

Virology

Where viruses may have come from, what a virion is actually made of, how phages choose between killing and hiding, and the biology behind the flu, hepatitis, and HIV.

Viral Structure Baltimore Classification Lytic/Lysogenic Cycle Interferon

Influenza Hepatitis HIV-AIDS

- Origin
- Structure
- Classification
- Phage Cycles
- Interferon
- Rhino/Flu/Hep

1 Origin & Evolution of Viruses

Three competing hypotheses — likely no single answer for all viruses

Hypothesis	Core idea	Supporting evidence
Regressive (degeneracy)	Free-living cells became obligate intracellular parasites, progressively losing genes	Complex viruses (poxvirus, mimivirus) with many genes; parallels obligate intracellular bacteria
Progressive (escaped gene)	Mobile genetic elements (plasmids/transposons) acquired protein coats and cell-to-cell mobility	Retrovirus–transposon similarities
Coevolution (virus-first)	Viruses coevolved alongside cellular life from a pre-cellular RNA world	RNA world hypothesis; RNA genome viruses' primacy

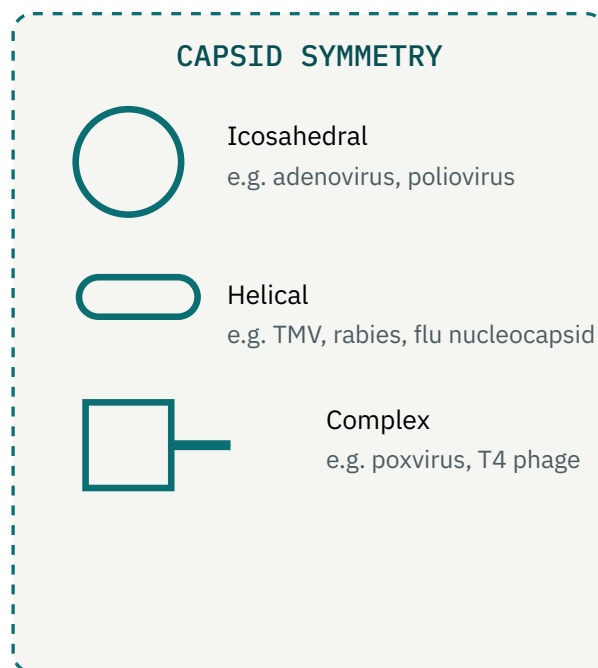
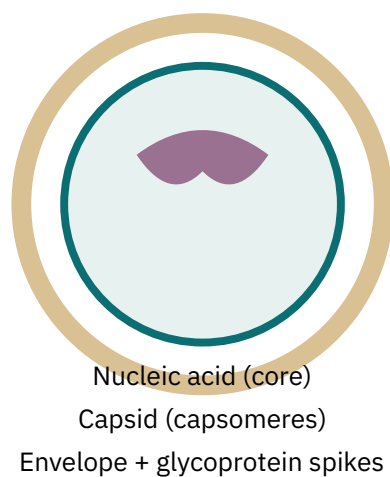
ARE VIRUSES "ALIVE"?

Viruses are acellular with no independent metabolism — by most definitions, not alive. Yet they replicate, mutate, and evolve under selective pressure, which is exactly why influenza needs a new vaccine every year.

