Microbiology

Unit 3 · Lesson 1 · Viruses and phages

The notes for Lesson 1 of Unit 3: chapters 3.1 to 3.2 of the unit book, with their figures, worked examples, exam traps and quizzes, in one file. The full unit book, with every chapter, the exam question bank and the glossary, is linked at the end.

Lesson 1 of 3 | 2 chapters | Chapters 3.1 to 3.2

About these notes

These are the notes for **Lesson 1 · Viruses and phages** of the University free n easy video lessons on Microbiology Unit 3, Viruses and Bacteriophages. They are chapters 3.1 to 3.2 of the unit book, unchanged, so a page here is the same page in the full book.

3.1 General properties, structure and classification of viruses

A virus is an infectious particle made of one kind of nucleic acid (DNA or RNA) wrapped in a protein coat, sometimes with a lipid envelope outside. It has no ribosomes and no energy metabolism of its own, so it can multiply only inside a living host cell, using that cell's machinery. Viruses infect every form of life, from bacteria to people, and they matter to a biotechnologist twice over: as causes of disease, and as tools for vaccines, gene therapy and cloning.

You need: the difference between prokaryotic and eukaryotic cells (Unit 1, Prokaryotic and eukaryotic cells compared) and the idea of a microscope's resolving limit (Unit 1, Fluorescence and electron microscopy).

What makes a virus a virus

The French microbiologist André Lwoff summed up the properties that set viruses apart from cells. Learn them as a list; examiners ask for it.

1. **One type of nucleic acid.** A virus particle carries DNA or RNA, never both. Cells carry both.
2. **No independent metabolism.** A virus has no ribosomes and makes no ATP. Outside a cell it is inert.

3. **Obligate intracellular parasite.** It multiplies only inside a living host cell.
4. **Replication from parts, not by division.** A virus does not grow and split. The cell makes many copies of the genome and the proteins separately, and these assemble into new particles.
5. **Small size.** Most viruses are about 20 to 300 nm across, below the resolving limit of a light microscope, so they are seen with an electron microscope. They pass through filters that hold back bacteria.
6. **Not affected by antibacterial antibiotics.** Penicillin and similar drugs act on bacterial walls and ribosomes, which viruses do not have.

The history follows from the size. In 1892 Dmitri Ivanovsky showed that sap from diseased tobacco stayed infectious after passing through a filter that held back bacteria. In 1898 Martinus Beijerinck called the agent a "contagium vivum fluidum", a living infectious fluid, and in the same year Loeffler and Frosch found the first animal virus, of foot-and-mouth disease. In 1935 Wendell Stanley crystallised tobacco mosaic virus (TMV), which showed that a virus could behave like a chemical.

The parts of a virus particle

The complete, infectious particle outside a cell is called a **virion**. Its parts are:

- **Genome:** DNA or RNA, single-stranded (ss) or double-stranded (ds), linear or circular, in one piece or several segments (influenza has eight RNA segments).
- **Capsid:** the protein shell around the genome, built from many copies of one or a few proteins. The visible units of the capsid are called **capsomeres**. The genome plus capsid is the **nucleocapsid**.
- **Envelope (in some viruses):** a lipid bilayer taken from a host membrane as the virus leaves, studded with viral glycoproteins called **spikes** or peplomers. Viruses without one are called **naked**. Enveloped viruses are destroyed by detergents, ether and alcohol, because these dissolve lipid; naked viruses resist them better.
- **Enzymes (in some viruses):** carried inside the virion because the host cannot supply them, for example the reverse transcriptase of HIV or the RNA-dependent RNA polymerase of influenza.

Capsids follow three plans of symmetry. In **helical** symmetry the protein subunits wind around the genome like a spiral staircase, giving a rod (TMV) or a flexible tube. In **icosahedral** symmetry the capsid is a 20-faced solid with 12 vertices and 30 edges (adenovirus, poliovirus); it is the most economical way to close a shell from identical subunits. **Complex** viruses fit neither plan: the poxviruses are brick-shaped, and the T-even phages join an icosahedral head to a helical tail (Chapter 3.2).

