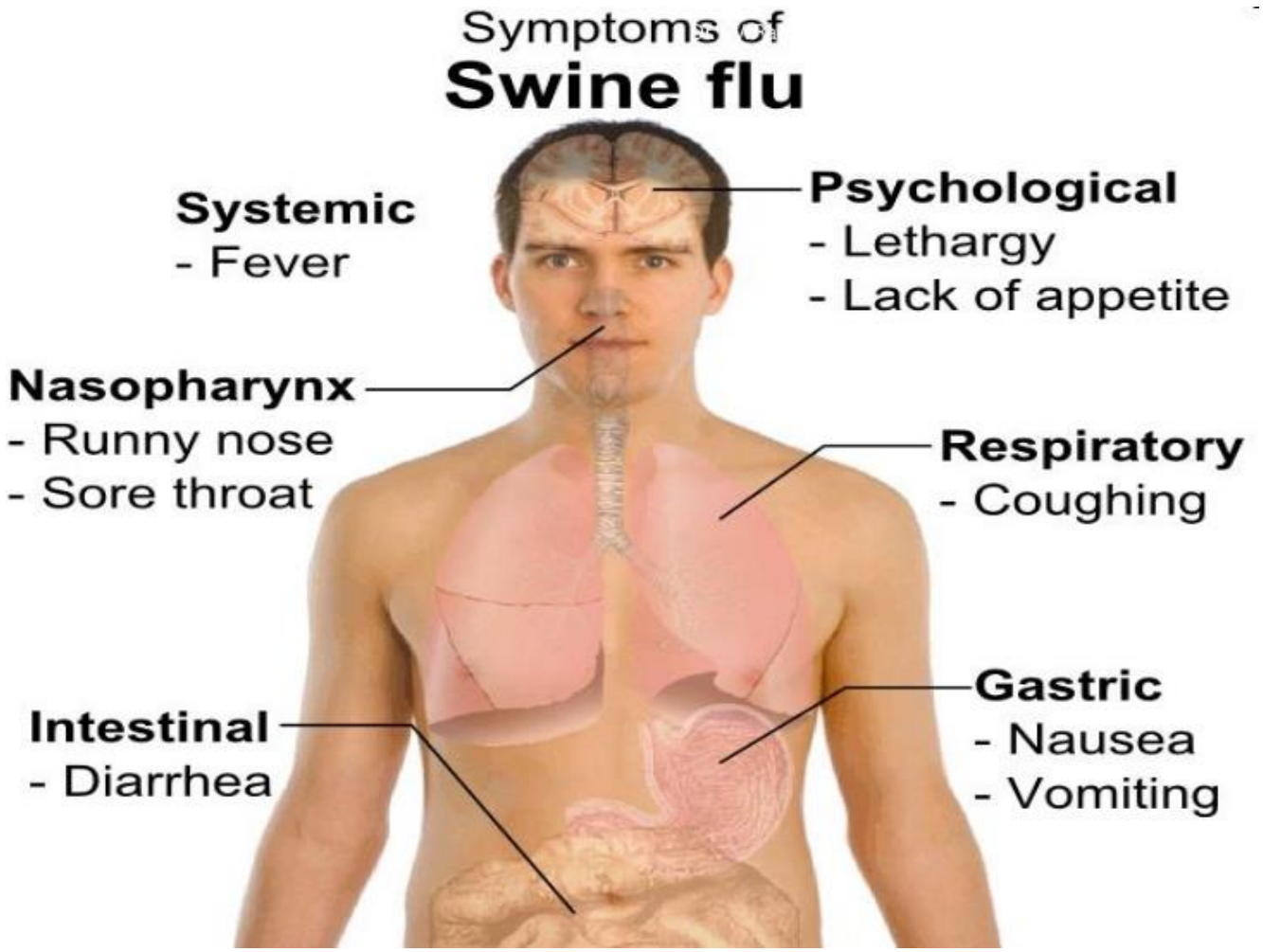
What is Bird Flu

- Avian Influenza in Animals
- Only birds get infected
- Less common Pigs
- Avian influenza is species specific
- Less common in Humans
- Can spread from poultry to Humans can produce sever disease

SWINE FLU 2009

- **Swine influenza** (also called **H1N1 flu**, **swine flu**, **hog flu**, and **pig flu**) is an infection by any one of several types of swine influenza virus. **Swine influenza virus** (**SIV**) is any strain of the influenza family of viruses that is endemic in pigs

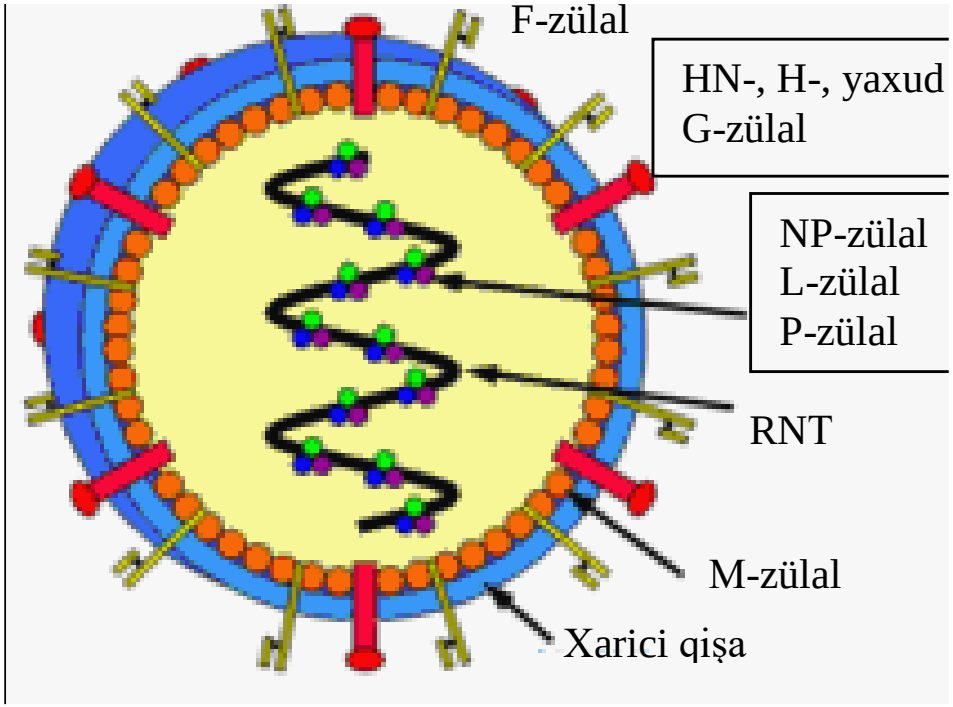



Symptoms of
Swine flu
Systemic
- Fever
Psychological
- Lethargy
- Lack of appetite
Nasopharynx
- Runny nose
- Sore throat
Respiratory
- Coughing
Intestinal
- Diarrhea
Gastric
- Nausea
- Vomiting

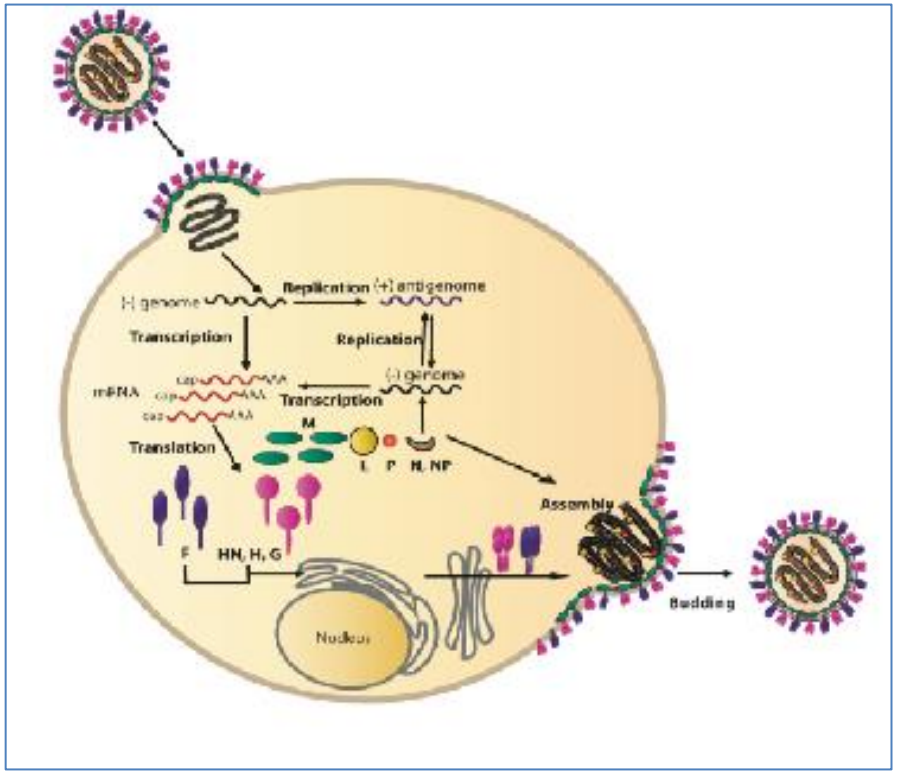
Paramyxoviridae family

- *Paramyxoviridae* consist of 2 subfamily - *Paramyxovirinae and Pnevmovirinae*
- *Paramyxovirinae* include: *Morbillivirus*, *Respirovirus*, *Rubulavirus, Avulovirus* and *Henipavirus*
- *Pnevmovirinae* include: *Pnevmovirus* and *Metapneumovirus*

Paramyxoviridae structure

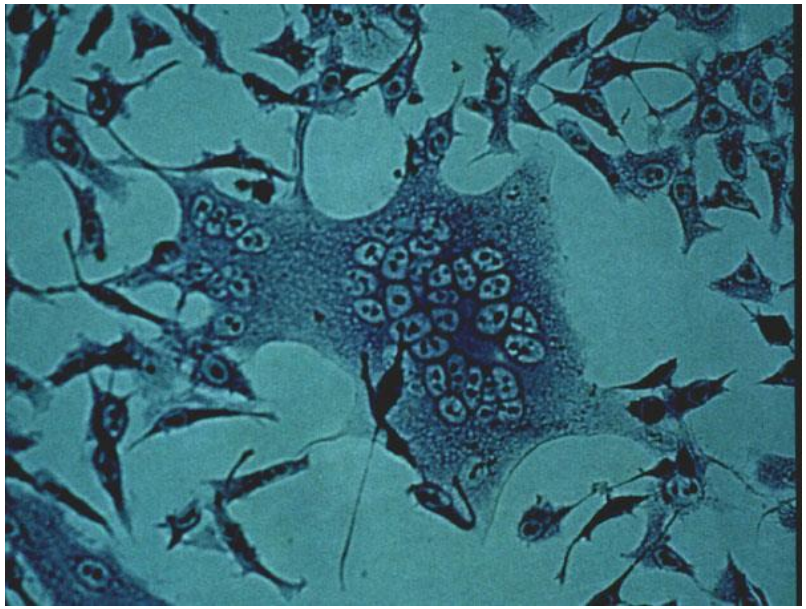


Paramyxoviridae replication

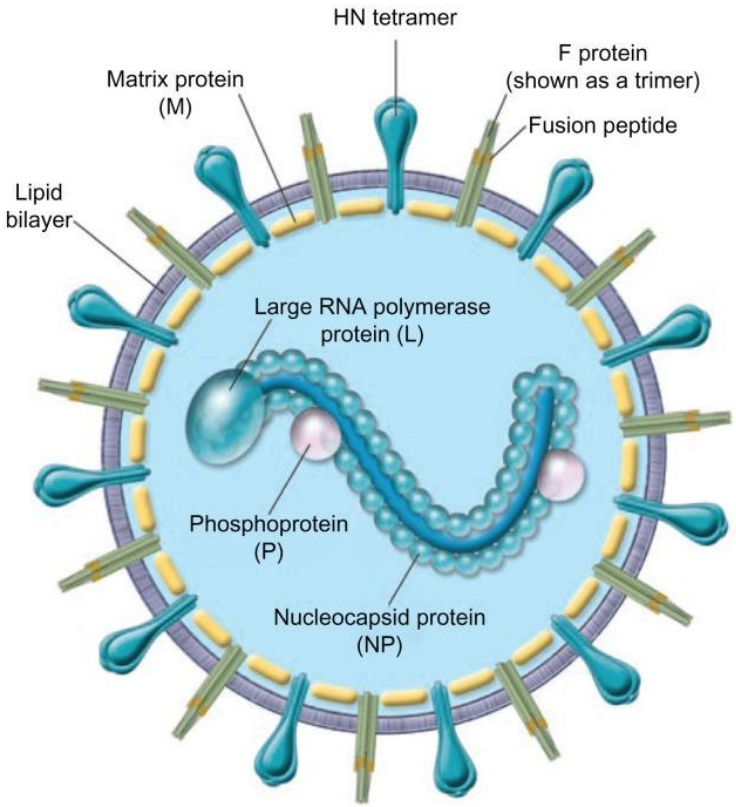


Paramyxoviridae cultivation

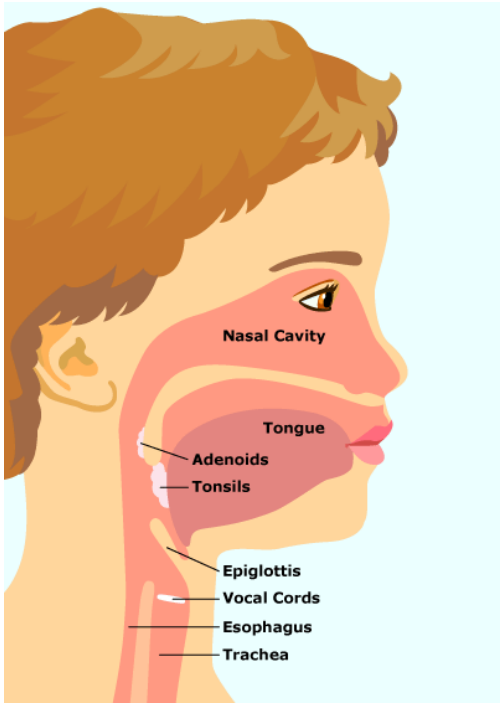
- Forms the cytopathic effect on cell culture as **inclusions** and **polikarions**

