Unit- 4 Classification, Multiplication, Cultivation and Impact of viruses

1) Classification and nomenclature of viruses

Viruses can be classified primarily on their phenotypic characteristics, core content, chemical composition, capsid structure, size, shape, genome structure and modes of replication.

The Baltimore classification is the most commonly used for studying the system of virus classification. This system was developed by an American biologist David Baltimore in the 1970s, for which he was awarded the Nobel Prize.

ICTV (International Committee on Taxonomy of Viruses)

The International Committee on Taxonomy of Viruses began to devise and implement rules for the naming and classification of viruses early in the 1970s, an effort that continues to the present. The ICTV is the only body charged by the International Union of Microbiological Societies with the task of developing, refining, and maintaining a universal virus taxonomy.

The below flowchart describes the classification of viruses based on their different criteria.

1) Classification based on the presence of nucleic acid

DNA virus

The virus, having DNA as its genetic material. There are two different types of DNA virus

Single-stranded (ss) DNA virus: e.g. Picornaviruses, Parvovirus, etc.

Double-stranded (ds) DNA virus: e.g. Adenovirus, Herpes virus, etc.

RNA virus

The virus, having RNA as its genetic material. There are two different types of RNA virus

Double-stranded (ds) RNA virus: e.g. Reovirus, etc.

Single-stranded (ss) RNA virus. It is further classified into two Positive sense RNA (+RNA) and negative sense RNA (-RNA). Poliovirus, Hepatitis A, Rabies virus, Influenza virus are examples of single-stranded RNA virus.

2) Classification based on the structure or symmetry

Complex virus. E.g Poxvirus

Radial symmetry virus.E.g.Bacteriophage

Cubical or icosahedral symmetry shaped virus. E.g. Reovirus, Picornavirus

Rod or Spiral shaped or helical symmetry virus.E.g. Paramyxovirus, orthomyxovirus

3) Classification based on the replication properties and site of replication

Here, viruses invade into the host cell, where it replicates and assembly within the cell organelles.

Replication within the cytoplasm of the host cell.

E.g. All RNA viruses except the Influenza virus.

Replication within the nucleus and the cytoplasm of the host cell.

E.g. Influenza virus, Poxvirus, etc.

Replication within the nucleus of the host cell.

All DNA viruses except Pox virus.

Replication of the virus through the double-stranded DNA intermediate.

E.g. All DNA viruses, Retrovirus and some tumour causing RNA virus.

Replication of the virus through a single-stranded RNA intermediate.

E.g. All RNA viruses except Reovirus and tumour-causing RNA viruses.

4) Classification based on the host range

Based on the type of host, there are four different types of viruses:

Animal viruses

These viruses infect by invading the cells of animals, including humans. Prominent examples of animal viruses include the influenza virus, mumps virus, rabies virus, poliovirus, Herpes virus, etc.

Plant viruses

These viruses infect plants by invading the plant cells. Well-known examples of plant virus include the potato virus, tobacco mosaic virus, beet yellow virus, and turnip yellow virus, cauliflower mosaic virus, etc.

Bacteriophages

The virus which infects bacterial cells is known as bacteriophage. There are many varieties of bacteriophages, such as DNA virus, MV-11, RNA virus, λ page, etc.

Insect virus

The virus which infects insects is known as Insect virus, also called the viral pathogen of insects. These viruses are considered as a powerful biocontrol agent in the landscape of modern agriculture. Ascovirusvirionsand Entomopox virus, are best examples for insect virus.

5) Classification based on the mode of transmission

Airborne infections – Transmission of the virus through the air into the respiratory tract. E.g. Swine flu, and Rhinovirus.

Fecal oral route– Transmission of the virus through the contaminated water or food.

E.g. Hepatitis A virus, Poliovirus, Rotavirus.

Sexually transmitted diseases – Transmission of the virus through sexual contacts with the infected person.E.g. Retrovirus, human papillomavirus, etc.

Transfusion-transmitted infections- Transmission of the virus through the blood transfusion.

E.g. Hepatitis B virus, Human Immunodeficiency Virus, etc.

Zoonoses -Transmission of the virus through the biting of infected animals, birds, and insects to human. E.g. Rabies virus, Alpha virus, Flavivirus, Ebola virus, etc.

❖ Classification systems of Viruses

1). Baltimore classification

2). Holmes classification

3). LHT System of Virus Classification

4). Casjens and Kings classification of virus

5). ICTV classification

1). Baltimore classification

Baltimore classification (first defined in 1971) is a classification system that places viruses into one of seven groups depending on a combination of their nucleic acid (DNA or RNA), strandedness (single-stranded or double-stranded), sense, and method of replication. Named after David Baltimore, a Nobel Prize-winning biologist, these groups are designated by Roman numerals.

Group I: double-stranded DNA viruses (dsDNA viruses) (e.g. Adenoviruses, Herpesviruses, Poxviruses)

Group II: single-stranded DNA viruses (ssDNA viruses) (+ strand or "sense") DNA (e.g. Parvoviruses)

Group III: double-stranded RNA viruses (dsRNA viruses) (e.g. Reoviruses)

Group IV: positive-sense single-stranded RNA viruses [(+)ssRNA viruses] (+ strand or sense) RNA (e.g. Coronaviruses, Picornaviruses, Togaviruses)

Group V: negative-sense single-stranded RNA viruses [(−)ssRNA viruses] (− strand or antisense) RNA (e.g. Orthomyxoviruses, Rhabdoviruses)

Group VI: reverse transcribing Diploid single-stranded RNA viruses (ssRNA-RT viruses) (+ strand or sense) RNA with DNA intermediate in life-cycle (e.g. Retroviruses)

Group VII: reverse transcribing Circular double-stranded DNA viruses (dsDNA-RT viruses) DNA with RNA intermediate in life-cycle (e.g. Hepadnaviruses)

DNA viruses

Group I: double-stranded DNA viruses (dsDNA viruses)

Viruses possess double-stranded DNA and include such virus families as Herpesviridae (examples like HSV1 (oral herpes), HSV2 (genital herpes), VZV (chickenpox), EBV (Epstein-Barr virus), CMV (Cytomegalovirus)), Poxviridae (smallpox) and many tailed bacteriophages. The mimivirus was also placed into this group.

Group II: single-stranded DNA viruses (ssDNA viruses) (+ strand or "sense")

Viruses possess single-stranded DNA and include such virus families as Parvoviridae and the important bacteriophage M13.

Virus Family	Virus Genus	Virion- naked/ enveloped	Capsid Symmetry	Type of nucleic acid
1.Adenoviridae	Adenovirus	Naked	Icosahedral	ds
2.Papovaviridae	Papillomavirus	Naked	Icosahedral	ds circular
3.Parvoviridae	B 19 virus	Naked	Icosahedral	ss
4.Herpesviridae	Herpes Simplex Virus, Varicella zoster virus, Cytomegalovirus, Epstein Barr virus	Enveloped	Icosahedral	ds
5.Poxviridae	Small pox virus, Vaccinia virus	Complex coats	Complex	ds

6.Hepadnaviridae	Hepatitis B virus	Enveloped	Icosahedral	ds circular
7.Polyomaviridae	Polyoma virus (progressive multifocal leucoencephalopathy)	?	?	ds

RNA viruses

Group III: double-stranded RNA viruses (dsRNA viruses)

Viruses possess double-stranded RNA genomes, e.g. rotavirus. These genomes are always segmented.

Group IV: positive-sense single-stranded RNA viruses [(+)ssRNA viruses] (+ strand or sense) RNA

Viruses possess positive-sense single-stranded RNA genomes. Many well known viruses are found in this group, including the picornaviruses (which is a family of viruses that includes well-known viruses like Hepatitis A virus, enteroviruses, rhinoviruses, poliovirus, and foot-and-mouth virus), SARS virus, hepatitis C virus, yellow fever virus, and rubella virus.

Group V: negative-sense single-stranded RNA viruses [(−)ssRNA viruses] (− strand or antisense) RNA

Viruses possess negative-sense single-stranded RNA genomes. The deadly Ebola and Marburg viruses are well known members of this group, along with influenza virus, measles, mumps and rabies.

Virus Family	Virus Genera	Virion-naked/ enveloped	Capsid Symmetry	Type of nucleic acid
1.Reoviridae	Reovirus, Rotavirus	Naked	Icosahedral	ds
2.Picornaviridae	Enterovirus, Rhinovirus, Hepatovirus, Cardiovirus, Aphthovirus, Parechovirus, Erbovirus, Kobuvirus, Teschovirus	Naked	Icosahedral	ss
3.Caliciviridae	Norwalk virus, Hepatitis E virus	Naked	Icosahedral	ss
4.Togaviridae	Rubella virus	Enveloped	Icosahedral	ss
5.Arenaviridae	Lymphocytic choriomeningitis virus	Enveloped	Complex	ss
6.Retroviridae	HIV-1, HIV-2, HTLV-I	Enveloped	Complex	ss
7.Flaviviridae	Dengue virus, Hepatitis C virus, Yellow fever virus	Enveloped	Complex	ss
8.Orthomyxoviridae	Influenzavirus A, Influenzavirus B, Influenzavirus C, Isavirus, Thogotovirus	Enveloped	Helical	ss
9.Paramyxoviridae	Measles virus, Mumps virus, Respiratory syncytial virus	Enveloped	Helical	ss
10.Bunyaviridae	California encephalitis virus, Hantavirus	Enveloped	Helical	ss
11.Rhabdoviridae	Rabies virus	Enveloped	Helical	ss
12.Filoviridae	Ebola virus, Marburg	Enveloped	Helical	ss

	virus			
13.Coronaviridae	Corona virus	Enveloped	Complex	ss
14.Astroviridae	Astrovirus	Naked	Icosahedral	ss
15.Bornaviridae	Borna disease virus	Enveloped	Helical	ss

Reverse transcribing viruses

Group VI: reverse transcribing Diploid single-stranded RNA viruses (ssRNA-RT viruses) (+ strand or sense) RNA with DNA intermediate in life-cycle

Viruses possess single-stranded RNA genomes and replicate using reverse transcriptase. The retroviruses are included in this group, of which HIV is a member.

Group VII: reverse transcribing Circular double-stranded DNA viruses (dsDNA-RT viruses) DNA with RNA intermediate in life-cycle

Viruses possess double-stranded DNA genomes and replicate using reverse transcriptase. The hepatitis B virus can be found in this group.

Virus Family	Examples (common names)	Capsid naked/ enveloped	Capsid Symmetry	Nucleic acid type	Group
1. *Retroviridae*	HIV	Enveloped		dimer RNA	VI
2. *Caulimoviridae*	Caulimovirus, Cacao swollen-shoot virus (CSSV)	Naked			VII
3. *Hepadnaviridae*	Hepatitis B virus	Enveloped	Icosahedral	circular, partially ds	VII

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2). Holmes classification

Holmes (1948) used Carolus Linnaeus system of binomial nomenclature classification system to viruses into 3 groups under one order, order virales. They are placed as follows:

Group I: phaginae (attacks bacteria)

Group II: phytophaginae (attacks plants)

Group III: zoophaginae (attacks animals)

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3). LHT System of Virus Classification [Provisional Committee on Nomenclature of Virus (PNVC)]

The LHT System of Virus Classification is based on chemical and physical characters like nucleic acid (DNA or RNA), Symmetry (Helical or Icosahedral or Complex), presence of envelope, diameter of capsid, number of capsomers. This classification was approved by the Provisional Committee on Nomenclature of Virus (PNVC) of the International Association of Microbiological Societies (1962).

❖ Phylum Vira (divided into 2 subphyla)

- Subphylum Deoxyvira(DNA viruses)
  - ➢ Class Deoxybinala(dual symmetry)

    Order Uroviridae

    - ✓ Family Phagoviridae
  - ➢ Class Deoxyhelica(Helical symmetry)

    Order Chitovirales

    - ✓ Family Poxviridae
  - ➢ Class Deoxycubica (cubical symmetry)

    Order Peplovirales

    - ✓ Family Herpesviridae(162 capsomeres)

    Order Haplovirales(no envelope)

    - ✓ Family Iridoviridae(812 capsomeres)
    - ✓ Family Adenoviridae(252 capsomeres)
    - ✓ Family Papiloviridae(72 capsomeres)
    - ✓ Family Paroviridae(32 capsomeres)

- ✓ Family Microviridae(12 capsomeres)

- Subphylum Ribovira(RNA viruses)
  - Class Ribocubica

    Order Togovirales

    - ✓ Family Arboviridae

    Order Lymovirales

    - ✓ Family Napoviridae
    - ✓ Family Reoviridae
  - Class Ribohelica

    Order Sagovirales

    - ✓ Family Stomataviridae
    - ✓ Family Paramyxoviridae
    - ✓ Family Myxoviridae

    Order Rbadovirales

    Suborder Felxiviridales

    - ✓ Family Mesoviridae
    - ✓ Family Peptoviridae

    Suborder Rigidovirales

    - ✓ Family Pachyviridae
    - ✓ Family Protoviridae
    - ✓ Family Polichoviridae

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4). Casjens and Kings Classification of virus

Casjens and Kings (1975) classified virus into 4 groups based on type of nucleic acid, presence of envelope, symmetry and site of assembly. It is as follows:

i. Single Stranded RNA Viruses
ii. Double Stranded RNA Viruses
iii. Single Stranded DNA Viruses
iv. Double Stranded DNA Viruses

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5). ICTV classification

The International Committee on Taxonomy of Viruses (ICTV) began to devise and implement rules for the naming and classification of viruses early in the 1970s, an effort that continues to the present. The ICTV is the only body charged by the International Union of Microbiological Societies with the task of developing, refining, and maintaining a universal virus taxonomy. The ICTV was created as a committee of the Virology Division of the International Union of Microbiological Societies (IUMS) and is governed by Statutes approved by the Virology Division. The system shares many features with the classification system of cellular organisms, such as taxon structure.

Viral classification starts at the level of realm and continues as follows, with the taxonomic suffixes in parentheses:

Realm (-viria)

Kingdom (-virae)

Phylum (-viricota)

Subphylum (-viricotina)

Class (-viricetes)

Order (-virales)

Suborder (-virineae)

Family (-viridae)

Subfamily (-virinae)

Genus (-virus)

Subgenus (-virus)

Species

As of 2019, 6 realms, 2 incertaesedis genera and 24 incertaesedis families, are recognized:

6 Realms:

- Duplodnaviria,
- Monodnaviria,
- Adnaviria,
- Ribozyviria,
- Riboviria,
- Varidnaviria

2 Incertaesedis genera:

- Dinodnavirus,
- Rhizidiovirus

24 families

1. Alphasatellitidae
2. Ampullaviridae
3. Anelloviridae
4. Avsunviroidae
5. Baculoviridae
6. Bicaudaviridae
7. Clavaviridae
8. Finnlakeviridae
9. Fuselloviridae
10. Globuloviridae
11. Guttaviridae
12. Halspiviridae
13. Hytrosaviridae
14. Nimaviridae
15. Nudiviridae
16. Ovaliviridae
17. Plasmaviridae
18. Polydnaviridae
19. Portogloboviridae
20. Pospiviroidae
21. Spiraviridae
22. Thaspiviridae
23. Tolecusatellitidae
24. Tristromaviridae

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2) Multiplication of viruses

❖ Overview of virus replication /Replication cycle / Lytic cycle of viruses

Viral populations do not grow through cell division, because they are acellular. Instead, they use the machinery and metabolism of a host cell to produce multiple copies of themselves, and they assemble in the cell. The life cycle of viruses differs greatly between species but there are six basic stages in the life cycle of viruses-

1. Attachment / Adsorption
2. Penetration
3. Uncoating
4. Replication
5. Assembly
6. Release / Lysis

1. Attachment / Adsorption

It is a specific binding between viral capsid proteins and specific receptors on the host cellular surface. This specificity determines the host range of a virus. Here, the attachment proteins on the surface of the virus align to specific receptors on the surface of the animal cells. Apart from virus binding, cellular receptors usually have glycolipids or glycoprotein. The interaction between these specific attached proteins and cellular receptors determine the host range. This mechanism has evolved to favour those viruses that infect only cells in which they are capable of replication. Attachment to the receptor can induce the viral envelope protein to undergo changes that results in the fusion of viral and cellular membranes, or changes of non-enveloped virus surface proteins that allow the virus to enter.

