Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
B19 (A parvovirus, member of the erythrovirus genus)	-ssDNA -Naked -capsid is made of vp1 and vp2(major)	1–2 weeks but may extend to 3 weeks	• **-Erythema infectiosum** (-**5th disease-**, cutaneous rash in children (slapped cheek appearance) arthralgia-arthritis in adults) • **Transient aplastic crisis** (severe acute anemia) • **Purple red cell aplasia** (chronic anemia) • **Hydrops fetalis** (fatal anemia)	-PCR, probe hybridization of serum or tissue extracts, and in situ hybridization of fixed tissue -Serologic assays: IgM indicates recent infection; IgG indicates past infection.	-Fifth disease and transient aplastic crisis are treated symptomatically -No antiviral drug therapy -No vaccine -Antibodies are mainly against vp2	• 5th disease symptoms: -1st phase: fever, malaise, myalgia, chills, and itching coinciding with viremia and reticulocytopenia and with detection of circulating IgM–parvovirus immune complexes. -2nd Phase: appearance of an erythematous facial rash and a lacelike rash on the limbs or trunk may be accompanied by joint symptoms, especially in adults. Specific IgG antibodies appear about 15 days post-infection. -Infects progenitor erythrocytes because it can interact with P antigen found on their surfaces -transmission: respiratory droplets, blood, vertically (torch agent).
Bocavirus (A parvovirus)	-ssDNA -Naked		• Prevalent among children with **acute wheezing** • Has been detected in about 3% of stool samples from children with **acute gastroenteritis**	PCR	-No treatment for human bocavirus infections -No antiviral drug therapy -No vaccine	-parvoviruses can't be cultured -During blood transmission we make sure it's parvo-negative
Herpes simplex virus type 1	-dsDNA -Enveloped	~3–5 days, range of 2–12 days.	• **Cold sores** (fever blisters) near the lip • Fever, sore throat, vesicles, ulcers and **gingivostomatitis**	-PCR -Virus isolation (using Giemsa's stain) -Enzyme immunoassays	-Treatment: acyclovir, valacyclovir, and vidarabine (inhibit DNA synthesis)	-Spread by contact, usually involving infected saliva -Site of latency: neurons (trigeminal ganglia)

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
HSV 1 (Cont.)	(enveloped derived from the nuclear membrane)		• **pharyngitis and tonsillitis.** • Localized **lymphadenopathy** may occur • May cause severe **keratoconjunctivitis** • Progressive involvement of the corneal stroma, can cause **permanent opacification and blindness** • **Eczema herpeticum** in a person with chronic eczema	-Serologic assays (limited by the multiple antigens shared by HSV-1 and HSV-2) -Dendritic ulcers are pathognomonic of HSV keratitis		-Primary HSV infections are mostly asymptomatic or mild -HSV-1 infections are the second cause of corneal blindness in the US -Can also cause clinical episodes of genital herpes. - HSV-1 is the most common cause of sporadic, fatal encephalitis in US, with high mortality rate, and those who survive often having residual neurologic defects. About half of patients with HSV encephalitis appear to have primary infections, and the rest appear to have recurrent infection
Herpes simplex virus type 2	-dsDNA -Enveloped		• Primary genital herpes infections can be severe (very painful and associated with fever, dysuria, and inguinal lymphadenopathy). • Genital herpes is characterized by vesiculoulcerative lesions of the penis of the male or of the cervix, vulva, vagina, and perineum of the female.	-PCR -Virus isolation (using Giemsa's stain) -Enzyme immunoassays -Serologic assays (limited by the multiple antigens shared by HSV-1 and HSV-2)	-Treatment: acyclovir, valacyclovir, and vidarabine (inhibit DNA synthesis)	-Transmitted sexually or from a maternal genital infection to a newborn -Site of latency: neurons (sacral ganglia) -Immunocompromised patients are at increased risk of developing severe HSV infections - Complications include extragenital lesions (~20% of cases) and aseptic meningitis (~10% of cases) -Torch agent