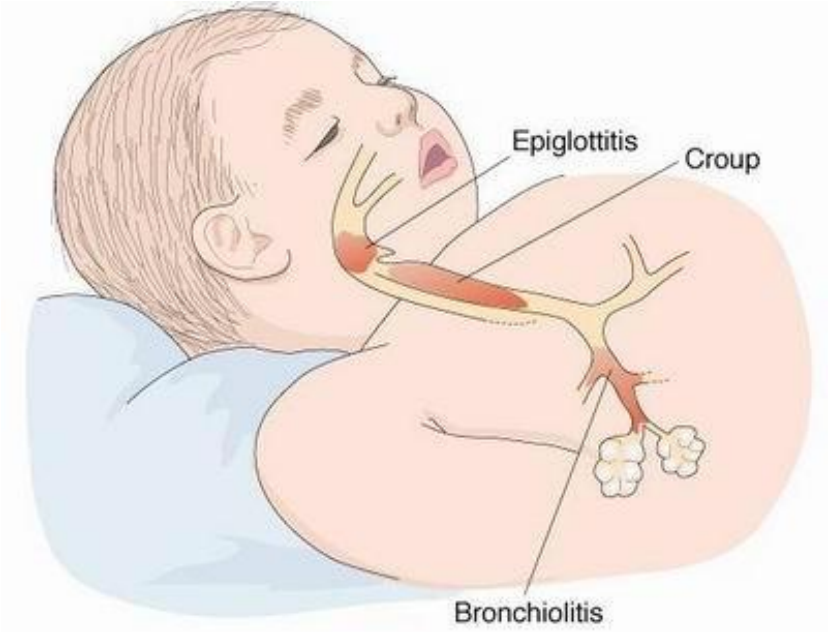


Parainfluenza virus

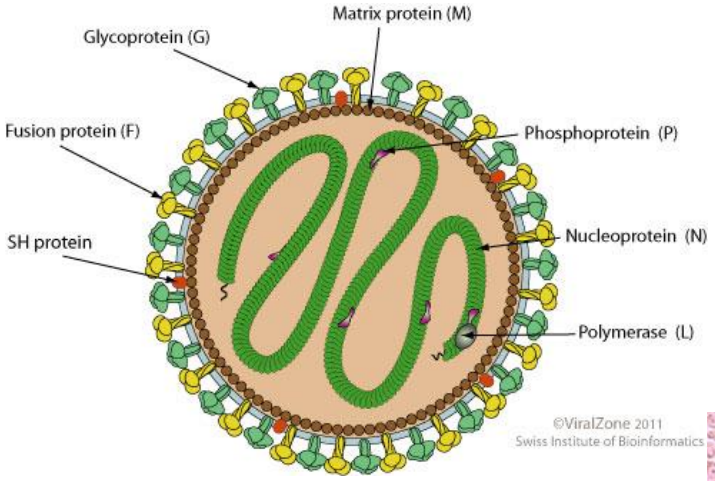


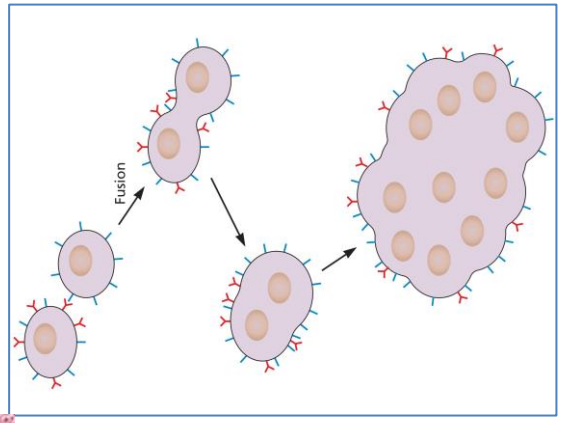
Parainfluenza: clinical manifestations

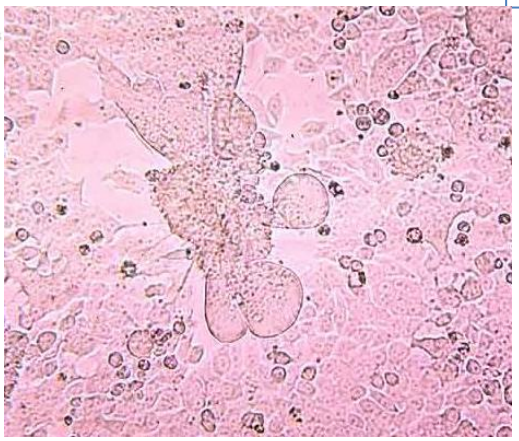




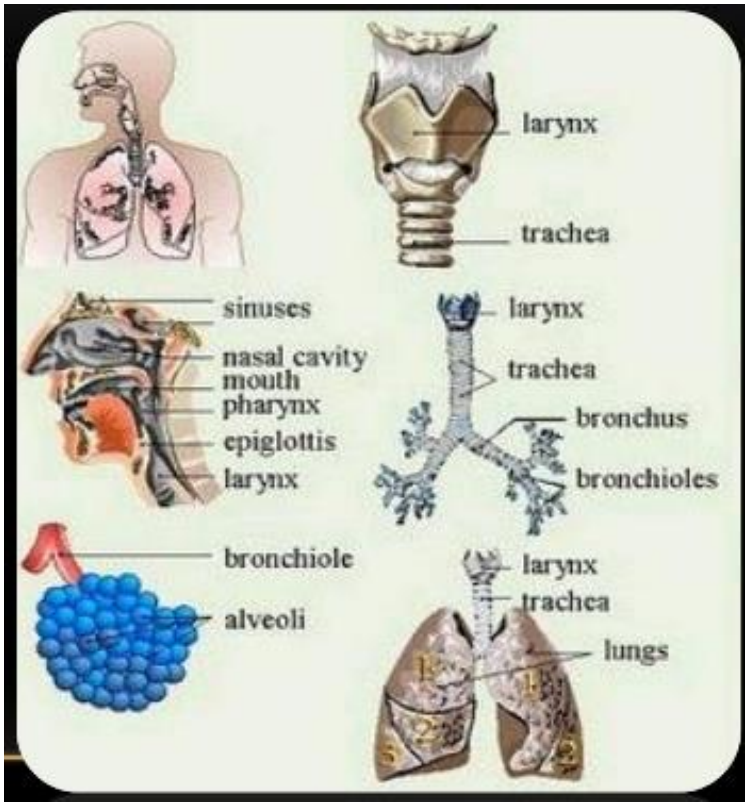
Respiratory syncytial virus (RS-virus)





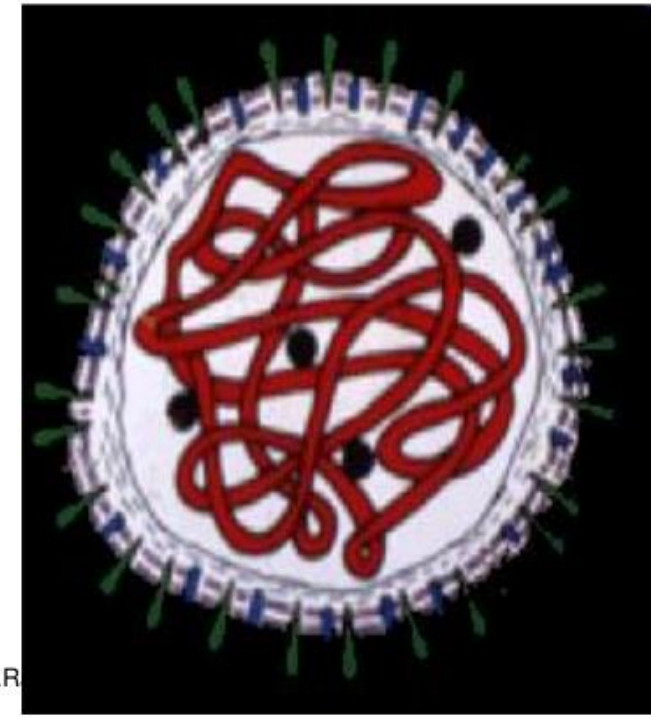


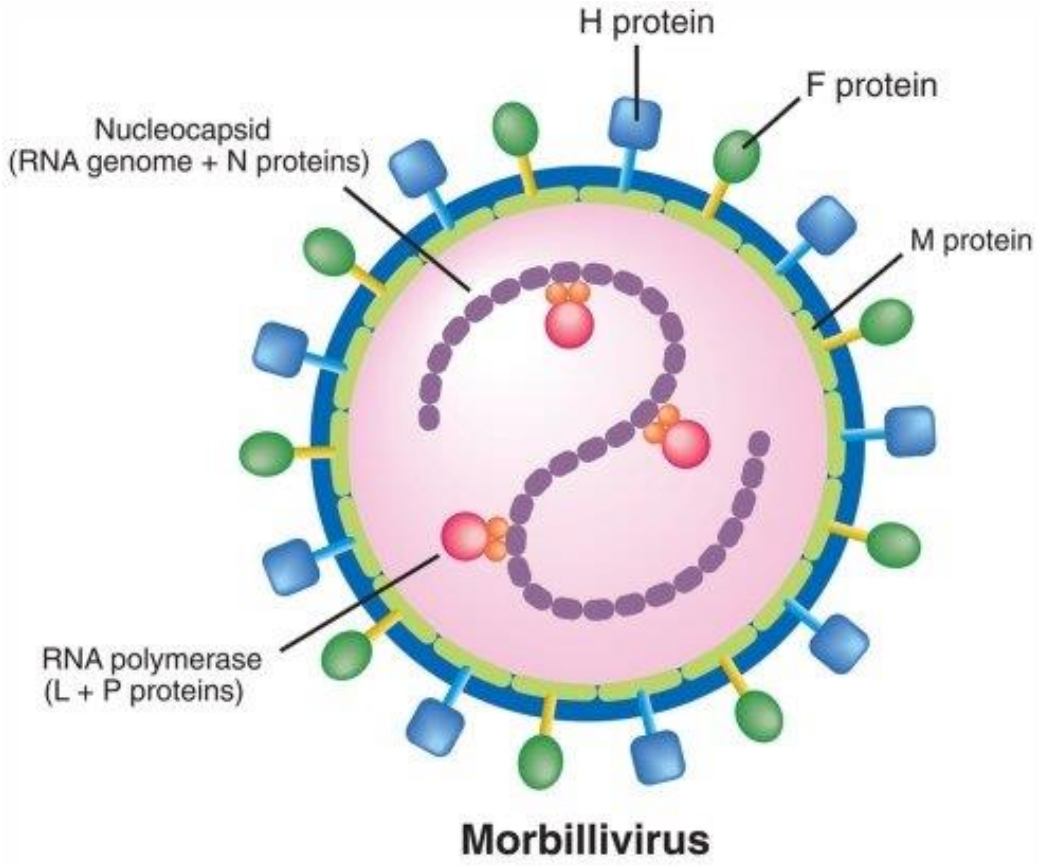
Pathogenesis of RS-virus infection



Measles - Paramyxoviridae

- **Measles** is an infection of the respiratory system caused by a virus, specifically a Paramyxovirus of the genus *Morbillivirus* Morbilliviruses, like other paramyxovirus, are enveloped, single-stranded, negative-sense RNA viruses.




H protein
F protein
Nucleocapsid
(RNA genome + N proteins)
M protein
RNA polymerase
(L + P proteins)
Morbillivirus

Measles a Childhood Infection

- Age-specific attack rates may be highest in susceptible infants younger than 12 months, school-aged children, or young adults, depending on local immunization practices and incidence of the disease.

EPIDEMIOLOGY

- Infection sources
  - Patients of acute stage and viral carriers of atypical measles
- Transmission
  - Highly contagious, approximately 90% of susceptible contacts acquire the disease.
  - Respiratory secretions: maximal dissemination of virus occurs by droplet spray during the prodromal period (catarrhal stage).
  - Contagious from 5 days *before symptoms*, 5 days *after onset of rash*
  - Seasons: in the spring, peak in Feb-May

PATHOGENESIS AND PATHOLOGY

- Portal of entry
  - Respiratory tract and regional lymph nodes
  - Enters bloodstream (primary viraemia) → monocyte – phagocyte system → target organs (secondary viraemia)
- Target organs
  - The skin; the mucous membranes of the nasopharynx, bronchi, and intestinal tract; and in the conjunctivae, ect

Resulting In-----

1) Koplik spots and skin rash: serous exudation and proliferation of endothelial cells around the capillaries
2) Conjunctivis

PATHOGENESIS AND PATHOLOGY

3) Laryngitis, croup, bronchitis :general inflammatory reaction

4) Hyperplasia of lymphoid tissue: multinucleated giant cells (Warthin-Finkeldey giant cells) may be found

5) Interstitial pneumonitis: Hecht giant cell pneumonia.

6) Bronchopneumonia: due to secondary bacterial infections

7) Encephalomyelitis: perivascular demyelinization occurs in areas of the brain and spinal cord.

