Microbiology

Unit 3 · Lesson 3 · Oncoviruses, HIV and titres

The notes for Lesson 3 of Unit 3: chapters 3.5 to 3.7 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 3 of 3 | 3 chapters | Chapters 3.5 to 3.7

About these notes

These are the notes for **Lesson 3 · Oncoviruses, HIV and titres** of the University free n easy video lessons on Microbiology Unit 3, Viruses and Bacteriophages. They are chapters 3.5 to 3.7 of the unit book, unchanged, so a page here is the same page in the full book.

3.5 Oncoviruses

An oncovirus (oncogenic or tumour virus) is a virus that can turn a normal cell into a cancer cell. In 1911 Peyton Rous showed that a cell-free filtrate of a chicken sarcoma caused new tumours in healthy chickens; the agent is now called Rous sarcoma virus. Viruses are linked to a large share of human cancers worldwide, mainly cervical and liver cancers, and some of these cancers can now be prevented by vaccines. This shorter chapter covers the mechanisms and the human viruses.

How a virus can cause cancer

Cell division is held in balance by two kinds of genes. **Proto-oncogenes** (c-onc) push the cell to divide, like an accelerator. **Tumour suppressor genes**, such as *p53* and *Rb*, stop division or trigger cell death when DNA is damaged, like brakes. A virus causes cancer by pressing the accelerator or cutting the brakes.

1. **Carrying an oncogene.** Acute transforming retroviruses carry a viral oncogene (v-onc), a mutated copy of a host proto-oncogene picked up during evolution. The *src* gene of Rous sarcoma virus was the first. J. Michael Bishop and Harold Varmus showed that *src* came from a normal cell gene, and shared the 1989 Nobel Prize for it.

2. **Insertional activation.** A slow retrovirus with no oncogene integrates its DNA next to a cell proto-oncogene, such as *c-myc*; the strong viral promoter or enhancer then switches that gene on too much.
3. **Viral oncoproteins that block tumour suppressors.** DNA tumour viruses make proteins that bind and disable p53 and Rb. In high-risk human papillomavirus (HPV), the E6 protein marks p53 for destruction and the E7 protein binds Rb, freeing the cell to enter S phase. SV40 large T antigen and adenovirus E1A/E1B act the same way in laboratory cells.
4. **Chronic inflammation.** Hepatitis B and C viruses infect the liver for decades; repeated cell death and regrowth, with inflammation, builds up mutations that lead to liver cancer.

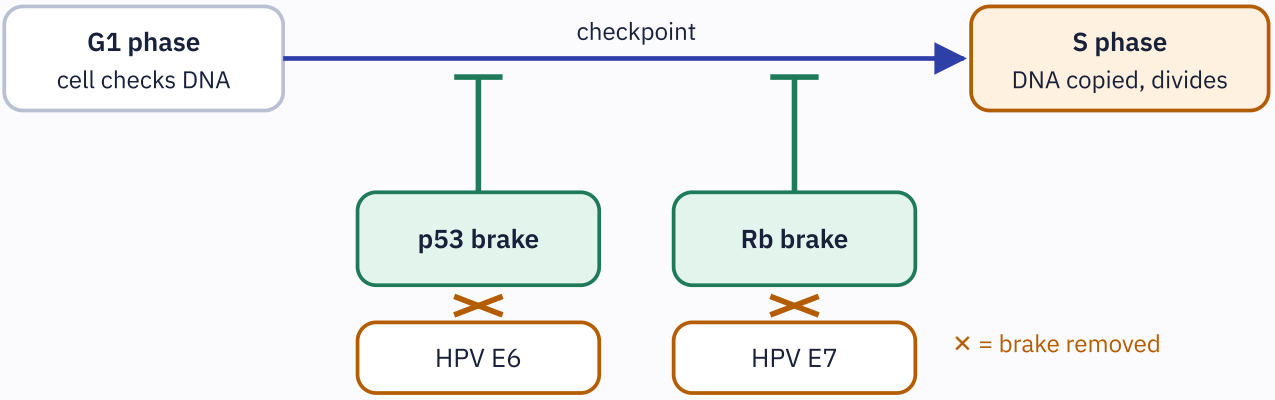


Figure 3.5.1 How high-risk HPV cuts the brakes. p53 and Rb normally block the move from G1 to DNA synthesis (S phase) when something is wrong. E6 removes p53 and E7 disables Rb, so the cell divides with damaged DNA and mutations pile up.