Note! Herpes viruses bind to heparan sulfate

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Varicella-zoster virus (type 3)	-dsDNA -Enveloped		• **Varicella** (chickenpox); primary response • **Zoster** (shingles); reactivation of VZV • **Encephalitis** occurs in rare cases and can be life threatening -**Neonatal varicella** can be fatal -**Varicella pneumonia** (rare in healthy children), responsible for VZV related deaths -can cause pneumonia		-Previous infection with varicella can confer lifelong immunity to varicella -A live attenuated varicella vaccine is available	-Site of latency: neurons -Complications are rare in normal children, and the mortality rate is very low -Varicella spreads by airborne droplets and by direct contact
Cytomegalovirus (type 5)	-dsDNA -Enveloped	4-8 weeks incubation period	-Most common cause of **congenital infections** -An infectious **mononucleosis-like syndrome (triad: fever, pharyngitis, cervical lymphadenopathy)** -Causes disseminated disease in untreated AIDS patients**; gastroenteritis and chorioretinitis** are common problems, the latter often leading to progressive blindness -most common complication is pneumonia	-PCR -Virus isolation -Serologic detection (not informative for immunocompromised people)	-**Treatment**: -Ganciclovir to treat life-threatening CMV infections in immunosuppressed patients -CMV retinitis: Foscarnet - Acyclovir and valacyclovir have shown some benefits in bone marrow and renal transplant patients	-Most CMV infections are subclinical -A high percentage of babies with this disease will exhibit developmental defects and mental retardation -Site of latency: glands, kidneys (causes retinitis)

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Human herpesvirus 6	-dsDNA -Enveloped		-Causes roseola infantum (sixth disease)			- T-lymphotropic (The virus grows well in CD4 T lymphocytes) -Site of latency: lymphoid tissue -Transmitted via oral secretions -Receptor: CD46
Human herpesvirus 7	-dsDNA -Enveloped		-Causes roseola infantum (sixth disease)			-Site of latency: lymphoid tissue -Receptor: CD4
Epstein Barr Virus (type 4)	-dsDNA -Enveloped	30–50 days	-Infectious **Mononucleosis** (triad: fever, pharyngitis, cervical lymphadenopathy) (headache, fever, malaise, fatigue, and sore throat occur. Enlarged lymph nodes and spleen are characteristic. Some patients develop signs of hepatitis) -Cancer: EBV is associated with **Burkitt lymphoma, nasopharyngeal carcinoma, Hodgkin and NHLs, and gastric carcinoma** -**Oral hairy leukoplakia** -**Pharyngitis**	- NAH is the most sensitive means of detecting EBV in patient materials -Hybridization (EBER RNA) -Serologic detection		-During infectious mononucleosis, there is an increase in the number of circulating white blood cells, with a predominance of lymphocytes. Many of these are large, atypical T lymphocytes - Low-grade fever and malaise may persist for weeks to months after acute illness -Site of latency: lymphoid tissue -heterophile antibodies -Main target cells are B lymphocytes (CD21) receptors of CR2

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Human herpesvirus 8 (KSHV)	-dsDNA -enveloped		-The cause of **Kaposi sarcomas, vascular tumors** of mixed cellular composition -Patients have cachexia	- Serologic assays are available to measure persistent antibody to KSHV using indirect immunofluorescence, Western blot, and ELISA formats	- Foscarnet, famciclovir, ganciclovir, and cidofovir have activity against KSHV	- KSHV was first detected in Kaposi sarcoma specimens - It is lymphotropic and is more closely related to EBV -Site of latency: lymphoid tissue -Involved in the pathogenesis of body cavity-based lymphomas (primary effusion lymphoma) occurring in AIDS patients and of multicentric Castleman disease -transmission: oral secretions, sexually, vertically.
Herpes B virus					-No specific treatment -Treatment with acyclovir is recommended immediately after exposure. -No vaccine	-It is designated cercopithecine herpesvirus 1, replacing the older name of Herpes simiae -From monkeys to humans
Adenoviruses	-dsDNA -Naked		-**Follicular conjunctivitis** -**Keratoconjuctivitis** **-Acute febrile pharyngitis** **-Acute respiratory disease** **-Viral pneumonia** **-Infantile gastroenteritis** (Ad40, Ad41): long-lasting diarrhea, less frequent vomiting, frequent development of dehydration, and	-Ag detection or PCR	-No specific treatment for adenovirus infections -Live adenovirus vaccine containing types 4 and 7 is available -Careful hand washing is the easiest way to prevent infections	-Adenoid tissue (tonsils) -Infect and replicate in epithelial cells of the respiratory tract, eye, gastrointestinal tract and urinary bladder -Cause localized infection and do not spread systemically -Respiratory disease (Ad1, 2, 3, 5, and 7), are transmitted via the respiratory route -The GI disease is transmitted via the fecal-oral route

