Microbiology

Unit 3 · Lesson 2 · Animal and plant viruses

The notes for Lesson 2 of Unit 3: chapters 3.3 to 3.4 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 2 of 3 | 2 chapters | Chapters 3.3 to 3.4

About these notes

These are the notes for **Lesson 2 · Animal and plant viruses** of the University free n easy video lessons on Microbiology Unit 3, Viruses and Bacteriophages. They are chapters 3.3 to 3.4 of the unit book, unchanged, so a page here is the same page in the full book.

3.3 Animal viruses: life cycle and cultivation

Animal viruses follow the same plan as phages (attach, enter, copy, assemble, leave), but the details change because an animal cell has no wall, has a nucleus, and takes things in by endocytosis. The whole virion usually enters the cell and must be **uncoated** inside. Because viruses cannot grow on a nutrient medium, a laboratory must supply living cells: a live animal, a fertilised egg, or a cell culture. These cultivation methods are how viruses are isolated from patients, studied and made into vaccines.

You need: envelope, capsid and the Baltimore groups (Chapter 3.1) and the lytic cycle of phages (Chapter 3.2) for comparison.

The life cycle in six steps

1. **Attachment (adsorption).** A viral attachment protein binds a specific receptor on the cell. Influenza haemagglutinin binds sialic acid; HIV gp120 binds CD4; poliovirus binds CD155. The receptor explains host range and tissue tropism: polio infects primates because only primates carry the matching receptor.
2. **Penetration.** Three routes. In **receptor-mediated endocytosis** the cell swallows the bound virion into an endosome (influenza, adenovirus). In **membrane fusion** the envelope fuses

with the plasma membrane and the nucleocapsid is released into the cytoplasm (HIV). Some naked viruses **translocate** or open a pore to pass their genome through the membrane (poliovirus).

3. **Uncoating.** The capsid is removed to free the genome. Often the acid pH of the endosome triggers it: in influenza, the M2 ion channel lets protons into the virion, which loosens the core, and haemagglutinin fuses the envelope with the endosome membrane.
4. **Biosynthesis.** Viral mRNA, proteins and genomes are made. A rule of thumb: DNA viruses replicate in the nucleus, where the host's DNA and RNA polymerases are, except poxviruses, which carry their own enzymes and replicate in the cytoplasm. RNA viruses replicate in the cytoplasm, except influenza and the retroviruses, which use the nucleus. Early proteins (enzymes, regulators) come first, late proteins (capsid parts) later.
5. **Assembly (maturation).** Capsid proteins gather round new genomes. Herpesviruses assemble in the nucleus; picornaviruses in the cytoplasm.
6. **Release.** Naked viruses usually leave when the cell lyses. Enveloped viruses **bud**: viral glycoproteins gather in a patch of host membrane, the nucleocapsid pushes against it, and the membrane wraps it. The envelope comes from the plasma membrane (influenza, HIV), the nuclear membrane (herpes) or internal membranes (coronaviruses, Chapter 3.6). Budding need not kill the cell at once.

The result of infection varies. A **lytic** (cytocidal) infection kills the cell. A **persistent** infection releases virus slowly for a long time (hepatitis B). A **latent** infection keeps the genome silent for years and reactivates later, as herpes simplex does in nerve ganglia to cause cold sores, or varicella-zoster to cause shingles. A **transforming** infection changes cell growth (Chapter 3.5).

Cultivation method 1: experimental animals

Animals were the first hosts used in virology. Suckling (newborn) mice injected into the brain or abdomen are still used for viruses that do not grow well in culture, such as coxsackieviruses and some arboviruses (viruses carried by mosquitoes and ticks). The disease, death or antibody response shows that the virus is present. Animal use is costly, slow and raises welfare concerns, so it is now a last resort.

Cultivation method 2: embryonated eggs

Ernest Goodpasture and colleagues showed in the early 1930s that viruses grow in fertilised hen's eggs. The egg is sterile, cheap and has several tissues, so different viruses can be put in different places. A hole is drilled in the shell over the air sac, the virus is injected, the hole is sealed with wax, and the egg is incubated. The four routes are shown in Figure 3.3.1.