Classification: two systems you must know

The official names are set by the **International Committee on Taxonomy of Viruses (ICTV)**. It uses ranks like those of cellular life, from realm down through order (-virales), family (-viridae) and genus (-virus) to species. Family and genus names are written in italics, for example *Retroviridae* and *Lentivirus*. Older books also describe the LHT system of Lwoff, Horne and Tournier (1962), which grouped viruses by nucleic acid, symmetry, envelope and size.

The **Baltimore classification**, proposed by David Baltimore, groups viruses by how they make messenger RNA (mRNA), because every virus must make mRNA for the host ribosome to read. Positive-sense (+) RNA has the same sequence as mRNA and can be translated at once; negative-sense (–) RNA is its complement and must first be copied.

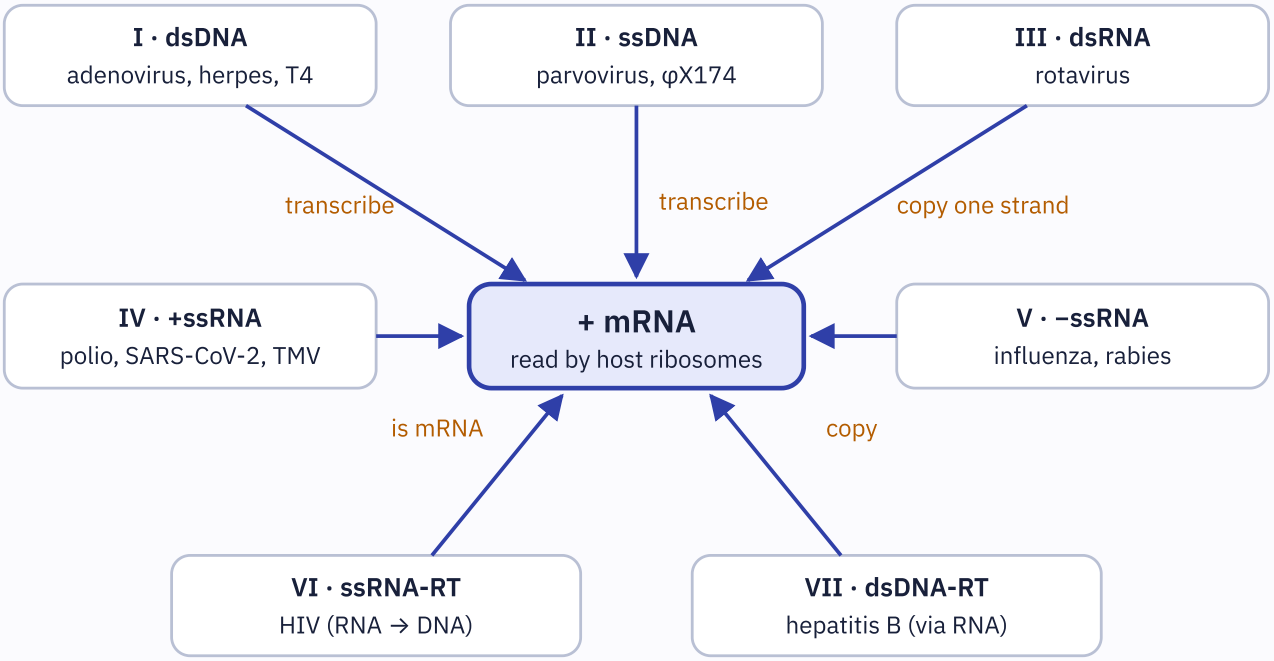


Figure 3.1.1 The Baltimore classification. Seven genome types, each with its own route to + mRNA in the centre. Notice that group IV genomes already are mRNA, group V must copy theirs first (so they carry their own polymerase), and the two RT groups pass through a DNA–RNA switch using reverse transcriptase.

Pathogenicity in brief

A virus causes disease by killing the cells it grows in (the lytic damage seen as cytopathic effect, Chapter 3.3), by making infected cells targets of the immune system, by suppressing immunity (HIV), or by changing cell growth (Chapter 3.5). Its host range and tissue tropism depend mainly on which cell-surface receptor its attachment protein binds. The infections and their spread belong to Unit 6 (Microbial pathogenicity).

