An Introduction to Viruses /1/2020

Viruses are a unique group of biological entities known to infect every type of cell, including bacteria, algae, fungi, protozoa, plants, and animals viruses are unable to exist independently from the host cell, so they are not living things but are more akin to large, infectious molecules. it is best to describe viruses as infectious particles (rather than organisms) and as either active or inactive (rather than alive or dead). They are a type of **obligate intracellular parasite** that cannot multiply unless it invades a specific host cell and instructs its genetic and metabolic machinery to make and release quantities of new viruses. Because of this characteristic, viruses are capable of causing serious damage and disease.

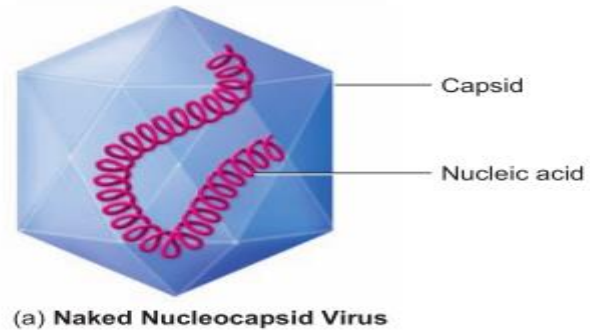
Size Range:- Their size places them in the realm of the ultramicroscopic . (0.2 μm) that an electron microscope is necessary to detect them .Animal viruses range in size from the small parvoviruse (20 nm in diameter) to poxviruses that are as large as small bacteria (up to 450 nm in length Viral architecture is most readily observed through special stains in combination with electron microscopy .

Shape of Viruses :-Viruses vary in shape from the simple helical and icosahedral to more complex structures. Spherical ,Rod-shaped ,Brick-shaped ,Tadpole-shaped ,Bullet-shaped and Filament

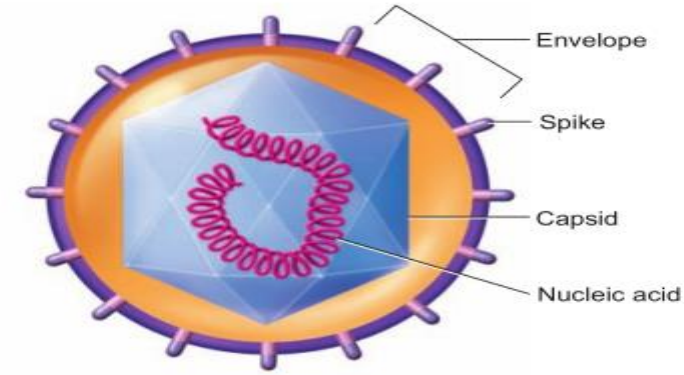
Viral Components: Capsids, Nucleic Acids, and Envelopes

viruses bear no real resemblance to cells and that they lack any of the synthetic machinery found in even the simplest cells. Viruses contain only those parts needed to **invade and control a host cell**: an external coating and a core containing one or more nucleic acid strands of either DNA or RNA. All viruses have a protein capsid, or shell, that surrounds the nucleic acid in the central core. Together, the **capsid** and the **nucleic acid** are referred to as the **nucleocapsid**. Viruses that consist of **only a nucleocapsid** are considered **naked viruses** viruses are **enveloped**, that is, they possess an additional **covering external to the capsid called an envelope**, which is usually a modified piece of the host's cell membrane the enveloped viruses also differ from the naked viruses in the way that they enter and leave a host cell.

Virion is the infectious particle composed of nucleic acid, protein capsid, +/- envelope may be extracellular or intracellular **Virus** is any stage of infection



(a) **Naked Nucleocapsid Virus**



(b) **Enveloped Virus**

The Viral Capsid: The Protective Outer Shell,

, the capsid of any virus is constructed from a number of identical protein subunits called capsomers. * The capsomers can spontaneously self-assemble into the finished capsid, this assembly results in two different types: helical and icosahedral.

The simpler **helical capsids** have rod-shaped capsomers that bind together to form a series of hollow discs resembling a bracelet. The nucleocapsids of naked helical viruses are very rigid and tightly wound into a cylinder- shaped package. This type of capsid(helical) is found in influenza, measles, and rabies.

Several virus for example, a poliovirus and adenovirus have capsids arranged in an **icosahedron** a three-dimensional, 20-sided The arrangements of the capsomers vary from one virus to another. During assembly of the virus, the nucleic acid is packed into the center of this icosahedron, forming a nucleocapsid.