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Adenoviruses (Cont.)			abdominal pains and distension. -**Hemorrhagic cystitis** (Ad11): gross hematuria			-Ocular infections are transmitted by direct inoculation of the eye
Polyomaviridae (BKV, JCV, Merkel cell polyomavirus)	-dsDNA -Naked		-BK virus: **Hemorrhagic cystitis** -JC virus: **Progressive multifocal** leukoencephalopathy (PML) -MCPyV: **Merkel cell carcinoma**	-Definitive diagnosis of BKV requires a biopsy to be taken for histopathology, BKV-specific qPCR in plasma or urine -MRI is the imaging modality of choice if PML is suspected. The confirmatory test for suspected PML is the demonstration of JCV DNA in the CSF or brain by PCR	-There is no antiviral therapy specifically licensed for the treatment of either JCV or BKV infections -MCC: Surgery, radiotherapy and chemotherapy	-Genus alphapolyomavirus -Able to establish latency -Primary polyomavirus infections are presumably subclinical -BKV and JCV are known to persist in the reno-urinary tract
Hepatitis A	-ssRNA -Naked	10–50 days (average, 25–30)	-Onset is abrupt (sudden) and fever is common -Complications are uncommon, no chronic state -Patients with inapparent or subclinical hepatitis **have neither symptoms nor jaundice.** -Other patients can develop **anicteric hepatitis or icteric hepatitis.**	-Occurrence of virus in blood: 2 weeks before to ≤1 week after jaundice -Occurrence of virus in stool: 2 weeks before to 2 weeks after jaundice. -Duration of aminotransferase elevation: 1–3 weeks	-No specific treatment for acute viral hepatitis exists, and hospitalization is not ordinarily indicated. -Formaldehyde inactivated vaccines are available worldwide.	-Family: picornaviridae -Genus: hepatovirus -Transmison: fecal-oral -HAV is stable at low pH, heating -Only one serotype is known -A relatively high concentrations of HAV are shed in the feces before the alanine aminotransferase (ALT) level initially becomes elevated and before the onset of clinical symptoms or jaundice

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Hepatitis A (Cont.)			-Most patients recover completely; however, some develop **fulminant hepatitis and die**	-Liver function tests, IgM anti-HAV		-Therapy should be supportive and aimed at maintaining comfort and adequate nutritional balance -Peaks in autmun
Hepatitis B	-dsDNA -Enveloped		-Can cause both acute and chronic infections -**Fulminant** hepatitis can follow acute infection -Could cause cancer (oncogenic)	-Viral DNA in the liver and plasma together with circulating HBsAg. -High levels of viremia is followed by rise in the level of markers of hepatocyte damage (mainly ALT) and the appearance of clinical features (fever, malaise and jaundice) -HBcAb appears within the first two weeks after the appearance of HBsAg and preceding HBsAb -NAT is also available for screening blood/blood products	-Treatment: IFNs and several nucleotide and nucleoside analogs -For prevention of HBV infection, an effective vaccine (recombinant HBsAg) is available	-Family: hepadnaviridae -Genus: orthohepadnavirus -Transmission: percutaneous (major route) , sexual spread and MTCT -hepatocyte damage occurs as a result of T cell mediated immune attack on hepatocytes expressing HBV antigens on the context of their HLA molecules -HBsAg becomes undetectable 1–2 months after the appearance of jaundice -The persistence of HBsAg beyond 6 months marks HBV chronicity -Clearance is associated with the appearance of HBsAb