2. Penetration

In this stage, the virus or its genetic material enters the cell. Viruses with envelopes usually enter through fusion with the membrane.Virions enter the host cell through receptor-mediated endocytosis or membrane fusion. This is often called viral entry. Some viruses have evolved mechanisms that inject their genome into the bacterial cell across the cell wall, while the viral capsid remains outside.

3. Uncoating

It is a process in which the viral capsid is removed. This may be by degradation by viral enzymes or host enzymes or by simple dissociation; the end-result is the releasing of the viral genomic nucleic acid.

4. Replication

It involves primarily multiplication of the genome. The genome replication of most DNA viruses takes place in the cell's nucleus. Most DNA viruses are entirely dependent on the host cell's DNA and RNA synthesizing machinery, and RNA processing machinery. Replication of RNA of RNA viruses usually takes place in the cytoplasm . All RNA viruses use their own RNA replicase enzymes to create copies of their genomes. Reverse transcribing viruses with RNA genomes (retroviruses), use a DNA intermediate to replicate.

Replication involves synthesis of viral messenger RNA (mRNA) from "early" genes, viral protein synthesis, possible assembly of viral proteins, then viral genome replication mediated by early or regulatory protein expression.

5. Assembly

Capsomers are the outer covering of proteins that protect the genetic information of a virus. In this stage, newly developed capsid proteins come together to form capsomers. Capsomers interact with other capsomers to form a fully developed capsid protein.
Viruses such as the head-tail viruses, first assemble an empty capsid and then store it with a viral genome. But, the rest of the viruses create the capsid around the viral genome.

Following the structure-mediated self-assembly of the virus particles, some modification of the proteins often occurs. In viruses such as HIV, this modification (sometimes called maturation) occurs after the virus has been released from the host cell.

6. Release / Lysis

This is the last stage in the life cycle of viruses, where they release newly created viruses from the host cell. Different kinds of viruses exit the cell in different methods.

- Some follow the process called lysis, where the virus bursts the host cell.

- The other viruses follow the process called exocytosis, where the virus exits from the cell’s own pathways.
- There are some other viruses which bud from the plasma membrane of the cell. Enveloped viruses (e.g., HIV) typically are released from the host cell by budding. During this process the virus acquires its envelope, which is a modified piece of the host's plasma or other, internal membrane.

When the new virus is released, it has the ability to kill the host cell. But some other viruses do not hinder the host cell, leave it as it is and continue to make more virus particles.

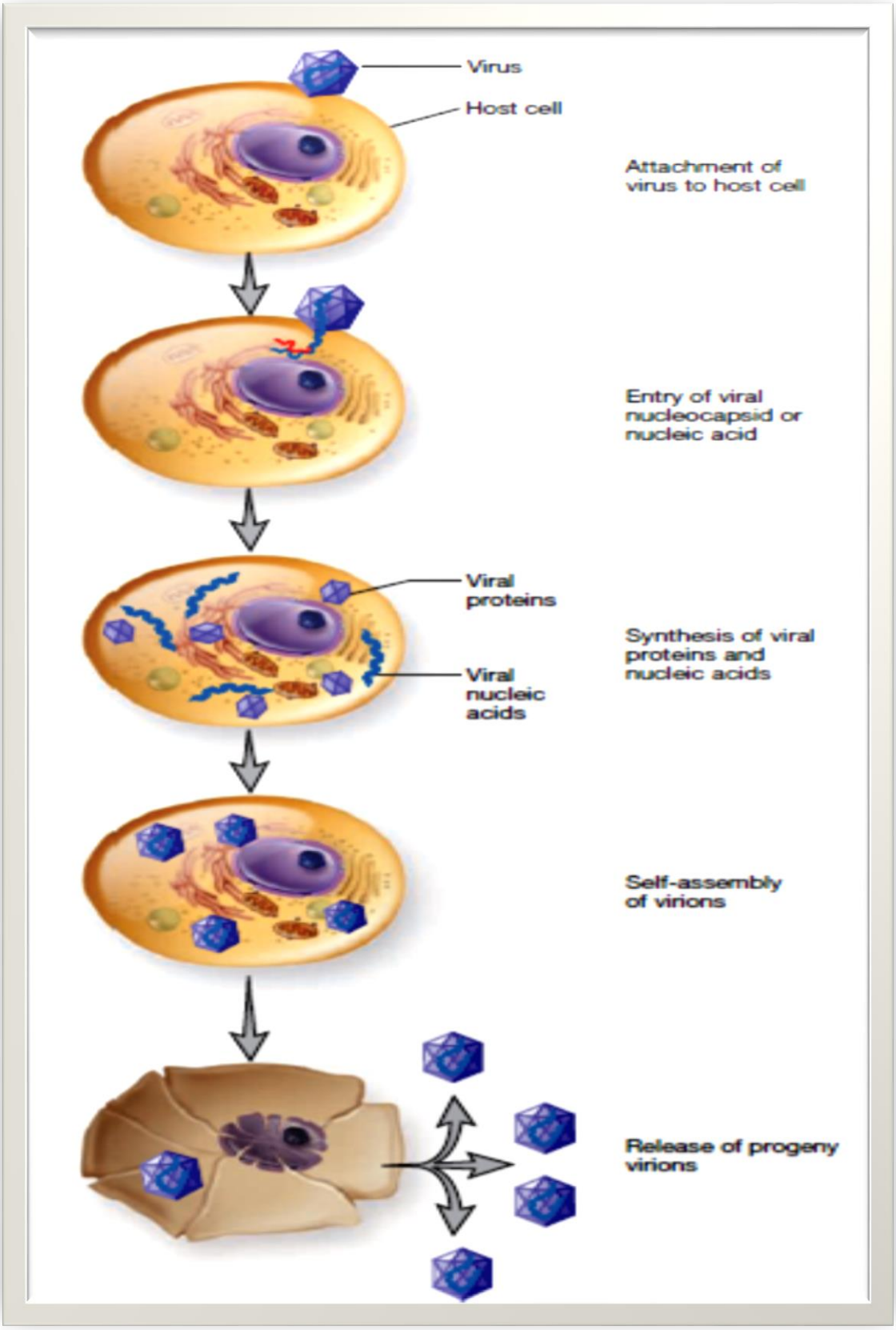


Figure - Generalized Illustration of Virus Reproduction.

❖ Replication of animal viruses

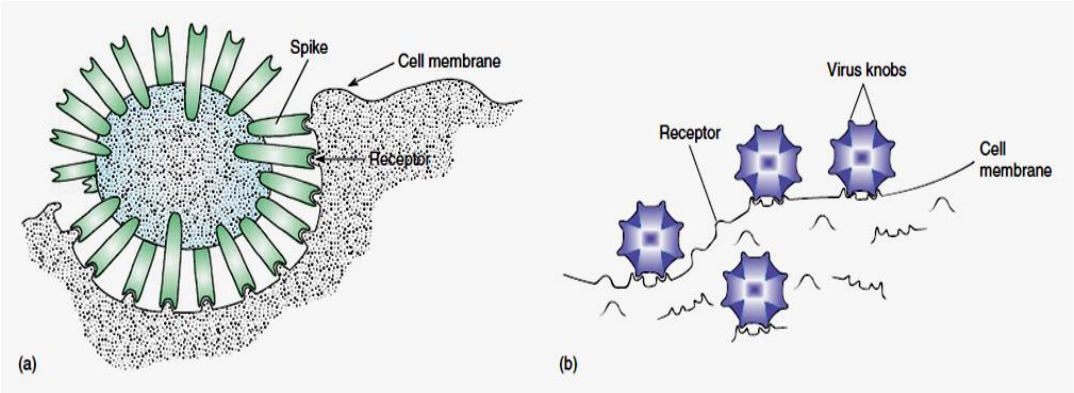
The generalphases in the cycle of animal viruses are

1) Adsorption
2) Penetration and Uncoating
3) Replication of genome and synthesis of protein capsids
4) Assemblyand maturation
5) Release of viruses from the host cell.

The length of the entire multiplication cycle varies from 8 hours inpolioviruses to 36 hours in herpesviruses.

1) Adsorption

Invasion begins when the virus encounters a susceptible host celland adsorbs specifically to receptor sites on the cell membrane. Themembrane receptors that viruses attach to are usually glycoproteins the cell requires for its normal function. For example, the rabiesvirus affixes to the acetylcholine receptor of nerve cells, and the humanimmunodeficiency virus (HIV or AIDS virus) attaches to theCD4 protein on certain white blood cells. The mode of attachmentvaries between the two general types of viruses. In enveloped formssuch as influenza virus and HIV, glycoprotein spikes bind to the cellmembrane receptors. Viruses with naked nucleocapsids (poliovirus,for example), possess surface molecules that adhere to cellmembrane receptors.



Host range / tropisms

Because a virus can invade its host cell only through makingan exact fit with a specific host molecule, the scope of hosts it caninfect in a natural setting is limited. This limitation, known as the**host range,** may be as restricted as hepatitis B, which infects onlyliver cells of humans;

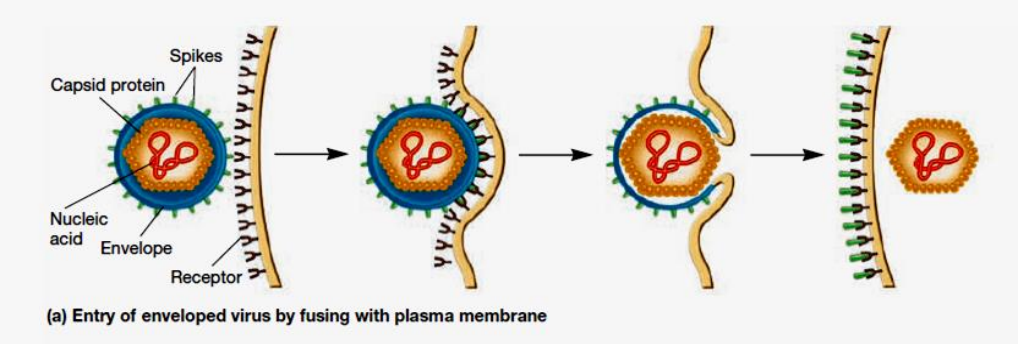
intermediate like the poliovirus, which infectsintestinal and nerve cells of primates (humans, apes, and monkeys);or as broad as the rabies virus, which can infect various cells of allmammals. Cells that lack compatible virus receptors are resistant toadsorption and invasion. This explains why, for example, humanliver cells are not infected by the canine hepatitis virus and dog livercells cannot host the human hepatitis A virus. It also explains whyviruses usually have tissue specificities called *tropisms*for certaincells in the body. The hepatitis B virus targets the liver, and themumps virus targets salivary glands.

2) Penetrationand Uncoating

Viruses penetrate the plasma membrane and enter a host cellshortly after adsorption. The mechanisms of penetration and uncoatingvary with the type of virus because viruses differ sogreatly in structure and mode of reproduction. For example, envelopedviruses may enter cells in a different way than naked virions.Furthermore, some viruses inject only their nucleic acid,whereas others must ensure that a virus-associated RNA or DNApolymerase, or even an organized core, also enters the host cell.The entire process of adsorption and uncoating may take frommінutes to several hours.

a) Fusion of the viral envelope with the host cell membrane—

Theenvelopes of paramyxoviruses, the *Retroviridae,* and someother viruses fuse directly with the host cell plasma membrane. Fusion may involve envelope glycoproteins thatbind to plasma membrane proteins. After attachment, several things happen: membrane lipids rearrange,the adjacent halves of the contacting membranesmerge, and a proteinaceous fusion pore forms. The nucleocapsidthen enters the host cell cytoplasm, where a viral polymerase,associated with the nucleocapsid, begins transcribingthe virus RNAwhile it is still within the capsid.



(a) Entry of enveloped virus by fusing with plasma membrane

b) Entry by endocytosis—

Nonenveloped viruses and some envelopedviruses enter cells by endocytosis. They may be engulfedby receptor-mediated endocytosis to form coatedvesicles. The virions attach to clathrin-coatedpits, and the pits then pinch off to form coated vesicles filled with viruses. These vesicles fuse with endosomes after theclathrin has been removed; depending on the virus, escape ofthe nucleocapsid or its genome may occur either before or aftervesicle fusion.

Endosomal enzymes can aid in virus uncoatingand low pHs often trigger the uncoating process. The viral envelope fuses with the endosomalmembrane, and the nucleocapsid is released into the cytoplasm(the capsid proteins may have been partially removedby endosomal enzymes).

Once in the cytoplasm, viral nucleicacid may be released from the capsid upon completion of uncoatingor may function while still attached to capsid components.

Naked viruses lack an envelope and thus cannot employthe membrane fusion mechanism. In this case,it appears that vesicle acidification causes a capsid conformationalchange. The altered capsid contacts the vesicle membraneand either releases the viral nucleic acid into thecytoplasm through a membrane pore (picornaviruses) or rupturesthe membrane to release the virion (adenovirus).

Virusesmay also enter the host cell by way of caveolae formation.

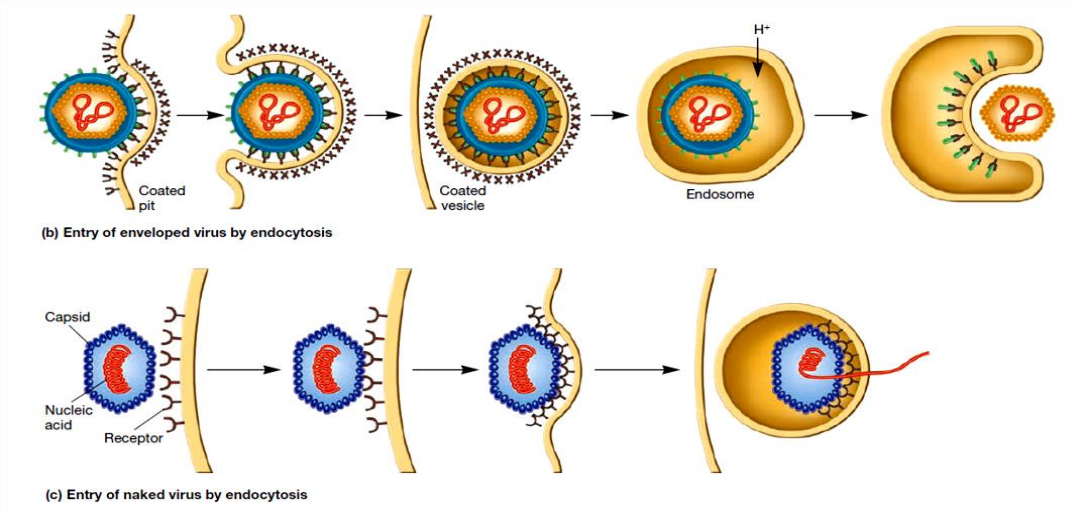


Figure -Animal Virus Entry. Examples of animal virus attachment and entry into host cells. Enveloped viruses can **(a)** enter afterfusion of the envelope with the plasma membrane, or **(b)** escape from the vesicle after endocytosis. **(c)** Naked viruses such as poliovirus, a picornavirusmay be taken up by endocytosis and then insert their nucleic

acid into the cytoplasm through the vesicle membrane. It also is possiblethat they insert the nucleic acid directly through the plasma membrane within a coated pit.

3) Replication of genome and synthesis of protein capsids

The replication strategy of any virus depends on the nature of its geneticmaterial. In this respect, all viruses can be divided into seven groups. Sucha scheme was first proposed by David Baltimore in 1971.