Some infectious agents are even simpler than viruses. **Viroids** are short, circular RNA molecules with no capsid that infect plants (Chapter 3.4). **Prions** are misfolded proteins with no nucleic acid at all; they cause slow brain diseases such as Creutzfeldt–Jakob disease. Satellite viruses need a helper virus to multiply.

WORKED EXAMPLE

Question (6 marks). The adenovirus capsid is an icosahedron built from 240 hexons (capsomeres of six protein units) and 12 pentons (capsomeres of five units). (a) Why are there exactly 12 pentons? (b) How many protein units make up the capsid shell? (c) Classify adenovirus in the Baltimore system and say what it needs from the host to make mRNA.

1. **(a)** An icosahedron has 12 vertices. A capsomere at a vertex has five neighbours around it, so it is a pentamer. Every icosahedral capsid therefore has exactly 12 pentons, however many hexons fill the faces.
2. **(b)** Protein units in hexons = 240 × 6 = 1440. Protein units in pentons = 12 × 5 = 60. Total = 1440 + 60 = **1500 units**.
3. **Check with the triangulation rule:** an icosahedral shell holds 60 × T units, where T is the triangulation number. 1500 ÷ 60 = 25, so T = 25, a known value for adenovirus.
4. **(c)** Adenovirus has a linear dsDNA genome, so it is **Baltimore group I**. It needs the host's DNA-dependent RNA polymerase II, which is in the nucleus, so adenovirus DNA must reach the nucleus to be transcribed.

EXAM TRAPS

- **Virion vs virus:** the virion is the complete particle outside the cell; inside the cell, during the eclipse, there is no intact virion at all.
- **Capsid vs capsomere:** the capsid is the whole shell; capsomeres are its visible units. Writing "the capsomere encloses the genome" loses the mark.
- **"Viruses have both DNA and RNA"** is wrong for the virion. The genome is one or the other; an RT virus makes the other form only inside the cell.
- **+ and − sense:** + sense means "same as mRNA", not "double-stranded". Influenza is − ssRNA; polio is + ssRNA.
- The favourite question: "Are viruses living?" Give both sides: they have genes and evolve, but they have no metabolism and cannot reproduce outside a cell.

CHECK YOURSELF

– REMEMBER **Name the three types of capsid symmetry with one example each.** 3 marks

Helical (TMV), icosahedral (adenovirus or poliovirus), complex (poxvirus or T4 phage).

– REMEMBER **Who first showed that a plant disease agent passes through a bacterial filter, and in which year?** 1 mark

Dmitri Ivanovsky, 1892, with tobacco mosaic disease.

– UNDERSTAND **Why does a – ssRNA virus have to carry its own polymerase in the virion?** 3 marks

Its genome cannot be translated, and host cells have no enzyme that copies RNA from an RNA template. Without a packaged RNA-dependent RNA polymerase, no mRNA would be made and the infection could not begin.

– UNDERSTAND **Why is 70% ethanol effective against an enveloped virus but less so against a naked virus?** 2 marks

Alcohol disrupts the lipid envelope, which carries the attachment spikes, so the enveloped virus can no longer bind cells. A naked virus has only a protein capsid, which is more resistant.

– APPLY **An icosahedral capsid has T = 3. How many protein subunits does it contain, and how many of the capsomeres are pentamers?** 3 marks

60 × 3 = **180 subunits**. There are always **12 pentamers**, one at each vertex; the remaining 180 – 60 = 120 subunits form 20 hexamers.

– APPLY **Place hepatitis B virus, rotavirus and rabies virus in Baltimore groups.** 3 marks

Hepatitis B: group VII (dsDNA-RT). Rotavirus: group III (dsRNA). Rabies: group V (– ssRNA).

– ANALYZE **A purified RNA from virus X infects cells when injected; RNA from virus Y does not. What can you conclude about their genomes?** 4 marks

Virus X has + sense RNA: the cell can translate it directly and make the viral polymerase. Virus Y is probably – sense (or dsRNA): its RNA needs a polymerase packed in the virion, which was lost when the RNA was purified.

