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Viral Organization and Functioning: A Simple Unit with Complex Machinery

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Abstract:

An understanding of viral structure and behavior is essential to appreciate the complexity of viral diseases that afflict human hosts. It helps clinicians to effectively apply preventive strategies and available treatment options. In this commentary viral classification, capsid, design, life cycle, and metabolism are described.

Keywords: virus; virion; capsid; nucleic acid; viral metabolism

Introduction:

Viruses are filtrable non-living agents capable of causing disease. A virion is an infective viral unit of nucleoproteins, comprising of a genome of nucleic acids and multiple copies of one or more proteins. However, virions differ in size, shape, form, makeup, and intricacy. Although diverse, viruses share common properties such as their size, which ranges from 80-100 nm in diameter, and their dependency on internal cellular realm for survival (obligate parasites). They are composed of either an RNA or a DNA but not both (1).

Viral Classification:

Viruses are classified as follows: (2)

1. Classification based on the presence or absence of an outer lipid covering, ie. enveloped or non-enveloped
2. Classification based on nucleic acid genome, which consists of 7 different classes, namely, 1. Double stranded DNA (dsDNA), 2. Single stranded DNA (ssDNA), 3. Double stranded RNA (dsRNA), 4. Single stranded RNA+ (ssRNA+), 5. ssRNA-, 6. ssRNA whose copying involves reverse transcriptase (RT) that produces DNA from an RNA template, 7. dsDNA whose creation requires RT.
3. Classification based on the International Committee on Taxonomy of Viruses (ICTV) where taxonomy groups or taxa are created that include order, family, subfamily, genus, and species. Among these groups, the family assumes importance and only twenty-two out of ninety-four families are grouped into six orders consisting of Caudovirales, Herpesvirales, Mononegavirales, Nidovirales, Picornavirales, and Tymovirales.
4. Classification based on infected host cells:

Depending on the kind of cells the virus infects, they are called either prokaryotic or eukaryotic viruses. Those infecting prokaryotic bacteria are called bacteriophages or phages.

5. Classification as simple or complex viruses:

Viruses that do not require scaffolding proteins are classified as simple viruses and those that require scaffolding proteins belong to complex viruses.

6. Classification based on positive-strand or negative-strand viruses:

Positive-strand also called positive-sense consists of RNA that readily is translatable into protein (mRNA in a cell is positive-strand), Negative-strand, or negative-sense viruses are unable to translate RNA into proteins. Negative-strands viruses must undergo transformation into positive-strands for them to translate RNA into proteins.

Description of classified viruses:
Enveloped viruses:

The bilipid layer of enveloped viruses consists of simple or complex structure. The simple structure includes proteins, and the more complex structure includes multiple layers of proteins, lipid, and or nucleoproteins.

Non-enveloped or naked viruses:

In the absence of a bilipid outer covering, a protein shell coat, or capsid (CP) surrounds the virion consisting of copies of one or more proteins containing nucleic acid (3).

The viral coat:

The viral coat or protein shell CP, or box plays a role in the structure and function of a virus. The nucleic acid and CP form a nucleocapsid. It is a proportional and cavernous or hollow protein structure consisting of either oligomers (few repeating units) or multimers (multiple protein molecules) made from the occasional to multiple copies of polypeptides called the capsid protein subunits which are either helical or icosahedral or complex in structure.Helical CP forms ten percent of virus families and are simple in structure holding unlimited genetic information. It requires less energy to assemble and only one gene is needed for the storing and expression of genetic information, resulting in a reduced length of the nucleic acid. The viral nucleic acid coils into a helical (spiral) shape and the CP proteins curl outside or inside of the nucleic acid forming a long tube-like structure. The helical virus is described as enveloped e.g., influenza, measles, mumps etc. or non-enveloped e.g., tobacco mosaic virus. Icosahedral CPs,on the other hand, are limited by inherent geometrical constraints. The amount of genetic information stored is limited, although structural solutions have evolved to overcome such limitations. Icosahedral CPs occur in about fifty percent of viral families. It is more common than the helical structure. An icosahedron has twenty sides, each composed of an equilateral triangle. They can be enveloped or non-enveloped. Examples of icosahedral viruses include human papilloma virus, hepatitis B, rhino, and herpes. It requires less energy to assemble and is developmentally favored. Complex viral design is neither helical nor icosahedral. Pox and some bacteriophages are examples of viruses with complex design (4).

Virion design:

The basic design of the virion in non-enveloped virus is determined by the design of the CP. It is more complicated in enveloped viruses which are flexible and commonly spheroidal in design [1].