8) Subacute sclerosing panencephalitis(SSPE):

degeneration of the cortex and white matter with intranuclear and intracytoplasmic inclusion bodies

CLINICAL MANIFESTATION

Typical Manifestation:

patients havn't had measles immunization, or vaccine failure with normal immunity or those havn't used immune globulin

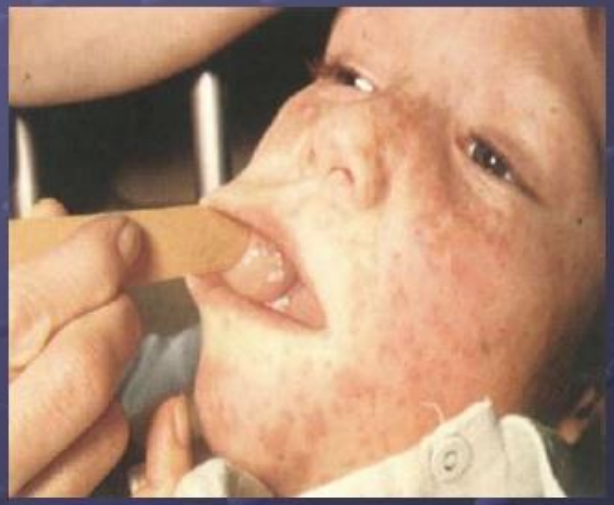
1. Incubation period (infection to symptoms) :
   6-18days (average 10 days)

2. Prodromal period:
   - 3-4 days
   - Non-specific symptoms: fever, malaise, anorexia, headache
   - Classical triad: cough, coryza, conjunctivitis (with photophobia, lacrimation)

CLINICAL MANIFESTATION

Enanthem (Koplik spots):

- *Pathognomonic for measles*
- 24-48 hr before rash appears
- 1mm, grayish white dots with slight, reddish areolae
- Buccal mucosa, opposite the lower 2nd molars
- increase within 1day and spread
- fade soon after rash onset



CLINICAL MANIFESTATION

3. Rash period

3-4days

Exanthem:

Erythematous, non-pruritic, maculopapular

- Upper lateral of the neck, behind ears, hairline, face → trunk → arms and legs →feet

- The severity of the disease is directly related to the extent and confluence of the rash

,

CLINICAL MANIFESTATION

Temperature:

- Rises abruptly as the rash appears
- Reaches 40°C or higher
- Settles after 4-5 days – if persists, suspect secondary infection

Coryza, fever, and cough:

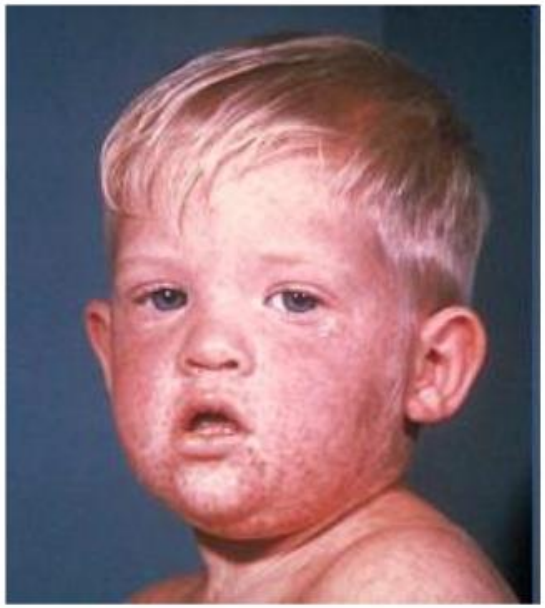
- Increasingly severe up to the time the rash has covered the body

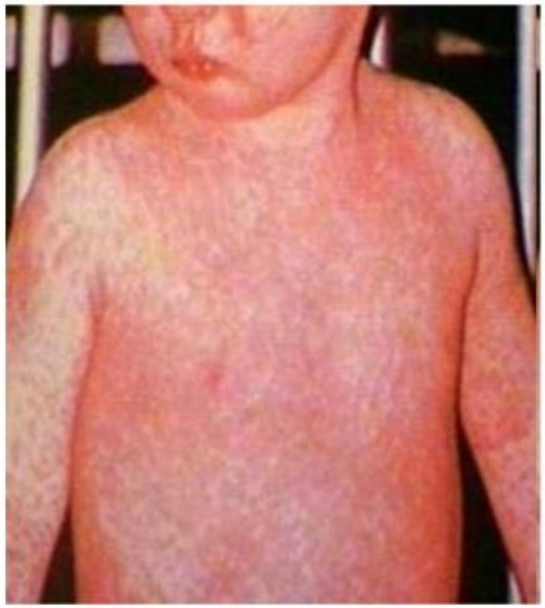
Lymphadenopathy (posterior cervical region, mesenteric) splenomegaly, diarrhoea, vomiting

Chest X ray:

- May be abnormal, even in uncomplicated cases

Rash is a Prominent Feature





CLINICAL MANIFESTATION

4. Recovery period

3-4days

Exanthem:

- Fades in order of appearance
- Branny desquamation and brownish discoloration

Entire illness – 10 days

LABORATORY EXAMINATION

- Isolation of measles virus from a clinical specimen (e.g., nasopharynx, urine)
- Significant rise in measles IgG by any standard serologic assay
- Positive serologic test for measles IgM antibody
- Immunofluorescence detects Measles antigens
- Multinucleated giant cells in smears of nasal mucosa

- Low white blood cell count and a relative lymphocytosis in PB
- Measles encephalitis – raised protein, lymphocytes in CSF

DIAGNOSIS

characteristic clinical picture:

- Measles contact
- Koplik spot
- Features of the skin rash
- The relation between the eruption and fever

Laboratory confirmation is rarely needed

TREATMENT

- Supportive, symptom-directed
  - Antipyretics for fever
  - Bed rest
  - Adequate fluid intake
  - Be protected from exposure to strong light
- Antibiotics for otitis media, pneumonia
- High doses Vitamin A in severe/ potentially severe measles/ patients less than 2 years
  - 100,000IU—200,000IU

PREVENTION

- 1. Quarantine period

  5 days after rash appears, longer for complicated measles

- 2. Vaccine

  The initial measles immunization is recommended at 8mo of age

  A second immunization is recommended routinely at 7yr of age

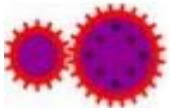
- 3. Postexposure Prophylaxis

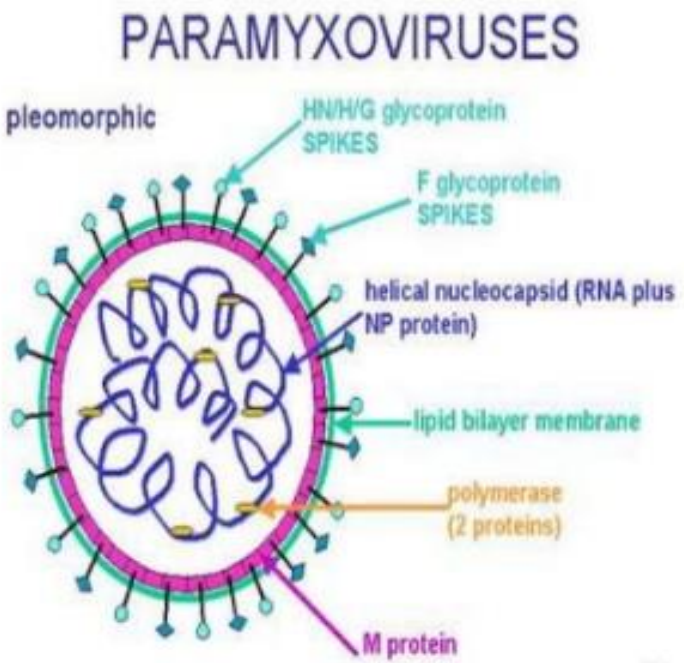
  Passive immunization with immune globulin (0.25mL/kg) is effective for prevention and attenuation of measles within 5 days of exposure.

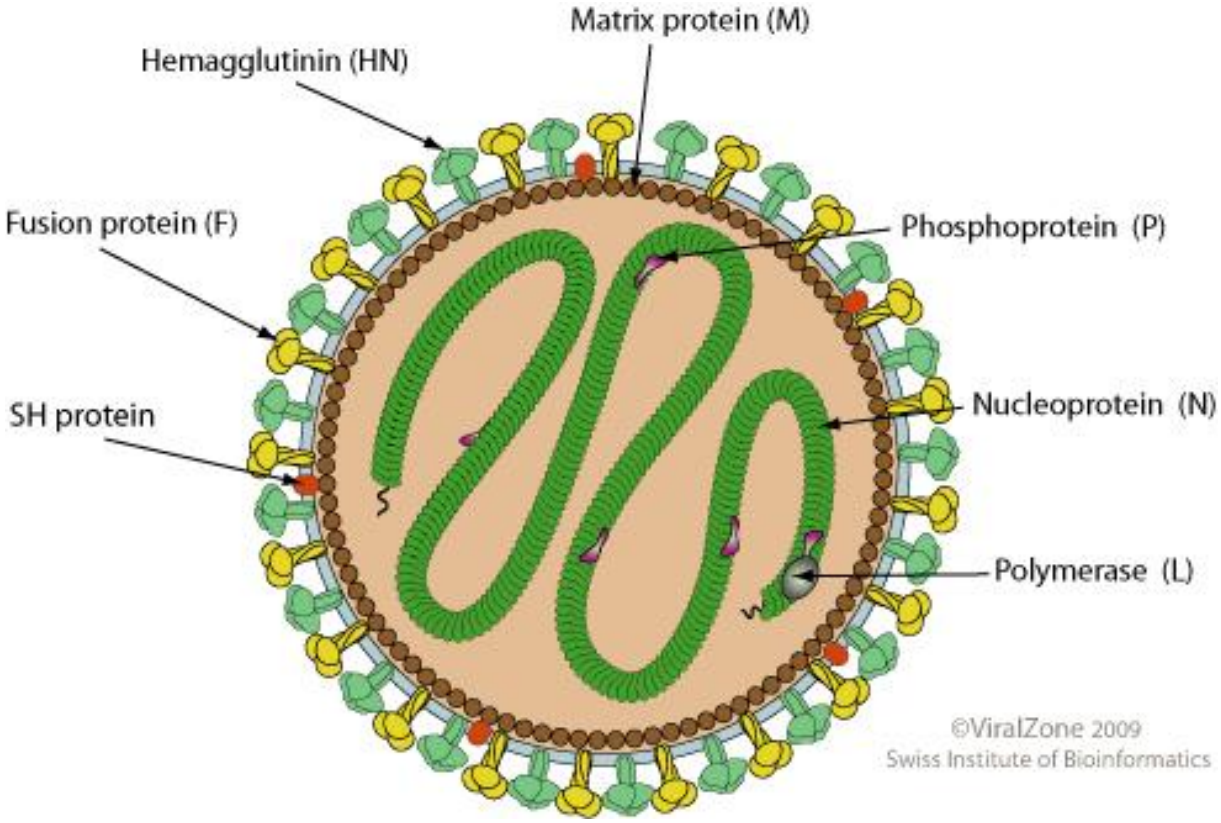
MUMPS

Properties of MUMPS virus.

- Posses HN and F properties.
- Growth in Chick Embryos, in the Amniotic cavity, Adopts in allantoic cavity,
- Cell cultures – Primary Monkey kidney,
- Typical Paramyxoviruses, produce cytopathic effects.






Matrix protein (M)
Hemagglutinin (HN)
Fusion protein (F)
Phosphoprotein (P)
SH protein
Nucleoprotein (N)
Polymerase (L)
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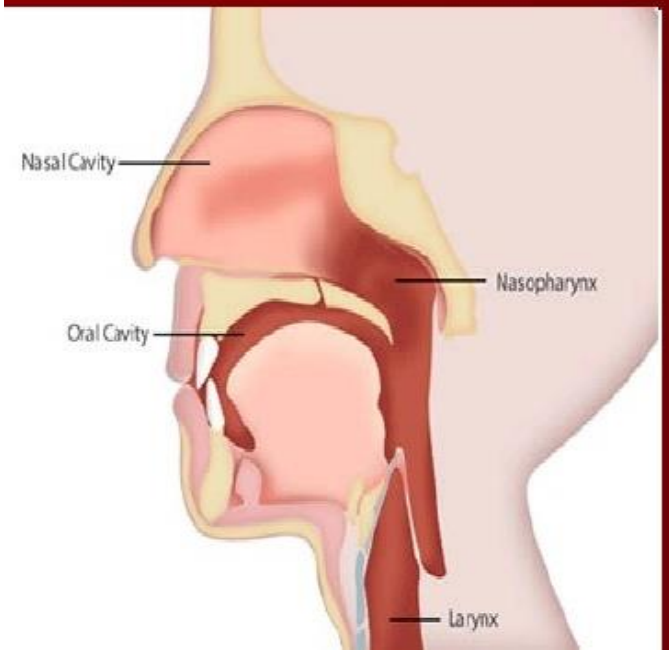
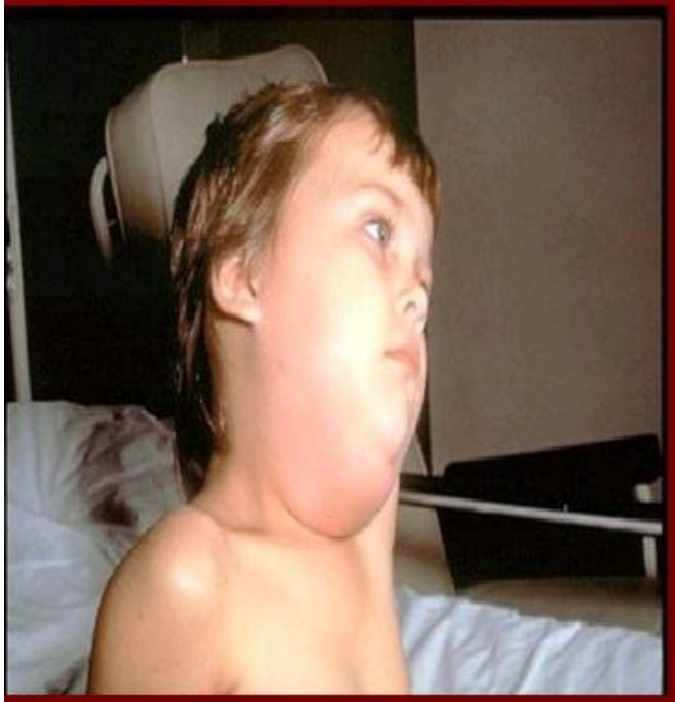
Pathogenesis - Mumps



- Respiratory transmission of virus
- Replication in nasopharynx and regional lymph nodes
- Viremia 12-25 days after exposure with spread to tissues
- Multiple tissues infected during viremia

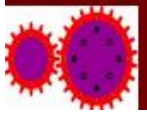
Mumps Clinical Features



- Incubation period 14-18 days
- Nonspecific prodrome of myalgia, malaise, headache, low-grade fever
- Parotitis in 30%-40%
- Up to 20% of infections asymptomatic

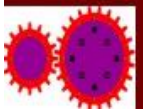
Complication with MUMPS.