Note! Treatment of chronic hepatitis infection: INF-a and ribavirin

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Hepatitis C	-ssRNA -Enveloped (Genome can be viewed as an ORF)		-Could cause cancer (oncogenic)	-Diagnosis relies on nucleic acid testing which is also used to monitor response to treatment	-No vaccine -Therapeutic option: direct-acting antivirals (DAAs).	-Family: flaviviridae -Genus: Hepacivirus -Transmission: parenteral, the major route of HCV transmission worldwide is the exposure to contaminated blood mainly through IDU particularly in the high-income countries -lower-risk modes of transmission include high risk sexual behaviour, vertical transmission, health-care associated infections, intrafamilial spread, tattooing, piercing and acupuncture. -HCV receptors: CD81, claudin, occludin and scavenger receptor class b type I.
Hepatitis D	-ssRNA -Enveloped (internal neuclocapsid surrounded by delta antigens enveloped by coat of HBsAG)			-Intrahepatic HDV Antigen or anti-HDV Ab. Circulating HDV antigen -Tests for HDV RNA to determine ongoing HDV replication and relative infectivity	-Delta hepatitis can be prevented by vaccinating HBV susceptible persons with hepatitis B vaccine	-Genus: deltavirus -Transmission: parenteral -is known to be defective and require a helper function from HBV for its transmission
Hepatitis E	-ssRNA -Naked	2 weeks to 2 months	-The most common cause of **acute hepatitis** -Causes mortality in pregnant women	-Individual cases of hepatitis E cannot be differentiated from other cases of hepatitis on the basis	-No specific treatment exists for acute hepatitis E.	-Family: hepeviridae -Genus: hepevirus -Transmission: fecal-oral

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Hepatitis E (Cont.)				of clinical presentation -Diagnosis can be achieved using serology and RT-PCR.	-Candidate recombinant vaccines in trials.	
Variola virus	-dsDNA -Enveloped	The incubation period was 10–14 days	-Was the cause of **smallpox** -One to 5 days of fever and malaise preceded the appearance of the exanthems, which began as macules, then papules, then vesicles, and finally pustules -The case-fatality rate reached 40%		-Smollpox was eradicated by a vaccine (Vaccinia vaccine)	-A poxvirus: only viruses that replicate in the cytoplasm -Poxviruses are the only viruses that replicate in the cytoplasm -Are transmitted aerogenically (highly contagious) - Deaths were related bleeding, cardiovascular collapse, and secondary infections
Molluscum contagiosum virus (MCV)	- dsDNA -Enveloped		-Causes **Molluscum contagiosum** (small, discrete, skin-coloured, dome-shaped papules)	-Diagnosis is based on observation of the characteristic umbilicated papules (punctum) - PCR	-Treatment: Physical therapy (curettage, cryotherapy), chemical agents or antiviral therapy (Cidofovir)	-A poxvirus -Transmission occurs by direct contact, through contaminated fomites, or sexual activity -Cidofovir is a DNA polymerase inhibitor -Portal of entry: mucosa of the upper respiratory tract
Human-papillomavirus	-dsDNA -Naked		- **Oral Squamous Papilloma** (HPV6, HPV11, and HPV16) - **Oral Verruca Vulgaris** -Common wart-, (caused by HPV types 1, 2, 4 and 7) - **Oral Condyloma Acuminatum** (HPV 2, 6, and 11)	- Visual inspection. -Pap smear	-Treatment of warts generally involves surgical removal or mechanical destruction of the wart tissue with: Liquid nitrogen,	-Family: papillomaviridae -Genus: Alphapapillomavirus -Transmission: Sexual, indirect& direct contact - Tropism: Epithelial cells of skin, mucous membranes -Receptor: heparin sulfate - Oncovirus