The Viral Envelope

When enveloped viruses (mostly animal) are released from the host cell, they take with them a bit of its membrane system in the form of an envelope. Some viruses **bud off** the

cell membrane, nuclear envelope or the endoplasmic reticulum. Some proteins form a binding layer between the envelope and capsid of the virus, and glycoproteins (proteins bound to a carbohydrate) remain exposed on the outside of the envelope. These protruding molecules, called spikes or **peplomers**, are essential for the **attachment** of viruses to the next host cell.

Functions of the Viral Capsid/Envelope

1 - **Protects** the nucleic acid from the effects of various enzymes and chemicals when the virus is outside the host cell.

2- The capsids of enteric (intestinal) viruses such as polio and hepatitis A, which are **resistant to the acid- and protein-digesting** enzymes of the gastrointestinal tract.

3- Capsids and envelopes are also responsible for helping to **introduce** the viral DNA or RNA into a suitable host cell, first by binding to the cell surface and then by assisting in penetration of the viral nucleic acid .

4- parts of viral capsids and envelopes **stimulate the immune system** to produce **antibodies** that can **neutralize viruses** and protect the host's cells against future infections .

Complex Viruses: Atypical Viruses

Two special groups of viruses, termed complex viruses are more intricate in structure than the helical, icosahedral, naked, or enveloped viruses .The poxviruses (including the agent of smallpox) are very large DNA viruses . Some members of another group of very complex viruses, the bacteriophages(virus that infect bacteria) .

Nucleic Acids: At the Core of a Virus

- The sum total of the genetic information carried by an organism is known as its genome (nucleic acids) (DNA, RNA) viruses contain either **DNA or RNA** but **not both**. Because viruses must pack into a tiny space all of the genes necessary to instruct the host cell to make new viruses. Nine genes inhuman immunodeficiency virus (HIV) to hundreds of genes in some herpes viruses. Viruses possess only the genes needed to invade host cells and redirect their activity to make new viruses. DNA viruses can have single-stranded (ss) or double-stranded (ds) DNA; the dsDNA can be arranged linearly or in circles. RNA viruses can be double-stranded

but are more often single-stranded. **Single-stranded RNA genomes** that are **ready for immediate translation** into **proteins** are called **positive-strand RNA*(sense strand)*. RNA genomes that** have to be **converted** into the **proper form for translation** are called **negative-strand RNA*(antisense)*** .

- **Viruses produce two type of proteins**
- **structural proteins- Capside ,**
- **non-structural virion proteins enzymes transcriptase, protease ,integrase**

Viruses and cancer:

About 15% of human cancers are caused by viruses. Certain persistent viruses survive in the host by transforming the cells they infect (inducing infected cells to proliferate). However, the virus infection is only the first step in the pathway to malignancy and only a small percentage of infected people actually get cancer.

Common virus-induced cancers include: carcinoma of the cervix (Human papillomavirus), liver cancer (hepatitis B and C), Kaposi sarcoma (human herpesvirus 8) and Burkitts lymphoma (Epstein Bar virus).

Viruses spread in many ways.

1-Viruses ***influenza*** are spread through the ***air*** by droplets of moisture when people cough or sneeze.

2- Viruses such as ***norovirus*** are transmitted by the ***fecal–oral route***, which involves the contamination of hands, food and water.

3- ***Rotavirus*** is often spread by ***direct contact*** with infected children.

4-The human immunodeficiency virus, ***HIV***, is transmitted by bodily fluids transferred ***during sex***.

5- ***Dengue virus***, are spread by ***blood-sucking insects***.

Disinfection and inactivation of viruses:

Heat	**Most are inactivated at 56 °C for 30 minutes or at 100 °C for a few seconds**

Drying	**Variable; enveloped viruses are rapidly inactivated.**
Ultra-violet irradiation	**Inactivates viruses**
Organic solvents (Chloroform, Ether, Alcohol)	**Enveloped viruses are inactivated; those without are resistant.**
Oxidizing and reducing agents	**Viruses are inactivated by formaldehyde, chlorine, iodine and hydrogen peroxide**
Phenols	**Most viruses are resistant**

Multiplication Cycles in Animal Viruses

The general phases in the life cycle of animal viruses are adsorption, * penetration, synthesis, assembly, and release from the host cell.