The human oncoviruses

VIRUS	GENOME	MAIN CANCER
Human papillomavirus 16, 18 (HPV)	dsDNA	Cervical cancer; some throat and anal cancers
Hepatitis B virus (HBV)	dsDNA-RT	Hepatocellular carcinoma
Hepatitis C virus (HCV)	+ ssRNA	Hepatocellular carcinoma
Epstein–Barr virus (EBV)	dsDNA (herpes)	Burkitt lymphoma, nasopharyngeal carcinoma
Kaposi sarcoma herpesvirus (HHV-8)	dsDNA (herpes)	Kaposi sarcoma, mainly with HIV
Human T-lymphotropic virus 1 (HTLV-1)	ssRNA-RT	Adult T-cell leukaemia
Merkel cell polyomavirus	dsDNA	Merkel cell carcinoma of skin

Infection alone rarely causes cancer. Most HPV infections clear by themselves; cancer follows only in the few that persist for years, together with other factors. That long gap is what makes screening (Pap smear, HPV DNA test) and vaccination work.

WORKED EXAMPLE

Question (4 marks). Three tumours are studied. Tumour A has the full genome of a retrovirus integrated beside *c-myc*; the virus has no oncogene. Tumour B contains a retrovirus carrying a gene almost identical to a cell kinase gene. Tumour C contains integrated HPV-16 DNA expressing E6 and E7. Name the mechanism in each.

1. **A:** insertional activation; the viral promoter drives *c-myc*.
2. **B:** transduced viral oncogene (v-onc), like *src* in Rous sarcoma virus.
3. **C:** oncoproteins that inactivate tumour suppressors: E6 degrades p53, E7 binds Rb.

EXAM TRAPS

- **v-onc vs c-onc:** c-onc is the normal cell gene (proto-oncogene); v-onc is the viral copy. Normal cells carry c-onc, so having it is not cancer.
- Oncoviruses include RNA and DNA viruses; do not say "only retroviruses cause cancer".
- Write "E6 → p53, E7 → Rb", not the other way round.

CHECK YOURSELF

– REMEMBER **Who first transmitted a tumour by a cell-free filtrate, and when?** 1 mark

Peyton Rous, 1911, with a chicken sarcoma.

– REMEMBER **Name two viruses linked to liver cancer.** 1 mark

Hepatitis B virus and hepatitis C virus.

– UNDERSTAND **Why does losing p53 make a cell more likely to become cancerous?** 3 marks

p53 stops the cell cycle or causes cell death when DNA is damaged. Without it, damaged cells keep dividing and pass on mutations, which build up to cancer.

– APPLY **Explain why an HPV vaccine given at age 10 can prevent a cancer that appears at age 45.** 3 marks

The vaccine prevents the persistent high-risk HPV infection that must last for years before cancer develops. No persistent infection, no E6/E7 activity, so the chain is broken at the start.

– ANALYZE **Why do acute transforming retroviruses cause tumours in weeks, while slow ones take months?** 3 marks

Acute viruses carry an oncogene that transforms every infected cell at once. Slow viruses must, by chance, integrate next to a proto-oncogene in some cell, and further mutations are usually needed.

BEYOND THE SYLLABUS

India launched its own quadrivalent HPV vaccine, Cervavac from the Serum Institute of India, in 2023. Oncolytic viruses turn the idea round: engineered viruses that grow in and kill cancer cells, such as the herpes-based talimogene laherparepvec approved for melanoma. Search "oncolytic virotherapy" to go further. This is not for the exam.

3.6 Human viruses: HIV and SARS-CoV-2

Two RNA viruses show every idea of this unit at work. HIV, the human immunodeficiency virus, is a retrovirus that writes its RNA into the DNA of immune cells and slowly destroys the immune system, causing AIDS. SARS-CoV-2, the coronavirus behind COVID-19, is a fast respiratory virus that copies its RNA in the cytoplasm and spreads before symptoms show. Each step of their life cycles is the target of a drug or a test, which is why examiners love them.

You need: Baltimore groups IV and VI (Chapter 3.1) and the animal virus life cycle (Chapter 3.3).