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
			- Focal epithelial hyperplasia (**heck disease**, HPV13 or HPV32) - **Oropharyngeal cancer** -**Skin warts** (HPV types: 1, 2, 3, 4, 7 among others) - **Laryngeal papillomatosis** (HPV-6 or HPV-11) - **Cervical Cancer** (HPV-16 or HPV-18, sometimes HPV-31 or HPV-33 as well) - **Anogenital Warts** - **condylomas**-(HPV6 or HPV11)		Laser vaporization, Cytotoxic chemicals such as podophyllin or trichloroacetic acid - A quadrivalent HPV vaccine (HPV types 6, 11, 16, and 18) -A bivalent vaccine (types 16 and 18)	- HPV16 accounts for about 90% of the HPV-positive tumors - Genital HPV infection is the most common sexually transmitted viral infection - High-risk HPV types can cause vulvar, vaginal, penile or anal cancer - Pap smear screening has decreased the frequency of cervical cancer in industrialized countries -Vaccines contain HPV L1 proteins -Able to establish latency
Enteroviruses	-ssRNA -Naked		-Can cause CNS disease; major cause of acute aseptic meningitis -EV 70 is a main cause of acute hemorrhagic conjunctivitis EV70 and 71 are associated with severe CNS disease -EV71 is associated with HFM disease			-A picornavirus -Infection by ingestion of contaminated food or water or via respiratory droplets - Stable at the low pH of the stomach, replicate in the GI tract, and excreted in the stool (fecal-oral spread)
Rhinovirus	-ssRNA -Naked	from 2 to 4 days	-The common cold viruses -Symptoms in adults include sneezing, nasal obstruction, nasal discharge, and sore throat; other symptoms may include headache, mild	-No distinctive clinical findings that permit an etiologic diagnosis of colds caused by rhinoviruses versus colds caused by other viruses	-No specific prevention method or treatment is available	-A picornavirus -Rhinoviruses are acid-labile. -Replicate in the nasal passages (replicate efficiently at temperatures several degrees below body temperature) -Cellular receptors: (ICAM-1)

Note!

Aseptic meningitis is mainly cause by enteroviruses, echoviruses and coxaschieviruses (T cell mediated)

Septic meningitis involves neutrophils and is mainly caused by strep. pneumonia

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
			cough, malaise, and a chilly sensation. -There is little or no fever -The nasal and nasopharyngeal mucosa become red and swollen			-Secondary bacterial infection may produce acute otitis media, sinusitis, bronchitis, or pneumonitis, especially in children
Poliovirus	-ssRNA -Naked	usually 1-2 weeks, but it may range from 3 to 35 days	-**Mild disease** (fever, malaise, drowsiness, headache, nausea, vomiting, constipation, and sore throat) -**Nonparalytic poliomyelitis (aseptic meningitis)** **-Paralytic poliomyelitis** and **postpoliomyelitis muscle atrophy** **-Respiratory paralysis** may occur		-Live attenuated vaccine (given orally and provides Herd immunity) -killed vaccines (not as effective)	-A picornavirus -Poliovirus receptor (PVR; CD155) -The mouth is the portal of entry of the virus, and primary multiplication takes place in the oropharynx or intestine -It is believed that the virus first multiplies in the tonsils, the lymph nodes of the neck, Peyer patches, and the small intestine -Most infections are subclinical, only 1% result in clinical illness -Replicate inside lower motor neurons not inside muscles
Coxsackieviruses **Coxsackieviruses (Cont.)**	-ssRNA -Naked		-**Aseptic meningitis** -**Respiratory and acute febrile illnesses** -Group A causes**:** **Herpangina** (vesicular pharyngitis), **hand-foot-and-mouth disease**, and **acute hemorrhagic conjunctivitis** -Group B causes: **Pleurodynia**, **myocarditis** and **pericarditis**			-Picornaviruses -They are divided into two groups: A and B -The main causes of hand-foot-and-mouth disease are A10, A16 and EV71(Enterovirus 71)