Adsorption and Host Range

Invasion begins when the virus encounters a **susceptible** host cell and adsorbs specifically to **receptor sites** on the cell membrane. The membrane receptors that viruses attach to are usually **glycoproteins** the cell requires for its normal function. For example, the rabies virus affixes to receptors found on mammalian nerve cells, and the human immunodeficiency virus (HIV or AIDS virus) attaches to the CD4 protein on certain white blood cells. The mode of attachment varies between the two general types of viruses. In enveloped forms such as influenza virus and HIV, glycoprotein spikes bind to the cell membrane receptors. Viruses with naked nucleocapsids (adenovirus, for example) use molecules on their capsids that adhere to cell membrane receptors **Because a virus can invade its host cell only through making an exact fit with a specific host molecule**, the range of hosts it can infect in a natural setting is **limited**. This limitation, known as the **host range**, can vary from one virus to another. For example, hepatitis B infects only liver cells of humans; the poliovirus infects primarily intestinal and nerve cells of primates (humans, apes, and monkeys); and the rabies virus infects nerve cells of most mammals. Cells that lack compatible virus receptors are resistant to adsorption and invasion by that virus. This explains why, for example, human liver cells are not infected by the canine hepatitis virus and dog liver cells cannot host the human hepatitis A virus.

Penetration/ Uncoating of Animal Viruses

Viruses must penetrate the cell membrane of the host cell and deliver the viral nucleic acid into the host cell's interior. This occurs by form of **fusion** or **endocytosis**

Fusion, the viral envelope fuses directly with the host cell membrane, so it can occur only in enveloped viruses .

Endocytosis version of penetration, the virus can be either enveloped or naked and it is engulfed entirely into a vesicle after its initial attachment. Once inside the cell,

the virus is uncoated ; that is, its nucleic acid or nucleocapsid is released by the actions of enzymes in the cytoplasm that dissolve the vesicle wall.

Synthesis: Replication and Protein Production

. Free viral nucleic acid exerts control over the host's metabolism and synthetic machinery.. In general, the **DNA** viruses (except poxviruses) enter the host cell's **nucleus** and are replicated and assembled there. **RNA** viruses are replicated and assembled in the **cytoplasm**, HIV depends on the expression of 250 human genes to complete its multiplication cycle. First, the RNA of the virus synthesizing viral proteins (translation). The viruses with **positive**-strand RNA molecules already contain the correct message for **translation** into proteins. Viruses with **negative**-strand RNA molecules must first be **converted** into a positive-strand message. Some viruses come equipped with the necessary enzymes for synthesis of viral components; others utilize those of the host. During the final phase, the host's replication and synthesis machinery produces new RNA, proteins for the capsid, spikes , and viral enzymes.

Assembly of Animal Viruses: Host Cell as Factory

the capsid is first laid down as an empty shell that will serve as a receptacle for the nucleic acid strand.

