Viruses

A2.3 · HL only

How can a small particle redirect a whole cell?

Host dependence and evolution connect the unit

A virus carries a genome and protective structures, but uses an infected cell to make components and assemble progeny.

A2.3 is HL only. Compare viral types, lambda pathways, origin hypotheses, influenza and HIV evolution.

Numerical examples are invented, not patient or outbreak measurements.
Prerequisites: gene expression, membranes and selection.

A genome inside a capsid is not a cell

Distinguish particle, genetic material and host

A virion is a complete particle outside a host. Its genome is nucleic acid; its capsid is protein.

It has no cellular cytoplasm containing ribosomes and a complete metabolic system. It does not grow and divide like a bacterium.

New particles assemble from components made during infection. Some carry enzymes, but still depend on the host.

A packaged genome is not every replication intermediate

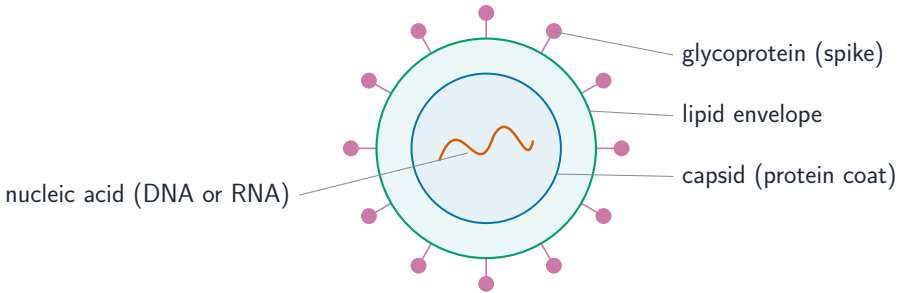
DNA and RNA viruses follow different pathways

Viral genomes can be DNA or RNA and single- or double-stranded, depending on the virus.

HIV packages RNA but makes a DNA intermediate by reverse transcription inside an infected cell.

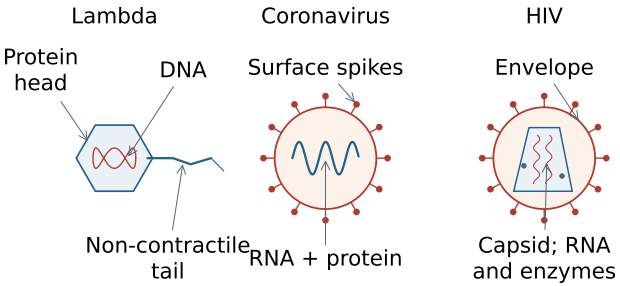
A lipid envelope comes from host membranes and contains viral proteins. It is not a cell wall or a complete cellular system.

The structure of a virus



No cytoplasm, no ribosomes, no metabolism

Compare organization without assuming a common scale



Schematic organization; no shared scale.
Envelope proteins and genomes are simplified.

Lambda has a DNA-containing head and non-contractile tail; coronavirus and HIV have envelopes.

Compare packaged genomes and envelopes

Virus	**Model used in this unit**
Lambda	Double-stranded DNA; head and non-contractile tail; no envelope
Coronavirus	Single-stranded RNA with protein; envelope and spikes
HIV	Two RNA copies plus enzymes; capsid and envelope

A suitable receptor is necessary but may be insufficient

Host range depends on several steps

Viral surface components interact with suitable host receptors. Failure to attach can prevent infection.

Entry, expression and genome copying also need compatible cell conditions. Binding alone may not permit reproduction.

A pathogen is a disease-causing agent. Not every virus infects humans or immediately causes visible illness.

Measure the particle before describing its size

1. **Invented image.** 9.0 mm represents a particle of diameter 90 nm.
2. **Convert.** $I = 9.0 \times 10^{6}\,\mathrm{nm}$.
3. **Ratio.** $M = (9.0 \times 10^{6})/90 = 1.0 \times 10^{5}$.

Decision

A large print does not establish that the original instrument resolved internal details.

Connect host resources to viral production

Requirement	Connection to the infected cell
Ribosomes	Translate viral proteins
Building materials	Supply amino acids, nucleotides and other components
Energy	Supports component synthesis and cellular processes
Enzymes	Viral and host contributions differ among viruses

Assembly is different from a particle dividing

Follow information, synthesis and release

Viral gene expression produces proteins; genomes are copied and packaged into new particles.