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Echoviruses	-ssRNA -Naked		-**Aseptic meningitis**, **encephalitis**, **febrile illnesses** with or without rash, **common colds**, and **ocular disease**			-A picornavirus
Orthomyxoviruses	-Segmented ssRNA -Enveloped		-Influenza attacks mainly the upper respiratory tract -Symptoms of classic influenza: chills, headache, and dry cough followed closely by high fever, generalized muscular aches, malaise, and anorexia -Influenza C rarely causes the influenza syndrome, causing instead a common cold illness -Complication: pneumonia	-Definitive diagnosis cannot be made on clinical grounds except in an epidemic situation -PCR	-1st -generation antiviral agents effective against influenza A include two related drugs, amantadine and rimantadine (inhibit M2) -2nd -generation antiviral agents effective against influenza A and B include zanamivir and oseltamivir (inhibit viral neuraminidase) -Inactivated viral vaccines are the primary means of prevention of influenza	-They are divided into 3 types: influenza A, B, and C -A and B contain eight separate RNA segments; C contains seven (lacks neuraminidase gene) -The antigenic protein: hemagglutinin (HA) -Cleavage of HA is necessary for the virus particle to be infectious and is mediated by cellular proteases (usually found in the respiratory tract) - Pneumonia, can be viral, secondary bacterial (S. aureus, S. pneumonia) , or a combination of the two -Antigenic drift of influenza A results in epidemic waves of the virus (2–3 years) -Antigenic shift of influenza A (a new subtype) has pandemic results (10-40 years)

Note!

Common cold is localized → Rhinovirus, coronaviruses and Influenza C virus

Flu is systemic → Influenza A and B

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Alphavirus	-ssRNA -Enveloped		-The majority of infections are **subclinical** -**Acute encephalitis** (equine encephalitis viruses) -**Acute arthropathy** (Chikungunya virus) -**A febrile illness** with a flulike syndrome			-Togaviridae family -Transmitted to humans and domestic animals by mosquitoes.
Rubivirus	-ssRNA -Enveloped		-**German measles:** -Infection during early pregnancy causes serious **congenital malformations and mental retardation of fetus** -The consequences of rubella in utero are referred to as **the congenital rubella syndrome (Catarats, deafness and cardiac abnormalities)** -**Transient arthralgia and arthritis** are commonly seen in adults, especially women.	-The disease is difficult to diagnose clinically because the rash caused by other viruses (e.g. enteroviruses) is similar	-One attack of the disease confers lifelong immunity because only one antigenic type of the virus exists -A rubella vaccine is available	-Togaviridae family -Rubella usually begins with malaise, low-grade fever, and a morbilliform (red macules) rash appearing on the same day. The rash starts on the face, extends over the trunk and extremities, and rarely lasts more than 3 days -Rubella antibodies appear in the serum of patients as the rash fades
Paramyxoviruses	-ssRNA -Enveloped		-The most important agents of **respiratory infections** of infants and young children -Causes childhood diseases (mumps and measles)			-All of them initiate infection via the respiratory tract -Divided into two subfamilies: Paramyxovirinae and Pneumovirinae

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Measles virus	-ssRNA -Enveloped		∝ **(Rubeola, First Disease):** -Measles begins with a prodrome of fever, upper respiratory tract symptoms, and conjunctivitis. ¬ A few days later, specific signs develop; first, Koplik spots (small white spots on bright red mucous membranes of the mouth and throat) and then a generalized macular rash ∝ **(Hard Measles, 10-day measles):** -Soon after the rash appears, the patient is no longer infectious. -Complications of infection: pneumonia and encephalitis and the most important of these is post-infectious encephalomyelitis	-Diagnosis can be achieved clinically, especially in an epidemic situation -The presence of Koplik spots provides a definitive diagnosis	-Measles is usually a disease of childhood, and is followed by lifelong immunity (single serotype) -A live attenuated measles vaccine is available.	-A morbillivirus -Subfamily: Paramyxovirinae -The cellular receptor for measles virus is CD46 -Transmitted by sneeze- or cough-produced respiratory droplets -The virus is extremely infectious, and almost all infected individuals develop a clinical illness -Measles virus replicates initially in the respiratory epithelium and then in various lymphoid organs -Morbidity is related to pneumonia and encephalitis
Mumps virus	-ssRNA -Enveloped		-More than one-third of all mumps infections are asymptomatic -Cause mild disease in children -In adults, complications include meningitis and orchitis	- diagnosis revolve around infection and swelling of the salivary glands, primarily the parotid glands	-Immunity is permanent after a single infection -An effective attenuated live-virus vaccine is available	-A rubulavirus - Subfamily: Paramyxovirinae - Mumps is an acute contagious disease involving salivary glands - The virus is spread by respiratory droplets