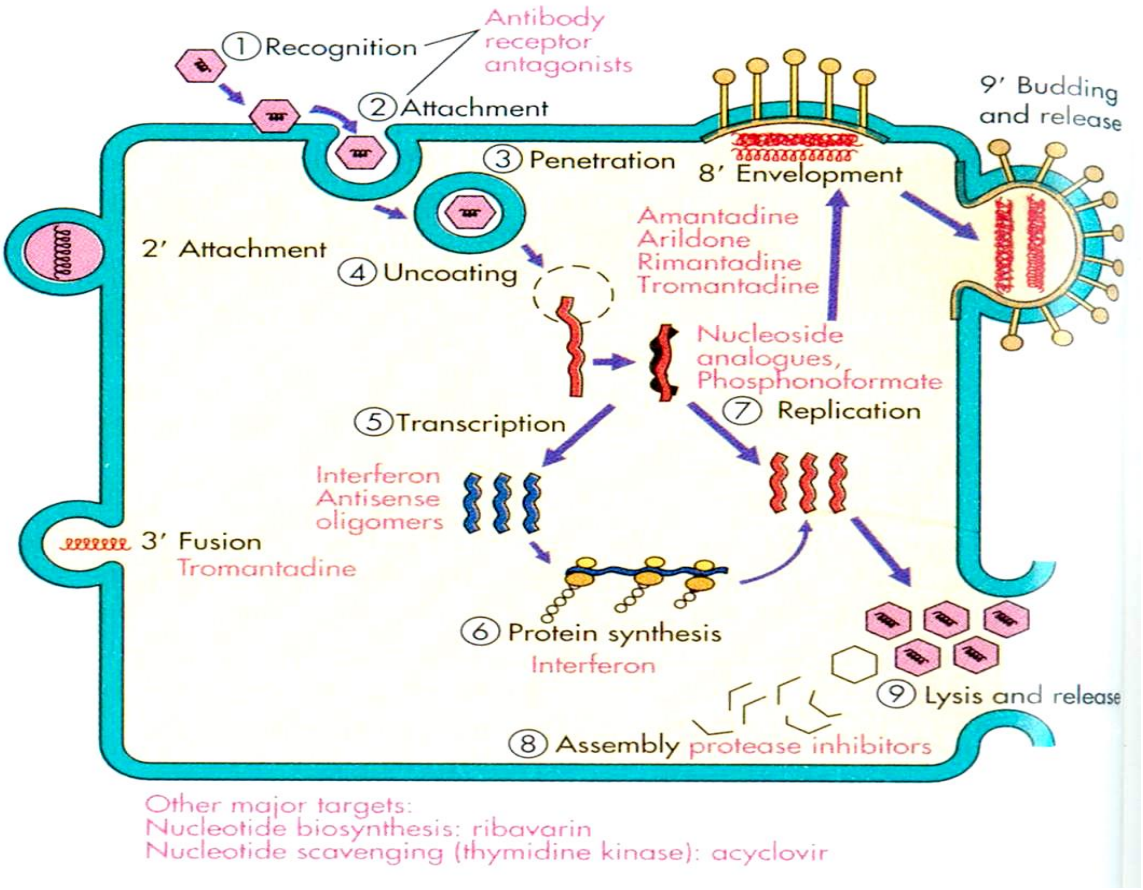
Release of Mature Viruses

To complete the cycle, assembled viruses leave their host in one of two ways.

- **Non enveloped** and complex viruses that reach maturation in the cell nucleus or cytoplasm are released through cell lysis or **rupturing.**

-**Enveloped** viruses are liberated by **budding** or **exocytosis** .most active viral infections are ultimately lethal to the cell because of accumulated damage. Lethal damages include a permanent shutdown of metabolism and genetic expression, destruction of cell membrane and organelles, toxicity of virus components, and release of lysosomes.

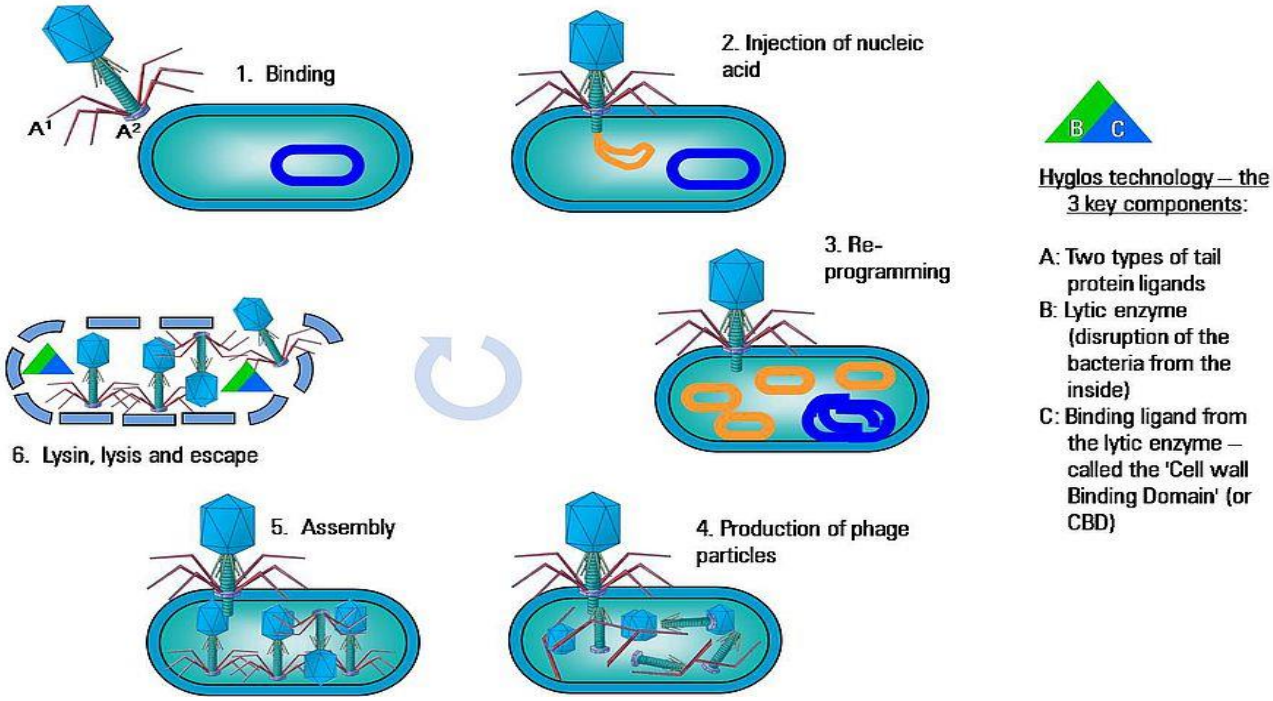
As a general rule, a virus infection kills its host cell, but some cells escape destruction by harboring the virus in some form. These so-called ***persistent infections*** can last from a few weeks to years and even for the life of the host. the **measles virus, remains hidden in brain** cells for many years , causing progressive damage and disease. Several viruses remain in a latent state, they are inactive over long periods . like herpes simplex viruses (cold sores and genital herpes) and herpes zoster virus (chicken pox and shingles). Both viruses go into latency in nerve cells and later emerge under the influence of various stimuli to cause recurrent symptoms.