HIV: structure

HIV belongs to the family *Retroviridae*, genus *Lentivirus* ("slow virus"). It was identified in 1983 at the Pasteur Institute by Françoise Barré-Sinoussi and Luc Montagnier, who shared the 2008 Nobel Prize. There are two types: HIV-1, the worldwide cause of AIDS, and HIV-2, mainly in

West Africa, which progresses more slowly. In India the first cases were found in 1986 in Chennai by Dr Suniti Solomon and her team.

The virion is roughly spherical, about 100 nm across. From outside in:

- A lipid **envelope** from the host cell, carrying spikes of **gp120** (binds the receptor) on **gp41** (anchors in the membrane and drives fusion). The numbers are the protein mass in kilodaltons.
- A **matrix** of p17 protein lining the envelope.
- A cone-shaped **capsid** of p24 protein, holding **two copies** of + ssRNA (about 9.7 kb each) and three enzymes: **reverse transcriptase** (RT), **integrase** and **protease**.

The genome has three main genes, *gag* (core proteins), *pol* (the enzymes) and *env* (envelope glycoproteins), plus regulatory and accessory genes such as *tat*, *rev* and *nef*.

HIV: life cycle

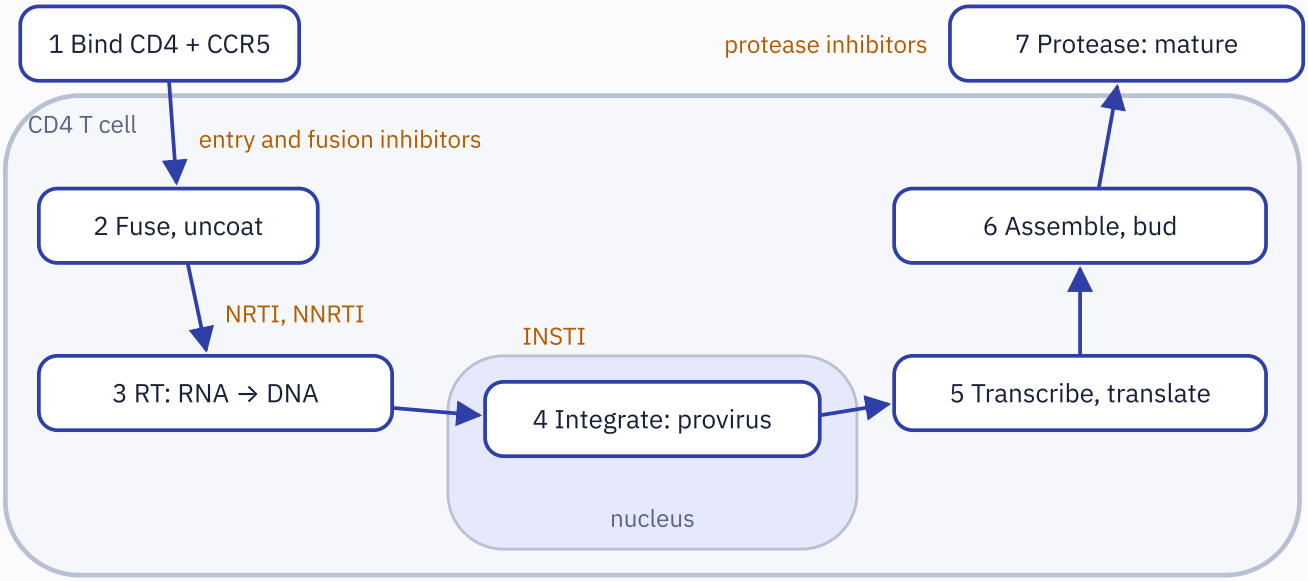


Figure 3.6.1 The HIV life cycle in a CD4 T cell, with the drug class that blocks each step in orange. Notice that the provirus in the nucleus (step 4) is a permanent copy; drugs stop new rounds of infection but cannot remove it, which is why treatment is lifelong.

1. **Binding.** gp120 binds the **CD4** receptor on helper T cells, macrophages and dendritic cells, then a **co-receptor**, CCR5 or CXCR4.
2. **Fusion and uncoating.** gp41 pulls the two membranes together; the core enters the cytoplasm.
3. **Reverse transcription.** RT copies the RNA into dsDNA. RT has no proofreading, so it makes many errors; this is the source of HIV's fast evolution and drug resistance.