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Mumps virus (Cont.)			-Orchitis may cause sterility		-Mumps vaccine is in combination with measles and rubella (MMR) live-virus vaccines. -Two doses of MMR vaccine are recommended for school entry	-Infection is widespread in the body and may involve not only the salivary glands but also the pancreas, CNS, and testes
Parainfluenza viruses (PIVs 1-4)	-ssRNA -Enveloped		-The infection may involve only the nose and throat, resulting in a harmless "**common cold**" syndrome -Infection may be more extensive, may involve the larynx and upper trachea, resulting in croup (l**aryngotracheobronchitis**) (1&2) - Primary infection usually results in rhinitis and pharyngitis, often with fever -Bronchiolitis and pneumonia (3) -The most common complication of PIVs infection is **otitis media**			-PIV1, PIV3 are respiviruses -PIV2, PIV4 are rubulaviruses -Subfamily: Paramyxovirinae -Replication is limited to respiratory epithelia - They are major pathogens of severe respiratory tract disease in infants and young children - PIV type 4 does not cause serious disease. -Mainly lower respiratory tract

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Respiratory Syncytial Virus	-ssRNA -Enveloped		-RSV is the most important cause of lower respiratory tract illness in infants and young children - The cause of bronchiolitis and pneumonia in infants -Most common cause of respiratory infections in infants	- DFA and RT-PCR can be used for laboratory diagnosis -Diagnosis can be made on the basis of the clinical syndrome combined with the time of year and other epidemiologic features	-Treatment of serious RSV infections depends primarily on supportive care (e.g. removal of secretions, administration of oxygen) -Treatment by antiviral drug ribavirin (aerosol) -Monoclonal Ab (palivizumab) against RSV has been shown to reduce viral shedding	-A Pneumovirus -Subfamily: Pneumovirinae
Metapneumovirus	- ssRNA -Enveloped		- Asymptomatic infections are more common than for influenza virus or RSV		- No specific therapy for human metapneumovirus infections, and no vaccine is available.	-Subfamily: Pneumovirinae
SARS Coronavirus	-ssRNA -Enveloped	2-11 days	- Fever, myalgia, headache, chills, cough, dyspnea, respiratory distress, diarrhea			-Transmission: civets to humans -Coronaviruses are the second most common cause of common cold and they show fatality -Receptor: angiotensin converting enzyme

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
MERS Coronavirus	-ssRNA -Enveloped	2-13 days	- Fever, myalgia, headache, chills, cough, dyspnea, pneumonia, vomiting, diarrhea			-Transmission: bats to camels, camels to humans --Coronaviruses are the second most common cause of common cold and they show fatality
Norovirus	-ssRNA -Naked		-most common cause of adult viral gastroenteritis			-Commonly transmitted through door handles.
Rotavirus	-Only family of double stranded segmented RNA		-most common cause of viral infantile gastroenteritis	-Rotavirus antigen detection -RT-PCR -Virus isolation	-No specific treatment -Rotarix vaccine	-A reovirus
Astrovirus			-Affects the gastrointestinal system			
Arbovirus						-Arthropod virus -After ingestion, the virus replicates in the gut of the arthropod and then spreads to other organs, including the salivary glands. -Only the female of the species serves as the vector of the virus, because only she requires a blood meal in order for progeny to be produced -Humans are dead-end hosts
Dengue virus			-Dengue (breakbone fever)		-Control depends on antimosquito measures	- is a mosquito-borne infection that is characterized by fever, severe headache, muscle and joint pain, nausea and vomiting, eye pain, and rash