- Epididymo orchids.
- May lead to atrophy, sterility, Low sperm counts.
- CNS involvement in 60% cases
- May manifest with Aseptic meningitis,
- Deafness,
- Arthritis,Oopharitis,Nephritis and Myocarditis,



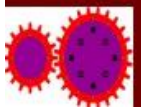
Complication with MUMPS.

- **Orchitis.** This inflammatory condition causes swelling of one or both testicles. Orchitis is painful.
- **Pancreatitis..**
- **Encephalitis.** A viral infection, such as mumps, can lead to inflammation of the brain (encephalitis). Although it's serious, encephalitis is a rare complication of mumps.



Complication with MUMPS.

- **Meningitis.** Meningitis is infection and inflammation of the membranes and fluid surrounding your brain and spinal cord.
- **Inflammation of the ovaries.** Pain in the lower abdomen in women may be a symptom of this problem. Fertility doesn't seem to be affected.
- **Hearing loss.**
- **Miscarriages.**



Laboratory Diagnosis

- **No Laboratory confirmation needed.**
- Atypical infection needs laboratory Diagnosis.
- Virus isolated from

  Saliva

  Urine

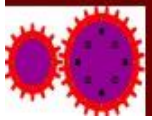
  CSF.

Culturing in **Human amnion, He la cells.**

Immunoflorecence Methods. Isolation in Chick Embryos

ELISA, Complement fixation tests,

Doctortvrao's 'e' learning



MEASLES, MUMPS & RUBELLA (MMR) VACCINE:

2 D

Measles vaccine is available as a monovalent preparation or combined as measles-mumps-rubella (MMR) vaccine.

MMR:

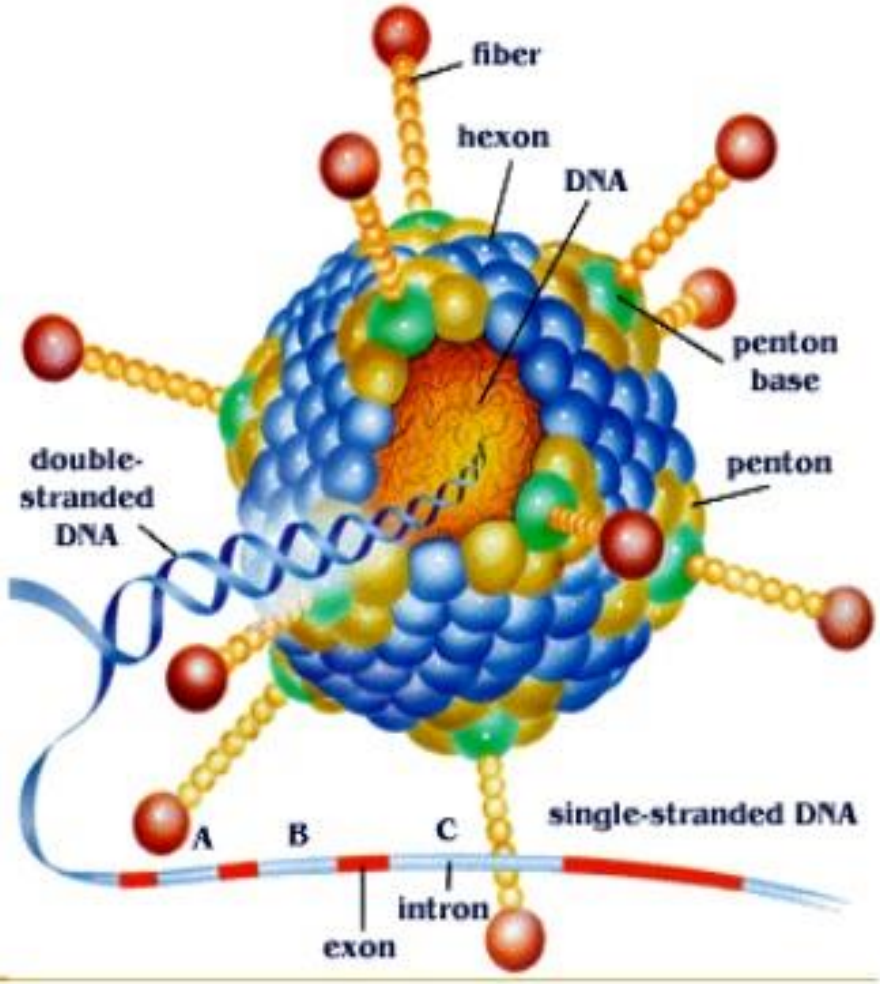
- Minimum age 12 mo.
- The current recommendations include a first dose at **12-15 mo** followed by a second dose at **4-6 yr** of age. **Seroconversion** is slightly lower in children who receive the first dose before or at 12 mo of age because of persisting maternal antibody.
- MMR may be administered **before** age 4-6 yr, provided **≥ 4wk** have elapsed since the 1st dose.
- For children who have ***not*** received 2 doses by 11-12 yr of age, a second dose should be provided.
- Infants who ***receive a dose before 12 mo*** of age should be given 2 additional doses at 12-15 mo and 4-6 yr of age.

Measles vaccine:

- For children 6-11 mo of age in epidemic situations or prior to international travel.

What are Adenoviruses

- Adenoviruses are a group of medium sized, nonenvelopedd, double stranded DNA viruses that share a common complement fixing antigen
- They infect humans and animals



Chaterstics of Adenovirus

- **Adenoviruses** are medium-sized (90–100 nm, nonenveloped (naked) **icosahedral** viruses composed of a nucleocapsid and a double-stranded linear DNA genome. There are over 52 different serotypes in humans, which are responsible for 5–10% of upper respiratory infections in children, and many infections in adults as well.

CLASSIFICATION

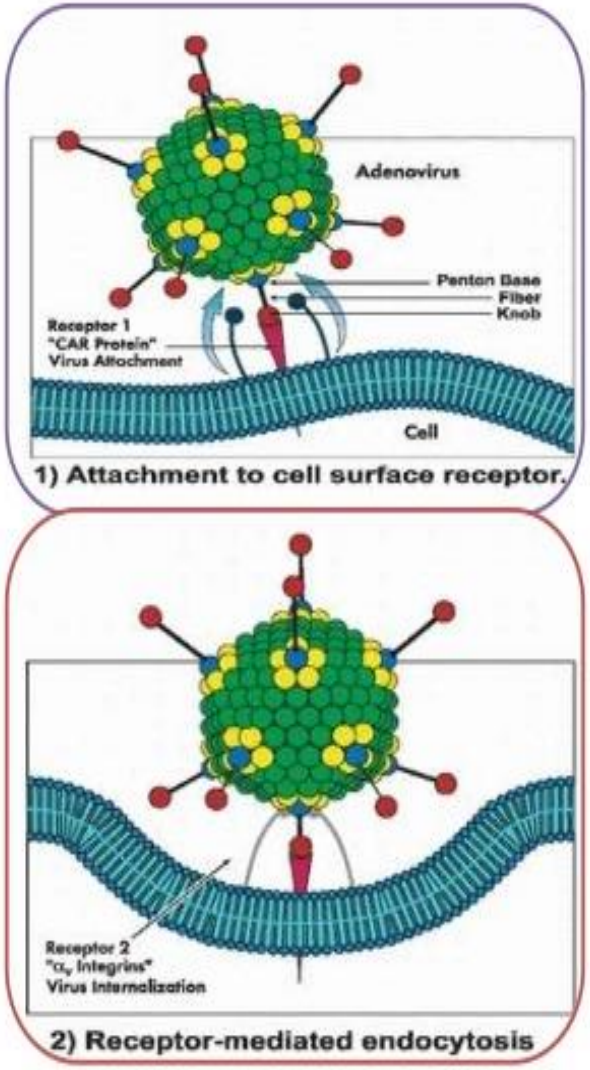
- Family Adenoviridae : 2 genera
  - Aviadenovirus : adenoviruses of birds;
  - Mastadenovirus : infect mammals;
    - **47 serotypes** of human origin;
    - **6 subgroups (A-F)** based on genome homology;
    - **Subgroup A : serotypes 12,18 and 31 : highly oncogenic;**

DISEASE ASSOCIATIONS

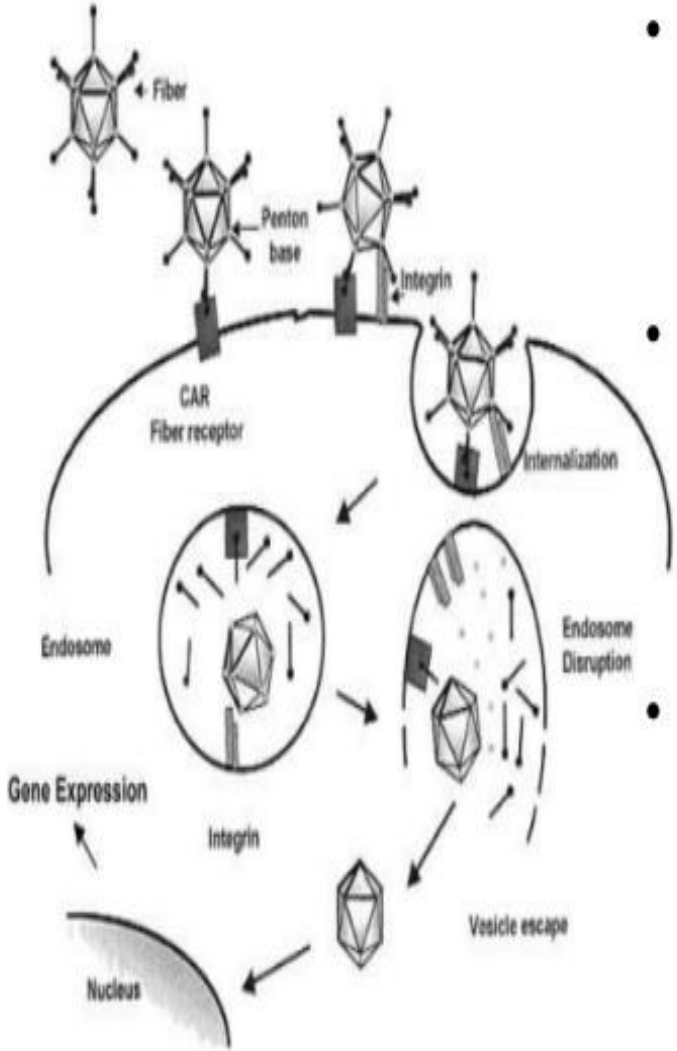
Serotype	Disease	At risk
1-7	Acute febrile pharyngitis	Children
3,7,14	Pharyngoconjunctival fever	Older children
8,9,37	Epidemic keratoconjunctivitis (shipyard eye)	Adults
3,4,11	Acute follicular conjunctivitis	Any age
40,41	Diarrhoea	Infants, young children
11,21	Hemorrhagic cystitis	Children

VIRAL REPLICATION

- Adenoviruses attach to surface of the cells by their fibers, then penetrate the cell, and once inside the cell, uncoat the viral DNA.
- The viral DNA is then transported into the nucleus of the cell and initiates replication cycle.
- Host cell DNA-dependent RNA polymerase transcribes the early genes leading to formation of functional mRNA.



VIRAL REPLICATION

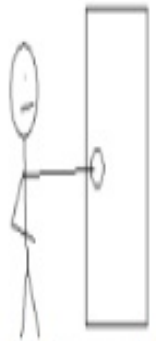


- Then in the cytoplasm, the early mRNA is translated into nonstructural proteins.
- In the nucleus, after viral DNA replication, late mRNA is transcribed and then translated into structural virion proteins.
- This is followed by assembly of virions in the nucleus and release of virions by lysis of the cells, but not by budding.

PATHOGENESIS

- Adenoviruses are transmitted mainly by respiratory or feco-oral contact from humans.
- They infect the conjunctiva or the nasal mucosa.
- They may multiply in conjunctiva, pharynx, or small intestine, and then spread to preauricular, cervical and mesenteric lymph nodes, where epithelial cells are infected.

respiratory transmission



fomite transmission

water-borne transmission

fecal-oral transmission

PATHOGENESIS

- Adenoviruses may cause three different types of interaction with the infected cells.
- These are (a) lytic infection, (b) latent infection, and (c) transforming infection.
- lytic infection: Adenoviruses infect mucoepithelial cells in the respiratory tract, gastrointestinal tract, and conjunctiva or cornea, causing damage of these cells directly.
- After local replication of the virus, viremia follows with subsequent spread to visceral organs.
- Dissemination occurs more commonly in immunocompromised patients than in the immunocompetent individuals.