4. **Integration.** The DNA enters the nucleus and integrase inserts it into a host chromosome. The integrated DNA is the **provirus**; it can stay silent in resting cells (the latent reservoir).
5. **Expression.** Host RNA polymerase II transcribes the provirus; Tat boosts transcription and Rev exports unspliced RNAs. Gag and Gag-Pol polyproteins are made.
6. **Assembly and budding** at the plasma membrane, taking envelope with gp120/gp41.
7. **Maturation.** Viral protease cuts Gag and Gag-Pol into their final proteins, forming the cone-shaped core. Without this step the particle is not infectious.

Disease and diagnosis. HIV spreads by sexual contact, blood (shared needles, unsafe transfusion) and from mother to child. After an acute flu-like phase, years of clinical latency follow while CD4 T cells fall. AIDS is diagnosed when the CD4 count falls below 200 cells/µL or when opportunistic infections appear, such as tuberculosis, *Pneumocystis* pneumonia and oral candidiasis. Tests detect antibodies (ELISA and rapid tests), antibody plus p24 antigen (fourth-generation tests) or viral RNA (nucleic acid tests, used for viral load). **Antiretroviral therapy (ART)** combines drugs from different classes, so a virus would need several mutations at once to escape.

SARS-CoV-2: structure and life cycle

The WHO declared COVID-19 a pandemic on 11 March 2020. The virion is enveloped, roughly spherical, about 100 nm, with a crown ("corona") of spikes. Its genome is + ssRNA of 29,903 nucleotides in the reference sequence, among the largest RNA genomes known. Four structural proteins: **S** (spike), **E** (envelope), **M** (membrane) and **N** (nucleocapsid, which coats the RNA).

1. **Attachment:** the receptor-binding domain of the S1 part of the spike binds **ACE2** on cells of the nose, airway and lung.
2. **Entry:** a host protease must cut the spike to expose the fusion machinery of S2: TMPRSS2 at the cell surface, or cathepsins inside endosomes.
3. **Translation:** being + sense, the genome is translated at once into two large polyproteins (pp1a and pp1ab, the second made by a ribosomal frameshift). Two viral proteases, the main protease (Mpro) and the papain-like protease (PLpro), cut them into 16 non-structural proteins.
4. **Replication:** these proteins build a replication complex inside double-membrane vesicles made from the endoplasmic reticulum. The RNA-dependent RNA polymerase (nsp12) makes − strand copies, then new genomes and a set of shorter **subgenomic mRNAs** for the structural proteins. Unusually for an RNA virus, an exonuclease (nsp14) proofreads, which keeps the long genome stable.
5. **Assembly and release:** virions assemble in the ER–Golgi compartment and leave by exocytosis.

Tests target the genome (RT-PCR) or the N protein (rapid antigen tests). Drugs target the polymerase (remdesivir, molnupiravir) and the main protease (nirmatrelvir). India used three kinds of vaccine: Covishield (a chimpanzee adenovirus vector carrying the spike gene), Covaxin (whole inactivated virus, Bharat Biotech with ICMR) and ZyCoV-D (a DNA vaccine).

FEATURE	HIV-1	SARS-COV-2
Baltimore group	VI (ssRNA-RT)	IV (+ ssRNA)
Receptor	CD4 + CCR5 or CXCR4	ACE2 (+ TMPRSS2)
Genome copy made in	Nucleus (as provirus)	Cytoplasm only
Proofreading	No	Yes (nsp14)
Infection	Lifelong	Acute, usually cleared

WORKED EXAMPLE

Question (6 marks). (a) Two RT-PCR samples for SARS-CoV-2 give cycle threshold (Ct) values of 20 and 29. Assuming 100% efficiency (the target doubles every cycle), how many times more viral RNA did the first sample hold? (b) A patient's CD4 count is 150 cells/µL. What does this mean?

1. **(a)** Ct is the cycle at which the signal crosses the threshold; less starting RNA needs more cycles. Difference = 29 − 20 = 9 cycles.
2. Each cycle doubles the target, so the ratio = 2^9 = **512 times more RNA** in the first sample. A *lower* Ct means *more* virus.
3. **(b)** Normal CD4 counts are several hundred to over a thousand cells/µL. Below 200 cells/µL defines AIDS in a person with HIV, with high risk of opportunistic infections; ART and preventive drugs (such as co-trimoxazole) are urgent.