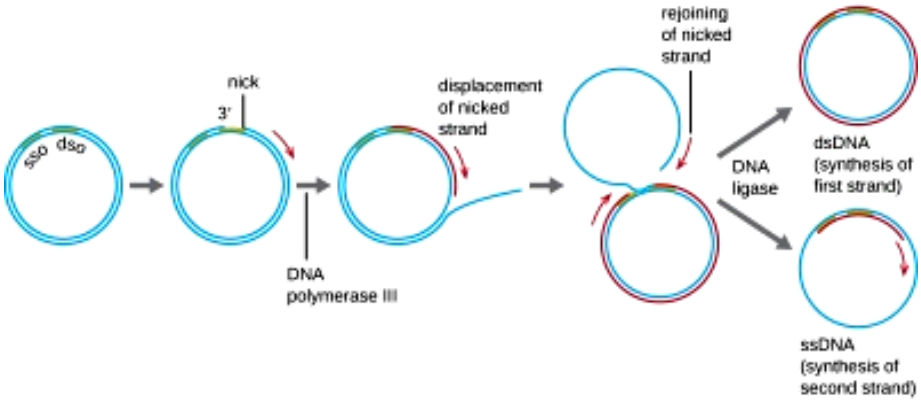
Class I:Double-stranded DNA viruses

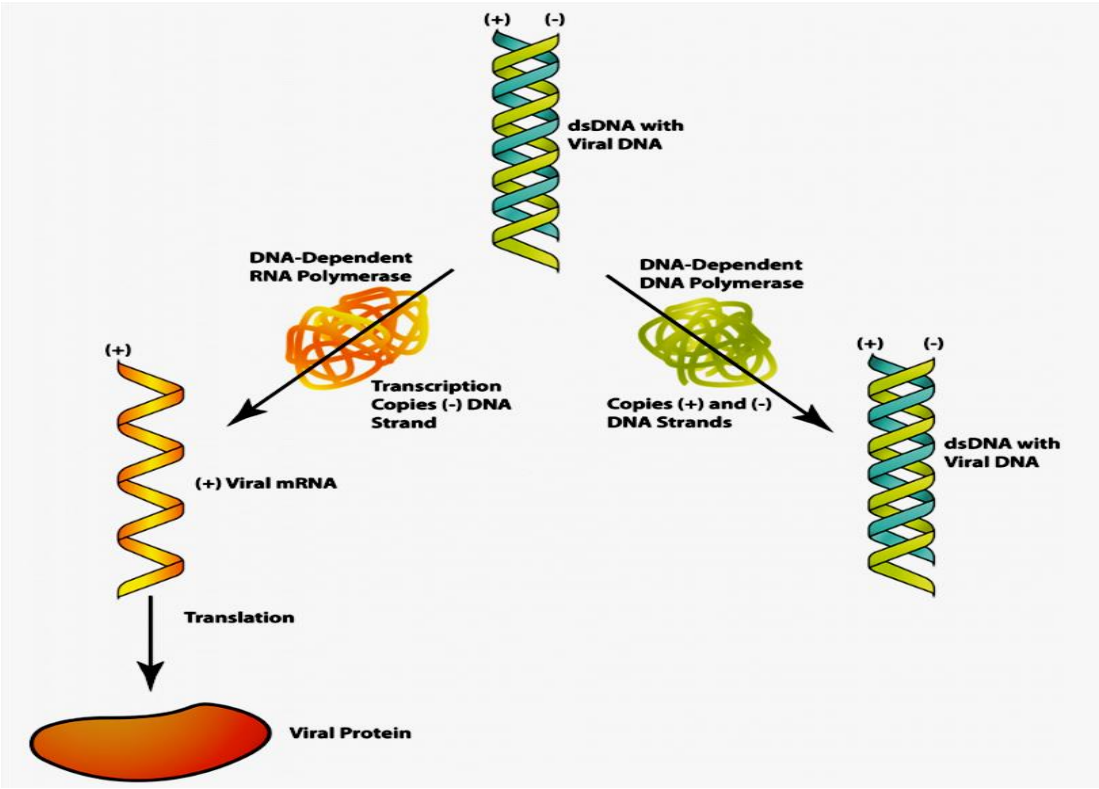
DNA viruses with a dsDNA genome, like bacteriophages T4 and lambda, have a genome exactly the same as the host cell that they are infecting. For this reason, many host enzymes can be utilized for replication and/or protein production. The flow of information follows a conventional pathway: dsDNA → mRNA → protein, with a DNA-dependent RNA-polymerase producing the mRNA and the host ribosome producing the protein. The genome replication, dsDNA → dsDNA, requires a DNA-dependent DNA-polymerase from either the virus or the host cell.

mRNA is transcribed from the DS DNA viruses in a similar fashionto host cellular DNA replication. These viruses can therefore completely dependupon the host cellular process to replicate. The genome of these viruses needs to carry information to codefor the virus specific proteins only. Regulatory proteins and those required for viralDNA synthesis are coded early on and the later proteins are generally structuralproteins.

This class can be subdivided into two further groups:
(a) replication isexclusively nuclear, meaning that replication of these viruses is relatively dependent on cellular factors;
(b) replication occurs in cytoplasm(e.g., the Poxviridae), in which case the viruses have evolved (or acquired) allthe necessary factors for transcription and replication of their genomes andare therefore largely independent of the cellular machinery.

The replicative form can be used for rolling-circle replication, where one strand is nicked and replication enzymes are used to extend the free 3’ end. As a complementary strand is synthesized around the circular DNA, the 5’ end is peeled off, leading to a displaced strand that continues to grow in length.



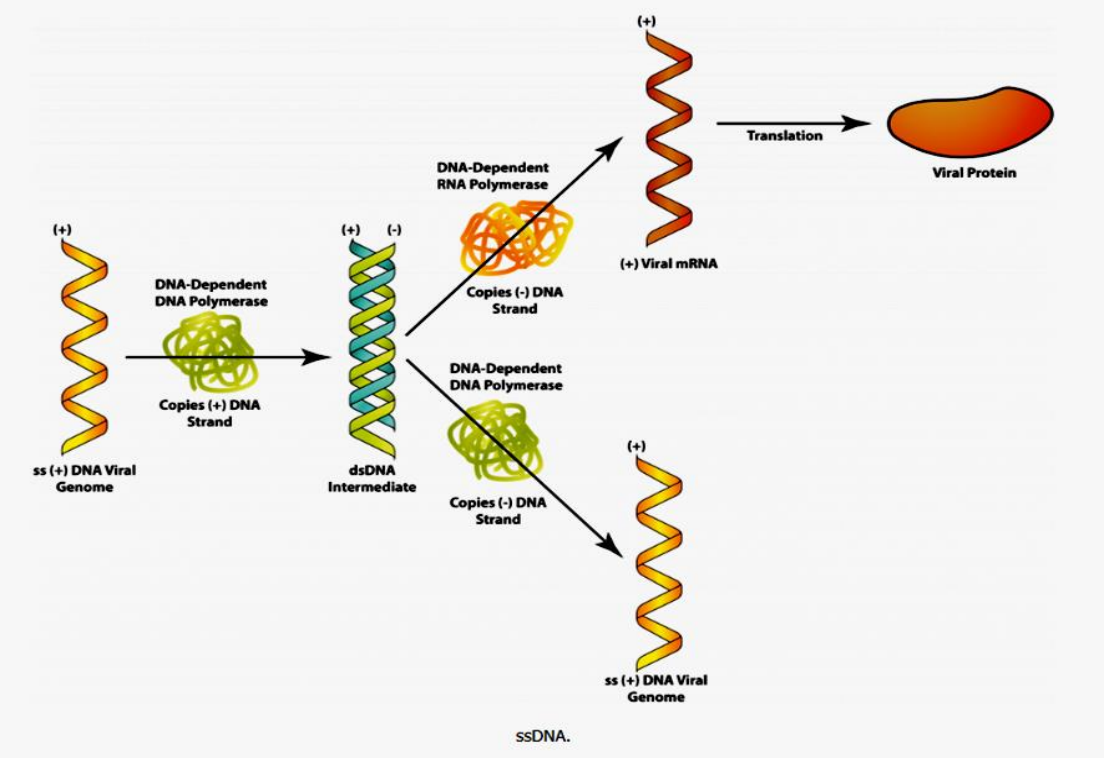


Class II: Single-stranded DNA

Replication occurs in the nucleus, involving the formation of a double strandedintermediate that serves as a template for the synthesis of single strandedprogeny DNA.Single stranded DNA viruses are first converted into double stranded, and thenmRNA is transcribed as for the DS DNA viruses.

The flow of information for ssDNA viruses, such as the parvoviruses, will still follow the conventional pathway, to a certain extent: DNA → mRNA → protein. But the viral genome can either have the same base sequence as the mRNA (plus-strand DNA) or be complementary to the mRNA (minus-strand DNA). In the former case, a DNA strand that is complementary to the viral genome must be manufactured first, forming a double-stranded

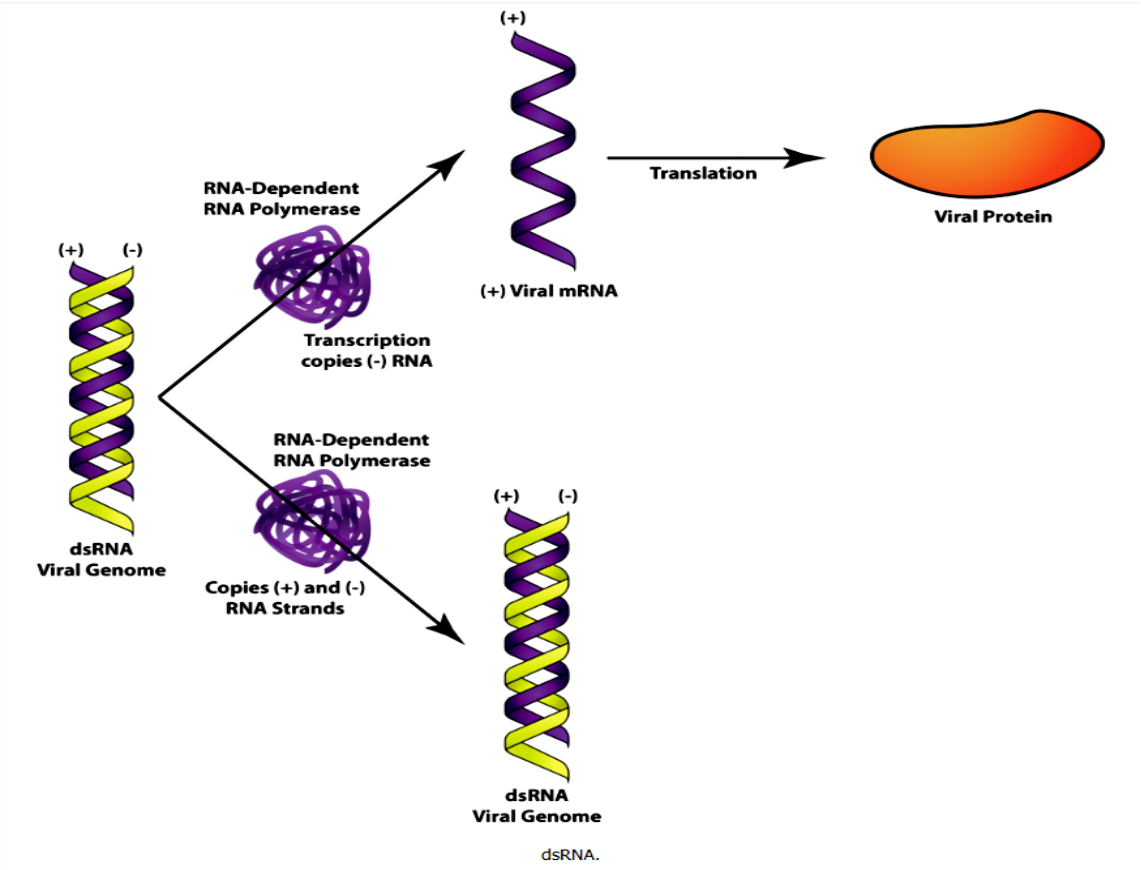
replicative form (RF). This can be used to both manufacture viral proteins and as a template for viral genome copies. For the minus-strand DNA viruses, the genome can be used directly to produce mRNA but a complementary copy will still need to be made, to serve as a template for viral genome copies.



Class III: Double-stranded RNA

Double-stranded RNA viruses infect bacteria, fungi, plants, and animals, such as the rotavirus that causes diarrheal illness in humans. But cells do not utilize dsRNA in any of their processes and have systems in place to destroy any dsRNA found in the cell. Thus the viral genome, in its dsRNA form, must be hidden or protected from the cell enzymes. Cells also lack RNA-dependent RNA-polymerases, necessary for replication of the viral genome so the virus must provide this enzyme itself. The viral RNA-dependent RNA polymerase acts as both a transcriptase to transcribe mRNA, as well as a replicase to replicate the RNA genome.

For the rotavirus, the viral nucleocapsid remains intact in the cytoplasm with replication events occurring inside, allowing the dsRNA to remain protected. Messenger RNA is transcribed from the minus-strand of the RNA genome and then translated by the host ribosome in the cytoplasm. Viral proteins aggregate to form new nucleocapsids around RNA replicase and plus-strand RNA. The minus-strand RNA is then synthesized by the RNA replicase within the nucleocapsid, once again insuring protection of the dsRNA genome.



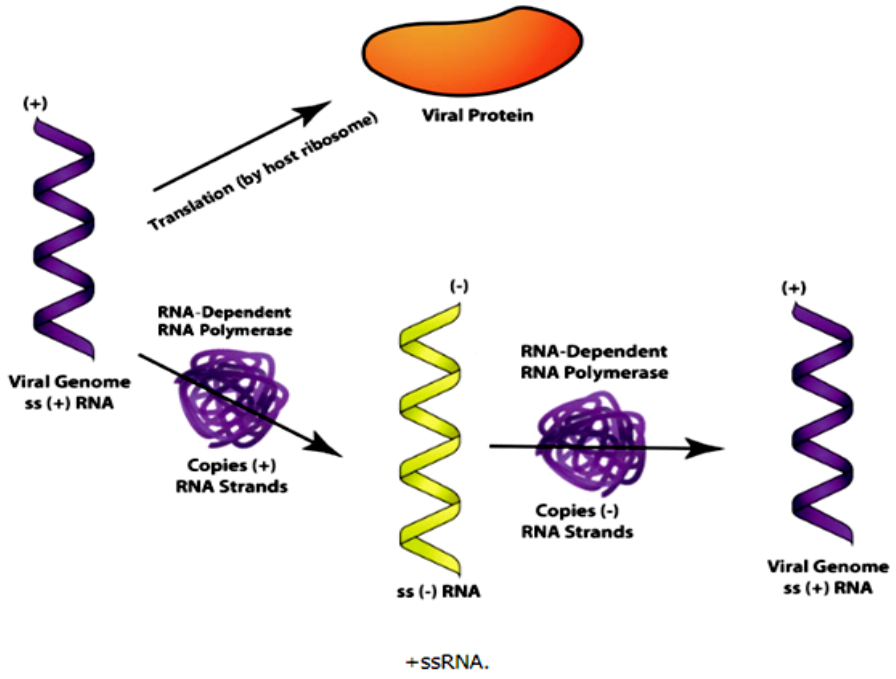
dsRNA.

Class IV: Single-stranded (+) sense RNA.

Viruses with plus-strand RNA, such as poliovirus, can use their genome directly as mRNA with translation by the host ribosome and enzymes occurring as soon as the unsegmented viral genome gains entry into the cell. One of the viral genes expressed yields an RNA-dependent RNA-polymerase (or RNA replicase), which creates minus-strand RNA from the plus-strand genome. The minus-strand RNA can be used as a template for more plus-strand RNA, which can be used as mRNA or as genomes for the newly forming viruses.

Theseviruses, because they can subvert the cellular system for their own replication, donot need to carry the information for the initial replication enzymes within theirgenome.

These can be subdivided into two groups: (a) viruses with polycistronicmRNA as with all the viruses in this class, the genome RNAforms the mRNA and is translated to form a polyproteinproduct, whichis subsequently cleaved to form the mature proteins; (b) viruses withcomplex transcription, for which two rounds of translation (e.g., togavirus)or subgenomic RNAs (e.g., tobamovirus) are necessary to produce thegenomic RNA.

