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Replication of Viruses: A Review

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Abstract

Viruses are among the smallest parasitic organisms in our world. They are non-living because they cannot reproduce without the cells of other living organisms, whether plants, animals, humans, or bacteria. Viruses are transmitted to living organisms in many ways and are found all around us in enormous numbers. Despite the differences between viruses, there are some basic steps similar in the viral life cycle. Antibiotics have no effect on viruses, so a few antiviral drugs have been developed. However, since there are relatively few targets for these drugs to interfere with, they are ineffective. This is because the virus reprograms its host cell to produce new viruses, making almost all the proteins used in this process part of the host's own tissue, with only a few being viral proteins.

Keywords: Virus, living organisms, replication.

Introduction

Virus is a Latin word that means poison or slimy liquid. The parasite is a tiny one, and has no ability to replicate itself: but when it gets inside a host cell [1], it can order the cell to churn out huge numbers of new viruses. genetic material (DNA or RNA) in a protein coat [2]. They are not able to reproduce independently, so they invade living cells (host cells) to form copies of themselves and sometimes destroy the host cell in the process. They are three orders of magnitude smaller than bacteria, and are invisible under the light microscope. Viruses infect every living organism and are a source of such diseases as influenza and COVID-19 [3, 4]

The full virus particle that is infectious within the host organism consists of the nucleic acid and the outer shell. It is composed of DNA and an external protein shell. A pathogenic particle with nothing but DNA is a viroid [5]. The virus depends on the host body because it doesn't have a store of energy to create, and it takes this power from the infected host cell. It also hijacks the host cell's nucleotides, amino acids and fatty acid for synthesizing its own nucleic acids, protein and membrane [6].

Host cell RNA, including ribosomal RNA, occurs in virions but there is no proof that it plays a functional role in virus replication [7]. For enveloped viruses containing membrane glycoproteins, glycoproteins are the most abundant protein type exposed on the outer surface of the membrane [8]. The existence/presence of a lipoid envelope provides an operational basis for dividing viral agents into two classes—those which are inactivated by organic solvents. Multiplication of DNA viruses Since DNA, being a virus, carries neutral charge, thus its goes through duplicate to mRNA under the action of transcriptase enzyme. The mRNA moves to the ribosomes, which translate (early and late) proteins responsible for the manufacture of a new DNA strand. Multiplier Logic for RNA Viruses RNA viruses are either –ve or as +ve charge [9, 10]. In positively charged nucleic acid viruses the nucleic acid per se functions as mRNA and the multiplication proceeds as described above [11]. In case of a–ve charge viruses, the strand had to have another +ve charge strand by transcription process performed through the transcriptase enzyme [12].

Humans as well as animals have natural protections against viruses. Fever, for instance, is a key mechanism employed by the human body to defend itself against viruses: when an infected person's temperature goes up it can put certain viruses into remission [13]. The immune system in the body also combats viruses by producing white blood cells and proteins called antibodies to fight them. After these cells and antibodies have fought and killed the virus, they stick around so that the body is ready to fight off the same virus later. [14].

Replication of viruses

All of the dynamic events associated with the virus (transcription & replication of genomes) occurs within the living host cell. All DNA viruses multiply inside the host cell nucleus (except poxvirus) and all RNA viruses multiply in the cytoplasm [15, 16]. As shown in Fig.1, replication steps in all types of viruses are:

1- **Attachment**

Attachment to cells is one of the earliest significant steps in viral replication, but successful attachment and entry do not ensure infection of that cell [17]. How the virus attaches and enters largely depends on a number of variables involving both the virus and the cell. Attachment and entry are mediated by the virus's outer protein, or envelope, and the wall of the host cell. The viral nucleic acid induces viral replication and synthesis of its constituents in the cell [18].

The virus attaches to a receptor site on the host cell membrane through attachment proteins in the viral capsid (or protein envelope), or via adhesion precursors within the viral envelope. The specificity of this interaction is what defines which host cell (or cells within it) a given virus may infect. This can be demonstrated with keys and locks; imagine a set of keys (which cannot be duplicated) each opening one lock, table 1 shows targets and receptors for some types of viruses. [19, 20].

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Table 1. Target cells and receptors type for some types viruses.

Virus	Target cell	Receptor
Epstein Bar virus	**B- cell**	**CD21**
Human Immuno-deficiency virus	**Helper T cell**	**CD4**
Rhinovirus	**Epithelial cell**	**ICAM-1 Intracellular adhesion molecule**
Rabies cell	**Neuron cell**	**Acetylcholine receptor**
Influenza virus	**Epithelial cell**	**Sialic acid**
B19 Parvovirus	**Erythroid precursor**	**Erythrocyte P antigen (globoid)**

2- **Penetration**

Following attachment, virions can enter cells by one of the following ways [21]:

A- Translocation of virion across plasma membrane.

B- Endocytosis: The virus accumulates inside the cell.

C- Fusion with Plasma Membrane: The virus fuses directly with the cell's plasma membrane and then enters the host cell.

3- **Uncoating**

This is a crucial stage in viral replication, occurring after the virus enters the host cell. Its envelope (whether protein or lipid) is broken down by enzymes from either the virus or the host, releasing viral DNA/RNA so it can replicate and take control of the cell to create new viruses. Uncoating required acidic pH in the endosome. The infectivity of the parental virus is lost at the uncoating stage [22, 23] .

4- **Gene expression and biosynthesis**:

Virus cannot replicate by binary fission or mitosis, but they replicate by complex process called Replication [24]. Once the virus takes control of the host cell's mechanisms, the viral genetic material is then copied, copies of its nucleic acid are made, and finally, viral proteins, including envelope proteins, are made using the host cell's ribosomes and their knowledge [25, 26].