The Multiplication Cycle in Bacteriophages

A. Bacteriophages are viruses that attack bacteria. They penetrate by injecting their nucleic acid and are released as virulent phage upon lysis of the cell.

B. Some viruses go into a latent, or lysogenic, phase in which they integrate into the DNA of the host cell and later may be active and produce a lytic infection.



Techniques in Cultivating and Identifying Animal Viruses

1- **Using Cell (Tissue) Culture Techniques**- Cultures of animal cells usually exist in the primary or continuous form. Embryonic, fetal, adult, and even cancerous tissues have served as sources of primary cultures. To detect the growth of a virus in culture is to observe degeneration and lysis of infected cells in the monolayer of cells. The areas where virus-infected cells have been destroyed show up as clear, well-defined patches in the cell sheet called **plaques.**

2- **Using Live Animal Inoculation** Specially bred strains of white mice, rats, hamsters, guinea pigs ,and rabbits are the usual choices **Using Bird Embryos** Chicken, duck, and turkey eggs are the most common choices for inoculation. Certain viruses can also be detected by their ability to agglutinate red blood cells (form big clumps) or by

their reaction with an antibody of known specificity that will affix to its corresponding virus, if it is present.

Viral Infection, Detection

Immunoflorescence techniques ,Direct examination with an electron microscope

Detected the antigens from the virus ,polymerase chain reaction (PCR) ,

Host response to viral infection

Humoral Immunity: Virus and/or virus-infected cells can stimulate B lymphocytes to produce antibody (specific for viral antigens) Antibody neutralization is most effective when virus is present in large fluid spaces (e.g., serum) or on moist surfaces (e.g., the gastrointestinal and respiratory tracts). IgG, IgM, and IgA have all been shown to exert antiviral activity. Antibody can neutralize virus by: 1) blocking virus-host cell interactions or 2) recognizing viral antigens on virus-infected cells which can lead to antibody-dependent cytotoxic cells (ADCC) or complement-mediated lysis. IgG antibodies are responsible for most antiviral activity in serum, while IgA is the most important antibody when viruses infect mucosal surfaces.

Cell-Mediated Immunity: The term cell-mediated immunity refers to (1) the recognition and/or killing of virus and virus-infected cells by leukocytes and (2) the production of different soluble factors (cytokines) by these cells when stimulated by virus or virus-infected cells. Cytotoxic T lymphocytes, natural killer (NK) cells and antiviral macrophages can recognize and kill virus-infected cells. Helper T cells can recognize virus-infected cells and produce a number of important cytokines. Cytokines produced by monocytes (monokines), T cells, and NK cells (lymphokines) play important roles in regulating immune functions and developing antiviral immune functions.

I-Innate immune mechanisms (interferon, NK cells, and macrophages) restrict the early stages of infection and delay spread of virus.

Innate (non-specific) immune response to viral infection ,Body surface , Natural killer (NK) cells ,Interferon (IFN) ,Macrophages

II-Adaptive (specific) immune response to viral infection.

- Cytotoxic T lymphocytes (CTLs), Helper T (Th) cells , Antiviral antibodies

Cytotoxic T (T C) Cells: Cells That Kill Other Cells When CD8 cells are activated by antigen/MHC I, they differentiate into T cytotoxic cells (killer T cells Virally infected cells Cytotoxic cells recognize these because of virus peptide MHC combinations expressed on their surface. Cytotoxic defenses are an essential protection against viruses

(figure 1)

Viruses are highly adaptable, and have developed ways to avoid detection by T cells. Some viruses stop MHC molecules from getting to the cell surface to display viral peptides. If this happens, the T cell doesn't know there's a virus inside the infected cell.