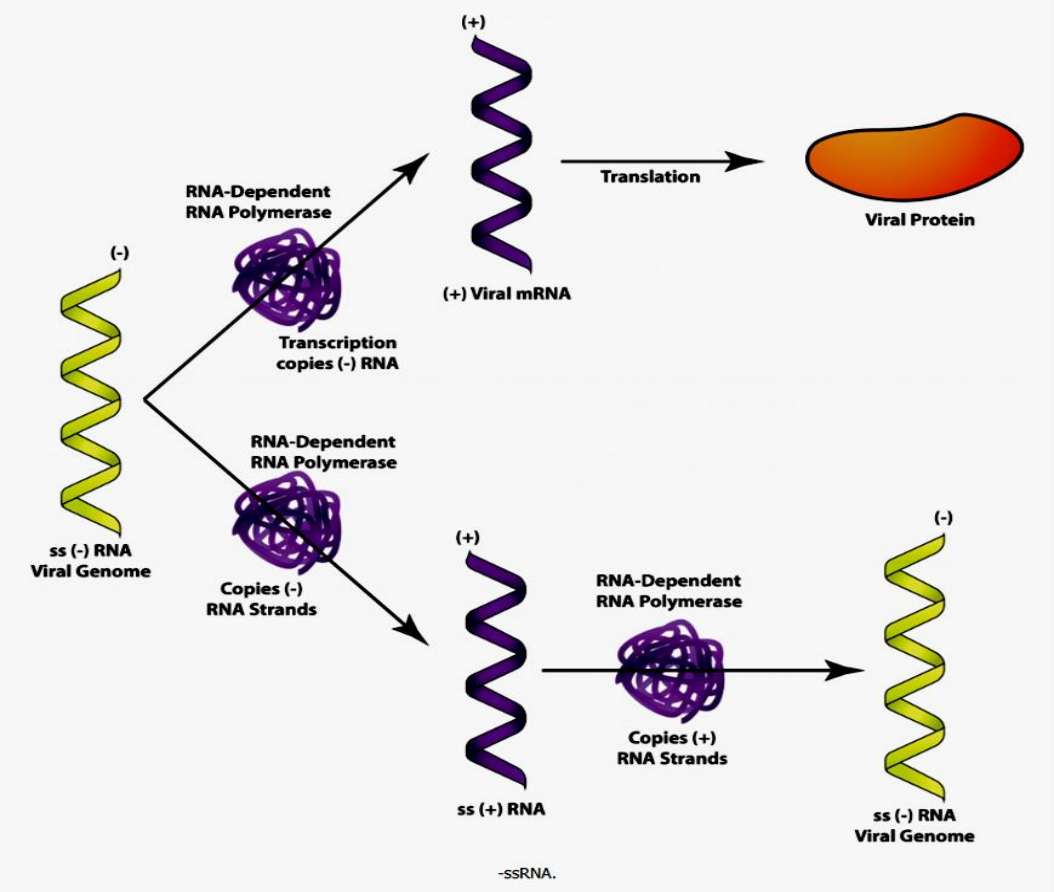


+ssRNA.

Class V: Single-stranded (-)sense RNA.

Minus-strand RNA viruses include many members notable for humans, such as influenza virus, rabies virus, and Ebola virus. Since the genome of minus-strand RNA viruses cannot be used directly as mRNA, the virus must carry an RNA-dependent RNA-polymerase within its capsid. Upon entrance into the host cell, the plus-strand RNAs generated by the polymerase are used as mRNA for protein production. When viral genomes are needed the plus-strand RNAs are used as templates to make minus-strand RNA.

The genomes of these viruses can bedivided into two types: (a) nonsegmented genomes for which the first step in replication is transcription of the (-)sense RNA genome by the virion RNA-dependent RNA polymeraseto produce monocistronic mRNAs, which also serve as the template forsubsequent genome replication. Eg.(orderMononegvirales)(b) segmented genomes forwhich replication occurs in the nucleus, with monocistronic mRNAs foreach of the virus genes produced by the virus transcriptase from thefull-length virus genome e.g. (Orthomyxoviridae)

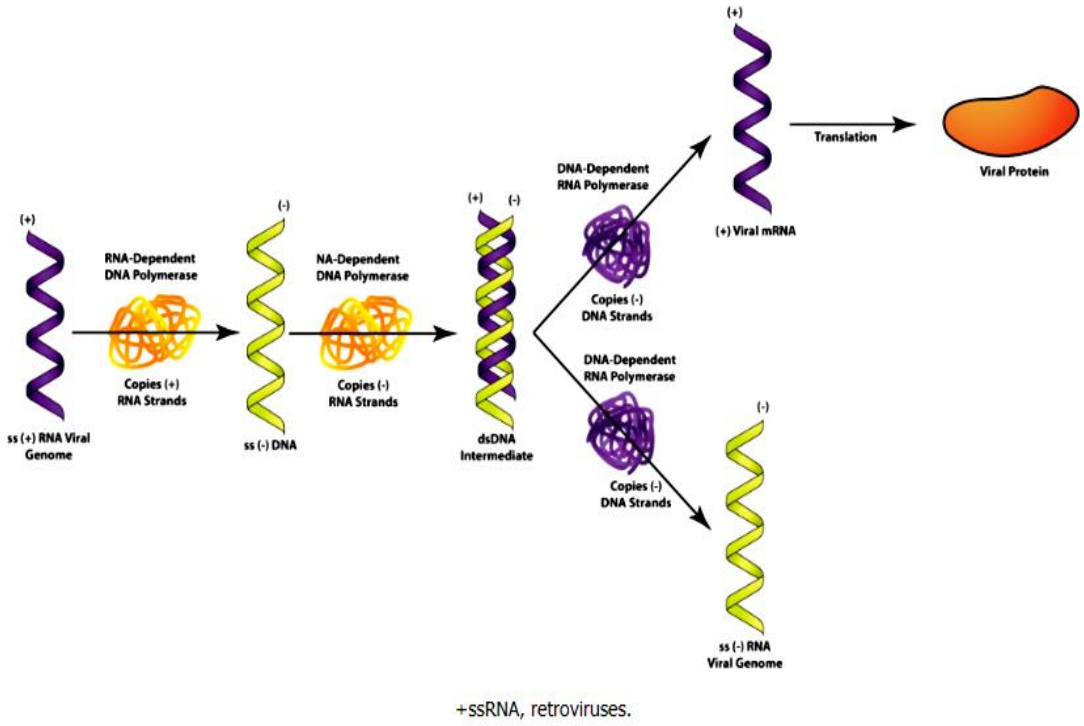


Class VI: Single-stranded (+)sense RNA with DNA intermediate/Retroviruses / Reverse Transcription

Despite the fact that the retroviral genome is composed of +ssRNA, it is not used as mRNA. Instead, the virus uses its reverse transcriptase to synthesize a piece of ssDNA complementary to the viral genome. The reverse transcriptase also possesses ribonuclease activity, which is used to degrade the RNA strand of the RNA-DNA hybrid. Lastly, the reverse transcriptase is used as a DNA polymerase to make a complementary copy to the ssDNA, yielding a dsDNA molecule. This allows the virus to insert its

genome, in a dsDNA form, into the host chromosome, forming a provirus. Unlike a prophage, a provirus can remain latent indefinitely or cause the expression of viral genes, leading to the production of new viruses. Excision of the provirus does not occur for gene expression.

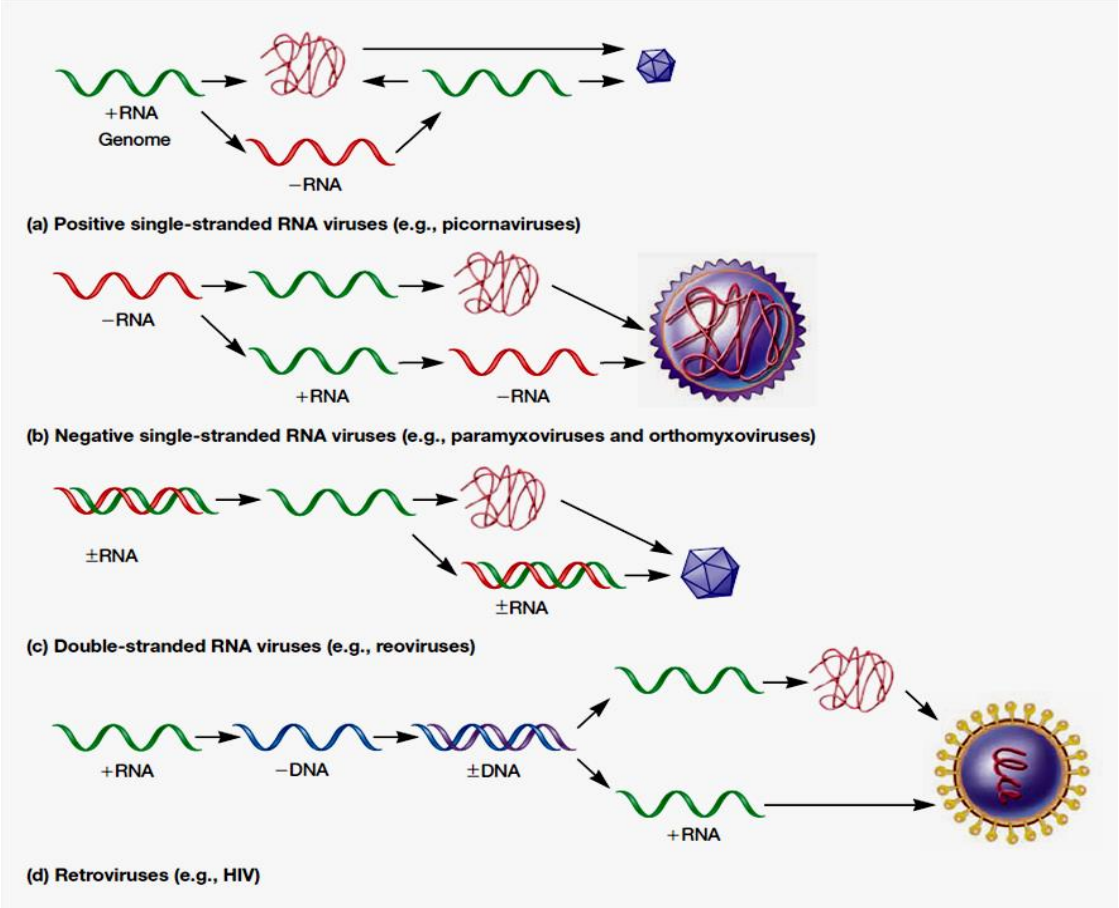
Retroviruses are unique SS +RNA viruses. Instead of using the SS +RNA asan mRNA template, the RNA is first transcribed into complementary DNA byan RNA-dependent DNA polymerase in a process called reverse transcription(hence the name, retro =reverse). The normal transcription is always fromDNA to RNA.



+ssRNA, retroviruses.

Class VII: Double-stranded DNA with RNA intermediate

This group of viruses also relies on reverse transcription, but, unlike theretroviruses (class VI), this process occurs inside the virus particle duringmaturation. On infection of a new cell, the first event to occur is repair ofthe gapped genome, followed by transcription.



4) Assembly and maturation

The assembly process involves the collection of all the components necessaryfor the formation of the mature virionat a particular site in the cell. Duringassembly, the basic structure of the virus particle is formed. The site of assemblydepends on the site of replication within the cell and on the mechanism bywhich the virus is eventually released from the cell and varies for differentviruses. For example, in picornaviruses, poxviruses, and reoviruses, assemblyoccurs in the cytoplasm; in adenoviruses, polyomaviruses, and parvoviruses, itoccurs in the nucleus.

The first step in viral maturation is the assembly of the proteincapsid; this assembly is usually a spontaneous process. The capsidsof many animal viruses are enclosed by an envelope consistingof protein, lipid, and carbohydrate, as noted earlier. Examplesof such viruses include orthomyxoviruses and paramyxoviruses.

The envelope protein is encoded by the viral genes and is incorporatedinto the plasma membrane of the host cell. The envelopelipid and carbohydrate are encoded by host cell genes and arepresent in the

plasma membrane. The envelope actually developsaround the capsid by a process called budding.

Some **late genes** direct the synthesis of capsid proteins, and thesespontaneously self-assemble to form the capsid. The assemblyof enveloped virus capsids is generally similar to that of nakedvirions, except for poxviruses. These are assembled in the cytoplasmby a lengthy, complex process that begins with the enclosureof a portion of the cytoplasmic matrix through construction of a newmembrane. Then newly synthesized DNA condenses, passesthrough the membrane, and moves to the center of the immaturevirus. Nucleoid and elliptical body construction takes place withinthe membrane.

Maturation

Maturation is the stage of the replication cycle at which the virus becomesinfectious. This process usually involves structural changes in the virus particlethat may result from specific cleavages of capsid proteins to form the matureproducts or conformational changes that occur in proteins during assembly.Such events frequently lead to substantial structural changes in the capsid thatmay be detectable by measures such as differences in the antigenicity ofincomplete and mature virus particles, which in some cases (e.g., picornaviruses)alters radically. Alternatively, internal structural alterationsdforexample,the condensation of nucleoproteins with the virus genomedoften result in suchchanges. As already stated, for some viruses assembly and maturation occurinside the cell and are inseparable, whereas for others maturation events mayoccur only after release of the virus particle from the cell. In all cases, the processof maturation prepares the particle for the infection of subsequent cells.

Virus-encoded proteases are frequently involved in maturation, although cellularenzymes or a mixture of virus and cellular enzymes are used in some cases.

5) Release of viruses from the host cell

To complete the cycle, assembled viruses leave their host in one oftwo ways. Nonenveloped and complex viruses that reach maturationin the cell nucleus or cytoplasm are released when the celllyses or ruptures. Enveloped viruses are liberated by **budding** or**exocytosis** from the membranes of the cytoplasm, nucleus, endoplasmicreticulum, or vesicles. During this process, the nucleocapsidbinds to the membrane, which curves completely around it andforms a small pouch. Pinching off the pouch

releases the virus with its envelope. Budding of enveloped viruses causes them to be shed gradually, without the sudden destruction of thecell. Regardless of how the virus leaves, most active viral infectionsare ultimately lethal to the cell because of accumulated damage.Lethal damages include a permanent shutdown of metabolismand genetic expression, destruction of cell membrane and organelles,toxicity of virus components, and release of lysosomes.

Mechanisms of virion release differ between naked and envelopedviruses. Naked virions appear to be released most often by host celllysis. In contrast, the formation of envelopes and the release of envelopedviruses are usually concurrent processes, and the host cellmay continue virion release for some time. All viral envelopes arederived from host cell membranes by a multistep process. First,virus-encoded proteins are incorporated into the plasma membrane.Then the nucleocapsid is simultaneously released and the envelopeformed by membrane budding

The number of viruses released by infected cells is variable,controlled by factors such as the size of the virus and the health ofthe host cell. About 3,000 to 4,000 virions are released from a singlecell infected with poxviruses, whereas a poliovirus-infected cellcan release over 100,000 virions.

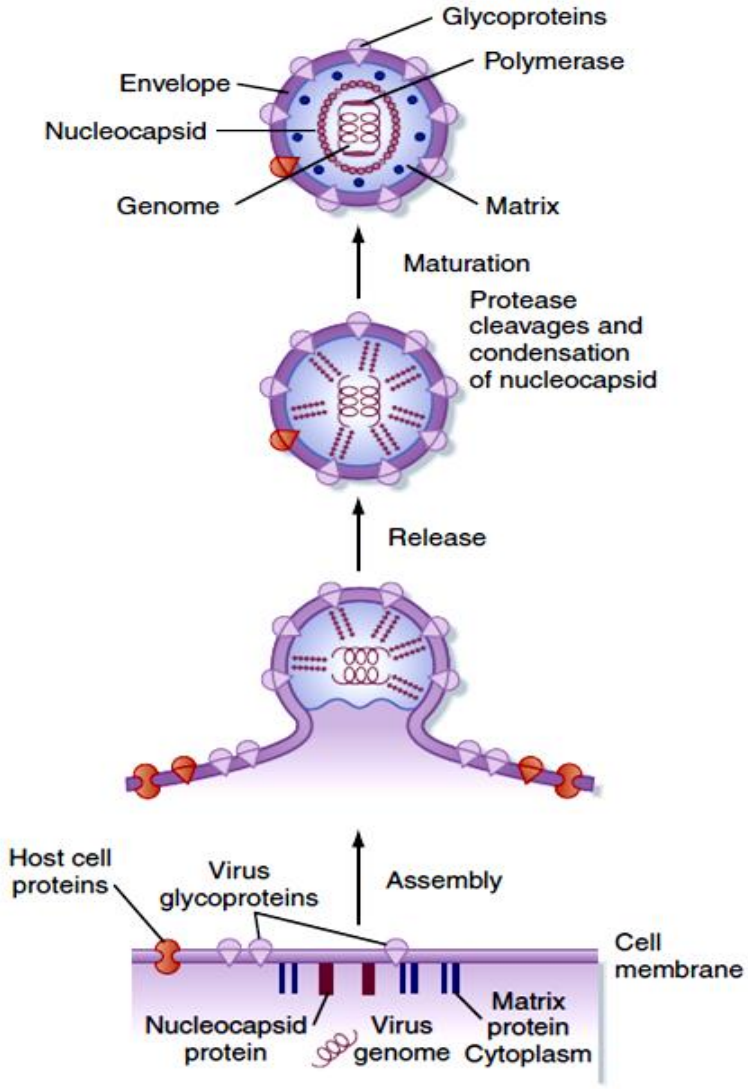


FIGURE - Virus release by budding.Budding is the process by which enveloped virus particles acquire their membranes and associated proteins,as well as how they are released for the host cell.