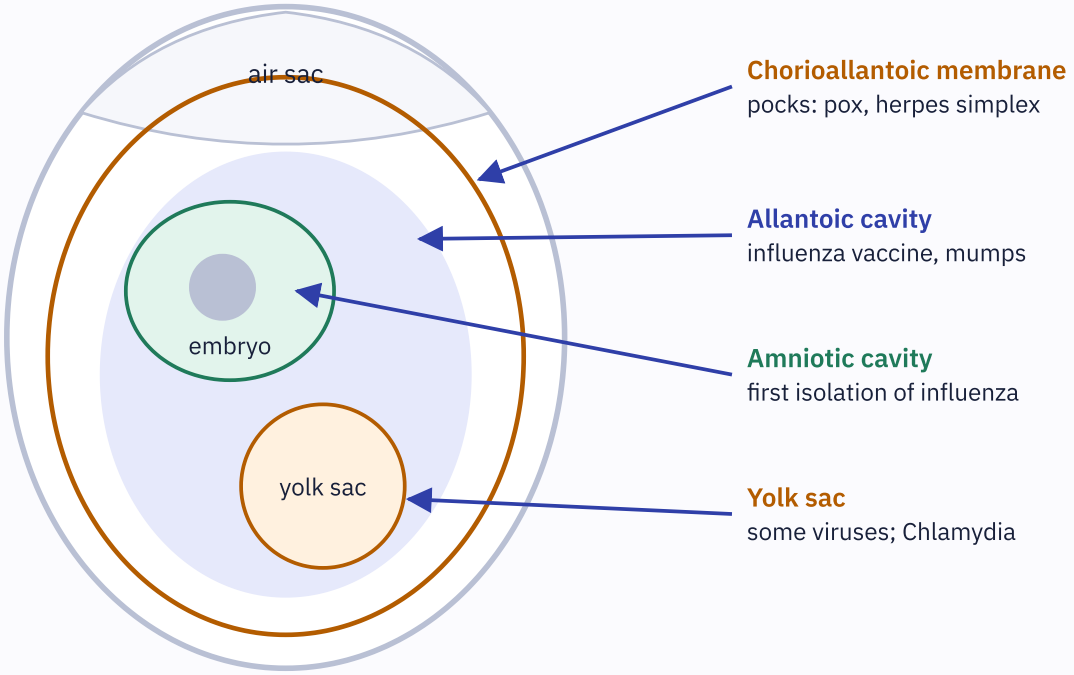


Figure 3.3.1 An embryonated hen's egg (simplified, not to scale) and its four inoculation routes. The orange ring stands for the chorioallantoic membrane, the blue space is the allantoic cavity, the green sac round the embryo is the amnion. Notice that each route matches a group of viruses and a way of reading the result.

- **Chorioallantoic membrane (CAM):** poxviruses and herpes simplex form visible white spots called **pocks**. Counting pocks gives a rough titre, and their look helps identify the virus.
- **Allantoic cavity:** influenza and mumps viruses grow to high yield. Most influenza vaccine has been made this way, using many millions of eggs each year.
- **Amniotic cavity:** used for the first isolation of influenza virus from a patient's sample.
- **Yolk sac:** used for some viruses and for the obligate intracellular bacteria *Chlamydia* and *Rickettsia*.

Growth in an egg is shown by death of the embryo, pocks on the CAM, or haemagglutinin in the harvested fluid (Chapter 3.7).

Cultivation method 3: cell culture

Today most viruses are grown in cells cultured in flasks as a single layer (a **monolayer**) on plastic, fed with a medium containing salts, glucose, amino acids, vitamins and serum. Three kinds are used:

TYPE	WHAT IT IS	EXAMPLES AND USE
Primary culture	Cells taken straight from an organ; survive only a few subcultures	Monkey kidney, chick embryo fibroblasts

TYPE	WHAT IT IS	EXAMPLES AND USE
Diploid cell strain	Normal chromosome number; divide about 50 times, then die (the Hayflick limit)	WI-38, MRC-5 (human foetal lung); used for some human vaccines
Continuous cell line	Grow indefinitely, often from tumours; abnormal chromosomes	HeLa (human cervical cancer), Vero (monkey kidney), HEp-2, BHK-21, MDCK (dog kidney, for influenza)

Virus growth in culture is detected by:

- **Cytopathic effect (CPE):** visible damage seen under the microscope, such as rounding and detachment of cells, giant multinucleate cells (**syncytia**, in measles and respiratory syncytial virus), or **inclusion bodies** (Negri bodies of rabies in nerve cells).
- **Haemadsorption:** red blood cells stick to cells infected by influenza or parainfluenza viruses, because viral haemagglutinin sits on the cell surface before budding. This shows the virus even when there is no CPE.
- **Interference:** a non-cytopathic virus is revealed when it blocks the CPE of a second virus added later.
- **Immunofluorescence or PCR** on the cells, for viral antigen or genome.