PATHOGENESIS

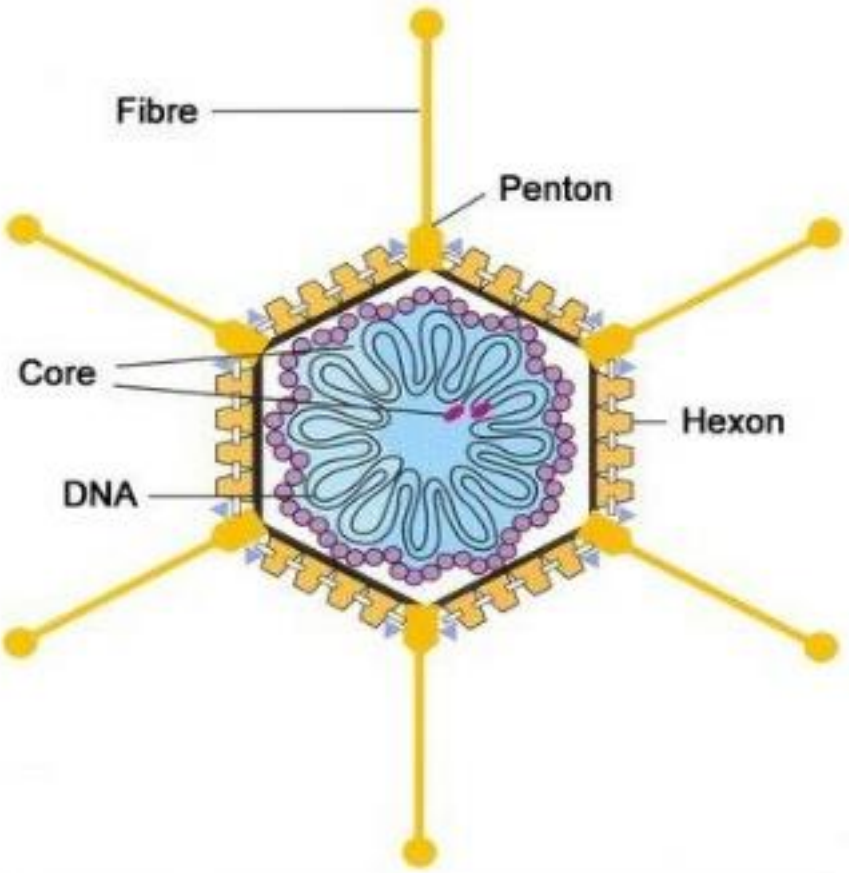
- latent infection: The adenovirus has a unique ability to become latent in lymphoid and other tissues such as adenoids, tonsils, and Payer's patches.
- The exact mechanism of latency of adenoviruses in these tissues is not known.
- These latent infections can be reactivated in patients infected with other agents or in the patients who are immunocompromised.

PATHOGENESIS

- Oncogenic transformation: Some adenoviruses belonging to groups A and B have the property for oncogenic transformation in rodent cells.
- During oncogenesis the multiplication of adenovirus is inhibited followed by integration of viral DNA into the host DNA.
- After integration adenoviruses produce E1A proteins which target rodent cells by altering cellular transcription, finally leading to transformation of rodent cells.
- However, oncogenesis of human cells has not been demonstrated.

Morphology of Adenovirus

- Adenovirus are 70-75 nm in size
- The capsid contains 252 capsomers arranged as icosahedrons with 20 triangular facets and 12 vertices
- 240 are called as hexons
- 12 are called as pentons



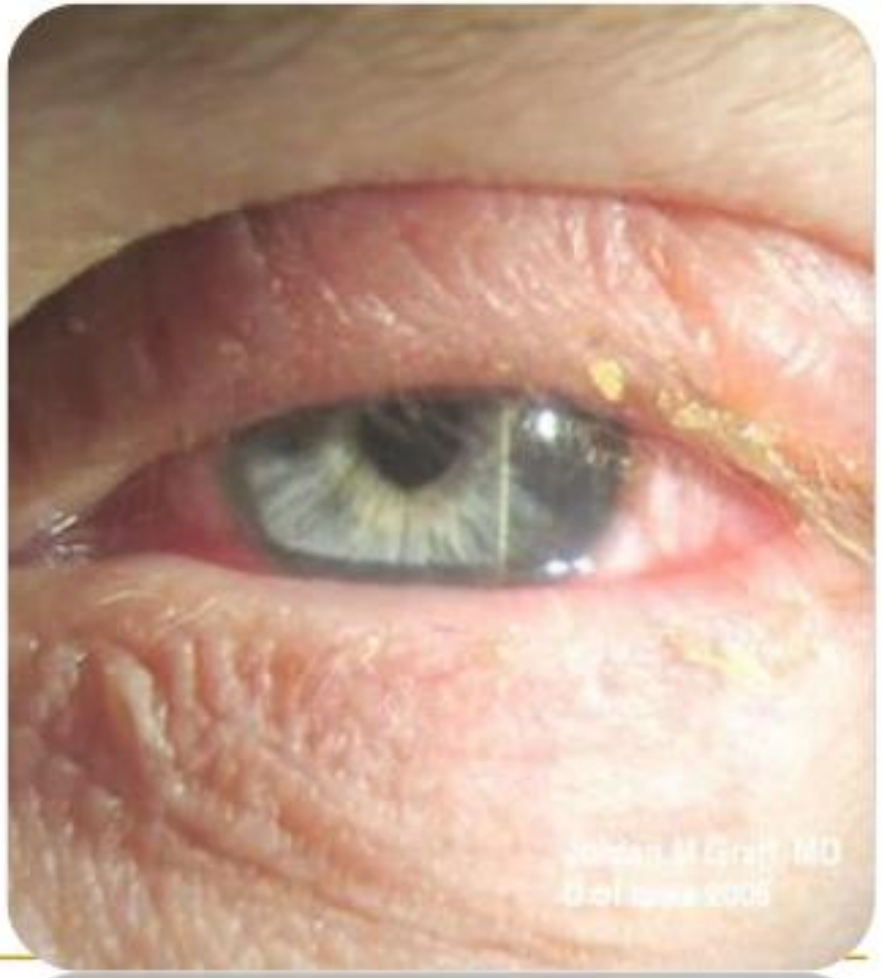
Pathogenesis:

- Adenoviruses infect and replicate in the epithelial cells of the:
  - pharynx,
  - conjunctiva,
  - urinary bladder
  - small intestine.

  They usually do not spread beyond the regional lymph nodes **EXCEPT IN THE IMMUNE COMPROMIZED HOST.**

Pathogenesis

- Adenovirus cause infections in
- Respiratory tract
- Eye, Urinary bladder, and Intestines
- More than one type of virus may cause clinically different diseases



CLINICAL SYNDROMES

A. Respiratory diseases:

B. Eye infections:

C. Gastrointestinal disease

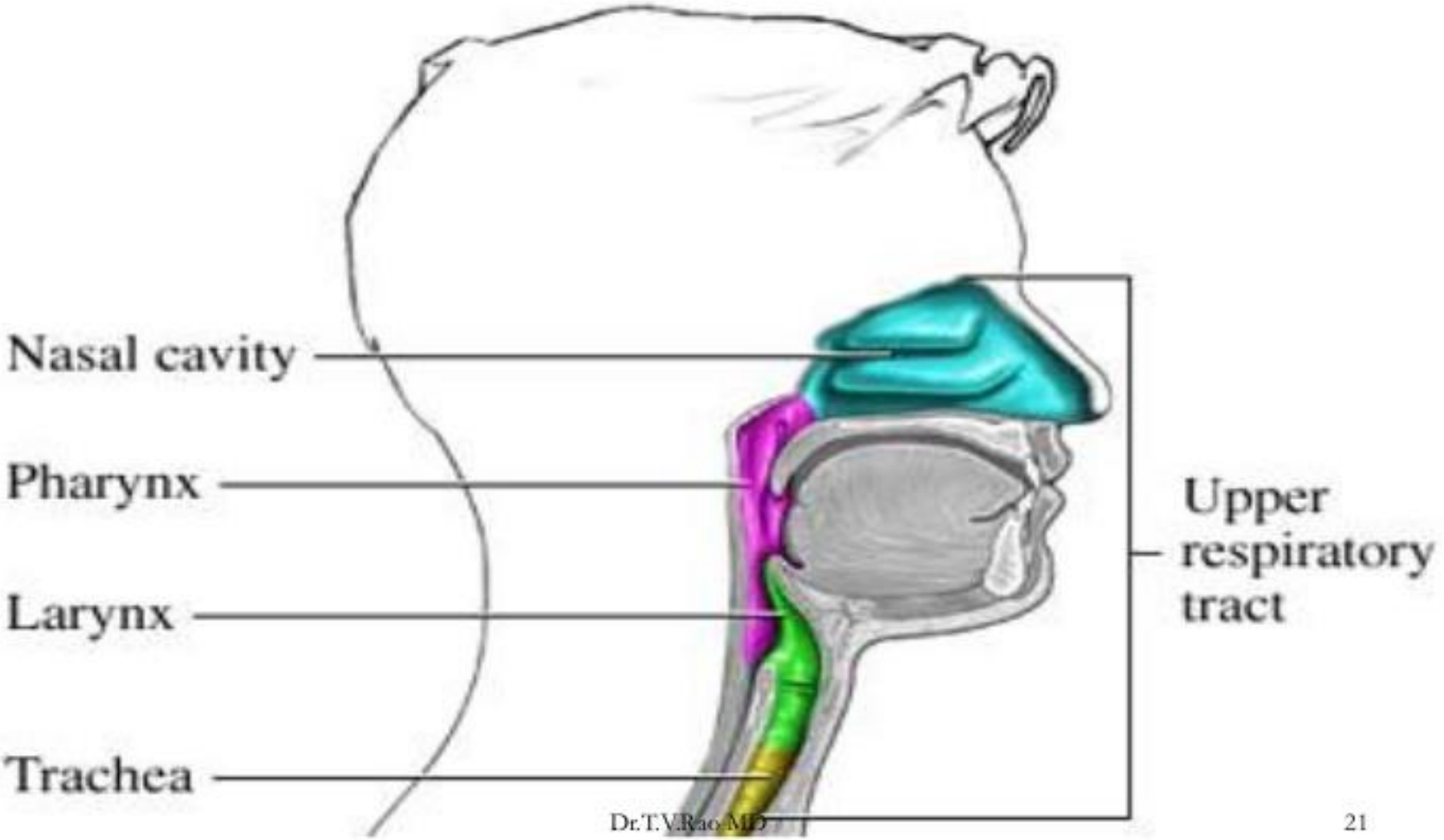
D. Other diseases:

E. Adenoviral infections of the immune compromised host

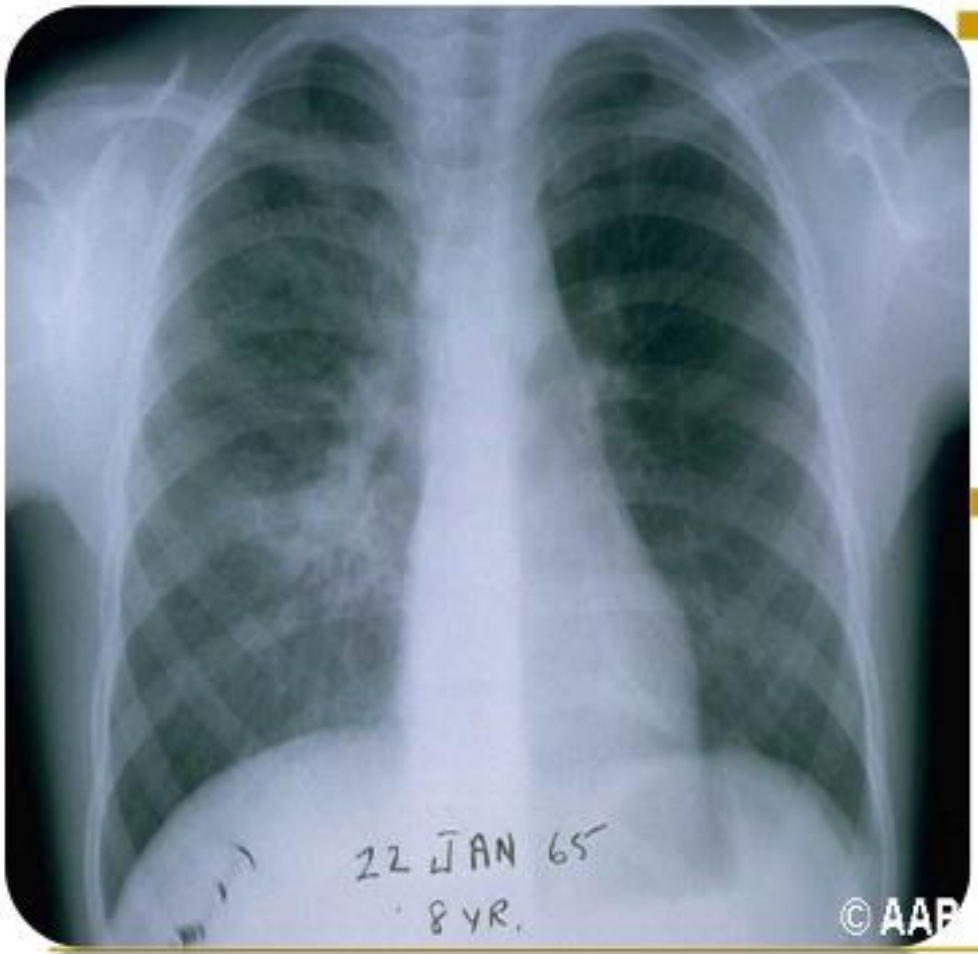
Adenovirus - Pharyngitis

- Major cause of infections associated with nonbacterial pharyngitis and tonsillitis
- Causes febrile common cold
- Types 1 – 7 are common types