Nucleic acid component of the virus:

RNA or Ribo viruses use Ribonucleic Acid (RNA) as genetic material (5). ssRNA viruses use ssRNA and dsRNA viruses use ds RNA as genetic material. ssRNA viruses are divided into ssRNA+ and ssRNA -ve based on the polarity (divergence) of RNA genome string or strand. The term +ve polarity is used when the single stranded nucleic acid sequence corresponds to the mRNA sequence and the term -ve polarity is used when the nucleic acid sequence corresponds to the complementary of the mRNA sequences. Most viruses contain RNA genetic material. DNA viruses are divided into ssDNA or ds DNA viruses. Viruses belonging to the same family exhibit equivalent structural, functional, virion, capsid, and nucleic acid genome although they may differ significantly in some of the above-described features. Even striking differences in phenotype may occur between the same clone of viruses. Sometimes viruses from different families may share similar structure and functionality.

Techniques used to study viruses:

Viruses are studied using techniques such as transmission electron microscopy, cryo-electron microscopy, cryo-electron tomography, X-ray crystallography, nuclear magnetic resonance (NMR) spectroscopy, atomic force microscopy, and combine methods [2].

Viral life cycle:
Forms of viral infection:

1. Lytic infection wherein copious amounts of virion overwhelms the host cells,
2. Persistent virus infection occurs by the continuous generation of a small number of viruses that endure in cells without getting killed,
3. Latent infection arises when viral genome propagated in host cells is copied along with the genome of the host cells and transmitted by passing onto the progeny cells. Latent infection can become active infection leading to infection of other host cells.

Thus, a viral cycle involves a viral particle infecting a host cell, producing progeny viral particles in host cells and the eventual release of virus particles by lysis. Alternatively, a viral particle may remain dormant as a latent infection. Recognition and entry of a viral particle into a host cell involves recognition of receptor and co-receptors on the surface of host cells, internalization of the viral particle or some of its parts including the viral genome into the host cell and transportation of viral particle or one of its components in the host cell. This is followed by the uncoating of the viral genome and thereby releasing the viral nucleic acid. An eclipse phase is entered when the parental virus loses its identity and progeny viral particles are generated within the host cell. Viral genome expression and replication takes place next, and the host cellular metabolism is diverted for this purpose. Viral genes are transcribed into mRNAs followed by the synthesis of structural and functional proteins required for the formation of progeny viral particles. The replication involves a polymerase of the host cell or viral particle. Transportation of viral particles into intracellular sites of the host cell occurs next, to enable the assembling into a virion. The formation of a virion involves the assembly of the virus CP from CP subunits, the arranging of nucleic acid genome inside the CP which is followed by the maturation of a virus particle into an

infectious virion. Finally, the infectious virion is released from the host cell. As the virion navigates through extracellular environment it must survive many factors that may inactivate or neutralize it (6).

Metabolism of the virus:

Many new metabolic events are initiated by viral genetic material entering the host cell as well as altering structural and metabolic events of the host cell. Several virions are eventually assembled in the host cell. Viral gene expression requires genomic transcription which involves the synthesis of mRNA, involving a polymerase. mRNA undergoes translation into early or later viral proteins that are needed to sabotage host cell metabolism (7) and for the formation of structural viral proteins, respectively. The next process involves the formation of virions which consists of CP assembly, nucleic acid packaging, and maturation of the virus particles. CP assembly may either be self-assembly, scaffolding protein-assisted assembly, or nucleic acid assisted assembly.CP assembly leads to viral particle maturation which involves a series of steps that converts a non infectious virus into a mature infectious virus. The maturation process leads to the physical stabilization of the CP. Host factors that assist in viral morphogenesis include macromolecular overcrowding in the host cell, supplementary viral, and cellular factors. To achieve stability in an aggressive cellular environment, a virus needs many different strategies to protect itself from being inactivated or neutralized. Such strategies include noncovalent interactions between CP subunits, covalent bonds between CP subunits, cementing proteins, capsid-viral nucleic acid interactions, hysteresis to dissociation, entropic stabilization, stabilizing ligands, covalent attachment of functional groups, and mechanical stability. Finally, for a virus to infect a host cell, it must be recognized by a cell receptor, which is followed by the entry of a virus into the cell, and ultimately genomic uncoating of the virus (8).

Conclusion:

Viral disease with a pandemic potential is always lurking around the corner. As our environment changes, the ability of viruses to infect and mutate has grown. Although viruses have a simple structure, they can cause devastating diseases resulting in severe morbidity and mortality. Understanding viral anatomy and physiology helps to effectively implement management strategies to successfully overcome their untoward health effects.

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