2 Viral Structure — Virion, Prion & Bacteriophage

What's actually inside an infectious particle

The virion, and the three capsid symmetries



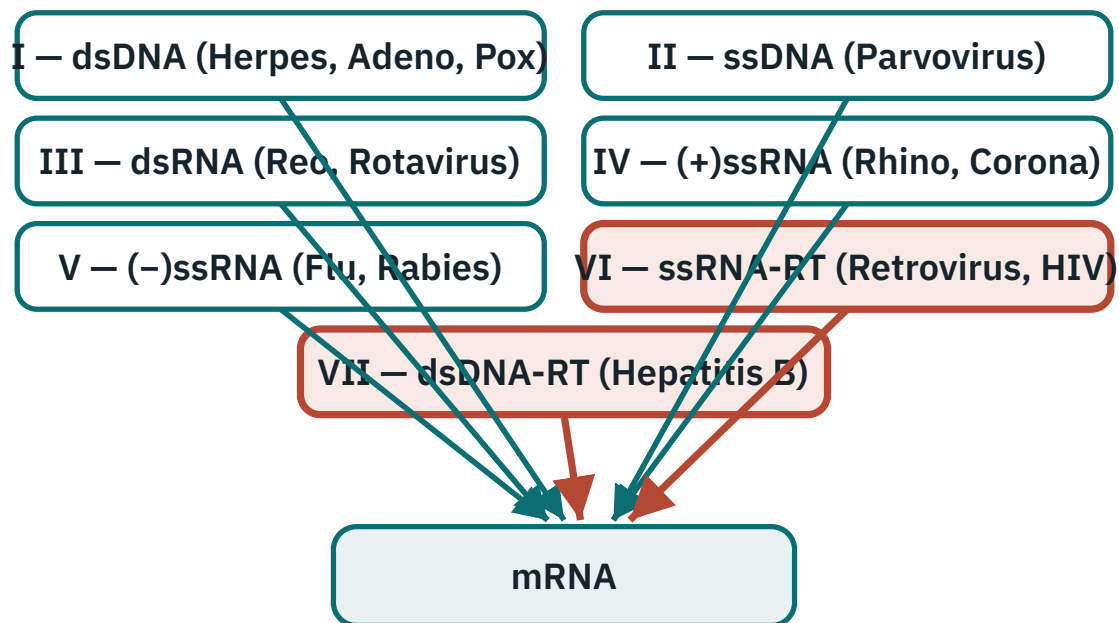
Particle	Genome	Key feature
Virion	DNA or RNA (never both)	Complete infectious particle — capsid ± envelope
Prion	None	Misfolded host protein (PrP ^{Sc}) templates normal PrP ^C to misfold; causes CJD, kuru, BSE; resists standard sterilization; no immune response (self protein)

Bacteriophage	DNA or RNA	Infects bacteria; often complex structure — icosahedral head + tail + tail fibres (e.g. T4)
---------------	------------	---

3 Classification of Viruses

Baltimore classification — grouped by genome type and route to mRNA

Seven classes, one destination: mRNA



CLASSES VI & VII NEED REVERSE TRANSCRIPTASE

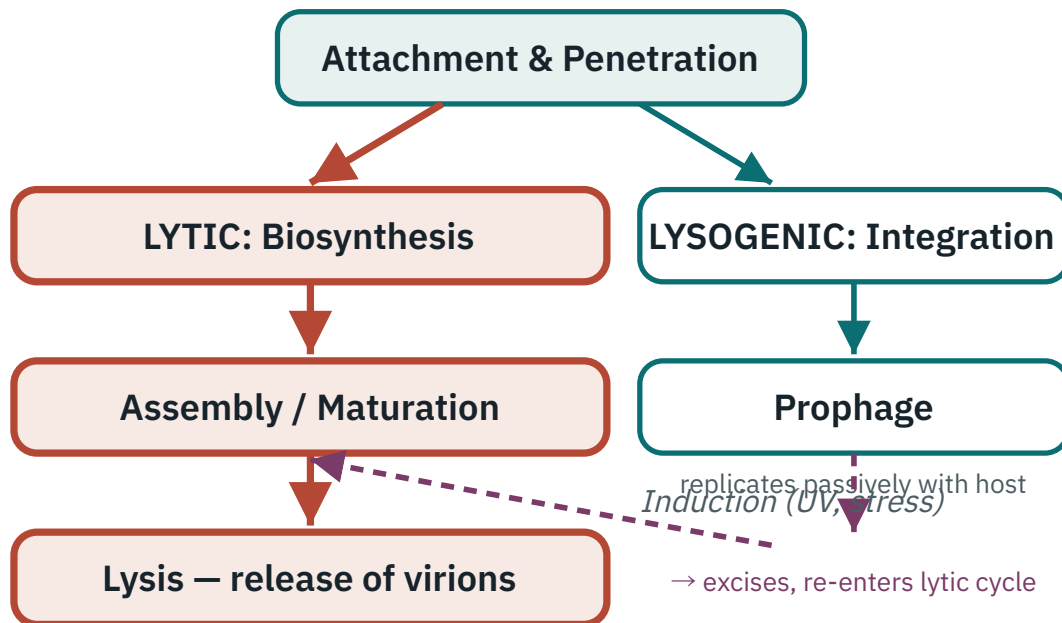
Class VI (retroviruses, e.g. HIV): RNA genome → reverse-transcribed to DNA → integrates as provirus