WORKED EXAMPLE

Question (8 marks). A throat swab from a child with fever is sent for virus isolation. Influenza is suspected. (a) Describe two ways to grow the virus. (b) After three days the cell culture shows little CPE. How would you still show influenza virus is present? (c) Why is the sample not put on nutrient agar?

1. **(a) Egg:** inoculate the amniotic cavity of an embryonated egg for first isolation; pass the harvested fluid into the allantoic cavity for higher yield. **Cell culture:** inoculate MDCK cells (with trypsin in the medium, which influenza needs to cleave its haemagglutinin).
2. **(b) Haemadsorption:** add guinea pig or chicken red cells to the monolayer; they stick to infected cells. Or test the culture fluid for **haemagglutination**: red cells clump. Confirm the type by immunofluorescence or RT-PCR.
3. **(c)** Viruses have no metabolism and need living cells; on agar they will not multiply at all.

EXAM TRAPS

- **Haemagglutination vs haemadsorption:** haemagglutination is red cells clumping with free virus in a tube; haemadsorption is red cells sticking to infected cells in a monolayer.
- **Cell strain vs cell line:** a diploid strain has a limited lifespan; a continuous line grows forever. HeLa is a line, not a strain.
- **"All DNA viruses replicate in the nucleus"**: poxviruses are the exception. "All RNA viruses replicate in the cytoplasm": influenza and retroviruses are the exceptions.
- Do not write "the animal virus injects its DNA". Only phages with tails inject; animal viruses enter whole.
- The favourite question: "Describe the cultivation of viruses", with a labelled egg diagram. Draw the four routes.

CHECK YOURSELF

– REMEMBER **Name the four routes of inoculation in an embryonated egg.** 2 marks

Chorioallantoic membrane, allantoic cavity, amniotic cavity, yolk sac.

– REMEMBER **What is cytopathic effect? Give two examples.** 2 marks

Visible damage to cultured cells caused by virus growth: cell rounding and detachment, syncytia, inclusion bodies.

– UNDERSTAND **Why does an enveloped virus usually not need to lyse its host cell to leave it?** 3 marks

It leaves by budding: viral glycoproteins gather in the host membrane and the nucleocapsid is wrapped by that membrane as it pushes out. The membrane reseals, so the cell can keep releasing virus.

– UNDERSTAND **Why does the receptor decide which tissues a virus infects?** 2 marks

The first step is binding of the viral attachment protein to a specific receptor. Cells that lack the receptor cannot be entered, so only tissues that carry it are infected.

– APPLY **Choose a cultivation method for (a) vaccinia virus to see pocks, (b) a rabies vaccine, (c) a coxsackievirus that grows poorly in cells.** 3 marks

(a) Chorioallantoic membrane of an egg. (b) A cell culture such as Vero cells or a diploid strain. (c) Suckling mice.

– APPLY **A drug blocks the influenza M2 ion channel. Which step of the life cycle does it stop, and why?** 3 marks

Uncoating. M2 lets protons from the acid endosome into the virion; this loosens the core so the genome can be released. Blocking M2 keeps the genome locked in the capsid. (Amantadine acts this way.)

– ANALYZE **Compare cell culture and embryonated eggs for making a vaccine for millions of people.** 4 marks

Eggs are proven and cheap per egg but need huge numbers of eggs months ahead, are hard to scale fast, and some people are allergic to egg protein. Cell culture in bioreactors scales faster, is closed and sterile, avoids egg protein, but needs certified cell lines and costly media. For a sudden outbreak, cell culture is more flexible.

BEYOND THE SYLLABUS

Vaccine plants now grow viruses in Vero or MDCK cells on microcarrier beads in stirred bioreactors of hundreds or thousands of litres, the same scale-up ideas taught in bioprocess engineering. Organoids, three-dimensional mini-organs grown from stem cells, let researchers grow viruses such as human norovirus that refused to grow in ordinary cell lines. The HeLa line also raises questions of consent, since the cells were taken in 1951 without the patient's permission. Search "Vero cell bioreactor vaccine" and "organoid virology" to go further. This is not for the exam.