BEYOND THE SYLLABUS

Virus taxonomy now leans on genome sequences more than on shape. ICTV has added ranks above order, such as the realm *Riboviria* for RNA viruses that use an RNA-dependent RNA polymerase, and has moved species names to a two-word format. Giant viruses such as mimivirus, discovered in amoebae, are larger than some bacteria and carry genes for parts of the translation system, which has reopened the "are viruses alive" debate. Capsid assembly is also engineered: virus-like particles (capsids with no genome) are the basis of the HPV and hepatitis B vaccines. Search "ICTV Master Species List" and "virus-like particle vaccine" to go further. This is not for the exam.

3.2 Bacteriophages: structure and lytic and lysogenic cycles

Bacteriophages ("phages") are viruses that infect bacteria. They were found by Frederick Twort in 1915 and by Félix d'Hérelle in 1917, who named them "bacteria eaters". A phage can follow one of two paths after it enters a cell: the **lytic cycle**, which makes new phages and bursts the cell, or the **lysogenic cycle**, in which the phage genome sleeps inside the bacterial chromosome and is copied with it. Phages taught us much of molecular biology, they are tools for cloning, and they are returning as a treatment for antibiotic-resistant infections.

You need: virion parts and capsid symmetry (Chapter 3.1) and the Gram-negative cell envelope (Unit 2, Bacterial cell wall).

Structure of phage T4

The T-even phages (T2, T4, T6) of *Escherichia coli* are the textbook example of complex symmetry. T4 has about 169 kb of linear dsDNA. Its parts, from top to bottom:

- **Head:** an elongated icosahedron of protein that holds the tightly packed DNA.
- **Collar and neck:** join the head to the tail.
- **Tail:** a hollow core tube surrounded by a **contractile sheath**. When the sheath contracts, the core is driven through the cell envelope like a syringe needle.
- **Baseplate:** a hexagonal plate at the tail tip with short tail pins (spikes) and an enzyme that makes a hole in the peptidoglycan.
- **Tail fibres:** six long, jointed fibres that recognise receptors on the bacterial outer membrane.

Other phages are simpler. Phage λ (lambda) has an icosahedral head and a long, flexible tail that does not contract; its genome is 48.5 kb of dsDNA. φX174 is a small icosahedral phage with circular ssDNA. M13 is a filamentous ssDNA phage that leaves the cell without killing it, and MS2 is a small ssRNA phage. Textbooks group tailed phages into three families by tail type: *Myoviridae* (contractile tail, T4), *Siphoviridae* (long non-contractile tail, λ) and *Podoviridae* (short tail, T7). Know these names for the exam, but note that the ICTV, in its 2022 taxonomy update (published 2023), abolished these three families as not reflecting real ancestry and now places tailed phages in the class *Caudoviricetes*, classified by genome sequence.

The lytic cycle

A **virulent** phage such as T4 can only follow the lytic cycle. The steps are:

1. **Adsorption.** Tail fibres bind specific receptors (for T4, outer-membrane proteins and lipopolysaccharide). The receptor decides the host range.
2. **Penetration.** The baseplate settles on the surface, the sheath contracts, the core pierces the envelope and the DNA is injected. The empty capsid stays outside as a "ghost".
3. **Early gene expression.** The host RNA polymerase reads phage early genes. Their products shut down host transcription and break down host DNA. T4 DNA contains hydroxymethylcytosine in place of cytosine, so phage nucleases cut host DNA and spare the phage's own.
4. **Replication and late genes.** The phage genome is copied many times; late genes make head, tail and fibre proteins.
5. **Assembly (maturation).** Heads, tails and fibres are made separately and join; DNA is pumped into the head by a packaging motor.
6. **Release.** A **holin** makes holes in the inner membrane, letting an **endolysin** reach and digest the peptidoglycan. The cell bursts and releases the progeny.