Class VII (HBV, a "pararetrovirus"): DNA genome, but replicates via an RNA intermediate + reverse transcriptase

4 Bacteriophage Replication

Lytic and lysogenic cycles

Two fates after infection



LYSOGENIC CONVERSION

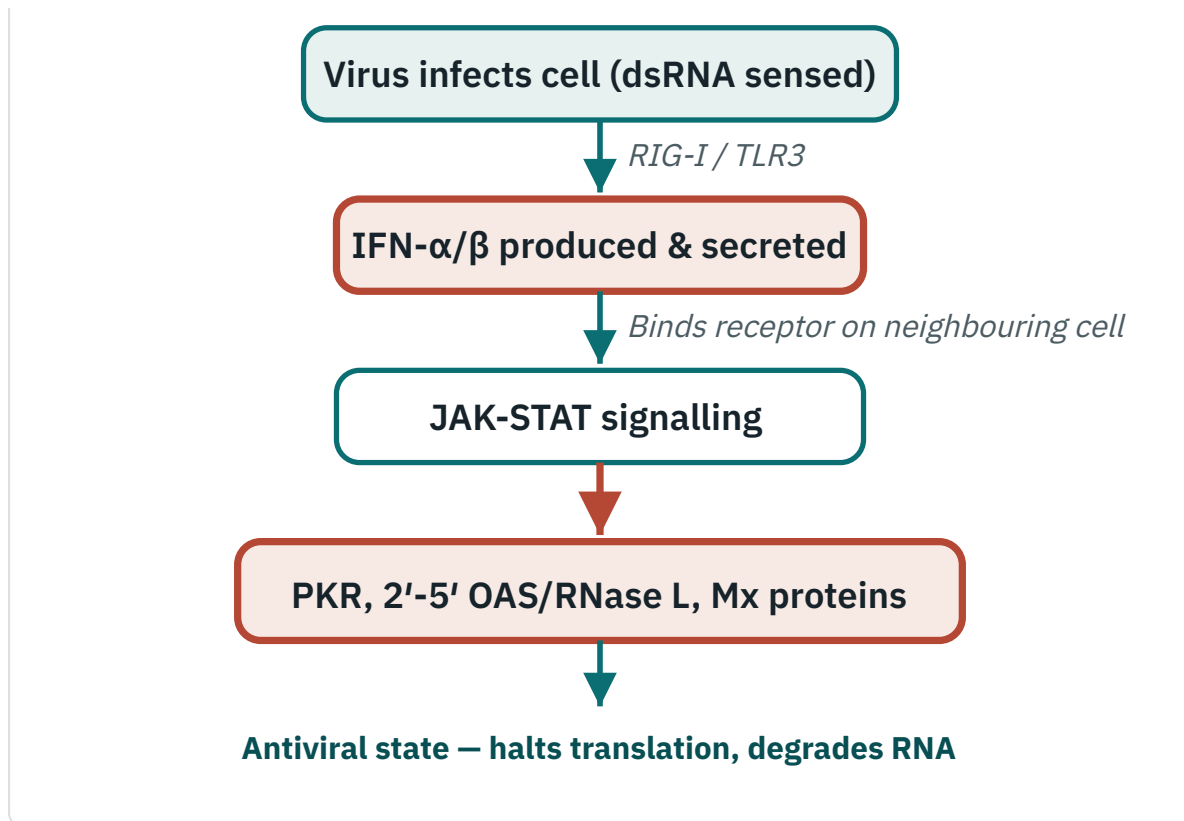
A prophage can hand its host new, sometimes pathogenic, traits — diphtheria toxin (