Some viruses release particles by lysis. Others bud through membranes, acquiring envelopes containing viral proteins.

HIV reverse transcriptase copies RNA into DNA. One carried enzyme does not supply autonomous metabolism or translation.

Different targets explain different interventions

Separate bacterial targets, viral stages and immune memory

Antibiotics aimed at peptidoglycan synthesis have no equivalent viral cell-wall target.

Antivirals target particular entry or replication stages; they are not interchangeable universal virus treatments.

Vaccination can establish antigen-specific memory supporting faster secondary responses. It is not an immediate cure for an established infection.

Bacterial antibiotic resistance evolves through selection

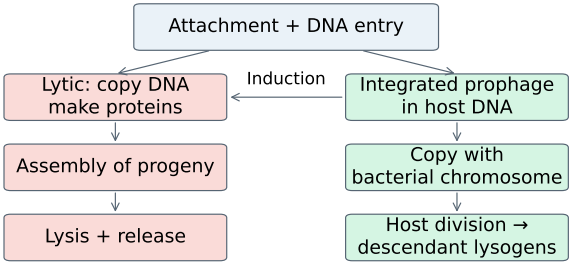
Resistance changes the bacterial population

Bacteria can differ in inherited antibiotic resistance before treatment.

An antibiotic inhibits susceptible bacteria more strongly; resistant variants can leave more descendants.

The resistant fraction can rise. An individual bacterium does not choose to become resistant because it needs to survive.

Follow lambda DNA along two possible pathways



Lysogeny copies integrated DNA with the host.
It does not itself require a burst of free particles.

> Lytic production ends in assembly and release; lysogeny copies integrated viral DNA with the host.

Order the events of a lytic infection

1. **Attach and enter.** The tail region binds a suitable bacterial surface receptor; phage DNA enters.
2. **Biosynthesize and assemble.** DNA is copied, proteins are produced and genomes are packaged into progeny particles.
3. **Release.** Phage-directed envelope disruption lyses the bacterium and releases progeny.

Decision

The whole head and tail are not injected as a ready-made cell.

Calculate burst size only under the stated model

1. **Invented single cycle.** 80 infected cells release 4,800 newly produced infectious particles.
2. **Average.** $B = 4800/80 = 60$ infectious particles per infected cell.
3. **Assumptions.** Input particles were removed; no second infection cycle contributes.

Decision

Infectious units need not count every physical particle, and an average need not describe every cell.

An integrated prophage is DNA, not a hidden virion

A lysogen carries phage DNA within its chromosome

Lambda DNA integrates as a prophage. The carrying bacterium is a lysogen; immediate lytic production is suppressed.

Chromosome copying and host division pass the prophage to descendant cells.

Bacteria have no membrane-bound nucleus. Lysogeny does not mean that the host stops growing or all viral genes are silent.

Count descendant bacteria separately from free particles

1. **Invented model.** One lysogen; no induction, loss or cell death.
2. **Four division rounds.** $N = 2^4 = 16$ prophage-carrying cells.
3. **Interpretation.** Integrated viral DNA is copied with host DNA; no free-particle burst is required.

Decision

The counted objects are bacteria carrying a prophage, not released virions.

Induction changes the regulated pathway

A temperate phage can enter lytic production

Suitable conditions, including particular DNA-damage responses, can induce a switch from lambda lysogeny.

Excision and viral gene expression allow DNA copying, assembly and lysis/release.

Not all viruses integrate. HIV integration is not automatically the lambda lysogenic cycle; compare mechanisms explicitly.

Several origins are hypotheses tested by combined evidence

Diversity does not establish one simple origin story

Genomes, replication systems and structural organization differ widely among viral groups.

Proposed routes include genetic elements escaping cells and cellular parasites losing functions as dependence increased.

Shared genetic-code meanings connect viral expression with cellular systems, but do not require identical viral genomes or genes.

Similar needs can favour similar structures independently

Convergence is an alternative to a simple ancestry claim

Protecting a genome and transferring it between hosts are useful functions for unrelated obligate parasites.

A protein shell alone is insufficient to prove one recent common origin of all viruses. Compare sequences and replication mechanisms.

Diversity alone also cannot identify one group's exact historical route. Evaluate an origin hypothesis with observations and limits.