The **one-step growth curve** (Ellis and Delbrück, 1939) measures this cycle. A culture is infected so that every cell gets a phage, diluted so that new phages cannot infect again, and sampled for plaque-forming units over time. Three numbers come from it. The **eclipse period** is the time when no infectious phage can be found even inside broken cells; the injected genome is not yet a virion. The **latent period** is the time from infection until the burst, when free phages first rise; for T4 at 37 °C it is around 25 minutes. The **burst size** is the average number of phages released per infected cell.

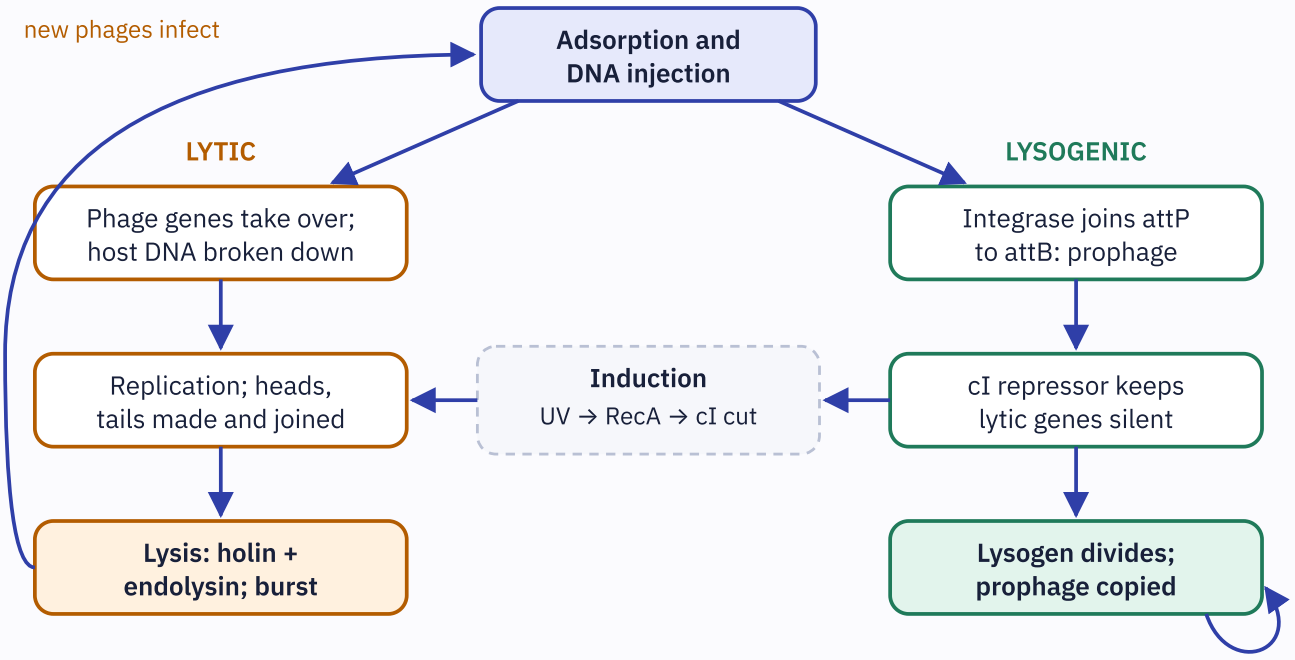


Figure 3.2.1 The two paths of a temperate phage such as λ. Left: the lytic cycle ends in a burst of new phages that infect more cells. Right: in lysogeny the genome integrates as a prophage and is passed to daughter cells; the loop shows many generations. The dashed box is induction: DNA damage switches a lysogen back to the lytic path.

The lysogenic cycle

A **temperate** phage can choose either path. Phage λ is the model. After its linear DNA enters, the two single-stranded **cos** ends pair up and the genome closes into a circle. Then a genetic switch decides the outcome.

- If the λ **cI repressor** wins, it binds the operators that control the lytic genes and keeps them off. The phage enzyme **integrase** joins the phage site *attP* to the bacterial site *attB* (between the *gal* and *bio* genes of *E. coli*). The integrated genome is a **prophage**; the cell carrying it is a **lysogen**.
- If the **Cro** protein wins, it blocks cI synthesis and the lytic genes run.