C. diphtheriae) and cholera toxin (*V. cholerae*) are both phage-encoded genes.

5 Interferon

The cell's early warning cytokine

From infected cell to an antiviral state next door



BEYOND THE ANTIVIRAL STATE

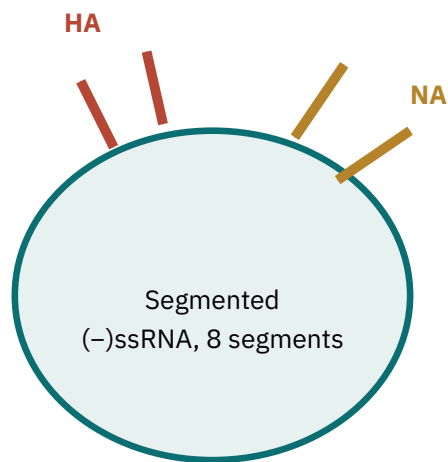
Interferons also activate NK cells and upregulate MHC I, improving CTL recognition of infected cells. Therapeutically, IFN-α treats hepatitis B/C and certain cancers, while IFN-β is used in multiple sclerosis.

6 Rhinoviruses, Influenza & Hepatitis Viruses

Structure, transmission, and control across three very different families

Rhinovirus — (+)ssRNA, non-enveloped, icosahedral (Picornaviridae). Spread by droplets and fomites (stable on surfaces, being non-enveloped). Causes the common cold; no vaccine exists because of enormous serotype diversity (>100 types) — control relies on hand hygiene.

Influenza — HA/NA and antigenic change



Spread: droplets/aerosols
Control: annual vaccine,
neuraminidase inhibitors

Antigenic DRIFT

Small HA/NA mutations —
gradual, seasonal epidemics,
needs yearly vaccine update

Antigenic SHIFT

Reassortment of segments
between strains (e.g. human +
avian/swine) — abrupt, can
cause pandemics (no prior immunity)

Hepatitis viruses

Virus	Genome	Transmission	Chronicity	Vaccine
HAV	(+)ssRNA, non-enveloped	Faecal-oral	None	Yes (inactivated)
HBV	dsDNA (partial), enveloped	Parenteral, sexual, vertical	Yes — esp. if perinatal	Yes (recombinant HBsAg)
HCV	(+)ssRNA, enveloped	Parenteral (blood-borne)	High (~75–85%)	No — but direct-acting antivirals cure >95%
HDV	Defective — needs HBsAg	Parenteral (HBV co/superinfection)	Worsens HBV severity	Prevented by HBV vaccine
HEV	(+)ssRNA, non-enveloped	Faecal-oral (water)	Usually none; severe in pregnancy	Limited availability