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❖ Replication of bacterial viruses /Bacteriophages – Lytic and lysogenic replication

Bacteriophages can multiply by two alternativemechanisms:
the lytic cycle
or the lysogenic cycle.

The lytic cycleends with the lysis and death of the host cell, whereas the hostcell remains alive in the lysogenic cycle. Because the T-

evellbacteriophages(T2, 14, and T6) have been studied most extensively,we will describe the multiplication of T-even bacteriophages intheir host, E. coli, as an example of the lytic cycle.

T-Even Bacteriophages: The Lytic Cycle

The virions of T-even bacteriophages are large, complex, andnonenveloped, with a characteristic head -and -tail structure. The length of DNA containedin these bacteriophages is only abou l 6% of that containedin E. coli, yet the phage has enough DNA for over 100 genes. Themultiplication cycle of these phages, like that of all viruses,occurs in five distinct stages:

1) Attachment
2) Penetration
3) Biosynthesis
4) Matu ration
5) Release.

1) Attachment

After a chance collision between phage particlesand bacteria, atfachmellt, or adsorption, occurs. During thisprocess, an attachment site on the virus attaches to a complementaryreceptor site on the bacterial cell. This attachment is achemical interaction in which weak bonds are formed betweenthe attachment and receptor sites. T-even bacteriophages usefibers at the end of the tail as attachment sites. The complementaryreceptor sites are on the bacterial cell wall.

2) Penetration

After attachment, the T-even bacteriophageinjects its DNA (nucleic acid) into the bacterium. To do this, thebacteriophage's tail releases an enzyme, phage lysozyme, whichbreaks down a portion of the bacterial cell wall. During theprocess of penetration, the tail sheath of the phage contracts, andthe ta il core is driven through the cell wall. When the tip of thecore reaches the plasma membrane, the DNA from the bacteriophage'shead passes through the tail core, through the plasmamembrane, and enters the bacterial cell. The capsid remains out - side the bacterial cell. Therefore, the phage particle functions likea hypodermic syringe to inject its DNA in to the bacterial cell.

3) Biosynthesis

Once the bacteriophage DNA has reached thecytoplasm of the host cell, the biosynthesis of viral nucleic acidand protein occurs. Host protein synthesis is stopped by virusinduced degradation of the host DNA, viral proteins that interferewith transcription, or the repression of translation.Initially, the phage uses the host cell's nucleotides and severalof its enzymes to synthesize many copies of phage DNA. Soonafter, the biosynthesis of viral proteins begins. Any RNA transcribedin the cell is mRNA transcribed from phage DNA for thebiosynthesis of phage enzymes and capsid proteins. The hostcell's ribosomes, enzymes, and amino acids are used for translation.

Genetic controls regulate when different regions of phageDNA are transcribed into mRNA during the multiplicationcycle. For example, early messages are translated into early phageproteins, the enzymes used in the synthesis of phage DNA. Also,late messages are translated into late phage proteins for the synthesisof capsid proteins.

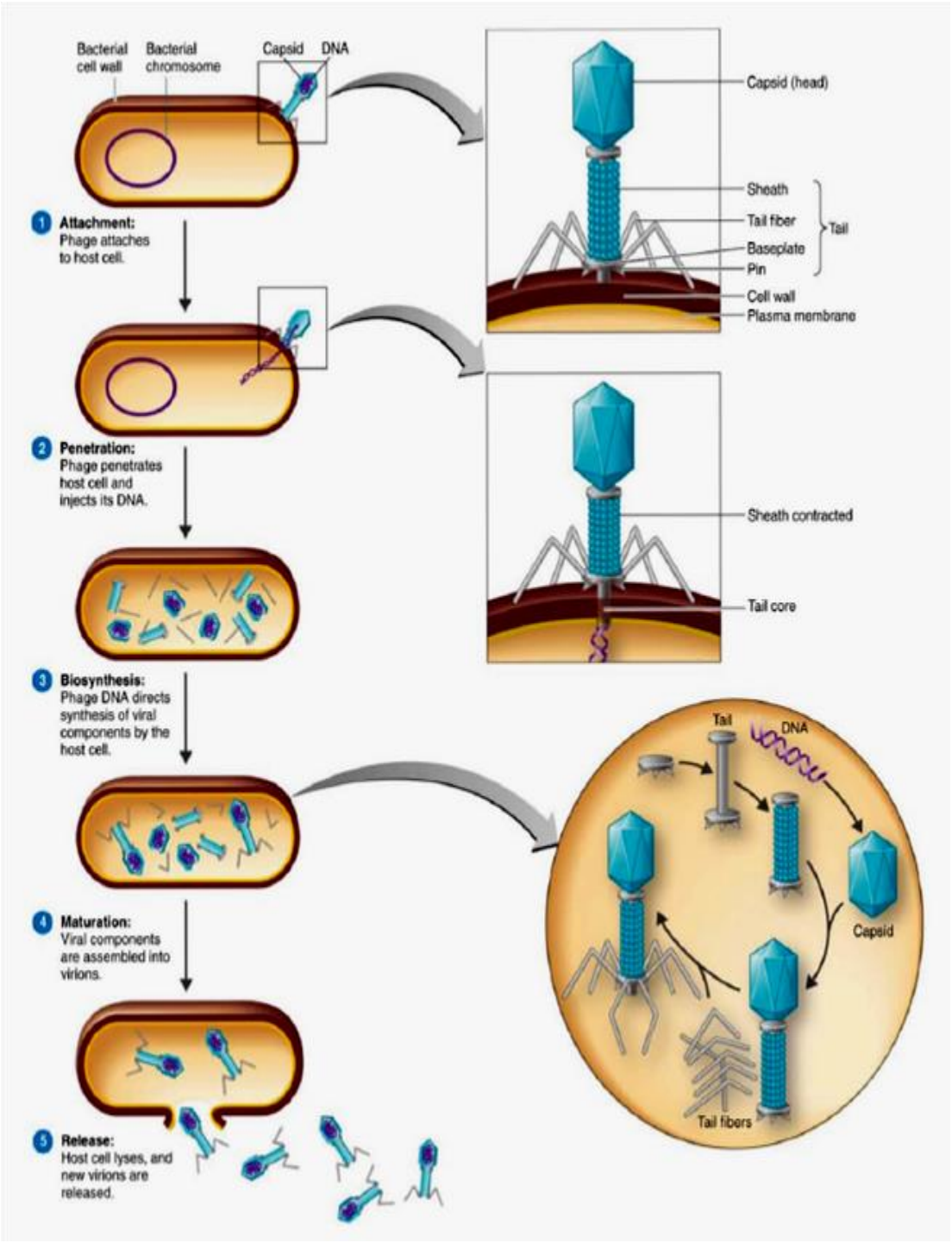
For several minutes following infection, complete phagescannot be found in the host cell. Only separate components-DNA and protein- can be detected. The period during viralmultiplication when complete, infective virions are not yet presentis called the eclipse period.

4) Maturation

In the next sequence of events, maturation occurs.In this process, bacteriophage DNA and capsids are assembledinto complete virions. The viral components essentially assembleinto a viral particle spontaneously, eliminating the need for manynonstructural genes and gene products. The phage heads and tailsare separately assembled from protein subunits, and the head isfilled with phage DNA and attached to the tail.

5) Release

The final stage of viral multiplication is the release ofvirions from the host cell. The term lysis is generally used for thisstage in the multiplication ofT-even phages because in this case, theplasma membrane actually breaks open (lyses). Lysozyme, which isencoded by a phage gene, is synthesized within the cell. Thisenzyme causes the bacterial cell wall to break down, and the newlyproduced bacteriophages are released from the host cell. Thereleased bacteriophages infect other susceptible cells in the vicinity,and the viral multiplication cycle is repeated within those cells.


Bacterial cell wall
Bacterial chromosome
Capsid
DNA
Capsid (head)
Sheath
Tail fiber
Baseplate
Pin
Tail
Cell wall
Plasma membrane
1 Attachment: Phage attaches to host cell.
2 Penetration: Phage penetrates host cell and injects its DNA.
Sheath contracted
Tail core
3 Biosynthesis: Phage DNA directs synthesis of viral components by the host cell.
Tail
DNA
Capsid
Tail fibers
4 Maturation: Viral components are assembled into virions.
5 Release: Host cell lyses, and new virions are released.

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3) Lysogeny / The Lysogenic Cycle

Bacteriophage Lambda (A.): The Lysogenic Cycle

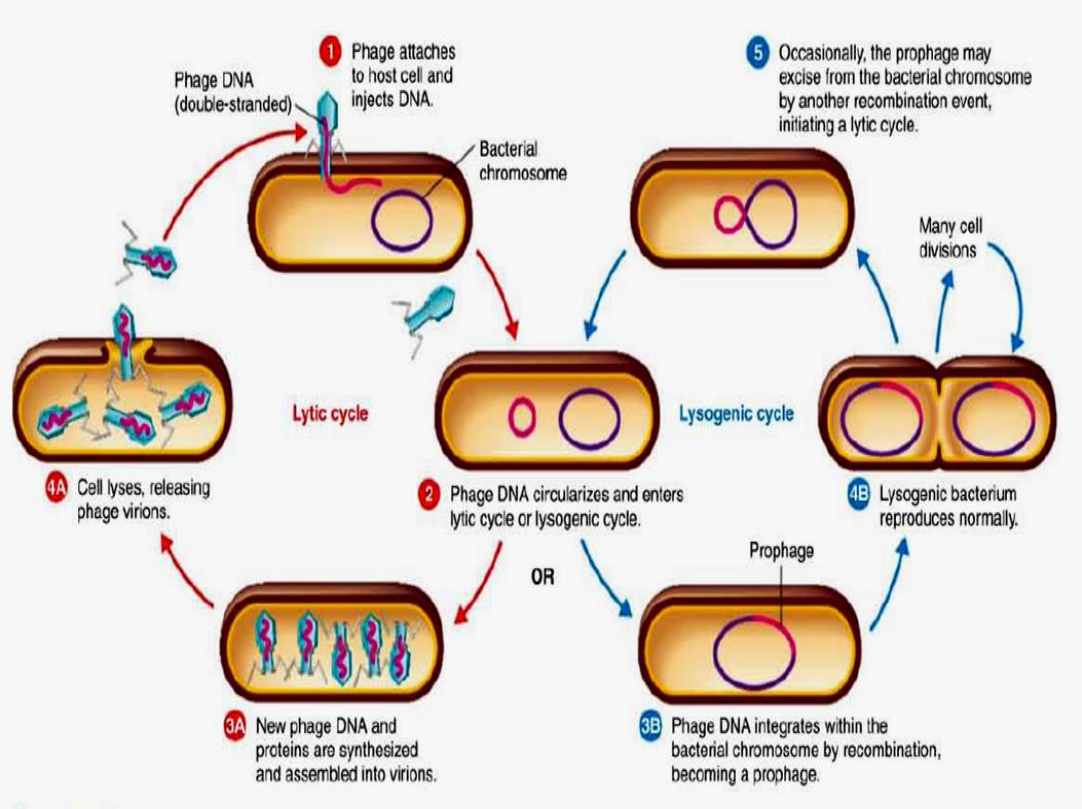
In contrast to T-even bacteriophages, some viruses do not causelysis and death of the host cell when they multiply. Theselysogenic phages (also called temperate phages) may indeed proceed through a lytic cycle, but they are also capable of incorporatingtheir DNA into the host cell's DNA to begin a lysogeniccycle. In lysogeny, the phage remains latent (inactive). The participatingbacterial host cells are known as lysogenic cells.
We will use the bacteriophage A (lambda), a well-studied lysogenic phage, as an example of the lysogenic cycle.

- Upon penetration into an E. coli cell,
- the originally linear phage DNA forms a circle.
- This circle can multiply and be transcribed,
- leading to the production of new phage and to cell lysis (the lytic cycle).

Alternatively, the circle can recombine with and becomepart of the circular bacterial DNA (the lysogenic cycle). Theinserted phage DNA is now called a prophage. Most of theprophage genes are repressed by two repressor proteins thatare the products of phage genes. These repressors stop transcriptionof all the other phage genes by binding to operators.

Thus, the phage genes that would otherwise direct thesynthesis and release of new virions are turned off, in muchthe same way that the genes of the E. coli lac operon areturned off by the lac repressor.

Every time the host cell's machinery replicates the bacterialchromosome,e it also replicates the prophage DNA. The prophage remainslatent within the progeny cells.However, a rare spontaneous event, or the action of UV lightor certain chemicals, can lead to the excision (popping-out)of the phage DNA, and to initiation of the lytic cycle.



There are three important results of lysogeny.

The first result

The lysogeniccells are immune to reinfection by the same phage.(However, the host cell is not immune to infection by otherphage types.)

The second result

The second result of lysogeny is phage conversion;that is, the host cell may exhibit new properties. For example, thebacterium *Corynebacteriumdiphtheriae*, which causes diphtheria,is a pathogen whose disease-producing properties are related to the synthesis of a toxin. The organism can produce toxin onlywhen it carries a lysogenic phage, because the prophage carries the gene coding for the toxin.

As another example, only streptococci carrying a lysogenic phage are capable of causing toxicshock syndrome.

The toxin produced by *Clostridium botulinum*,which causes botulism, is encoded by a prophage gene,

Shiga toxin produced by pathogenic strains of *E. coli*.

The third result

The third result of lysogeny is that it makes specializedtransduction possible. Bacterialgenes can be picked up in a phage coat and transferred to another bacterium in a process called generalized transduction. Any bacterial genes can be transferred bygeneralized transduct ion because the host chromosome is broken down into fragments, any of which can be packaged into aphage coat. In specialized transduction, however, only certainbacterial genes can be transferred.

Specialized transduction is mediated by a lysogenic phage,which packages bacterial DNA along with its own DNA in thesame capsid. When a prophage is excised from the host chromosome,adjacent genes from either side may remain attached to thephage DNA. Bacteriophage'" has picked up thegal gene for galactose fermentation from its galactose-positivehost. The phage carries this gene to a galactose-negative cell,which then becomes galactose-positive.

Certain animal viruses can undergo processes very similar tolysogeny. Animal viruses that can remain latent in cells for longperiods without multiplying or causing disease may becomeinserted into a host chromosome or remain separate from hostDNA in a repressed state (as some lysogenic phages). Cancercausingviruses may also be latent.