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
West Nile Virus			About 80% of West Nile infections are asymptomatic, with about 20% causing West Nile fever (fever, headache, skin rash, and LAP) and less than 1% causing neuro-invasive disease (meningitis, encephalitis)			-Sequence analysis of virus isolates showed that it originated in the Middle East; it probably crossed the Atlantic in an infected bird, mosquito, or human traveler
Zika virus			-Infection is likely asymptomatic in about 80% of cases. -When symptoms occur they include symptoms include rash, fever, arthralgia, myalgia, fatigue, headache, and conjunctivitis -Rash, a prominent feature, is maculopapular and pruritic in most cases.		-Spontaneous resolution within 1–4 days of onset	-Transmitted by mosquitos -Other non-vector modes of Zika virus transmission include congenital and sexual -Congenital infection association with microcephaly
Filovirus (Ebola virus)			-Characterized by fever, headache, sore throat, and myalgia followed by abdominal pain, vomiting, diarrhea, and rash, with both internal and external bleeding, often leading to shock and death			-Ebola virus has a tropism for cells of the macrophage system, dendritic cells, interstitial fibroblasts, and endothelial cells -Filoviruses have the highest mortality rates of all the viral hemorrhagic fevers -Transmission: resp. droplets, body contact, fomites

Viruses Summary

Virus	Structure	Incubation period	Diseases	Diagnosis	Treatment/Vaccine	Notes
Rabies virus	-Negative sense ssRNA -Enveloped -Bullet shape	typically 1–3 months but may vary from 1 week to 1 yea	-The virus multiplies in the CNS and progressive encephalitis develops -The clinical spectrum can be divided into three phases: 1. Prodrome: malaise, anorexia, headache, photophobia, nausea and vomiting, sore throat, and fever. 2. Acute neurologic phase: CNS signs including nervousness, apprehension, hallucinations, and bizarre behavior. sympathetic overactivity is observed, including lacrimation, pupillary dilatation, and increased salivation and perspiration. Hydrophobia and aerophobia are common as well. 3.Coma and death: The major cause of death is cardiorespiratory arrest.	-clinical through history of exposure -Rabies antigens or nucleic acid detection -Serology -Virus isolation	-There is no effective treatment -A killed rabies virus vaccine is available for prophylaxis -Postexposure prophylaxis refers to treatment after an animal bite or suspected of being rabid, and consists of thorough cleaning of the wound, passive immunization, and active immunization. -Prevention of initial exposure is, however, the most important mechanism for controlling human rabies.	-Causes rabies in humans and animals -Transmitted from infected animals to humans via a bite or a scratch -The organs with the highest titers of virus are the salivary gland -Rabies virus produces a specific eosinophilic cytoplasmic inclusion, the Negri body (pathognomonic)

Transmissible Spongiform Encephalopathies (Prion Diseases)

Cause	Incubation Period	Clinical Findings	Treatment	Notes	Diseases
Infectivity is associated with proteinaceous material devoid of detectable amounts of nucleic acid	Long incubation periods (months to decades) precede the onset of clinical illness and are followed by chronic progressive disease (weeks to years)	-The basic features are neurodegeneration and spongiform changes	-Prions are resistant to treatment with formaldehyde, dry heat, boiling, ethanol, proteases, and ionizing radiation. -Prions are sensitive to phenol, household bleach, ether and autoclaving (1 hour, 121°C). -Guanidine thiocyanate is highly effective in decontaminating medical supplies and instruments.	-Degenerative CNS diseases (confined to the nervous system) -The diseases are always fatal, with no known cases of remission or recovery -Prions are non-immunogenic -In humans, the PrP prion diseases occur in three different forms: sporadic, inherited, and infectious	-In humans: **Kuru and Creutzfeldt–Jakob Disease:** develops gradually, with progressive dementia, ataxia, and myoclonus, and leads to death in 5–12 months. **-**In cows(Cattle): **Bovine spongiform encephalopathy (BSE), or "mad cow disease:** similar to scrapie. **Note**! It is now accepted that the new variant forms of CJD and BSE are caused by a common agent, indicating that the BSE agent had infected humans.

Kuru occurred only in the eastern high lands of New Guinae and was spread by customs surrounding ritual cannibalism of dead relatives. Since the practice has ceased, the disease has disappeared.

Done by: Rama Abbady