3.4 Plant viruses

Plant viruses cause mosaic, leaf curl, yellowing and stunting in crops, and in India they cut the yield of banana, papaya, cotton, tomato and rice. Their biology is shaped by one obstacle: the thick plant cell wall. A plant virus cannot enter a cell by itself, so it needs a wound or a vector, usually an insect, to get in, and special movement proteins to spread from cell to cell. This shorter chapter gives the core that an exam answer needs.

Features and examples

Most plant viruses have + ssRNA genomes. Tobacco mosaic virus (TMV) is a rigid helical rod about 300 nm long and 18 nm wide, with about 2,130 copies of one coat protein wound round an RNA of about 6,400 nucleotides. Cauliflower mosaic virus (CaMV) has dsDNA copied through

RNA (Baltimore group VII), and the geminiviruses (twin icosahedra) have small circular ssDNA genomes. Indian examples worth naming:

- **Banana bunchy top virus**: ssDNA; spread by the banana aphid; leaves bunch at the top.
- **Papaya ringspot virus**: + ssRNA potyvirus; spread by aphids; ring spots on fruit and mosaic on leaves.
- **Cotton leaf curl and tomato leaf curl viruses**: geminiviruses (begomoviruses) spread by the whitefly *Bemisia tabaci*.
- **Rice tungro disease**: two viruses together, spread by green leafhoppers; yellow-orange, stunted plants.

Even smaller are **viroids**: naked circular RNAs of a few hundred nucleotides that code for no protein. Potato spindle tuber viroid, found by Theodor Diener in 1971, has 359 nucleotides.

How plant viruses spread

Between plants, viruses travel by **mechanical contact** (TMV on hands and tools), through **seed** and pollen, by **grafting** and vegetative propagation (banana suckers, potato tubers), and above all by **vectors**: aphids, whiteflies, leafhoppers, thrips, nematodes and soil fungi. Insect transmission is classed by how long the vector stays infective.

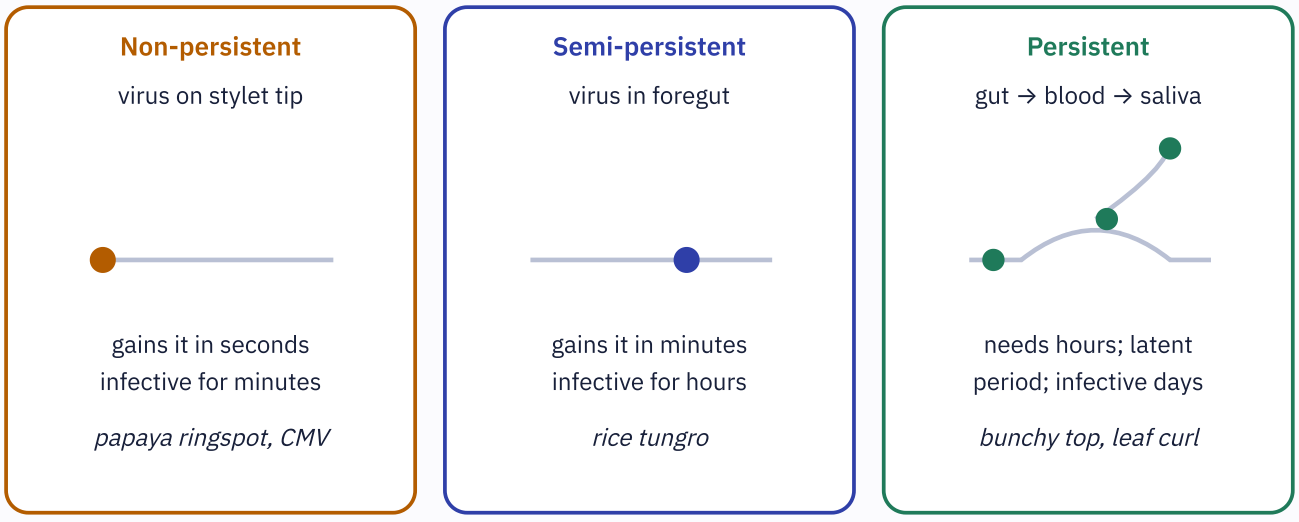


Figure 3.4.1 The three modes of insect transmission. The grey line is the insect's feeding tube from mouthparts (left) inward; the dots show where the virus is held. Notice that the deeper the virus goes into the insect, the longer it takes to pick up and the longer the insect stays infective.