Other Types of Killer Cells Natural killer (NK) cells are a type of lymphocyte related to T cells that are part of cytotoxic CMI(of cell-mediated immunity). They circulate through the spleen, blood, and lungs, and are probably the first killer cells to attack cancer cells and virus-infected cells. They destroy such cells by similar mechanisms as cytotoxic T cells. They are considered part of cell-mediated immunity because their activities are moderated by cytokines such as interleukin-12 and interferon.

Cytotoxic cells are armed with preformed mediators. Cytotoxic factors are stored inside compartments called granules, in both cytotoxic T cells and NK cells, until contact with an infected cell triggers their release. One of these mediators is perforin, a protein that can make pores in cell membranes; these pores allow entry of other factors into a target cell to facilitate destruction of the cell. Enzymes called granzymes are also stored in, and released from, the granules. Granzymes enter target cells through the holes made by perforin.

-Once inside the target cell, they initiate a process known as programmed cell death or **apoptosis**, causing the target cell to die.

- Released cytotoxic factor is granulysin, which directly attacks the outer membrane of the target cell, destroying it by lysis.

Cytotoxic cells synthesise and release cytokines, after making contact with infected cells. like interferon-g and tumour necrosis factor-a, and transfer a signal from the T cell to the infected, or other neighboring cells, to enhance the killing mechanisms.

Interferons

Virally infected cells produce and release small proteins called interferons, which play a role in immune protection against viruses. Interferon prevent replication of viruses within an

infected cell.its signaling molecules that allow infected cells to warn nearby cells of a viral presence – this signal makes neighboring cells increase the numbers of MHC class I molecules upon their surfaces, so that T cells surveying the area can identify and eliminate the viral infection .

Antibodies

Viruses can also be removed by antibodies before they get the chance to infect a cell.

Firstly, the antibodies neutralize the virus, meaning that it is no longer capable of infecting the host cell.

Secondly, many antibodies can work together, causing virus particles to stick together in a process called agglutination. Agglutinated viruses make an easier target for immune cells than single viral particles.

A third mechanism used by antibodies to eradicate viruses, is the **activation of phagocytes**. A virus-bound antibody binds to receptors, called Fc receptors, on the surface of phagocytic cells and triggers a mechanism of phagocytosis, by which the cell engulfs and destroys the virus, antibodies can also **activate the complement system**.

Scope of Infections

Viral diseases range from very mild or asymptomatic infections such as colds to destructive and life-threatening diseases such as rabies and AIDS.. The course of viral diseases starts with invasion at the portal of entry by a few virions and a primary infection. In some cases, the viruses replicate locally and disrupt the tissue, and in others, the virus enters the blood or travels along neuron pathways to tissues far from the initial entry point. Common manifestations of virus infections include rashes, fever, muscle aches, respiratory involvement, and swollen glands. Some diseases of unknown etiology are thought to have a viral basis. Examples include type I diabetes, multiple sclerosis, and chronic fatigue syndrome .

Viral Persistence, Latency, and Oncogenicity

Many human viral infections such as colds and influenza have a rapid course and result in a lytic cycle of infection the virus being eliminated by the body within a relatively short time. But a few important viruses establish themselves in longer-term persistent infections that last for many years and sometimes for life. Two types of persistence are I-Chronic viral

infections- the virus is detectable in tissue samples and is still multiplying at a slow rate ,but symptoms of infection are usually mild or absent. chronic effects are HIV (human immunodeficiency virus) and hepatitis B virus (HBV)

II- Latent viral infections, or latency In latent infections result when the virus enters a dormant phase inside host cells and becomes inactive after a lytic infection. Herpes viruses are prominent examples of latent viruses

Some persistent viruses are oncogenic

DNA Viruses :Epstein-Barr virus Burkitt lymphoma

Human herpesvirus-8 Kaposi sarcoma

Hepatitis B Hepatic carcinoma

Certain papillomaviruses Cervical and penile cancer

RNA Viruses

Retrovirus HTLV 1 Leukemia

Hepatitis C Liver cancer

Viruses cause cancer through a variety of genetic effects, Some may carry a viral oncogene into the human DNA. Others may insert into a site on the genome and activate a human oncogene. Some viruses do not insert into the genome but appear to regulate or promote the cell cycle in a way to stimulate uncontrolled cell growth. In all cases of virally induced cancers, a normal cell is transformed into a cancer cell and a tumor results

Pathogenesis :-Pathogenesis is the process by which an infection leads to disease. Pathogenic mechanisms of viral disease include (1) implantation of virus at the portal of entry, (2) local replication, (3) spread to target organs (disease sites), and (4) spread to sites of shedding of virus into the environment.

Factors that affect pathogenic mechanisms are (1) accessibility of virus to tissue, (2) cell susceptibility to virus multiplication, and (3) virus susceptibility to host defenses.