EXAM TRAPS

- HIV is **+ sense** ssRNA but is Baltimore group VI, not IV: its RNA is copied to DNA, not translated directly as the first step.
- **gp120 vs gp41:** gp120 binds CD4; gp41 drives fusion. Swapping them is the commonest slip.
- "ART cures HIV" is wrong: it suppresses replication; the provirus stays.
- **Ct value:** high Ct = little RNA. Students often reverse it.
- ACE2 is the receptor; TMPRSS2 is a protease, not a receptor.

CHECK YOURSELF

– REMEMBER **Name the three enzymes packed in the HIV virion.** 3 marks

Reverse transcriptase, integrase, protease.

– REMEMBER **Name the four structural proteins of SARS-CoV-2.** 2 marks

Spike (S), envelope (E), membrane (M), nucleocapsid (N).

– UNDERSTAND **Why does HIV develop drug resistance quickly, and how does combination ART counter it?** 4 marks

RT has no proofreading, so each round makes mutants; huge numbers of virions are made daily. Some mutants resist one drug. With three drugs of different classes, a virus would need several resistance mutations at once, which is very unlikely.

– UNDERSTAND **Why are people with two copies of a broken CCR5 gene (CCR5-Δ32) highly resistant to most HIV-1?** 2 marks

Most transmitted HIV-1 uses CCR5 as its co-receptor. Without CCR5 on their cells, gp120 cannot complete entry.

– APPLY **Match each drug to its target: dolutegravir, nirmatrelvir, maraviroc, tenofovir.** 4 marks

Dolutegravir: HIV integrase. Nirmatrelvir: SARS-CoV-2 main protease. Maraviroc: CCR5 (blocks HIV entry). Tenofovir: HIV reverse transcriptase (nucleotide analogue).

– APPLY **Ct values of 18 and 24 are seen in two patients. Find the fold difference in RNA.** 2 marks

24 − 18 = 6 cycles; 2^6 = **64-fold** more RNA in the Ct 18 patient.

– ANALYZE **HIV infection lasts for life, while most SARS-CoV-2 infections clear in weeks. Explain using their life cycles.** 4 marks

HIV integrates as a provirus in long-lived resting cells, hiding from immunity and drugs, and it destroys the CD4 T cells that coordinate immunity. SARS-CoV-2 stays in the cytoplasm, makes no DNA copy, and infected airway cells die or are cleared by an intact immune response.

BEYOND THE SYLLABUS

A few people have been cured of HIV after stem cell transplants from CCR5-Δ32 donors, done to treat their cancer; gene editing of CCR5 is being studied as a safer route. Long-acting injectable drugs such as cabotegravir and lenacapavir are changing HIV prevention. For coronaviruses, genomic surveillance (in India, the INSACOG network) tracked variants such as Delta and Omicron, and mRNA vaccine technology won the 2023 Nobel Prize in Physiology or Medicine. Search "HIV latent reservoir" and "SARS-CoV-2 variant surveillance". This is not for the exam.

3.7 Viral quantification assays

To make a vaccine, test an antiviral drug or follow a patient, you must know how much virus a sample holds: its **titre**. Viruses are too small to count under a light microscope, so virologists count what they *do*: holes in a lawn of cells, damaged wells, clumped red cells, or copies of their genome. This chapter gives the main assays and the arithmetic an exam expects.

You need: serial dilution and plate counts (Unit 4, Measuring growth), plaques from Chapter 3.2 and cell culture from Chapter 3.3.

Infectivity assays: plaques and TCID50

Plaque assay. A virus sample is serially diluted, usually tenfold. For phages, each dilution is mixed with host bacteria in soft top agar and poured over a nutrient agar plate (the double-agar overlay). For animal viruses, it is added to a cell monolayer, then covered with agarose or methylcellulose so new virus can only reach neighbouring cells. Each infectious particle starts a round of infection that spreads outward and leaves a clear zone, a **plaque**, in the lawn or monolayer (stained with crystal violet or neutral red). One plaque comes from one infectious unit, so:

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titre (PFU/mL) = number of plaques ÷ (dilution × volume plated in mL)
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Here PFU means plaque-forming units, the dilution is the fraction (10^{-6} for a millionfold dilution), and only plates with about 30 to 300 plaques are counted: fewer gives large random error, more and the plaques merge.