Adenovirus Common cause of Respiratory infections



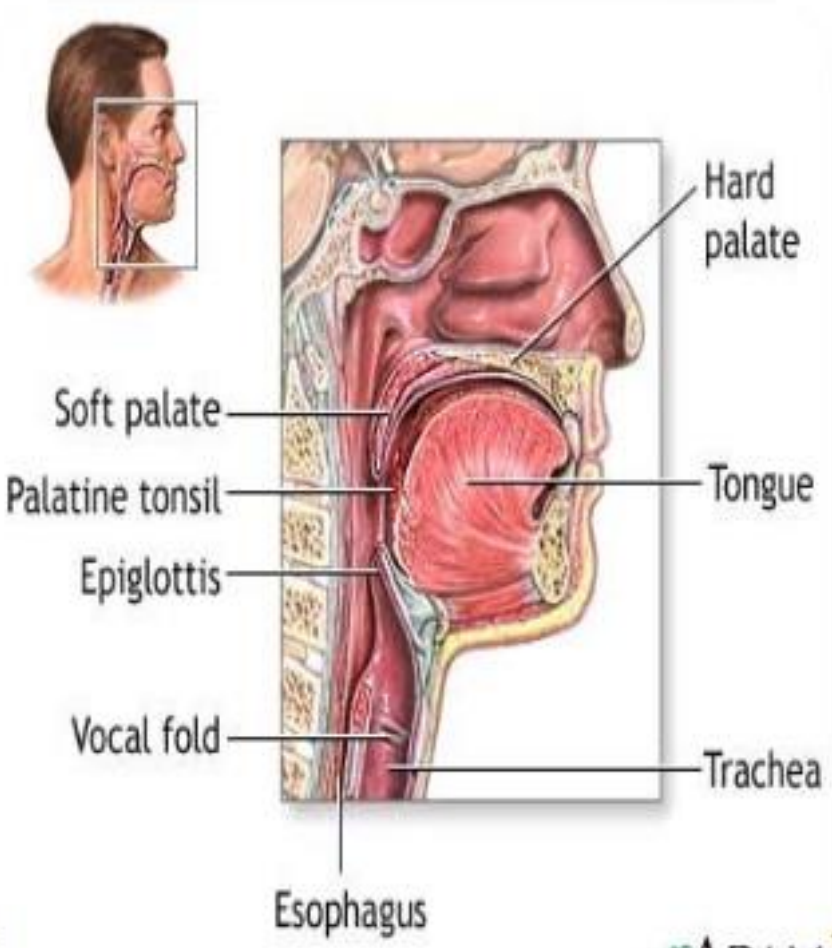
Pneumonia - Adenovirus



- Adenovirus types 3 and 7 are associated with pneumonia resembles like atypical pneumonia in adults.
- Type 7 causes serious and even fatal pneumonia in infants and young children

ARD – Acute respiratory disease with Adenovirus

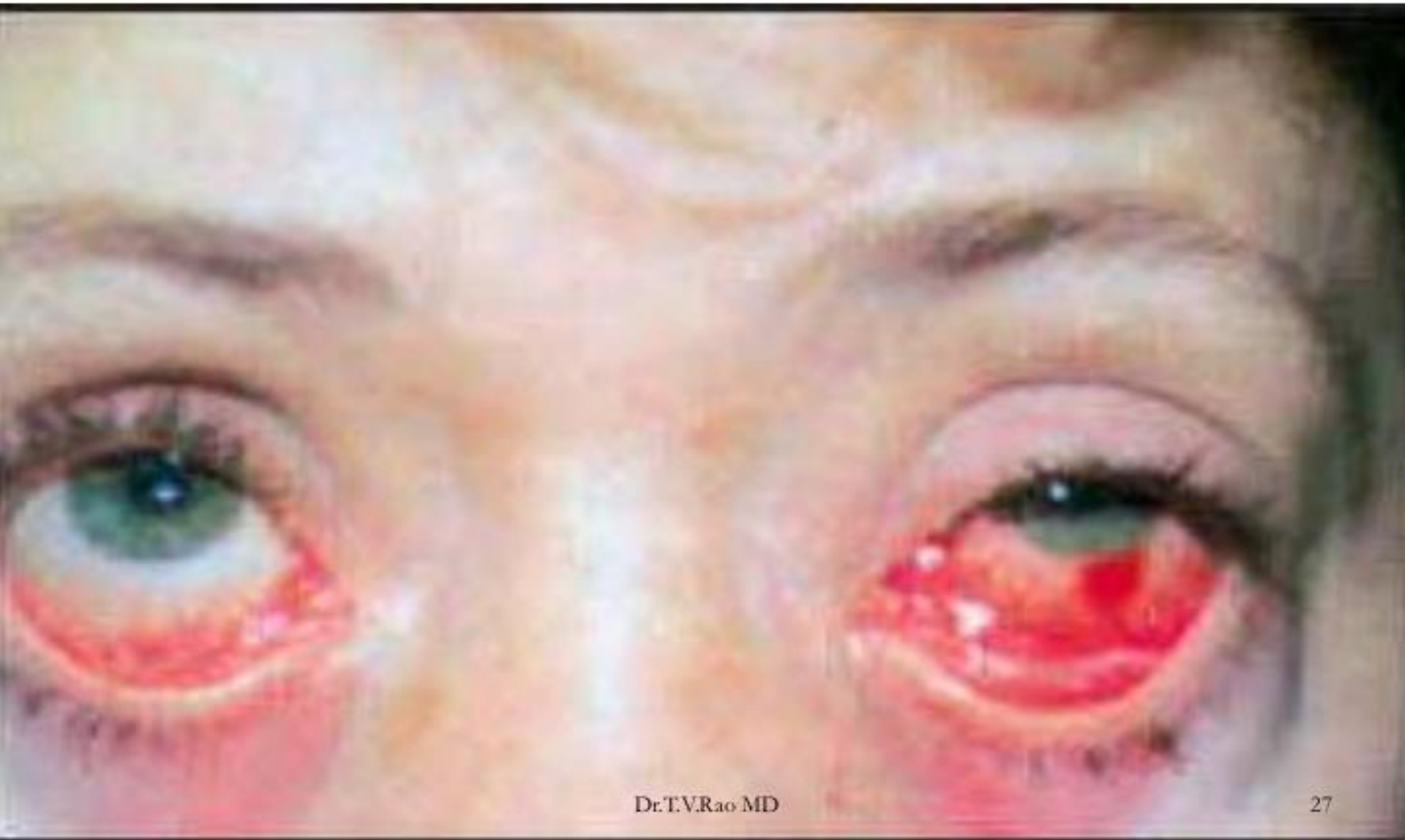
- Occurs usually in military recruits
- Serotypes 4,7,and 21 are agents commonly associated.



Epidemic keratoconjunctivitis

- Occurs with serious epidemic
- Caused by serotypes 8, and less frequently with 19, and 37.

A severe Adenoviral Infection



C. Gastrointestinal disease:

No disease association

- Many Adenoviruses replicate in intestinal cells and are **present in the stools withou being associated with GIT disease**.

Infantile gastroenteritis

- **Two serotypes (40, 41)** have been etiologically associated with infantile gastroenteritis.

CORONAVIRUS

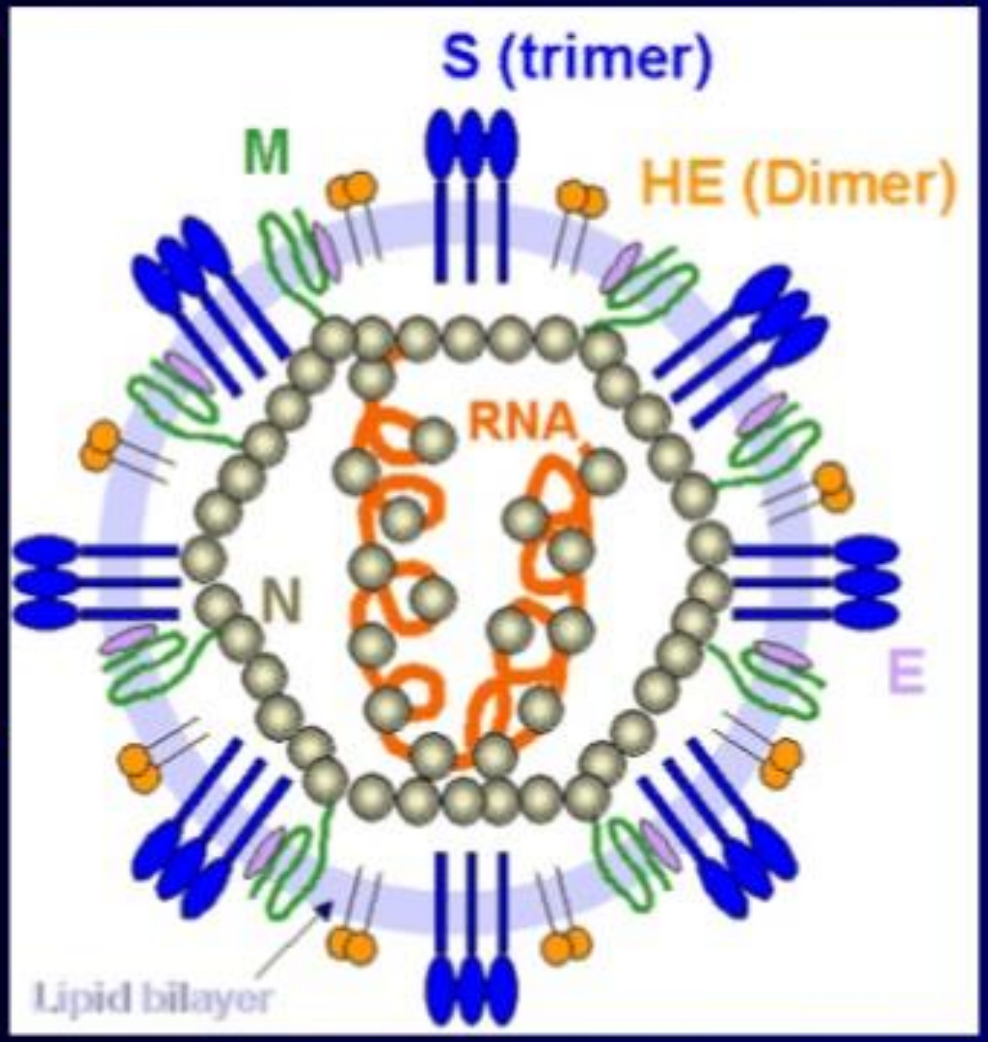
- Single stranded RNA
- Pleomorphic, 80 to 160 nm in diameter
- subfamilies *Coronavirinae* and *Torovirinae* in the family *Coronaviridae*, in the order Nidovirales.[1][2]
- Coronaviruses are enveloped viruses with a positive-sense RNA genome and with a nucleocapsid of helical symmetry

CORONAVIRUSES

The genome

- SS linear non segmented +ve sense RNA

- the largest among RNA viruses.

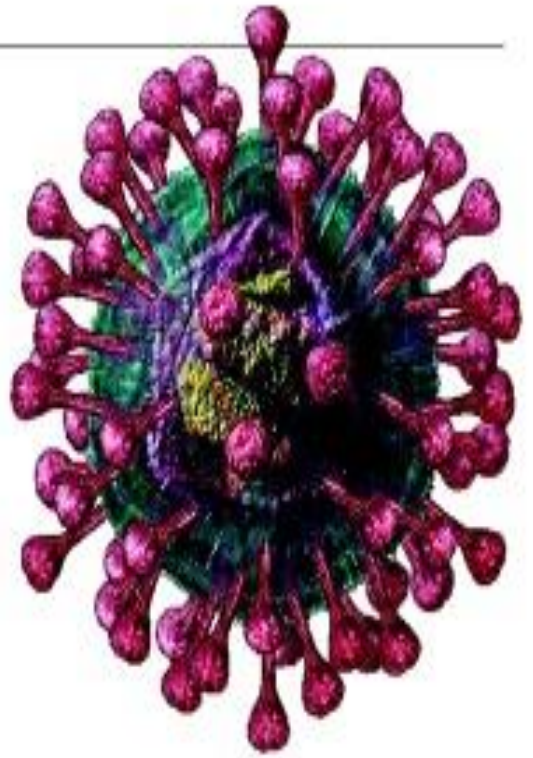


HUMAN CORONAVIRUS

- Human coronavirus were first identified in the mid-1960s
- Six coronavirus that can infect people are
- Alpha coronaviruses 229E
- Beta coronaviruses NL63
- Beta coronaviruses OC43
- Beta coronaviruses HKUI
- Beta coronaviruses SARS-CoV(causes severe acute respiratory syndrome)
- Beta coronaviruses MERS-CoV(Middle east respiratory syndrome)

Classification :

- Family: Coronaviridae
- • Gender: Coronavirus
- • Genome: linear single-stranded RNA +
- • pleomorphic, Wrapped
- • 80 to 220 nm
- • 30 serotypes



Characteristics:

- Protein S. Form projected responsible for stimulating neutralizing antibody and interaction with cellular receptors.
- Bind to cells via specific receptors.
- Enter through membrane fusion.
- In the cytoplasm, the viral RNA is translated by the host machinery.
- Viral proteins are synthesized, assembled, are fused to the cell membrane and the virus out.