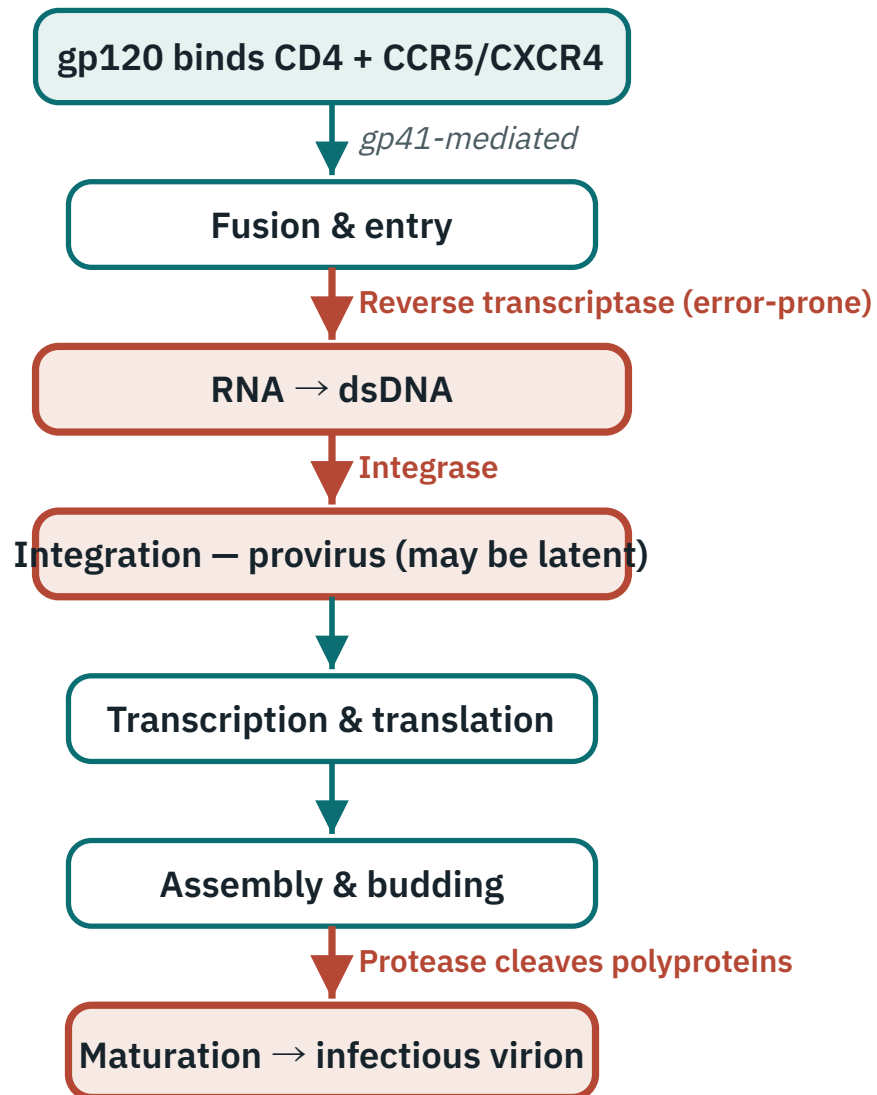
THE A-E SHORTCUT

"A and E hit your bowels" — both are faecal-oral, self-limited. B, C, and D are blood-borne/parenteral and carry the real risk of chronic liver disease.

7 HIV-AIDS

A retrovirus that dismantles the immune system it depends on

HIV replication cycle



Reverse transcriptase, integrase, protease — the three drug targets

Aspect	Detail
Pathogenesis	Progressive CD4 ⁺ T-cell depletion → immunodeficiency → AIDS (CD4 <200/μL or AIDS-defining illness, e.g. <i>Pneumocystis</i> pneumonia, Kaposi's sarcoma)

Transmission	Sexual contact, blood/blood products, vertical (in utero, delivery, breastfeeding) — not casual contact
Diagnosis	ELISA screening → confirmatory test; HIV RNA viral load; CD4 count monitoring
Treatment	Combination ART (NRTIs/NNRTIs, protease inhibitors, integrase inhibitors, entry/fusion inhibitors) — suppresses but does not cure (latent reservoir persists)
Prevention	Condoms, needle exchange, blood-supply screening, PrEP/PEP, antiretrovirals to prevent mother-to-child transmission

U = U

Effective ART that suppresses viral load to undetectable levels also makes the virus untransmissible sexually ("Undetectable = Untransmittable") — a cornerstone message of modern HIV care, even though the latent proviral reservoir means the infection is controlled, not cured.

8 Exam Corner

Practice question set — end-semester format

B.Sc. Biochemistry — End-Semester Examination

Paper: Virology

Full Marks: 30

Time: 1.5 Hrs

GROUP A — LONG ANSWER (ANSWER ANY TWO)

- Describe the lytic and lysogenic cycles of bacteriophage replication, and explain the concept of lysogenic conversion with an example. **10**
- Describe the replication cycle of HIV, and correlate each step with an antiretroviral drug class that targets it. **10**
- Explain the Baltimore classification of viruses, giving one example of each class and outlining its route to mRNA. **10**

GROUP B — SHORT NOTES (ANSWER ANY TWO)

- | | |
|---|---|
| a. Interferon and the antiviral state | 5 |
| b. Antigenic drift vs. antigenic shift in influenza | 5 |
| c. Prions and transmissible spongiform encephalopathies | 5 |