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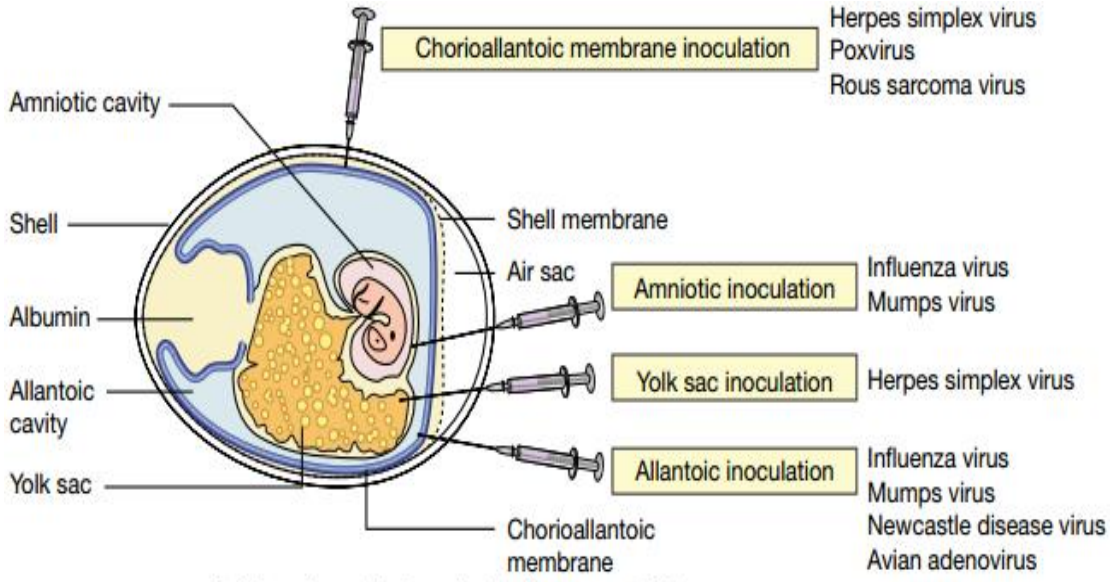
4) Cultivation of viruses: Egg inoculation and Tissue Culture

Cultivation of viruses: Egg inoculation(Inoculation into embryonated egg)

- Good pasture in 1931 first used the embryonated hen's egg for the cultivation of virus.
- The process of cultivation of viruses in embryonated eggs depends on the type of egg which is used.
- Viruses are inoculated into chick embryo of 7-12 days old.
- For inoculation, eggs are first prepared for cultivation, the shell surface is first disinfected with iodine and penetrated with a small sterile drill.
- After inoculation, the opening is sealed with gelatin or paraffin and incubated at 36°c for 2-3 days.
- After incubation, the egg is broken and virus is isolated from tissue of egg.

- Viral growth and multiplication in the egg embryo is indicated by the death of the embryo, by embryo cell damage, or by the formation of typical pocks or lesions on the egg membranes
- Viruses can be cultivated in various parts of egg like chorioallantoic membrane, allantoic cavity, amniotic sac and yolk sac.



Cultivation of virus in Embryonated Egg

1. Chorioallantoic Membrane (CAM):

- Inoculation is mainly for growing poxvirus.
- After incubation and incubation, visible lesions called pocks are observed, which is grey white area in transparent CAM.
- Herpes simplex virus is also grown.
- Single virus gives single pocks
- This method is suitable for plaque studies.

2. Allantoic cavity:

- Inoculation is mainly done for production of vaccine of influenza virus, yellow fever, rabies.
- Most of avian viruses can be isolated using this method.

3. Amniotic sac:

- Inoculation is mainly done for primary isolation of influenza virus and the mumps virus.

- Growth and replication of virus in egg embryo can be detected by haemagglutination assay.

4. Yolk sac inoculation:

- It is also a simplest method for growth and multiplication of virus.
- It is inoculated for cultivation of some viruses and some bacteria (Chlamydia, Rickettsiae)
- Immune interference mechanism can be detected in most of avian viruses.

Advantages of Inoculation into embryonated egg

1. Widely used method for the isolation of virus and growth.
2. Ideal substrate for the viral growth and replication.
3. Isolation and cultivation of many avian and few mammalian viruses.
4. Cost effective and maintenance is much easier.
5. Less labor is needed.
6. The embryonated eggs are readily available.
7. Sterile and wide range of tissues and fluids
8. They are free from contaminating bacteria and many latent viruses.
9. Specific and non specific factors of defense are not involved in embryonated eggs.
10. Widely used method to grow virus for some vaccine production.

Disadvantages of Inoculation into embryonated egg

1. The site of inoculation for varies with different virus. That is, each virus has different sites for their growth and replication.

Cultivation of viruses: Tissue CultureCell Culture

There are three types of tissue culture; organ culture, explant culture and cell culture.

Organ cultures are mainly done for highly specialized parasites of certain organs e.g. tracheal ring culture is done for isolation of coronavirus.
Explant culture is rarely done.
Cell culture is mostly used for identification and cultivation of viruses.

The following steps describe an overall strategyfor generating primary cell cultures. It is of utmostimportance that all work is done under sterile conditions.

1) The desired tissue is removed from the animal and is chopped or minced.
2) Tissue fragments are treated with enzymes such as collagenase to degrade the extracellular matrix and release single cells and small aggregates of cells.
3) Cells are pelleted by centrifugation and are resuspended in buffered saline or cell culture media.
4) Additional centrifugation steps may be performed to separate single cells from cell aggregates.
5) Cells and growth media are added to culture dishes and are maintained in a humidified incubator (37^0C, 5% CO_2).
6) Cells attach to the bottom of the dish where they grow and divide to form a monolayer.
7) The cells can be removed with trypsin, washed, and divided among new culture plates or dishes. This is called a passage, and is done to increase cell number.

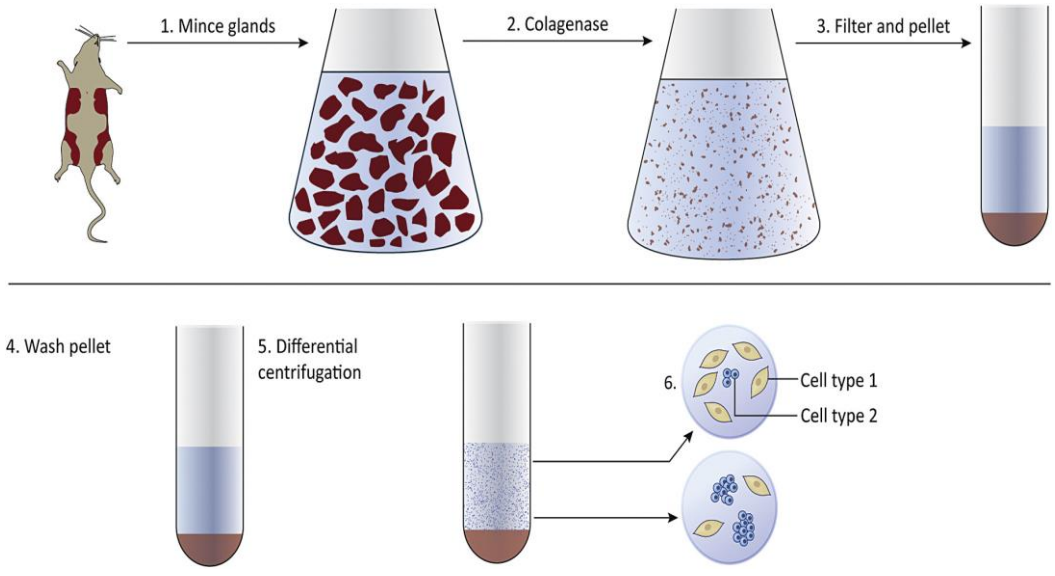


FIGURE -Generating cell cultures begins with removing tissues (normal or tumor) from an animal. Tissues are minced and treated withenzymes to degrade the extracellularmatrix. Centrifugation is used to pellet the cells. Cells are resuspended in media and placed in culturevessels

Types of cell culture

1. Primary cell culture:

- These are normal cells derived from animal or human cells.
- They are able to grow only for limited time and cannot be maintained in serial culture.
- They are used for the primary isolation of viruses and production of vaccine.
- Primary cells can be propagated for only a limited number of passages before the cells undergo a crisis and the culture dies.
- Examples: Monkey kidney cell culture, Human amnion cell culture

2. Diploid cell culture (Semi-continuous cell lines):

- They are diploid and contain the same number of chromosomes as the parent cells.
- Embryonic cells can be passaged many more times than cells taken from adults. Some types of cells (for example, fibroblasts) divide more readily than do cells that are normally nondividing in the adult animal (for example, neurons).
- They can be sub-cultured up to 50 times by serial transfer following senescence and the cell strain is lost.
- They are used for the isolation of some fastidious viruses and production of viral vaccines.
- Examples: Human embryonic lung strain, Rhesus embryo cell strain.

3. Tumor derived cells /Heteroploid cultures (Continuous cell lines):

- They are derived from cancer cells.
- They can be serially cultured indefinitely so named as continuous cell lines.
- Tumors provide another source of cells for virus culture. Tumor derived cells can often be passaged indefinitely. These immortalized cells are excellent tools for the virologist.
- They are relatively easy to culture, many types are commercially available and they can be genetically modified. Multiple genes can be introduced, mutated, or deleted to generate an unlimited supply of “designer” cells.
- They can be maintained either by serial subculture or by storing in deep freeze at -70°c.

- Due to derivation from cancer cells they are not useful for vaccine production.
- Examples: HeLa (Human Carcinoma of cervix cell line), HEP-2 (HummanEpithelioma of larynx cell line), Vero (Vervet monkey) kidney cell lines, BHK-21 (Baby Hamster Kidney cell line).

Susceptible Cell Lines

1. **Herpes Simplex** Vero Hep-2, human diploid (HEK and HEL),human amnion
2. **VZV** human diploid (HEL, HEK)
3. **CMV** human diploid fibroblasts
4. **Adenovirus** Hep2, HEK,
5. **Poliovirus** MK, BGM, LLC-MK2, human diploid, Vero, Hep-2,Rhadomyosarcoma
6. **Coxsackie B** MK, BGM, LLC-MK2, vero, hep-2
7. **Echo** MK, BGM, LLC-MK2, human diploid, Rd
8. **Influenza A** MK, LLC-MK2, MDCK
9. **Influenza B** MK, LLC-MK2, MDCK
10. **Parainfluenza** MK, LLC-MK2
11. **Mumps** MK, LLC-MK2, HEK, Vero
12. **RSV** Hep-2, Vero
13. **Rhinovirus** human diploid (HEK, HEL)
14. **Measles** MK, HEK
15. **Rubella** Vero, RK13

Advantages of cell culture

i. Relative ease, broad spectrum, cheaper and sensitivity

Disadvantage of cell culture

1. The process requires trained technicians with experience in working on a full time basis.
2. State health laboratories and hospital laboratories do not isolate and identify viruses in clinical work.
3. Tissue or serum for analysis is sent to central laboratories to identify virus.

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5) List of common viral diseases with causative agents and important symptoms in human beings, animals and plants,

List of common viral diseases with causative agents and important symptoms in human beings.

Sr. No.	Name of the viral disease	Name of the causative virus	Symptoms	Transmitted through
	Common Cold	Rhinovirus	Running nose, Sneezing, Temperature, Body pains	Air
	Influenza (Flu)	Influenza virus (RNA virus)	Temperature, Body pains	Air
	Covid-19 (Coronavirus disease 2019)	Severe Acute Respiratory Syndrome Corona Virus 2 (SARS-CoV-2).	Cough, Fever, Headache, Breathing difficulty, Loss of smell and taste, sometimes death	Air contaminated by droplets and small airborne particle s containing the virus. Contaminated fluids splashed or sprayed in the eyes, nose, or mouth, or, more rarely, via contaminated surfaces.
	Poliomyelitis (Polio)	Poliovirus	sore throat and fever, Headache, Paralysis	Food and water
	Rabies	Rabies lyssavirus	nausea, vomiting, violent movements, uncontrolled excitement, fear of water, an inability to move parts of the body, confusion, and loss of consciousness, death	infected animal bites or scratches commonly dogs
	Hepatitis A (Jaundice) Liver Infection	Hepatitis A virus (HAV)	Tiredness and weakness, Loss of appetite, Yellowing of the skin and the whites of eyes, nausea, vomiting and diarrhoea, Yellow urine	contaminated food and water or through direct contact with an infectious person.

	Hepatitis B Liver Infection		Loss of appetite, nausea, vomiting and diarrhoea, Dark urine	direct contact with infected blood or certain bodily fluids.unsterile medical or dental equipment, unprotected sex, or unsterile needles,
	Measles (Govar)	Rubeola virus	high fever, runny nose, a cough, red and watery eyes, and small white spots inside the cheeks, rashes on the skin,blindness, encephalitis (brain swelling), severe diarrhoea and dehydration, ear infections, or severe respiratory infections such as pneumonia.	Air
	Mumps (Tonsilitis)	Mumps Virus	fever, headache, malaise, muscle pain, and loss of appetite. painful swelling of the parotid glands,	respiratory secretions such as droplets and saliva, as well as via direct contact with an infected person.
	AIDS (Acquired immunodeficiency syndrome)	HIV (Human Immunodificiency Virus)	Weight loss, Pneuomonia. Dysentry, Typhoid, Many infections due to lowering of immune system	Unsafe sexual contact with AIDS partner, Through blood and body fluids of affected persons.
	Genital warts, and cervical cancer.	Human papillomaviruses (HPV)	skin or mucous membrane growths (warts). genital HPV can cause cancer of the lower part of the uterus, cancers of the anus, penis, vagina, vulva and back of the throat (oropharyngeal)	sexually or through other skin-to-skin contact.

	viral gastroenteritis.	Rota virus	fever, vomiting, and watery diarrhea.	ontact with feces or by contact with contaminated objects, food, or water.
	Small pox	Smallpox virus	fever and vomiting.[5] This was followed by formation of ulcers in the mouth and a skin rash.	inhalation of airborne variola virus, usually droplets expressed from the oral, nasal, or pharyngeal mucosa of an infected person. direct contact with infected bodily fluids or contaminated objects (fomites) such as bedding or clothing.

List of common viral diseases with causative agents and important symptoms in animals.

Sr. No.	Name of the viral disease	Name of the causative virus	Symptoms	Transmitted through
	Lumpy skin disease (LSD)	capripox virus	fever, enlarged superficial lymph nodes and multiple nodules (measuring 2–5 centimetres (1–2 in) in diameter) on the skin and mucous membranes, swelling in their limbs and exhibit lameness, chronic debility, reduced milk production, poor growth, infertility, abortion, and sometimes death.	blood-feeding insects, such as certain species of flies and mosquitoes, or ticks, blood, nasal discharge, lacrimal secretions, semen and saliva, infected milk to suckling calves
	Foot-and-mouth disease.	Coxsackievirus A16	close-contact, animal-to-animal spread, long-distance aerosol spread and fomites, or inanimate objects,	high fever, blisters inside the mouth that lead to excessive secretion of stringy or foamy saliva and to drooling, and blisters on the feet that may rupture and cause lameness, swelling in the testicles of mature

				males, milk production can decline
	Newcastle disease of poultry	para-myxo virus	direct contact between healthy birds and the bodily discharges of infected birds. The disease is transmitted through infected birds' droppings and secretions from the nose, mouth, and eyes.	loss of appetite, coughing, gasping, nasal discharge, watery eyes, bright green diarrhoea and nervous signs such as paralysis and convulsions
	Porcine epidemic diarrhoea in pigs	Porcine epidemic diarrhoea virus	Direct transmission occurs through ingestion of virus-contaminated faeces. Indirect transmission occurs through vehicles as well as personnel, equipment or other types of faeces-contaminated objects including feed.	Watery diarrhoea and mild systemic signs such as pyrexia, anorexia and lethargy.
	Rabies, hydrophobia ("fear of water")	Rabies virus	transmitted through the bite of a rabid animal	virus infects the central nervous system of mammals, ultimately causing disease in the brain and death. fever and tingling at the site of exposure, nausea, vomiting, violent movements, uncontrolled excitement, fear of water, an inability to move parts of the body, confusion, and loss of consciousness

List of common viral diseases with causative agents and important symptoms in Plants.