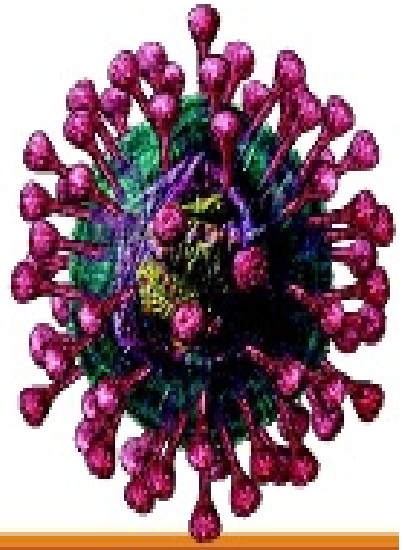
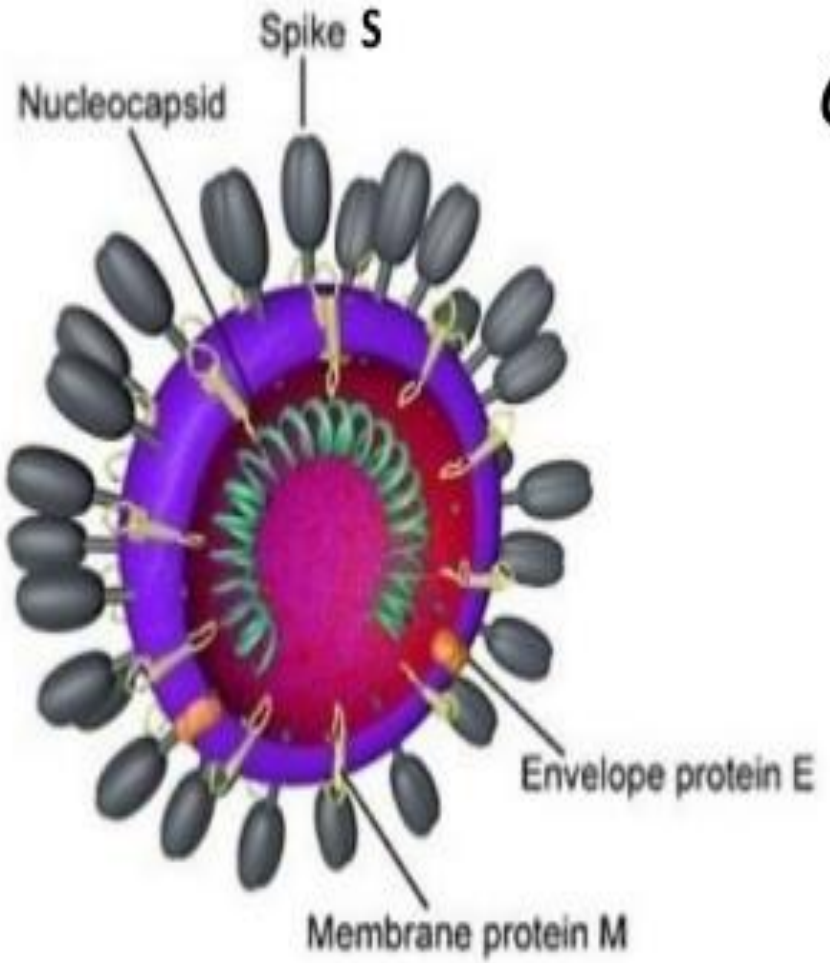
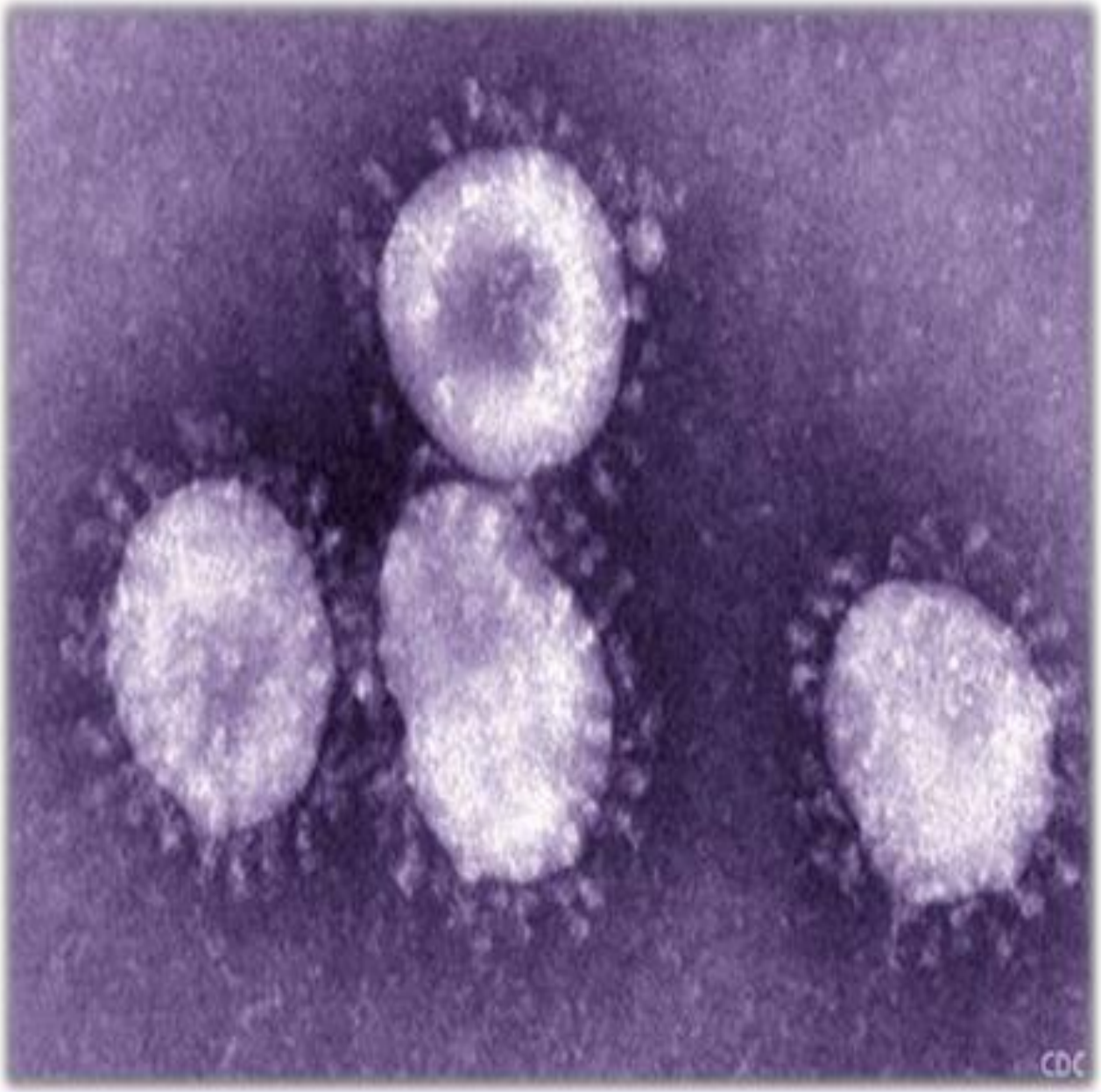


Diagram of **Coronavirus Virion**

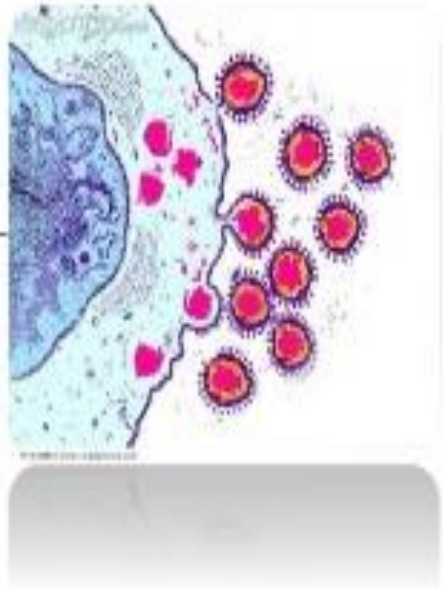




❖A **picture** of a coronavirus as seen through an "electron microscope"

Infection:

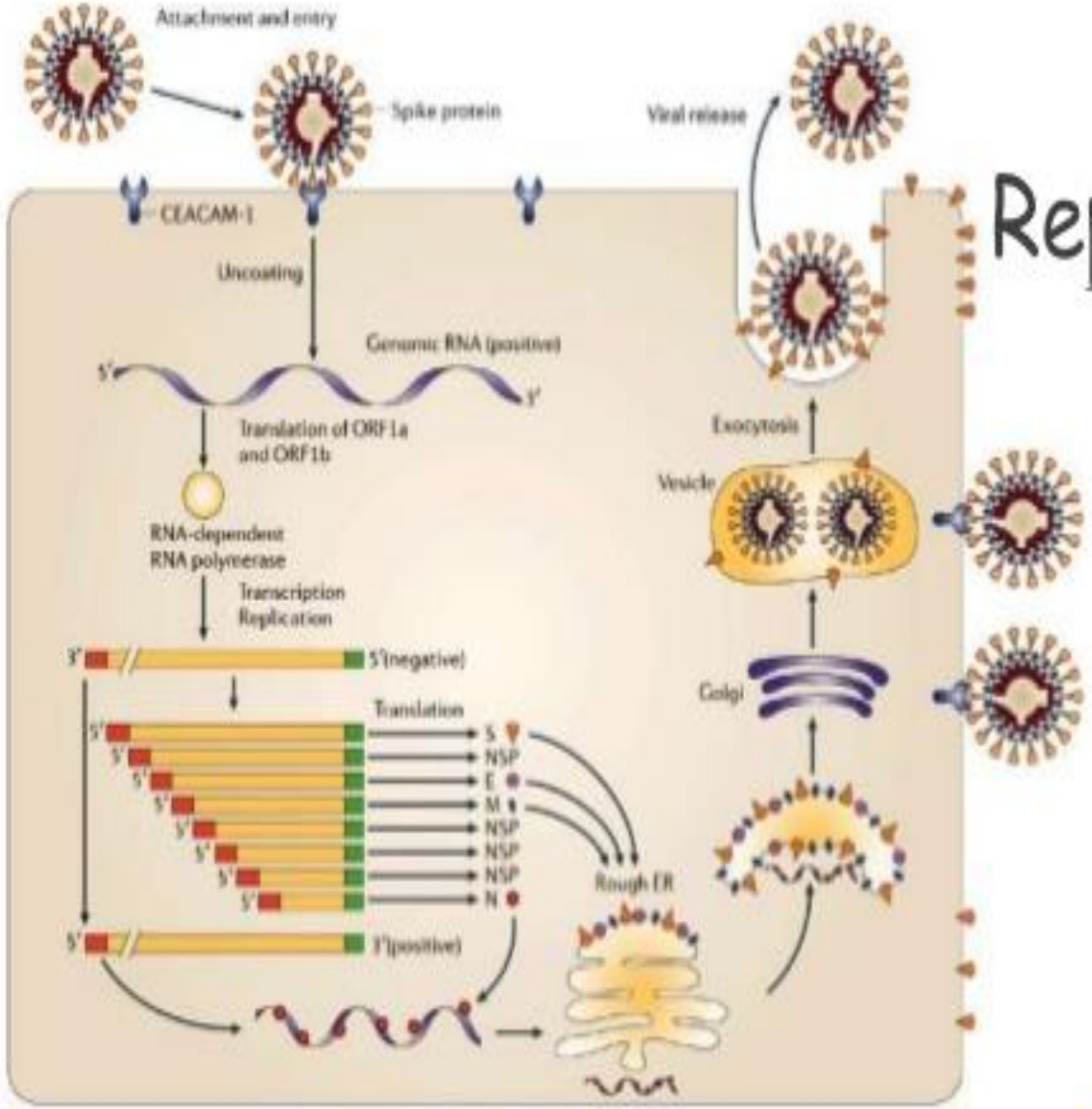
- They are transmitted by aerosols secretions respiratory.
- It has an incubation period of 2 days to a week, eliminating virus for one week.



Infection:

- The virus replicates in the cytoplasm of the cell host, mend in the endoplasmic reticulum and pass the Golgi cisternae until they are finally released by exocytose.
- Are responsible for up to 15% of colds, taking more often in the winter and beginning of the spring with a characteristic periodicity of 2-4 years

Replication:



PATHOGENESIS/SYMPTOM

- It causes mild to moderate upper-respiratory tract illnesses of short duration
- It shows severe symtoms in people with cardiopulmonary disease or compromised immune system, or the elderly
- Symtoms
- Runny nose
- Cough
- Sore throat
- Fever
- Lower respiratory tract illness, such as pneumonia
- MERS-CoV and SARS-CoV causes severe illness

Laboratory Diagnosis

ELISA

PCR

Rhinoviruses are ..

- Rhinoviruses are the most commonly isolated viruses from persons with mild upper respiratory illness.
- Rhinoviruses are a genus of picornaviridae
- In contrast to enteroviruses they do not replicate in the intestinal tract, they have an extreme species specificity and more fastidious growth requirements

RHINOVIRUS GROUP

- Produces Common Cold.
  - Mild respiratory Illness.
- More than 100 serotypes
- Nasal secretions are infective.