Mutation supplies variation; selection changes success

Useful changes are not produced by deliberate need

In some viruses, frequent copying, large populations and error-prone genome copying generate many variants quickly.

Many mutations are neutral or harmful. Some inherited variants reproduce more successfully under particular immune or drug conditions.

Copying accuracy, bottlenecks and selection differ. Not every RNA virus evolves at the same rate.

Influenza drift changes antigenic recognition

Small changes can affect surface proteins

Mutations accumulating in influenza genes can alter antigens, including haemagglutinin and neuraminidase.

Antibodies formed after an earlier infection or vaccination may then recognize some changed variants less effectively.

This antigenic drift motivates surveillance and assessment of vaccine strain matching; it does not mean all immune recognition disappears.

Reassortment is not a synonym for one small mutation

Segmented genomes allow new segment combinations

When different influenza viruses infect one cell, progeny can receive a new combination of genome segments.

In influenza A, major antigenic change can produce antigenic shift. Not every reassortment leads to such a change or to a pandemic.

Efficient transmission and population immunity also matter. Drift and shift are different mechanisms.

HIV variation can complicate continued suppression

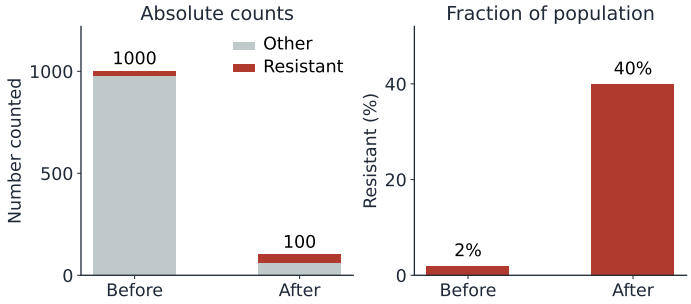
Copying, selection and integrated DNA matter

Reverse transcription is relatively error-prone; frequent replication generates variants and recombination can add variation.

Drug or immune conditions can favour variants already able to reproduce under them. Drugs do not instruct useful mutations.

Combination therapy suppresses replication through multiple targets. Suppression does not establish removal of every integrated viral genome.

Separate resistant count, fraction and total count



Invented data: resistant 20/1000 → 40/100.
Total count falls 90%; resistant count doubles.
Resistant fraction rises 20-fold.

Resistant: 20/1000 → 40/100. Total count falls; resistant fraction rises.

Recompute all three quantities

1. **Resistant fraction.** $20/1000 = 2\%$; $40/100 = 40\%$: a $40/2 = 20$-fold rise.
2. **Resistant count.** $20 \to 40$: a twofold rise.
3. **Total count.** $1000 \to 100$: $(1000 - 100)/1000 = 90\%$ reduction.

Decision

The pattern fits differential success, but does not identify the gene or rule out every sampling effect.

Use a common denominator for infection comparisons

1. **Invented group A.** 18 cases among 900 people in one month: $18/900 \times 1000 = 20$ per 1,000.
2. **Invented group B.** 10 among 250 in the same month: $10/250 \times 1000 = 40$ per 1,000.
3. **Interpretation.** B has fewer cases but a higher proportion. Different testing or exposure can still affect comparison.

Decision

Distinguish existing cases at one time from new cases during an interval. Use published data or simulations.

Try a burst-size and prophage-count problem

Name the counted objects before calculating

Invented single cycle: 90 infected bacteria release 6,300 new infectious particles after input removal. Find mean burst size.

Separately, one lysogen divides five times without induction, death or prophage loss. Find descendant carriers.

Then explain why the second calculation does not require release of any free particles.

Reveal: infectious output differs from carrier-cell number

1. **Burst size.** $B = 6300/90 = 70$ infectious particles per infected cell on average.
2. **Carrier cells.** $N = 2^5 = 32$ descendant lysogens.
3. **Pathway.** Integrated DNA is copied with host DNA; zero free-particle release is consistent with the second model.

Check yourself

Follow genetic material, mechanisms and denominators

Distinguish packaged genome, copied genome, integrated prophage and released particle.

Explain lambda's pathways and viral evolution without deliberate choices or need-directed mutation.

Keep absolute counts, fractions and rates separate. A higher resistant fraction can coexist with fewer total particles.