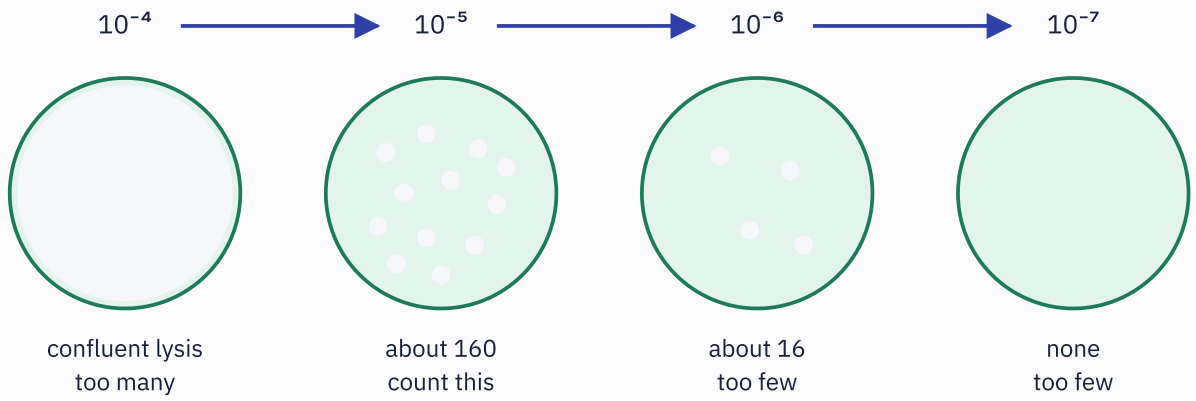


Figure 3.7.1 A plaque assay series (dots stand for plaques; not every plaque drawn). Each tenfold dilution cuts the plaque count tenfold. Only the plate in the 30–300 range is used for the titre; notice that the plate before it is fully cleared.

$TCID_{50}$ (50% tissue culture infectious dose). Some viruses do not form clear plaques. Instead, several wells of cells are inoculated with each dilution, and each well is scored as infected (shows CPE) or not. The $TCID_{50}$ is the dilution that infects half the wells, found by the **Reed–Muench** or the **Spearman–Kärber** method. The same idea in animals gives the LD_{50} (lethal dose) or ID_{50} (infectious dose), and in eggs the EID_{50}. From the Poisson distribution, 1 $TCID_{50}$ holds on average about 0.69 infectious units (ln 2), so PFU ≈ 0.7 × $TCID_{50}$ when both work on the same cells. The **focus-forming assay** is a plaque assay read by antibody staining, for viruses that do not kill cells.

Particle and genome assays

- **Haemagglutination (HA) assay.** Viruses such as influenza clump red blood cells. Twofold dilutions of virus are mixed with red cells in a U-bottom plate: a mat of cells means agglutination; a tight button means none. The **HA titre** is the reciprocal of the highest dilution with full agglutination. It measures particles, infectious or not. Its variant, **haemagglutination inhibition**, measures antibodies.
- **Quantitative PCR (qPCR or RT-qPCR).** Counts genome copies against a standard curve. Fast and sensitive, used for viral load in HIV and hepatitis patients, but it counts dead virus too.
- **Electron microscopy** counts particles directly; **ELISA** measures a viral protein, such as HIV p24.

The **particle-to-PFU ratio** compares total particles with infectious ones. It is usually well above 1, because many particles are defective or never start an infection. Dividing the number of infectious units added by the number of cells gives the **multiplicity of infection** (MOI). With MOI = m, the fraction of cells that get no virus is e^{-m} (Poisson); at m = 1 that is 37%.

WORKED EXAMPLE 1: PLAQUE ASSAY

Question (4 marks). A phage stock is diluted to 10^{-6} and 0.1 mL is plated. Three plates give 148, 162 and 155 plaques. Find the titre.

1. Mean plaques = (148 + 162 + 155) ÷ 3 = 465 ÷ 3 = 155. This is within 30–300, so it is valid.
2. Titre = 155 ÷ (10^{-6} × 0.1 mL) = 155 ÷ 10^{-7} mL = **1.55×10^{9} PFU/mL**.

WORKED EXAMPLE 2: $TCID_{50}$ BY SPEARMAN–KÄRBER

Question (6 marks). Tenfold dilutions 10^{-1} to 10^{-6} of a virus are each put into 8 wells, 0.1 mL per well. Wells with CPE: 8, 8, 8, 6, 2, 0. Find the titre in $TCID_{50}$/mL.