Conditions push the switch. Many phages per cell (high multiplicity of infection) and poor nutrition favour lysogeny, which makes sense: when hosts are scarce, it is safer to wait inside one.

A lysogen has three special properties. It is **immune to superinfection**: the cI repressor already present also silences any new λ genome that enters. It can be **induced**: UV light or DNA-damaging chemicals activate RecA (part of the SOS response), which triggers self-cleavage of cI; the prophage is cut out by integrase with excisionase (Xis) and enters the lytic cycle. And it may show **lysogenic conversion**: prophage genes change the host's properties. The diphtheria toxin of *Corynebacterium diphtheriae*, the cholera toxin of *Vibrio cholerae* and the Shiga toxins of

some *E. coli* are all coded by phage genes. Phages also move bacterial genes between cells by transduction, which belongs to Unit 4 (Bacterial recombination).

WORKED EXAMPLE

Question (6 marks). In a one-step growth experiment, *E. coli* at 2×10^8 cells/mL is infected with T4. Unadsorbed phages are removed and the culture is diluted 10^4-fold. A plaque assay of the diluted culture just after infection gives 1.8×10^4 PFU/mL (each infected cell forms one plaque). At 60 minutes the diluted culture gives 2.7×10^6 PFU/mL, and the count stops rising. Find the burst size and explain why the infected culture was diluted.

1. **Infective centres before the burst:** 1.8×10^4 per mL of diluted culture. Each is one infected cell.
2. **Phages after the burst:** 2.7×10^6 per mL of the same diluted culture.
3. **Burst size** = phages released ÷ infected cells = $2.7 \times 10^6 \div 1.8 \times 10^4$ = **150 phages per cell**. (Strictly, subtract the 1.8×10^4 starting count from the top; 150 becomes 149, which rounds the same.)
4. **Why dilute:** after a 10^4-fold dilution the bacteria are about 2×10^4/mL, so released phages rarely meet an uninfected cell. Only one cycle is measured; that is what "one-step" means.

EXAM TRAPS

- **Eclipse vs latent period:** the eclipse ends when new virions appear inside the cell; the latent period ends later, when they are released. The eclipse is always the shorter of the two.
- **Virulent vs temperate:** T4 is virulent (lytic only); λ is temperate (lytic or lysogenic). "λ is lysogenic" alone is incomplete.
- **Prophage vs lysogen:** the prophage is the integrated phage DNA; the lysogen is the bacterium carrying it.
- **Immunity:** a lysogen resists the same phage because of the repressor, not because of a missing receptor. The phage still adsorbs and injects.
- Do not say every phage lyses its host: filamentous phages such as M13 leak out through the envelope without killing the cell.

CHECK YOURSELF

– REMEMBER **List the structural parts of phage T4.** 3 marks

Icosahedral head with DNA, collar, tail core inside a contractile sheath, baseplate with tail pins, and six long tail fibres.

– REMEMBER **Which two scientists independently discovered bacteriophages?** 1 mark

Frederick Twort (1915) and Félix d'Hérelle (1917).

– UNDERSTAND **Why does a λ lysogen resist infection by another λ phage?** 3 marks

The prophage keeps making cI repressor. Any new λ genome that enters is bound by this repressor at once, so its lytic genes never switch on and it cannot integrate or multiply. This is immunity to superinfection.

– UNDERSTAND **How do holin and endolysin work together at the end of the lytic cycle?** 2 marks

Endolysin digests peptidoglycan but sits in the cytoplasm. Holin, at a set time, forms holes in the inner membrane so endolysin can reach the wall. The wall breaks and the cell bursts.

– APPLY **A culture with 5×10^5 infected cells/mL releases 4×10^7 PFU/mL after one cycle. What is the burst size?** 2 marks

$4 \times 10^7 \div 5 \times 10^5 =$ **80 phages per infected cell**.