Inside the plant, the virus moves from cell to cell through **plasmodesmata**, the channels that link neighbouring cells. A viral **movement protein** (the 30 kDa protein of TMV is the classic case) widens these channels so the genome can pass. For long-distance spread, the virus enters the

phloem and travels with the sugar flow to young leaves and roots; that is why symptoms often appear first in new leaves.

Diagnosis and control

Plant viruses are detected by symptoms, by **indicator plants** (hosts that show a clear reaction), by ELISA for coat protein, and by PCR or RT-PCR. In the **local lesion assay** (Francis Holmes, 1929), TMV rubbed on leaves of a resistant tobacco such as *Nicotiana glutinosa* gives dead spots, one per infection, which can be counted like plaques (Chapter 3.7).

There are no antiviral sprays for crops, so control is preventive: virus-free planting material (often from **meristem tip culture**, since the growing tip is frequently free of virus), control of vectors, removal of infected plants, resistant varieties, and **cross-protection** (a mild strain protects against a severe one). Genetic engineering added **coat-protein-mediated resistance**: transgenic papaya carrying the papaya ringspot virus coat protein gene has been grown in Hawaii since the late 1990s.

WORKED EXAMPLE

Question (4 marks). Aphids fed on an infected plant for 30 seconds transmit virus P at once, but lose the ability within an hour. Aphids fed for 30 seconds never transmit virus Q; those fed for a day transmit it only after a two-day wait, and then for the rest of their lives. Classify each mode and say which control works better against each.

1. **Virus P:** short acquisition, no waiting, short retention: **non-persistent** (stylet-borne).
2. **Virus Q:** long acquisition, a latent period, long retention: **persistent circulative**.
3. **Control:** insecticides kill too slowly to stop P, because a brief probe is enough; reflective mulches, barrier crops or oil sprays work better. For Q, killing vectors before the latent period ends does help.

EXAM TRAPS

- **Viroid vs virus:** a viroid has no capsid and codes for no protein; do not call it "a small virus".
- Plant viruses cannot "attach to a receptor" on an intact plant cell; the wall blocks them. Entry is through wounds or vectors.
- Do not write that insecticides control all insect-borne plant viruses; they fail for non-persistent ones.

CHECK YOURSELF

– REMEMBER **Name the vector of cotton leaf curl virus.** 1 mark

The whitefly *Bemisia tabaci*.

– UNDERSTAND **Why do plant viruses need movement proteins?** 2 marks

The cell wall blocks free passage between cells; movement proteins widen the plasmodesmata so the viral genome can pass to the next cell.

– UNDERSTAND **Why is meristem tip culture used to make virus-free plants?** 2 marks

The fast-dividing tip has no mature vascular tissue and the virus often has not reached it, so plants grown from it start free of virus.

– APPLY **TMV has about 2,130 coat protein subunits and each covers 3 nucleotides. Estimate the RNA length.** 2 marks

2,130 × 3 = 6,390 nucleotides, close to the measured length of about 6,400.

– ANALYZE **A farmer sprays insecticide weekly, yet papaya ringspot spreads. Explain and suggest a better plan.** 3 marks

The virus is non-persistent: an aphid probing for seconds transmits it before the insecticide kills it. Use virus-free seedlings, barrier crops, removal of infected plants, and resistant or transgenic varieties.

BEYOND THE SYLLABUS

The CaMV 35S promoter is one of the most used promoters in plant genetic engineering. Plant viruses are also being used as expression vectors to make vaccines and antibodies in tobacco leaves, and RNA interference sprays that target viral genes are under study. Search "virus-induced gene silencing" and "plant molecular farming". This is not for the exam.

Key terms of this lesson

TERM	MEANING OR FORMULA	CHAPTER
Cytopathic effect	Visible damage to cultured cells by virus	3.3
Movement protein	Widens plasmodesmata for cell-to-cell spread	3.4

Glossary (terms used in this lesson)

Capsid Protein shell around the viral genome.

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

Cytopathic effect Visible damage to cells caused by virus growth.

Embryonated egg Fertilised hen's egg with a developing embryo, used to grow viruses.

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

Haemadsorption Red cells sticking to virus-infected cells in culture.

Haemagglutination Clumping of red cells by virus particles.

Latent period Time from infection until progeny virus is released.

Movement protein Plant virus protein that lets the genome pass through plasmodesmata.

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

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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