1. Proportions infected: 1, 1, 1, 0.75, 0.25, 0. Their sum S = 4.0.
2. Spearman–Kärber: $\log_{10}$(endpoint dilution) = $x_0 - d(S - 0.5)$, where x_0 = log of the lowest dilution (all wells infected) = −1, and d = log step = 1.
3. = −1 − 1 × (4.0 − 0.5) = −4.5. The 50% endpoint is $10^{-4.5}$: it lies between 10^{-4} (75%) and 10^{-5} (25%), as expected.
4. So 0.1 mL of $10^{-4.5}$ dilution holds 1 $TCID_{50}$; the undiluted stock holds $10^{4.5}$ $TCID_{50}$ per 0.1 mL = $10^{5.5}$ $TCID_{50}$/mL ≈ **3.2×10^{5} $TCID_{50}$/mL**.

EXAM TRAPS

- Divide by the volume plated: forgetting 0.1 mL makes the answer ten times too small.
- PFU counts **infectious** units; HA, qPCR and EM count **particles** or genomes. They do not match.
- $TCID_{50}$ is not a number of particles; it is a dose that infects half the wells.
- Do not use a plate with 5 or 600 plaques for the titre.

CHECK YOURSELF

– REMEMBER **What does PFU stand for, and what is the countable plaque range?** 1 mark

Plaque-forming units; about 30 to 300 plaques per plate.

– UNDERSTAND **Why is an overlay of agarose put on cells in a plaque assay?** 2 marks

It stops new virus spreading through the liquid, so infection can only pass to neighbouring cells and each infectious unit forms one separate plaque.

– APPLY **0.2 mL of a 10^{-5} dilution gives 84 plaques. Find the titre.** 2 marks

$84 \div (10^{-5} \times 0.2) = 84 \div 2 \times 10^{-6} =$ **4.2×10^{7} PFU/mL**.

– APPLY **10^{6} cells are infected with 3×10^{6} PFU. What fraction of cells stay uninfected?** 2 marks

MOI = 3; fraction uninfected = $e^{-3} \approx$ **0.05 (5%)**.

– ANALYZE **A stock has 10^{11} particles/mL by EM but 10^{8} PFU/mL. Interpret this, and say which number a vaccine dose should be based on.** 3 marks

Particle-to-PFU ratio = 1000: most particles are defective or fail to infect. A live vaccine dose is set by infectivity (PFU or $TCID_{50}$), since only infectious units replicate; particle count matters for safety and for non-replicating vaccines.

BEYOND THE SYLLABUS

Digital PCR splits a sample into thousands of droplets and counts positive ones, giving absolute genome numbers without a standard curve, using the same Poisson logic as MOI. Wastewater surveillance used RT-qPCR on city sewage to follow COVID-19 trends, and is now used for other viruses. Search "digital droplet PCR" and "wastewater-based epidemiology". This is not for the exam.

Key terms of this lesson

TERM	MEANING OR FORMULA	CHAPTER
v-onc / c-onc	Viral oncogene / normal cell proto-oncogene	3.5
Provirus	HIV DNA integrated in the host chromosome	3.6
Ct fold change	$2^{\Delta Ct}$ (at 100% PCR efficiency)	3.6
Plaque titre	PFU/mL = plaques ÷ (dilution × mL plated)	3.7
Spearman–Kärber	log endpoint = $x_0 - d(S - 0.5)$	3.7
Uninfected fraction	e^{-MOI}	3.7

Glossary (terms used in this lesson)

ACE2 Angiotensin-converting enzyme 2; the cell receptor for SARS-CoV-2.

Capsid Protein shell around the viral genome.

CD4 Surface protein of helper T cells; the main receptor for HIV.

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

Haemagglutination Clumping of red cells by virus particles.

MOI Multiplicity of infection: infectious units added per cell.

Oncovirus A virus that can cause cancer.

Plaque Clear zone in a cell lawn or monolayer made by virus from one infectious unit.

Provirus Retroviral DNA integrated into the host chromosome.

Reverse transcriptase Enzyme that makes DNA from an RNA template.

$TCID_{50}$ Dose that infects 50% of inoculated cell cultures.

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.

Syllabus: the University free n easy universal syllabus, built from top universities' syllabi.

Built for the University free n easy YouTube channel from the universal syllabus of Microbiology (Oct 2026). Written with top universities' syllabi and standard textbooks; check a formula against your prescribed book before an exam, and report mistakes on the channel.