GENERAL

molecular and cellular biology of viruses

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Introduction

Viruses are the smallest biological entities that bridge the gap between chemistry and biology. Though they lack the cellular machinery to sustain life independently, their ability to hijack host cells and replicate with astonishing speed makes them a central focus of modern molecular and cellular biology. Understanding the **molecular and cellular biology of viruses** is essential not only for virology research but also for the development of vaccines, antiviral drugs, and gene therapy vectors. This comprehensive guide will walk you through the intricate details of viral structure, genome organization, replication strategies, host interactions, immune evasion tactics, and the cutting edge tools that scientists use to study and manipulate viruses.

1. Viral Structure: From Capsid to Envelope

1.1 Capsid Architecture

At the core of every virus lies the capsid, a protein shell that protects the genetic material. Capsids are built from repeating subunits called **capsomeres**, which assemble into geometric shapes such as icosahedrons, helical rods, or more complex structures. The symmetry of a capsid is defined by its **triangulation number (T number)**, which determines the number of capsomeres per face and, consequently, the size of the capsid.

1.2 Envelope and Glycoproteins

Many viruses acquire a lipid envelope derived from the host cell membrane during budding. Embedded in this envelope are viral glycoproteins that serve as the primary interface for receptor binding and membrane fusion. The envelope not only confers additional structural flexibility but also plays a critical role in immune evasion by masking underlying capsid proteins.

1.3 Genome Packaging

The viral genome can be either DNA or RNA, single stranded or double stranded, and may be linear, circular, or segmented. Packaging signals—short nucleotide or amino acid sequences—direct the genome into the capsid during assembly. Some viruses, like bacteriophages, use powerful molecular motors to inject their genome into host cells, while others rely on simple diffusion.

2. Viral Genome Diversity

2.1 DNA Viruses

- Double stranded DNA (dsDNA) viruses, such as Herpesviridae, possess large genomes that replicate in the host nucleus using host DNA polymerases.
- Single stranded DNA (ssDNA) viruses, like Parvoviridae, encode a minimal set of proteins and rely heavily on host replication machinery.

2.2 RNA Viruses

- Positive sense (+) RNA viruses (e.g., Picornaviridae, Flaviviridae) have genomes that function directly as mRNA.
- Negative sense (") RNA viruses (e.g., Orthomyxoviridae, Rhabdoviridae) must synthesize complementary

+RNA before translation.

- Segmented genomes allow reassortment events that generate viral diversity, a key factor in influenza evolution.

2.3 Reverse Transcribing Viruses

Retroviruses and hepadnaviruses encode reverse transcriptase, converting RNA or partially double stranded DNA into a stable DNA form that integrates into the host genome.

3. The Viral Life Cycle: From Attachment to Release

3.1 Attachment and Entry

Viruses initiate infection by recognizing specific receptors on the host cell surface. Binding can trigger receptor mediated endocytosis, direct fusion with the plasma membrane, or macropinocytosis. The choice of entry route often dictates the subsequent intracellular trafficking pathway.

3.2 Uncoating and Genome Release

Once inside the cell, the viral capsid must be disassembled to expose the genome. This process can be driven by host proteases, acidic pH within endosomes, or viral proteases that cleave capsid proteins.

3.3 Replication and Transcription

Depending on the genome type, replication strategies differ:

1. DNA viruses often replicate in the nucleus using host polymerases.
2. RNA viruses bring their own RNA dependent RNA polymerases (RdRp) or rely on host ribosomes to translate viral proteins.
3. Reverse transcribing viruses convert RNA into DNA, then integrate into the host genome.

3.4 Protein Synthesis

Viral mRNAs are translated by host ribosomes. Some viruses use unique mechanisms such as ribosomal frameshifting, leaky scanning, or internal ribosome entry sites (IRES) to maximize coding capacity.

3.5 Assembly and Budding

Structural proteins and genome components assemble into mature virions. For enveloped viruses, budding through the plasma membrane or intracellular membranes incorporates the viral envelope and glycoproteins.

3.6 Release and Spread

Virions are released by cell lysis or budding. They then encounter new cells, repeating the cycle. Some viruses also spread via cell to cell contact, forming virological synapses to evade immune detection.