– APPLY **A culture of λ lysogens is exposed to UV light. Predict what you see in the flask over the next hour, and why.** 3 marks

UV damages DNA, RecA becomes active and cI cleaves itself. The prophage is excised and enters the lytic cycle, so after the latent period the culture clears (lyses) and free λ phages appear.

– ANALYZE **A harmless *Vibrio cholerae* strain becomes toxigenic after exposure to a phage. Explain, and say why this matters for public health.** 4 marks

The cholera toxin genes are carried by a phage. When it establishes itself in the harmless strain, the strain gains the toxin genes: lysogenic conversion. Toxigenic strains can therefore arise from non-toxigenic ones in the environment, so surveillance must track the phage as well as the bacterium.

BEYOND THE SYLLABUS

Phage therapy, the use of lytic phages to treat infections, is being tested again for bacteria resistant to all antibiotics, and phage-derived endolysins are being developed as drugs. Phage parts are standard laboratory tools: λ for cloning vectors, M13 for phage display of antibodies (the work behind the 2018 Chemistry Nobel to George Smith and Gregory Winter), and T7 RNA polymerase in protein expression. CRISPR–Cas, the gene-editing tool, began as a bacterial immune system against phages. Search "phage therapy clinical trial" and "phage display" to go further. This is not for the exam.

Key terms of this lesson

TERM	MEANING OR FORMULA	CHAPTER
Virion	Complete infectious particle: genome + capsid (+ envelope)	3.1
Icosahedral capsid	20 faces, 12 vertices (pentons), 60 × T subunits	3.1
Baltimore groups	I dsDNA, II ssDNA, III dsRNA, IV +RNA, V –RNA, VI RNA-RT, VII DNA-RT	3.1
Burst size	phages released ÷ infected cells	3.2
Prophage / lysogen	Integrated phage DNA / the bacterium carrying it	3.2

Glossary (terms used in this lesson)

Burst size Average number of virions released from one infected cell.

Capsid Protein shell around the viral genome.

Capsomere Visible structural unit of a capsid.

Cytopathic effect Visible damage to cells caused by virus growth.

Eclipse period Time after infection when no infectious virions exist inside the cell.

Envelope Lipid membrane round some virions, taken from the host cell.

Induction	Switching a prophage from lysogeny into the lytic cycle.
Latent period	Time from infection until progeny virus is released.
Lysogen	A bacterium carrying a prophage.
Lysogenic conversion	Change in a host's properties caused by prophage genes.
Plaque	Clear zone in a cell lawn or monolayer made by virus from one infectious unit.
Prion	Misfolded infectious protein with no nucleic acid.
Prophage	Phage genome integrated into the bacterial chromosome.
Reverse transcriptase	Enzyme that makes DNA from an RNA template.
Temperate phage	Phage that can follow either the lytic or the lysogenic cycle.
Viroid	Small naked circular RNA that infects plants and codes for no protein.
Virion	Complete, infectious virus particle.

The full unit book

Every chapter of Unit 3, the unit summary, the exam question bank with model answers and the full glossary: Microbiology · Unit 3 · Viruses and Bacteriophages (one file, works offline).

Sources and further reading

- J. M. Willey, K. M. Sandman and D. H. Wood (earlier editions with L. M. Sherwood and C. J. Woolverton), *Prescott's Microbiology*, McGraw-Hill: the virus chapters (from memory; check).
- M. J. Pelczar, E. C. S. Chan and N. R. Krieg, *Microbiology*, Tata McGraw-Hill (from memory; check).
- Ananthanarayan and Paniker's *Textbook of Microbiology*, Universities Press: general virology, cultivation of viruses, HIV and oncogenic viruses (from memory; check).
- S. J. Flint and others, *Principles of Virology*, ASM Press, for the life cycles and assays in more depth (from memory; check).
- Turner and others, "Abolishment of morphology-based taxa and change to binomial species names: 2022 taxonomy update of the ICTV bacterial viruses subcommittee", *Archives of Virology* 168, 74 (2023); record at quadram.ac.uk.
- SARS-CoV-2 reference genome NC_045512 (Wuhan-Hu-1), 29,903 nucleotides, listed at the IIIT-Delhi coronavirus genome page.

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