Sr. No.	Name of the viral disease	Name of the causative virus	Symptoms	Transmitted through
	Discoloration of leaves.	Tobacco Mosaic Virus (TMV)	mottling; leaf distortion; and sometimes leaf death and defoliation.	contact between plants, and infected or contaminated seeds.
	Mottling, curling, and malformation of leavesin beans	Bean common mosaic virus	Mottling, curling, and malformation of leaves and a general stunting of the plant.	Aphid insects
	Mottling and black necrotic spots in cabbage, cauliflower,	Turnip mosaic virus	Mottling and black necrotic spots, leaf distortion and stunting	Aphid insects
	Chlorosis and blistering mottle of leaves in Capsicum; tomato; potato;	Cucumber mosaic and potato mosaic virus	Chlorosis and blistering mottle of leaves; plants are stunted.	Aphid insects
	Ringspots, line patterns, mottling, and chlorotic blotches on leaves in Capsicum; tomato; potatoes	Tomato Spotted Wilt Virus (TSWV)	Ringspots, line patterns, mottling, and chlorotic blotches on leaves.	Thrips insects
	capsicum: yellowing on leaf margins	Capsicum Chlorosis Virus (CaCV)	yellowing on leaf margins and between veins of young leaves; In tomato: chlorotic spots and blotches on leaves that become mottled.	Thrips insects
	Chlorosis or yellowing in Beet, Cucumber	Beet pseudoyellows virus (BPYV)	Chlorosis or yellowing between veins in older cucumber leaves,	Greenhouse whitefly

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6) Emerging human viruses

i) H1N1 influenza virus (Swine Flu)

ii) Avian influenza (Bird Flu)

iii) Ebola, also known as Ebola virus disease (EVD) and Ebola hemorrhagic fever (EHF)

iv) Chikungunya virus

v) Severe Acute Respiratory Syndrome (SARS)

vi) Nipah virus disease

vii) Zika Virus

i) H1N1 influenza virus (Swine Flu)

Swine flu (H1N1) is an infection that a type of flu (influenza) virus causes. It's called swine flu because it's similar to a flu virus that affects pigs (swine). The virus leads to a lung (respiratory) disease in pigs. Swine flu (H1N1) is a respiratory infection in humans.

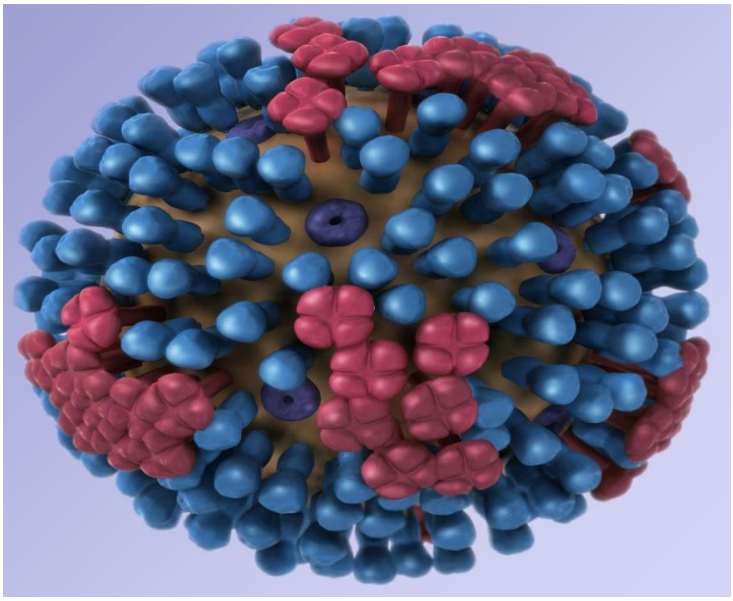
Influenza A virus subtype H1N1 (A/H1N1) is a subtype of influenza A virus. Major outbreaks of H1N1 strains in humans include the 1918 Spanish flu pandemic, the 1977 Russian flu pandemic and the 2009 swine flu pandemic.

In 1918, a deadly influenza pandemic caused by H1N1 influenza virus, also known as the Spanish flu, infected approximately 500 million people around the world and resulted in the deaths of 50 to 100 million people (3% to 5% of the world population) worldwide.

H1N1 influenza is a subtype of influenza A virus (a communicable viral disease), which causes upper, and potentially, lower respiratory tract infections in the host it infects, resulting in symptoms such as nasal

secretions, chills, fever, decreased appetite, and possibly lower respiratory tract disease.

The H1N1 influenza virus is an orthomyxovirus and produces virions that are 80 to 120 nm in diameter, with an RNA genome size of approximately 13.5 kb. It contains the glycoproteins hemagglutinin and neuraminidase. For this reason, they are described as H1N1, H1N2 etc., depending on the type of H or N antigens they express with metabolic synergy.



(H1N1) spreads from person to person. When a person coughs or sneezes, droplets go into the air. You can get the infection when you breathe in (inhale) the virus. You can also get the infection when you touch a contaminated surface and then touch your mouth, nose or eyes.

Symptoms of swine flu (H1N1)

The symptoms of swine flu (H1N1) are similar to the symptoms of regular flu. The symptoms may start three to five days after exposure to the virus. Symptoms may include:

Fever, Chills, Cough, Sore throat, Body or muscle aches, Headache, Fatigue,

Trouble breathing, Trouble waking up, Not drinking enough fluids, Fever with rash, Confusion.

Treatment

Get plenty of rest, Drink fluids, Eat a light diet, Stay home.

Take acetaminophen/ paracetamol to reduce fever and relieve aches and pains.

Antiviral drugs such as oseltamivir or zanamivir can kill the virus.

Prevention

Flu vaccine

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ii) Avian influenza (Bird Flu)

Avian influenza (AI), also called avian flu or bird flu, is caused by a group of influenza A viruses that spreads among birds. In rare cases, it can affect humans. Highly pathogenic avian influenza (HPAI) viruses can cause large numbers of illnesses and deaths in chickens and other poultry and sometimes in wild birds.

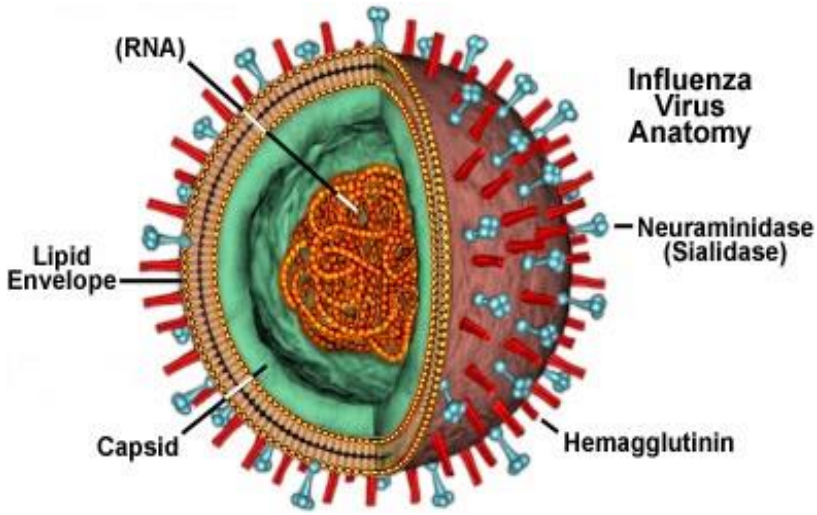
This complex disease is caused by viruses divided into multiple subtypes (i.e. H5N1, H5N3, H5N8 etc.)

The highly pathogenic influenza A virus subtype H5N1 is an emerging avian influenza virus that is causing global concern as a potential pandemic threat. It is often referred to simply as "bird flu" or "avian influenza". H5N1 has killed millions of poultry in a growing number of countries throughout Asia, Europe, and Africa.

Avian influenza virus belongs to the family Orthomyxoviridae. It is an enveloped virus, single-stranded RNA genome of negative polarity, with a length of about 13.5 kb in total.

Transmission

In birds, AI viruses are shed in the faeces and respiratory secretions. They can all be spread through direct contact with secretions from infected birds, especially through faeces or through contaminated feed and water.



Symptoms in birds

Temperature, swollen head, closed and runny eyes, sudden death

Symptoms of bird flu in humans

High temperature or feeling hot or shivery, aching muscles, headache, cough or shortness of breath, diarrhoea, sickness, stomach pain, chest pain, bleeding from the nose and gums, conjunctivitis.

Treatment

Get plenty of rest, Drink fluids, Eat a light diet, Stay home.

Take acetaminophen/ paracetamol to reduce fever and relieve aches and pains.

Antiviral drugs such as oseltamivir or zanamivir can kill the virus.

Prevention

H5N1 vaccine.

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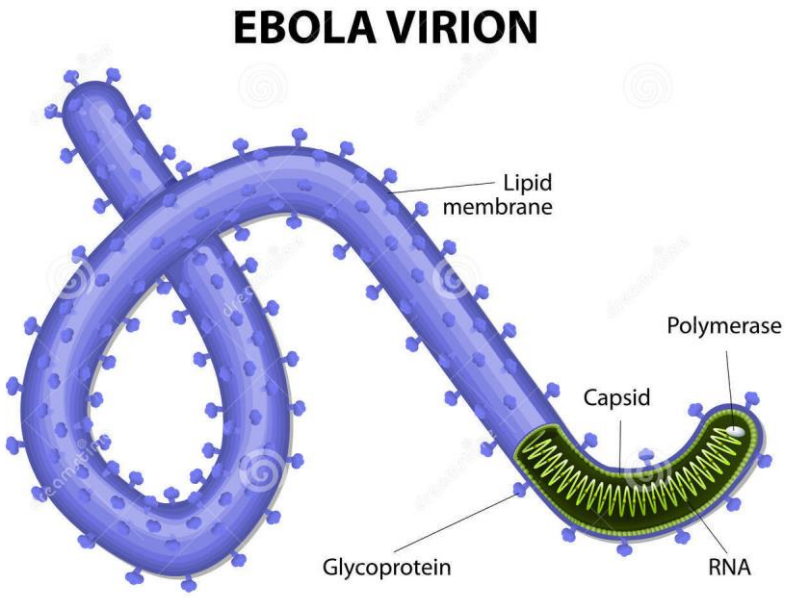
iii) Ebola, also known as Ebola virus disease (EVD) and Ebola hemorrhagic fever (EHF)

Ebola, also known as Ebola virus disease (EVD) and Ebola hemorrhagic fever (EHF), is a viral hemorrhagic fever in humans and other primates, caused by Ebola viruses.

Death rates from outbreaks of Ebola in the past have ranged from 25% to 90%. There are five types of Ebola virus. Four of them cause the disease in humans. The Ebola virus first appeared during two 1976 outbreaks in Africa. Ebola gets its name from the Ebola River, which is near one of the villages in the Democratic Republic of Congo where the disease first appeared.

Ebola is an enveloped, non-segmented, negative-stranded RNA virus. The ebola nucleoprotein wraps around the RNA, creating a helical complex.



Symptoms of EVD

Fever, Fatigue, Muscle pain, Headache, Sore throat, Vomiting, Diarrhoea, Rash, Symptoms of impaired kidney and liver function, In some cases, both internal and external bleeding (for example, oozing from the gums, or blood in the stools), Laboratory findings include low white blood cell and platelet counts and elevated liver enzymes.

Transmission

It is thought that fruit bats of the Pteropodidae family are natural Ebola virus hosts. Ebola is introduced into the human population through close contact with the blood, secretions, organs or other bodily fluids of infected animals such as fruit bats, chimpanzees, gorillas, monkeys, forest antelope or porcupines found ill or dead or in the rainforest.

Ebola then spreads through human-to-human transmission via direct contact (through broken skin or mucous membranes) with:

- Blood or body fluids of a person who is sick with or has died from Ebola
- Objects that have been contaminated with body fluids (like blood, feces, vomit) from a person sick with Ebola or the body of a person who died from Ebola

Treatment

Supportive care - rehydration with oral or intravenous fluids - and treatment of specific symptoms improves survival.

Two monoclonal antibodies (Inmazeb and Ebanga) were approved for the treatment of Zaire ebolavirus (Ebolavirus)

Prevention

Ebola vaccine

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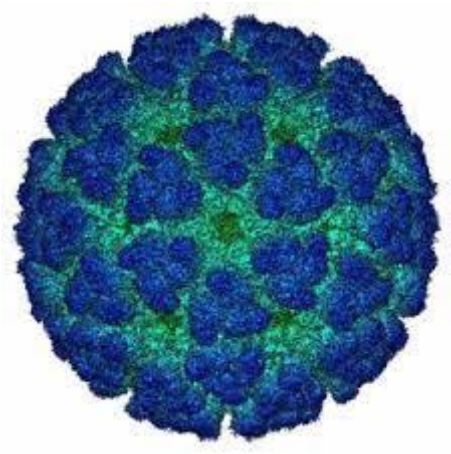
iv) Chikungunya virus

Chikungunya is an infection caused by the Chikungunya virus (CHIKV).

Chikungunya is a viral disease transmitted to humans by infected mosquitoes. The virus is primarily spread by the *Aedes aegypti* AND *Aedes albopictus* mosquito, which also transmits dengue fever and Zika virus. They mainly bite during the day. Chikungunya is not transmitted from one person to another.

Chikungunya word comes from the African Makonde language and means "bent over in pain."

The CHIKV genome consists of a single positive-stranded RNA molecule of 11.8 kb in length.



Symptoms

The symptoms of chikungunya usually appear between 3-7 days after being bitten by an infected mosquito and include fever, joint pain, headache, muscle pain, joint swelling, and rash. The joint pain associated with chikungunya can be severe and debilitating, and it can last for months or even years.

Treatment

There is currently no specific antiviral treatment for chikungunya, and the focus is on relieving the symptoms of the disease through pain relief and anti-inflammatory medication. Take paracetamol to reduce fever and pain. Do not take aspirin and other non-steroidal anti-inflammatory drugs (NSAIDS) until dengue can be ruled out to reduce the risk of bleeding.

Prevention

The best way to prevent chikungunya is to avoid mosquito bites by using mosquito repellent, wearing protective clothing, and staying indoors during peak mosquito hours.

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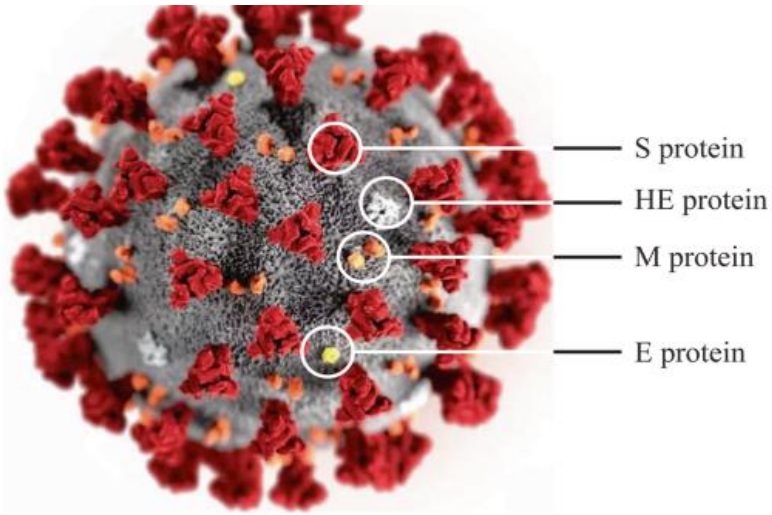
v) Severe Acute Respiratory Syndrome (SARS)

Severe acute respiratory syndrome (SARS) is a viral respiratory disease caused by a SARS-associated coronavirus (SARS-CoV or SARS-CoV-1). It was first identified at the end of February 2003 during an outbreak that emerged in China and spread to 4 other countries.

In December 2019, another strain of SARS-CoV was identified as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). This new strain causes coronavirus disease 2019 (COVID-19), a disease that brought about the COVID-19 pandemic.



Signs and symptoms

SARS produces flu-like symptoms which may include fever, muscle pain, lethargy, cough, sore throat, and other nonspecific symptoms. The only symptom common to all patients appears to be a fever above 38 °C (100 °F). SARS often leads to shortness of breath and pneumonia, which may be direct viral pneumonia or secondary bacterial pneumonia.

Transmission

When someone with SARS coughs or sneezes, infected droplets spray into the air. You can catch the SARS virus if you breathe in or touch these particles. The SARS virus may live on hands, tissues, and other surfaces for up to several hours in these droplets. It also spreads by hands and other

objects the droplets have touched. Airborne transmission is a real possibility in some cases.