4. Host Cell Entry Mechanisms

4.1 Receptor Binding Specificity

Viral attachment proteins (e.g., hemagglutinin, spike, gp120) have evolved to bind specific host receptors such as sialic acid, ACE2, or CD4. The affinity and distribution of these receptors influence tissue tropism and pathogenesis.

4.2 Endocytic Pathways

- Caveolae mediated endocytosis is used by some adenoviruses.
- Clathrin mediated endocytosis is common for many enveloped viruses.
- Macropinocytosis allows uptake of large volumes of extracellular fluid, utilized by some poxviruses.

4.3 Membrane Fusion

Fusion proteins undergo conformational changes triggered by low pH or receptor engagement, merging viral and cellular membranes to release the genome.

5. Intracellular Trafficking and Replication Strategies

5.1 Nuclear vs Cytoplasmic Replication

DNA viruses like adenoviruses replicate in the nucleus, whereas RNA viruses such as influenza replicate in the cytoplasm. The subcellular location influences the availability of host factors and the immune detection mechanisms.

5.2 Viral Replication Complexes

Many RNA viruses assemble replication complexes on intracellular membranes (endoplasmic reticulum, Golgi, mitochondria). These structures concentrate viral enzymes and substrates while shielding dsRNA intermediates from cytosolic sensors.

5.3 Host Factor Hijacking

Viruses recruit host proteins for replication, translation, and assembly. For example, poliovirus co opts the host eukaryotic initiation factor 4E (eIF4E), while hepatitis C virus uses the host lipid metabolism machinery for envelope formation.

6. Viral Transcription and Translation Mechanisms

6.1 Transcriptional Strategies

- Conventional transcription uses host RNA polymerase II for DNA viruses.
- RdRp mediated transcription creates subgenomic RNAs to maximize coding capacity.
- Cap snatching (influenza) steals host mRNA caps to initiate viral mRNA synthesis.

6.2 Translational Control

Viruses manipulate ribosome selection and initiation sites. The use of internal ribosome entry sites (IRES) allows translation under conditions where cap dependent initiation is inhibited.

6.3 Post Translational Modifications

Viral proteins often undergo host mediated modifications such as phosphorylation, glycosylation, and ubiquitination, which can influence protein stability, localization, and immune recognition.

7. Viral Assembly and Budding

7.1 Capsid Assembly

Capsid proteins self assemble into procapsids, which then undergo maturation steps including proteolytic cleavage and conformational rearrangements.

7.2 Envelopment

Enveloped viruses acquire their lipid bilayer by budding through cellular membranes. The budding process can involve the ESCRT (Endosomal Sorting Complex Required for Transport) machinery, especially in retroviruses.

7.3 Release Mechanisms

- Lytic release ruptures the host cell membrane.

- Budding allows continuous release while the cell remains viable.

8. Host Immune Response to Viral Infection

8.1 Innate Immunity

Pattern recognition receptors (PRRs) such as Toll like receptors (TLRs), RIG I like receptors (RLRs), and cGAS detect viral nucleic acids, triggering interferon production and antiviral gene expression.

8.2 Adaptive Immunity

Humoral responses involve neutralizing antibodies targeting viral envelope proteins, while cellular immunity relies on cytotoxic T lymphocytes (CTLs) that recognize viral peptides presented by MHC class I molecules.

8.3 Viral Evasion Strategies

- Inhibiting interferon signaling (e.g., influenza NS1 protein).
- Masking viral nucleic acids via nucleocapsid proteins.
- Decoy receptors that bind and neutralize antibodies.

9. Molecular Tools and Technologies in Virology

9.1 Reverse Genetics

Reverse genetics systems allow the generation of infectious virus from cloned cDNA, enabling the study of gene function and the creation of attenuated vaccine strains.

9.2 CRISPR/Cas Systems

CRISPR based antiviral strategies target viral genomes or host factors essential for viral replication, offering potential therapeutic avenues.

9.3 Next Generation Sequencing (NGS)

NGS provides high throughput sequencing of viral genomes, facilitating surveillance of emerging strains and tracking mutations associated with drug resistance.

9.4 Structural Biology

Cryo electron microscopy (cryo EM) and X ray crystallography reveal the atomic

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