Mistaken with Infections with

Corona viruses,

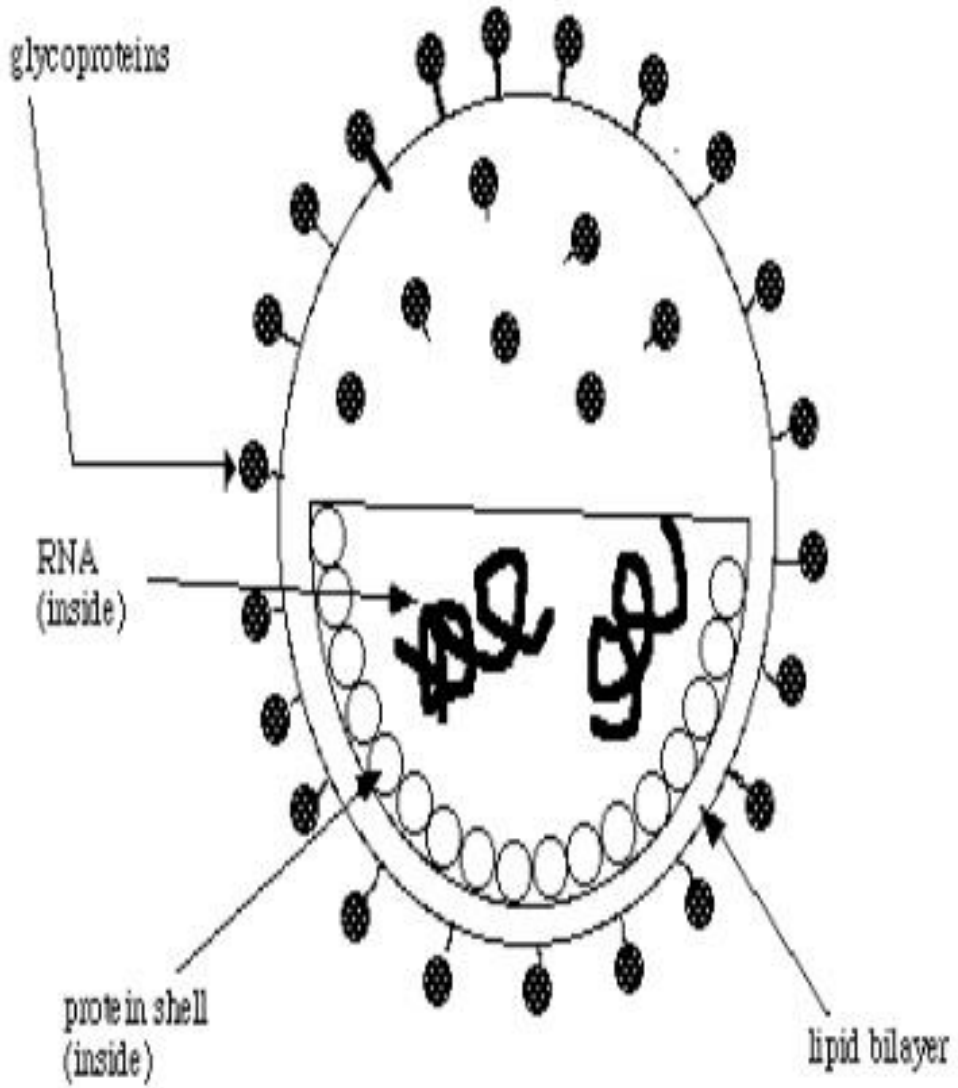
Adenovirus.

Para influenza viruses.

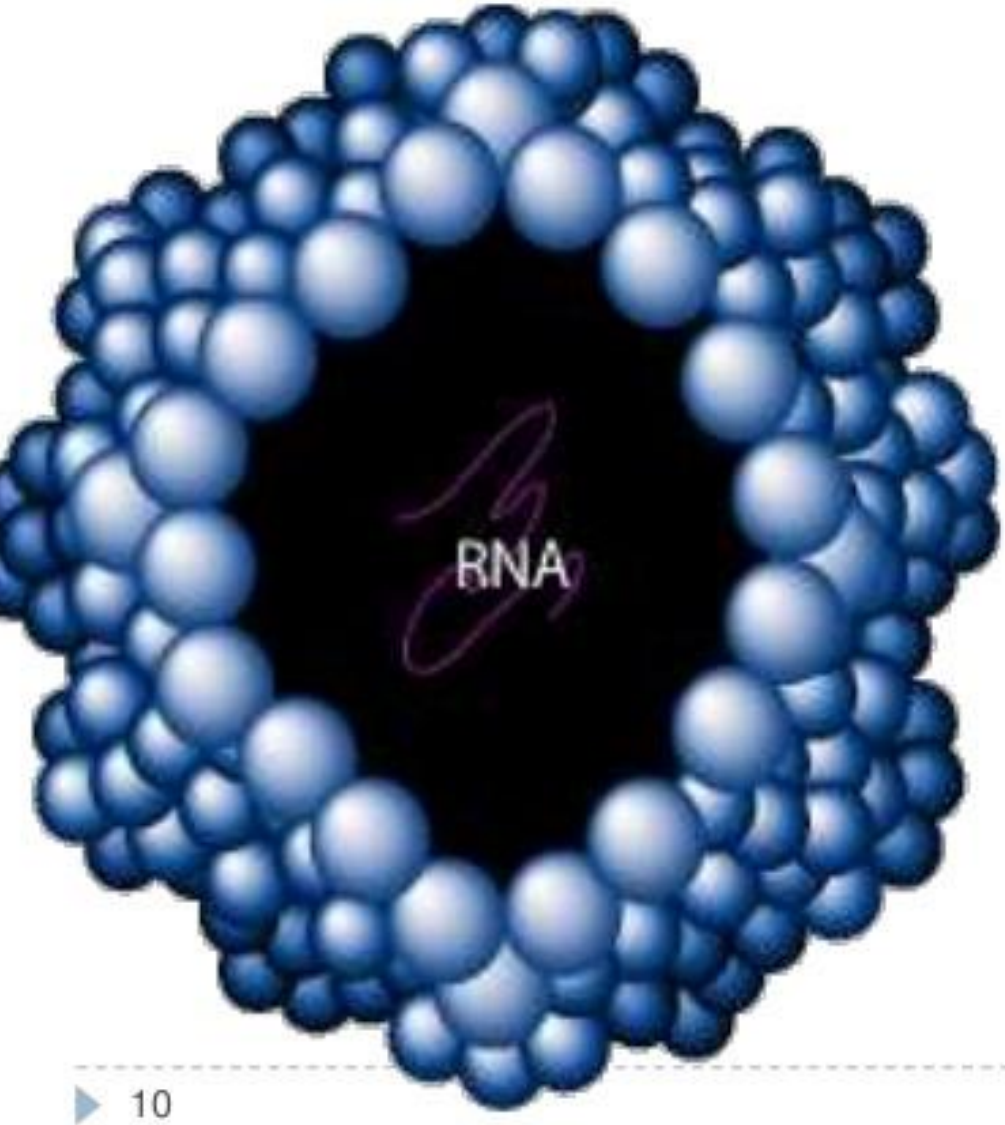
Influenza viruses

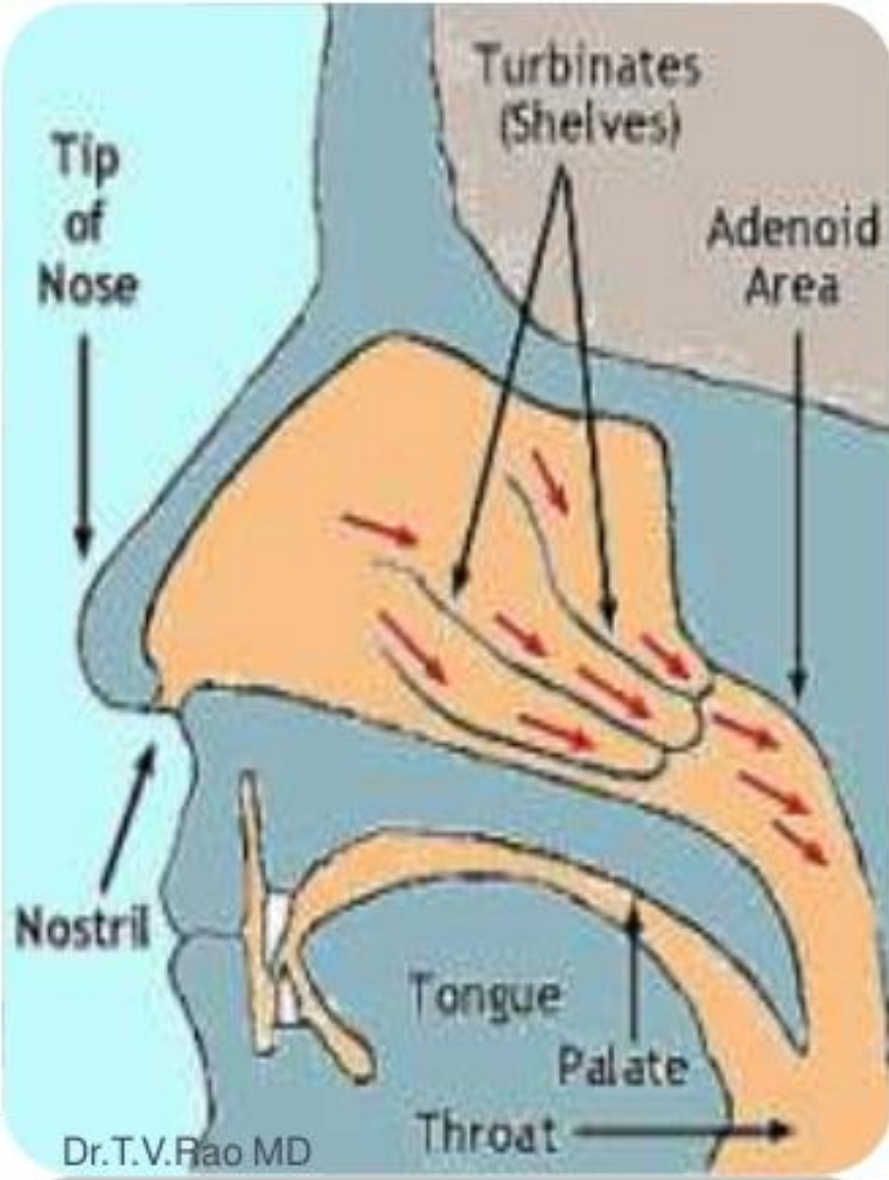
Rhinovirus

- Picornoviridae family
- Size
- Single stranded
- Incubation period of 1 to 3 days
- Optimum growth occurs between 33 and 34 deg Celsius (93 deg F)
- Not stable below the pH of 5-6

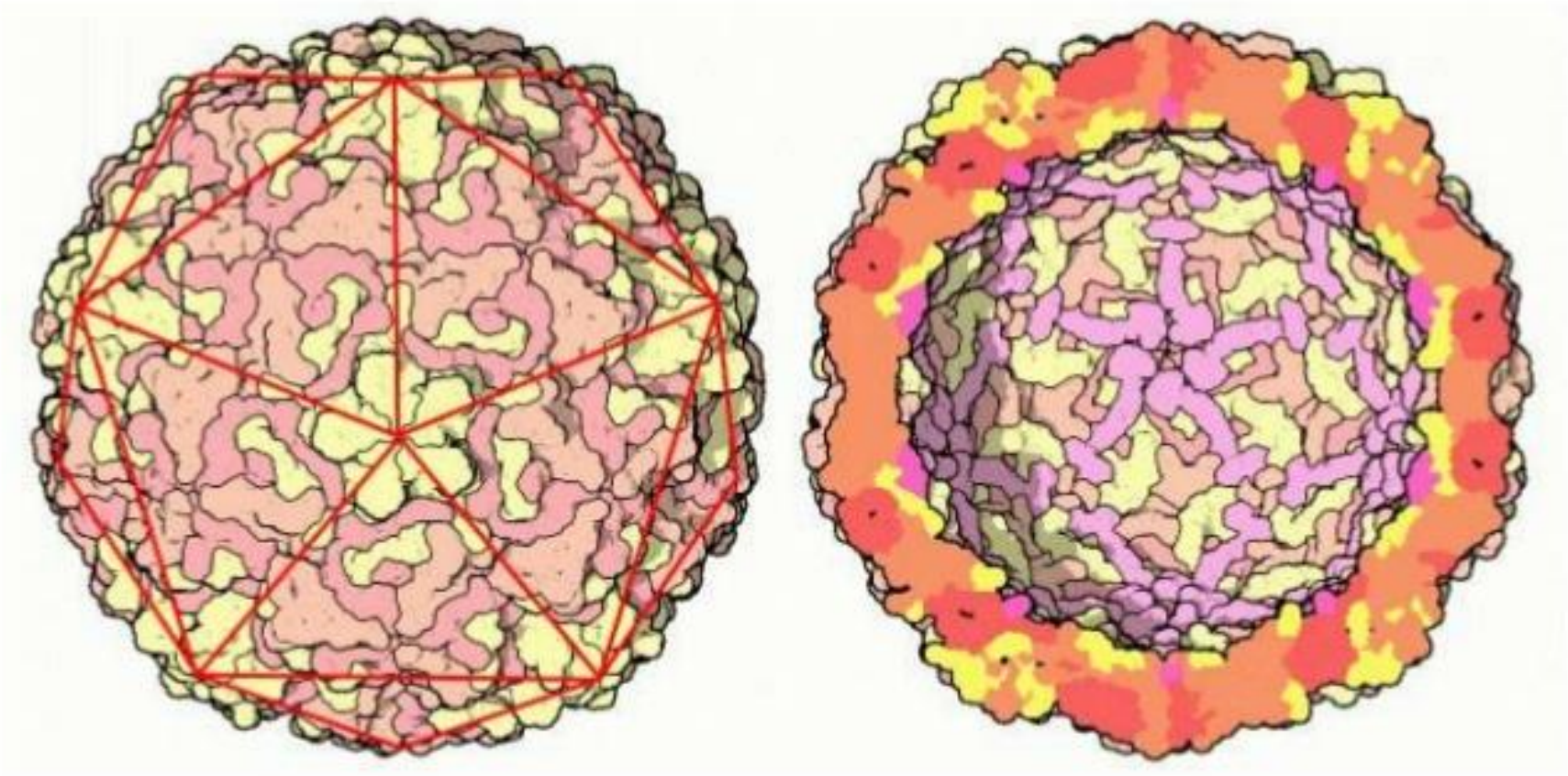


Rhino Virus





Structure



Pathogenesis - Rhinoviruses

- Entry through Respiratory tract.
- Nasal Mucosa, can infect Lower Respiratory tract.
- Chilling, wearing wet cloths do not produce infection.
- But common cold starts with chills.
- Local inflammation and cytokines may be responsible for the symptoms of common cold.
- Interferon production occurs early and specific antibody appears in nasal secretions