Prevention

There is no vaccine for SARS, but now vaccine is available for Covid-19.

Clinical isolation and quarantine remain the most effective means to prevent the spread of SARS. Other preventive measures include:

- Hand-washing with soap and water, or use of alcohol-based hand sanitizer
- Disinfection of surfaces of fomites to remove viruses
- Avoiding contact with bodily fluids
- Washing the personal items in hot, soapy water (eating utensils, dishes, bedding, etc.)
- Avoiding travel to affected areas
- Wearing masks and gloves

Treatment

As SARS is a viral disease, antibiotics do not have direct effect but may be used against bacterial secondary infection. Treatment of SARS is mainly supportive with antipyretics, supplemental oxygen and mechanical ventilation as needed.

There is currently no proven antiviral therapy. Tested substances, include ribavirin, lopinavir, ritonavir, type I interferon, that have thus far shown no conclusive contribution to the disease's course. Administration of corticosteroids, is recommended by the British Thoracic Society/British Infection Society/Health Protection Agency in patients with severe disease and O2 saturation of <90%.

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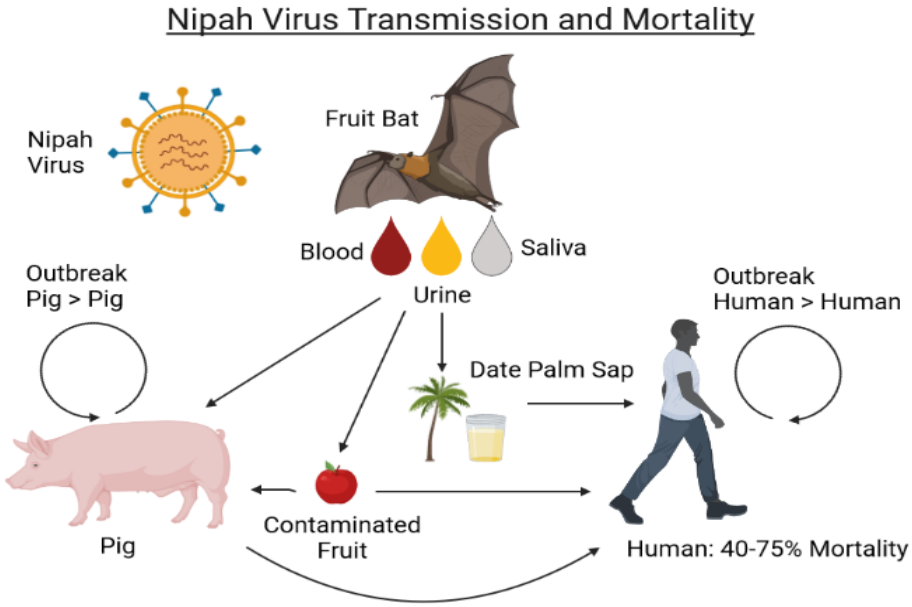
vi) Nipah virus disease

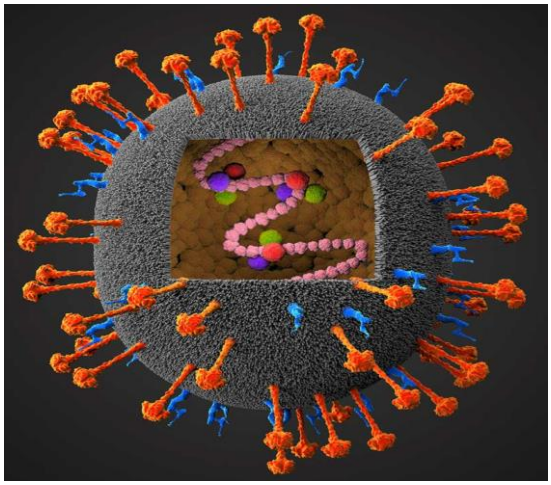
Nipah virus (NiV) causes emerging infectious diseases which first appeared in domestic pigs in Malaysia and Singapore in 1998 and 1999, when over 1 million pigs were destroyed to control the disease. Very closely related to Hendra virus (HeV), Nipah virus causes respiratory and occasionally nervous signs in pigs, and it has devastating zoonotic potential. In the Malaysian outbreak, most human infections presented as

an encephalitic syndrome and up to 50% of clinically affected human cases died. The disease is named after a village in Malaysia, Sungai Nipah.

Fruit bats of the Pteropodidae family are the natural host of Nipah virus.

The Nipah virus (NiV) is a type of RNA virus in the genus Henipavirus. The virus normally circulates among some fruit bats. It can both spread between people and from other animals to people.





Signs and symptoms

Infected people initially develop symptoms that include fever, headaches, myalgia, vomiting and sore throat. This can be followed by dizziness, drowsiness, altered consciousness, and neurological signs that indicate acute encephalitis. Some people can also experience atypical

pneumonia and severe respiratory problems, including acute respiratory distress. Encephalitis and seizures occur in severe cases, progressing to coma within 24 to 48 hours.

Transmission

Nipah virus can be transmitted to humans from animals (such as bats or pigs), or contaminated foods and can also be transmitted directly from human-to-human. Infection via bats can be caused by drinking raw palm sap (palm toddy) contaminated by bat excreta, eating fruits partially consumed by bats, and using water from wells infested by bats Subsequent human-to-human transmission of Nipah virus occurs via close contact with NiV-infected persons or exposure to NiV-infected body fluids (e.g., blood, urine, nasal secretions).

Treatment

There are currently no drugs specific for Nipah virus infection. The mainstay of treatment is supportive care. Acyclovir, favipiravir and remdesivir have been assessed as potential antivirals against Nipah virus.

Prevention

There are currently no vaccines specific for Nipah virus infection. Prevention through sanitary practices is the best protection. The infection through animal transmission can be reduced by avoiding exposure to sick pigs, and to bats where the disease is endemic.

Avoid palm toddy and contaminated fruits and water by bats because infection via bats can be caused by drinking raw palm sap (palm toddy) contaminated by bat excreta, eating fruits partially consumed by bats, and using water from wells infested by bats.

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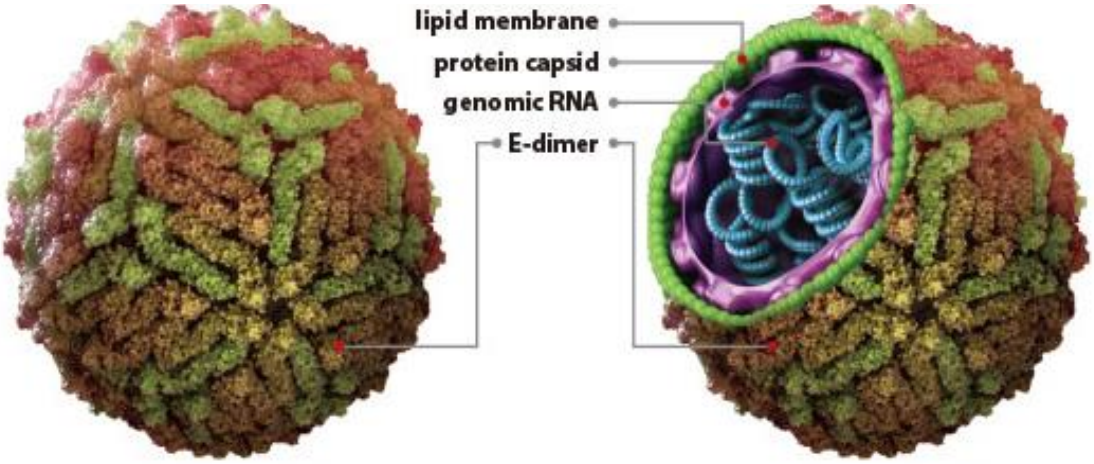
vii) Zika Virus

Zika virus is spread by daytime-active Aedes mosquitoes, such as *A. aegypti* and *A. albopictus.* Its name comes from the Ziika Forest of Uganda, where the virus was first isolated in 1947 in a Rhesus macaque monkey. Zika virus shares a genus with the dengue, yellow fever, Japanese encephalitis, and West Nile viruses.

Evidence of infection and disease in humans in other African countries occurred in the 1950s. From the 1960s to 1980s, sporadic human infections were detected across Africa and Asia. However, since 2007 outbreaks of Zika virus disease have been recorded in Africa, the Americas, Asia and the Pacific.

Zika virus belongs to the family Flaviviridae and the genus Flavivirus. is enveloped and icosahedral and has a nonsegmented, single-stranded, 10 kilobase, positive-sense RNA genome.



Symptoms

Most people infected with Zika virus do not develop symptoms. Among those who do, they typically start 3–14 days after infection, are generally mild including rash, fever, conjunctivitis, muscle and joint pain, malaise and headache.

Complications

Zika virus infection during pregnancy is a cause of microcephaly and other congenital malformations in the infant, including limb contractures, high muscle tone, eye abnormalities and hearing loss. These clinical features are collectively referred to as congenital Zika syndrome.

Transmission

Zika virus is primarily transmitted by infected mosquitoes of the Aedes genus, mainly *Aedes aegypti,* in tropical and subtropical regions. Aedes mosquitoes usually bite during the day. These mosquitoes also transmit dengue, chikungunya and urban yellow fever.

Treatment

There is no specific treatment available for Zika virus infection or disease. People with symptoms such as rash, fever or joint pain should get plenty of rest, drink fluids, and treat symptoms with antipyretics and/or analgesics. Nonsteroidal anti-inflammatory drugs should be avoided until dengue virus infections are ruled out because of bleeding risk.

Prevention

No vaccine is yet available for the prevention or treatment of Zika virus infection.

Protection against mosquito bites during the day and early evening is a key measure to prevent Zika virus infection, especially among pregnant women, women of reproductive age and young children.

Personal protection measures include wearing clothing (preferably light-coloured) that covers as much of the body as possible; using physical barriers such as window screens, mosquito nets and closed doors and windows; and applying insect repellent to skin or clothing.

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7) Viruses and cancer

Cancer develops when cells start to divide uncontrollably because the 'cell cycle machinery' that regulates this process stops working properly. These cancer cells can then invade other tissues. Cancer development is a complex process involving a series of genetic changes that disrupt the cell cycle machinery, interfering with cellular functions such as cell growth.

Some infectious agents, especially viruses, play a key role in the development of certain cancers by contributing to these genetic changes, although cancer itself is not an infectious disease. These viruses are known as tumour viruses or oncogenic viruses.

Other genetic, lifestyle, physical factors (such as x-rays, UV rays, Alpha, Beta and Gamma rays), Chemicals and environmental factors also contribute to cancer development.

Viruses can disrupt cell behavior in several different ways.

i. They can directly cause DNA damage (mutations) by inserting their genomes into the DNA of the host cell. The integration can disrupt important regulatory genes.
ii. The viruses may contain their own genes that disrupt the regulation of the cell. This process may be beneficial to the virus if it allows for rapid production of progeny but can be seriously detrimental to the host.
iii. Some viruses actually carry altered versions of genes that they have picked up from previous host cells. These altered genes no longer function properly, and when they are inserted into a new host cell, they cause disregulation and can lead to cancerous growth.
iv. Through their mutagenic activity or their effects on cell behavior, viruses play a significant role in the development of particular cancers in many different animals, including humans.

Seven human viruses have been linked to specific cancers. The involvement of these viruses in human cancer development means that the frequency of these cancers can be reduced either prophylactically by vaccinating against the viruses, or therapeutically by treating the infections.

It's important to note that not everyone who is infected with these viruses will develop cancer. However, infection with these viruses can increase a person's risk of developing cancer.

1) Epstein-Barr Virus (EBV) - Burkitt's Lymphoma
2) Hepatitis B Virus (HBV) - Liver Cancer
3) Hepatitis C Virus (HCV) - Liver Cancer
4) Human Herpes virus 8 (HH8) - Kaposi's Sarcoma
5) Human Papilloma virus (HPV) - Cervical Cancer, Head and Neck Cancers, Anal, Oral, Pharyngeal, and Penile Cancers
6) Human T-cell Lymphotropic Virus 1 (HTLV) - Adult T-cell Leukemia
7) Merkel Cell Polyoma virus - Skin Cancer (Merkel Cell Carcinoma)

1) Epstein-Barr Virus (EBV) - Burkitt's Lymphoma

B cell Infection is necessary for EBV mediated carcinogenesis. Only a small percentage of infections lead to cancer. These patients are especially susceptible because they lack sufficient immune function to inhibit the growth of infected B cells. Mechanism of transmission is possibly through saliva.

2) Hepatitis B Virus (HBV) - Liver Cancer

Hepatitis viruses (B and C) are responsible for 70-85% of primary liver cancers. HBV is transmitted via contact with contaminated blood, sweat, or tears. It can also be spread through sexual contact and from mother to child.

3) Hepatitis C Virus (HCV) - Liver Cancer

Hepatitis viruses (B and C) are responsible for 70-85% of primary liver cancers. HCV is transmitted primarily through blood-blood contact.

4) Human Herpes virus 8 (HH8) - Kaposi's Sarcoma

HHV8 primarily causes Kaposi's Sarcoma (KS), a type of cancer that affects the skin and soft organs. HHV8 is also associated with several blood disorders. HHV8 is most commonly spread through sexual contact and via saliva. Transmission also may occur via organ transplantation or blood transfusion.

5) Human Papilloma virus (HPV)

The human papilloma virus is the primary risk factor for cervical cancer. The human papilloma virus is transmitted via skin-skin contact.

Sexual intercourse is not necessary for transmission, but is the most common route. The virus can infect the genital, anal, and oral regions of the body.

6) Human T-cell Lymphotropic Virus 1 (HTLV)

HTLV causes Adult T-cell Leukemia (ATLL). HTLV can be transmitted via sexual or blood-blood contact. It can also be passed through breast milk and from mother to fetus.

7) Merkel Cell Polyoma virus (MCV)

MCV causes skin cancer that affects Merkel cells. Merkel cells are located in the outer layer of skin (epidermis).

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8) Viruses used in Recombinant DNA technology

Viruses can be used as vectors in recombinant DNA technology to introduce new genes into host cells. Viral vectors are modified forms of viruses that have been engineered to carry foreign DNA into cells. The DNA of interest is inserted into the viral vector, which then infects the host cell and delivers the foreign DNA into the cell's genome.

Vectors are essentially carrier molecules that are used to transfer genetic material, such as DNA, from one organism to another. In the case of recombinant DNA technology, the vectors are used to introduce specific genes into cells, so that the cells can then produce the protein encoded by the introduced gene.

Viruses are particularly useful as vectors because they have evolved to efficiently transfer genetic material into cells. By modifying the genetic material within a virus, scientists can use the virus to introduce foreign genes into cells. Once the virus has infected a cell, the viral DNA or RNA can be incorporated into the host cell's DNA, allowing the foreign gene to be expressed by the host cell.

There are several advantages to using viral vectors in recombinant DNA technology. First, viruses are naturally efficient at delivering their genetic material into cells, so they can be used to efficiently introduce new genes into target cells. Second, viral vectors can be designed to target specific types of cells, which can be useful for gene therapy applications.

Finally, viral vectors can be produced in large quantities relatively easily, making them an attractive option for large-scale gene therapy or genetic engineering projects.

Some examples of viruses that are used as vectors in genetic engineering include:

1) Adenovirus:

Adenovirus is a non-enveloped virus that can infect a wide range of mammalian cells, including human cells. It is commonly used as a vector for gene therapy and vaccine development.

2) Lentivirus:

Lentivirus is an enveloped virus that can integrate its genetic material into the host cell's genome. It is commonly used for long-term gene expression studies and gene therapy.

3) Retrovirus:

Retrovirus is an enveloped virus that can integrate its genetic material into the host cell's genome. It is commonly used for gene therapy and gene expression studies.

4) Herpes simplex virus (HSV):

HSV is a large, enveloped virus that can infect many different types of cells. It is commonly used as a vector for gene therapy and vaccine development.

5) Adeno-associated virus (AAV):

AAV is a non-enveloped virus that can integrate its genetic material into the host cell's genome. It is commonly used as a vector for gene therapy and gene editing.

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