5- Assembly of Viruses :

The infectious virus is simply the intact or functional viral fragments. At this stage, the newly synthesized genome (DNA) and proteins are assembled to form new viral components [27]. This stage may occur in the host cell nucleus, cytoplasm, or cell membrane for most evolving viruses [28].

6- **Release of virus** :

It is the final step in the virus's replication cycle, where new viral particles leave the host cell either by destroying it (lysis) or by budding without being destroyed, to move and

infect other cells, following key stages that begin with adhesion, penetration, removal of the envelope, replication and assembly [29, 30].

Some viruses are enveloped; they acquired their envelopes from cell membrane during releasing, while other enveloped viruses acquire their envelope from nuclear membrane of infected cell [31, 32].

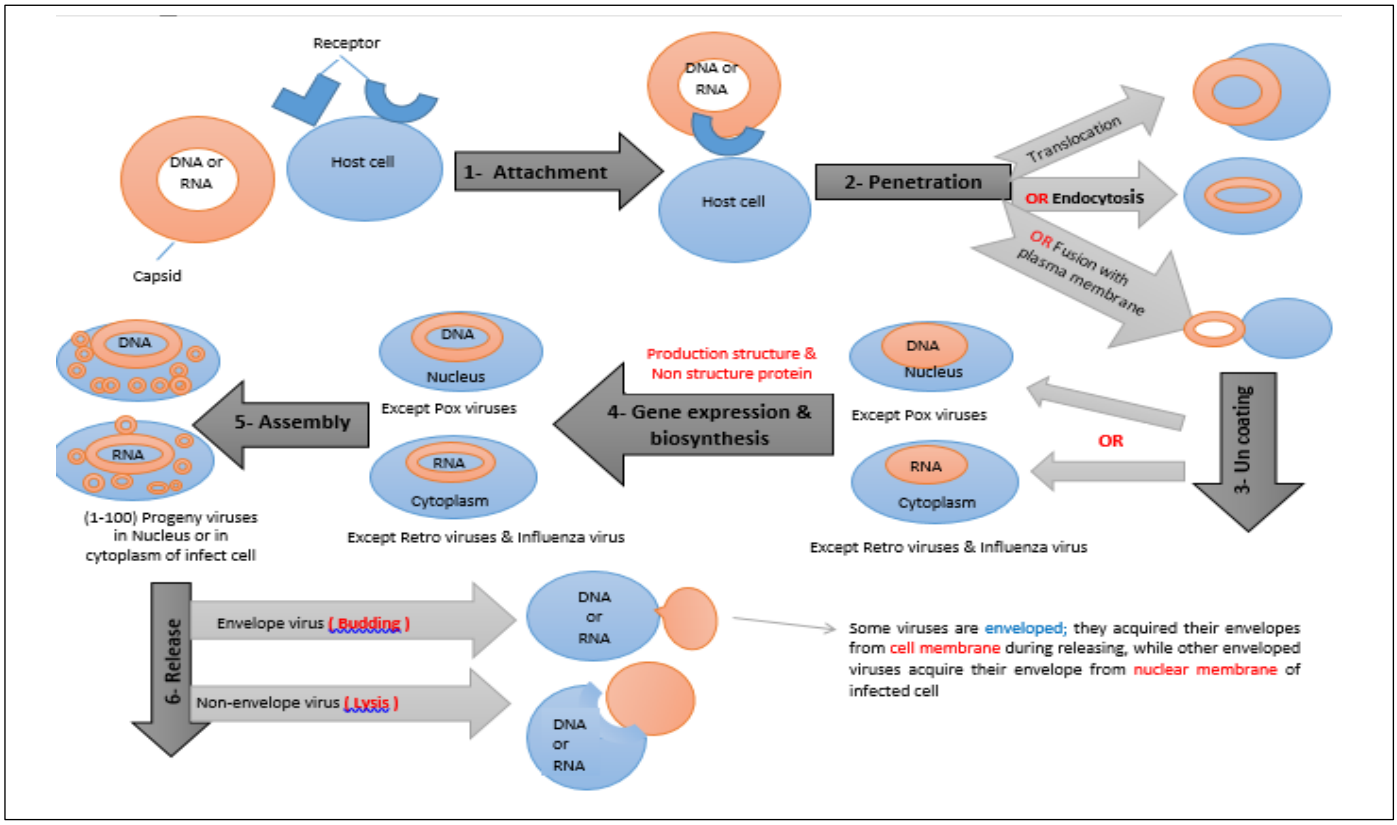

Figure 1. Replication steps in all types of viruses.

Conclusion

The virus is extremely small in size, yet the way it enters and "enslaves" a living cell is so ingenious that the virus is humanity's most lethal enemy and resists the most advanced efforts of modern science to eradicate it. Clinicians used vaccines to prevent many diseases caused by viruses. These vaccines are substances made from dead or weakened viruses that help prepare the immune system to fight active forms of the viruses.

ETHICAL APPROVAL

The research protocol was approved by the Ethical Research Committee of the Education Authority in Kirkuk.

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INFORMED CONSENT

Participants were aware of the purpose of the study and provided informed consent prior to the participations.

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STANDARDS OF REPORTING

STROBE guidelines were followed.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analyzed during this study are included in this published article.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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Declared none.

AUTHORS CONTRIBUTION

All the authors contributed in the Study conception and design, Data collection, Analysis and interpretation of results, Draft manuscript and all authors reviewed the results and approved the final version of the manuscript.

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