Cellular Pathogenesis

Direct cell damage and death from viral infection may result from

(1) diversion of the cell's energy.

(2) shutoff of cell macromolecular synthesis.

(3) competition of viral mRNA for cellular ribosomes.

(4) competition of viral promoters and transcriptional enhancers for cellular transcriptional factors such as RNA polymerases.

(5) Inhibition of the interferon defense mechanisms.

Indirect cell damage can result from integration of the viral genome, induction of mutations in the host genome, inflammation, and the host immune response..

Incubation Period

The incubation period is the time between exposure to virus and onset of disease. During this usually asymptomatic period, implantation, local multiplication, and spread (for disseminated infections) occur.

Viruses in Human Infections and Diseases

-Enveloped DNA Viruses: Poxviruses

A. Poxviruses are large, complex viruses that produce skin lesions called pox (pocks).

-. Variola is the agent of smallpox, the first disease to be eliminated from the world by vaccination.

-Enveloped DNA Viruses: The Herpes viruses

A. Herpes viruses are persistent latent viruses that cause recurrent infections and are implicated in neoplastic transformations.

1. Herpes simplex virus (HSV):

Both HSV-1 and HSV-2 create lesions of mucous membranes

-Type I affecting the lips, eyes, and oropharynx

- Type 2 usually affecting the genitals

. Habitat Humans are the only natural reservoir for herpes simplex virus

Herpetic whitlows. caused by the herpes simplex virus These painful, deep-set vesicles can become inflamed and necrotic and are difficult to treat. They occur most frequently in dental and medical personnel or people who carelessly touch lesions on themselves or others

2. Varicella-zoster virus (VZV): Responsible for

- Varicella (chickenpox) Chickenpox is the familiar childhood infection.

- Shingles (zoster).; zoster occurs when virus in the spinal/cranial nerves is reactivated.

A vaccine now offers protection against infection.

3. Cytomegalovirus is perhaps the most common herpes virus. Healthy adults may be asymptomatic; more serious disease is seen in neonates, newborns, and immunocompromised patients. Drug therapy is reserved for immunocompromised patients.

4. Epstein-Barr virus (EBV) is a herpes virus of the lymphoid and glandular tissue. Leads to mononucleosis and potentially Burkitt lymphoma or nasopharyngeal carcinoma. Disseminated EBV infection is a complication of AIDS and organ transplant patients.

5. Herpesviruses-6 and -7 are the cause of roseola infantum in children; herpesvirus-8 is the suspected viral agent of Kaposi sarcoma, a tumor of AIDS patients.

The Viral Agents of Hepatitis

A. Hepadna viruses: One cause of viral hepatitis, an inflammatory disease of liver cells that may result from several different viruses..

Hepatitis A

Clinical Features:-Incubation period 3-5 weeks (mean 28 days). There is no chronic form of the disease.

Milder disease than Hepatitis B; asymptomatic infections are very common, especially in children. Adults, especially pregnant women, may develop more severe disease(Complications) .

Fulminant hepatitis: rare; 0.3-1.8 % of cases

Highest risk: pregnant women, elderly, pre-existing liver disease, other chronic medical conditions

Pathogenesis

Virus enters via the gut; replicates in the alimentary tract and spreads to infect the liver, where it multiplies in hepatocytes. Viraemia is transient. Virus is excreted in the stools for two weeks preceding the onset of symptoms.

Transmission: Faecal-oral route, Contamination of food or water with sewage

Prevention

Active Immunization:- vaccine is available; it is recommended for travelers to third world countries and, indeed, all adults who are not immune. It is the recommended form of post-exposure prophylaxis if the exposure is identified early,. If there are such risk factors, or if prophylaxis is delayed, passive immunization in addition to vaccination is recommended.

Passive immunization:-Normal immunoglobulin (antibody prepared from pooled human serum) given to close contacts of acute cases.

Protection is short lived: three months

Hepatitis B virus (HBV)

Habitat Incubation period is long: 30-180 days, average 75 days

Hepatitis B virus is chronically carried, and this population serves as the reservoir for infection. The virus remains infective for days in dried blood, months in serum at room temperature, and decades if frozen. Transfusions or injections of blood products, such as plasma and clotting factors, served as prime means of infection prior to routine testing of donated blood. Currently, kidney dialysis equipment, reusable needles (such as those used

for acupuncture), tattooing and piercing equipment, and needleless vaccine guns have all been linked to infection. Other groups for whom prophylaxis is highly recommended are the sexual contacts of actively infected people or carriers and neonates born to infected mothers.

Pathogenesis:-Infection is parenterally transmitted. The virus replicates in the liver and virus particles, as well as excess viral surface protein, are shed in large amounts into the blood. Viraemia is prolonged and the blood of infected individuals is highly infectious. The host immune response to the virus is responsible for hepatocellular damage.

Dental health care professionals are said to be at a risk of infections caused by hepatitis B and hepatitis C viruses (HBV and HCV, respectively), herpes simplex virus type 1, HIV, mumps, influenza, and rubellaHBV and HCV infections are the major causes of liver disease worldwide and the health policy makers with their strategies try to control these infections in the community. dentists acquire the virus through a cut in the fingers contaminated by the patient's blood or saliva.

Virulence factors Persistence and oncogenicity.

Primary infections/Disease Most people infected with HBV show no symptoms and eventually develop immunity to the virus. Initial symptomatic infection is marked by malaise, fever, and discomfort, with rashes, arthritis, and jaundice occurring in some cases. Most patients experience complete recovery, whereas a small number may develop chronic liver disease as a result of necrosis or cirrhosis. Long-term complications include a greatly increased risk of hepatocellular carcinoma (liver cancer).

Control and treatment Mild HBV infections are treated by managing symptoms, whereas chronic infection may be treated with interferon, which stops virus multiplication. Passive immunization with hepatitis B immune globulin (HBIG) is administered to those who may have been exposed to the virus through needle punctures or other contact with blood. Extreme care when handling needles, broken blood vials, and splashed blood or serum is essential to reduce the risk of occupational exposure to the virus.

The primary prevention for HBV infection is vaccination. They are given in three doses over 18 months, with occasional boosters.. Vaccination is a must for medical and dental workers and students, patients receiving multiple transfusions, immunedeficient persons, and cancer patients. The vaccine is also now strongly encouraged for all newborns and infants as part of a routine immunization schedule and all older children and adolescents before age 18.

Hepatitis C virus (HCV) RNA virus , a flavivirus that causes many cases of transfusion hepatitis. HCV is involved in a chronic liver infection that can go undiagnosed, later leading to severe liver damage and cancer. It is spread primarily by exposure to blood and blood products It is considered one of the most common unreported agents of hepatitis (an estimated 3 million cases nationally).

.

An unusual virus, hepatitis D, or delta agent, is a defective RNA virus that cannot produce infection unless a cell is also infected with HBV. Hepatitis D virus has a circular RNA genome more closely related to viroids than other viruses. HDV infection can only occur simultaneously or subsequent to HBV infection because the delta agent lacks the genes for structural proteins and is reliant on hepatitis B virus for completing its multiplication cycle. When HBV infection is accompanied by the delta agent, the disease becomes more severe and is more likely to progress to permanent liver damage.

Hepatitis E

A newly identified RNA virus that causes a disease similar to HAV. It is associated with poor hygiene and sanitation and is spread through fecally contaminated foods

Clinical Features :-Incubation period: 45 days [2-9 weeks] Acute, self limiting hepatitis Most cases occur in young adults, 15-40 years.

Complications:-10 % develop fulminant hepatitis (more common in pregnant women). Mortality rate is high (20-40 %). Chronic hepatitis may develop in organ transplant patients and HIV-infected individuals and lead to cirrhosis.

Pathogenesis :-Acute hepatitis E is similar to hepatitis A; virus replicates in the gut initially, before invading the liver and virus is shed in the stool prior to the onset of symptoms. Viraemia is transient. A large inoculums of virus is needed to establish infection. Chronic hepatitis E infection seems similar to chronic hepatitis C infections, but much about the pathogenesis is unknown

Non enveloped DNA Viruses

A. Adenoviruses: Common infectious agent of the lymphoid organs, respiratory tract, eyes. Diseases include the common cold, kerato conjunctivitis (eye infections), and cystitis(urinary tract infections).

B. Papilloma and polyomaviruses.

1. Human papillomaviruses (HPVs) are the causative agent of papillomas, common warts, plantar warts, and genital warts,. Chronic latent infection with HPV is associated with cervical and penile cancer. Treatment includes surgery and interferon.

2. Polyomaviruses include, which causes complications in renal transplants.

C. Non enveloped Single-Stranded DNA Viruses:

The Parvoviruses: Human parvovirus (B19) causes erythema infectiosum, a relatively mild infection common in children

Reference :-Medical Microbiology. 18th edition

Foundation in microbiology 8th edition

Lecturer KaramaT. Altaee

B.S.c., M.Sc. Med .Microbiology

College of Dentistry / Dep.of Basic science