

A2.3 VIRUSES

Guiding Questions

How can viruses exist with so few genes?

In what ways do viruses vary?

Linking Questions

What mechanisms contribute to convergent evolution?

To what extent is the natural history of life characterized by increasing complexity or simplicity?

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Theme: Unity and Diversity

Level of Organization: Cells

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HL LEARNING OUTCOMES

A2.3.1	Structural features common to viruses	Relatively few features are shared by all viruses: small, fixed size; nucleic acid (DNA or RNA) as genetic material; a capsid made of protein; no cytoplasm; and few or no enzymes.
A2.3.2	Diversity of structure in viruses	Students should understand that viruses are highly diverse in their shape and structure. Genetic material may be RNA or DNA, which can be either single- or double-stranded. Some viruses are enveloped in host cell membrane and others are not enveloped. Virus examples include bacteriophage lambda, coronaviruses and HIV.
A2.3.3	Lytic cycle of a virus	Students should appreciate that viruses rely on a host cell for energy supply, nutrition, protein synthesis and other life functions. Use bacteriophage lambda as an example of the phases in a lytic cycle.
A2.3.4	Lysogenic cycle of a virus	Use bacteriophage lambda as an example.
A2.3.5	Evidence for several origins of viruses from other organisms	The diversity of viruses suggests several possible origins. Viruses share an extreme form of obligate parasitism as a mode of existence, so the structural features that they have in common could be regarded as convergent evolution. The genetic code is shared between viruses and living organisms.
A2.3.6	Rapid evolution in viruses	Include reasons for very rapid rates of evolution in some viruses. Use two examples of rapid evolution: evolution of influenza viruses and of HIV. Consider the consequences for treating diseases caused by rapidly evolving viruses.

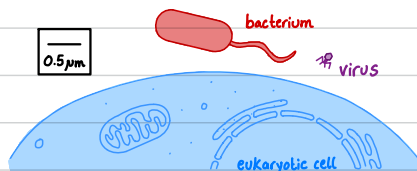
Virus non-cellular infectious agent/parasite which replicates only inside of a living host cell. Viruses infect all life forms (including bacteria, plants and animals), can be species-specific or may 'jump' from species to another

Viruses do not move or grow and rely on their hosts for metabolic processes and replication making them non-self sustaining and not considered alive ^{origin of cells A2.1} Viruses are obligate intracellular parasites

Viruses display enormous diversity in both form and function, but they all share common structural features:

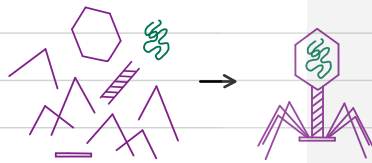
Small size

most viruses range from 20nm to 400nm, making them 100-1000x smaller than their hosts, allowing easier entry



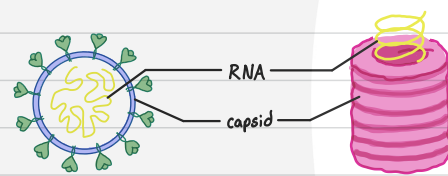
fixed size

viruses do not grow over time. They remain the same size and complexity after being assembled in their host



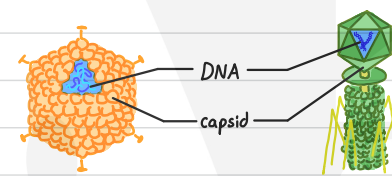
nucleic acid as genetic material

all viruses use DNA or RNA as genetic material using the same universal genetic code enabling translation to occur within host



capsid made of protein

genetic material is enclosed in a capsid (coat made of repeating protein subunits - capsomeres) before being released by host

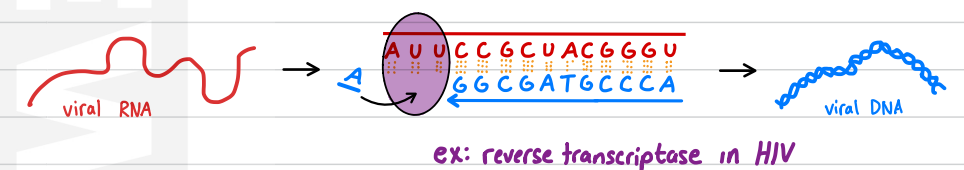


no cytoplasm

all viruses lack a cytoplasm as they do not carry out their own metabolism, rather relying on host's - contributing to their small size

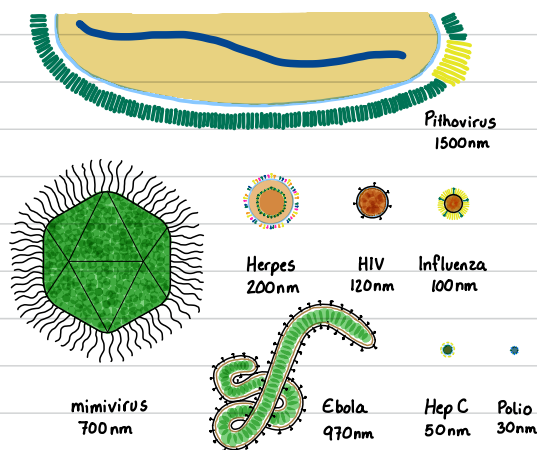
few or no enzymes

most enzymes lack their own enzymes, using those in their host instead. Some viruses have a few **enzymes** that are used to help in infection, lysis of host or replicating their genetic material



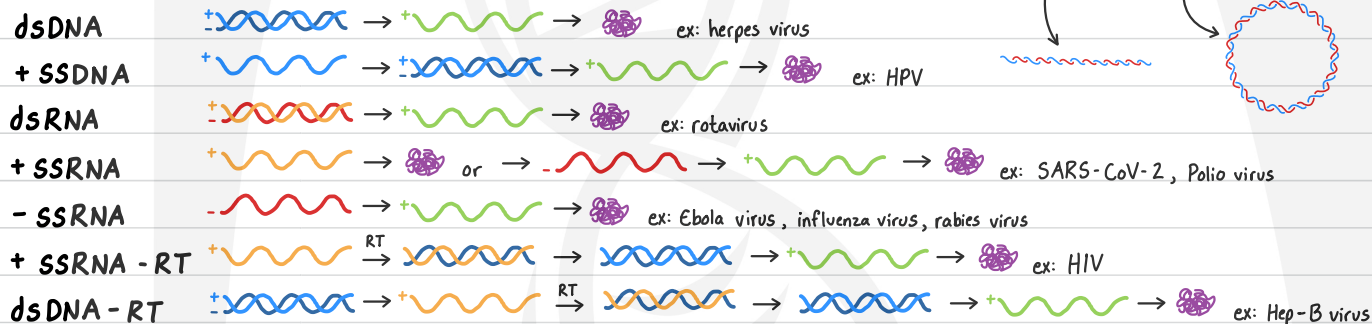
Viruses are highly diverse in shape and structure:

Vary in size



Vary in genetic material

Viral genetic material can be double-stranded (ds) or single-stranded (ss) which can be negative-sense (-) serving as a template to mRNA or positive-sense (+) and may be translated directly into proteins. Some also use reverse transcriptase (RT). ✗ genetic material can also be linear or circular

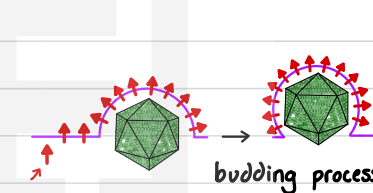


Vary in shape

viruses vary in their capsid structure and overall shape:



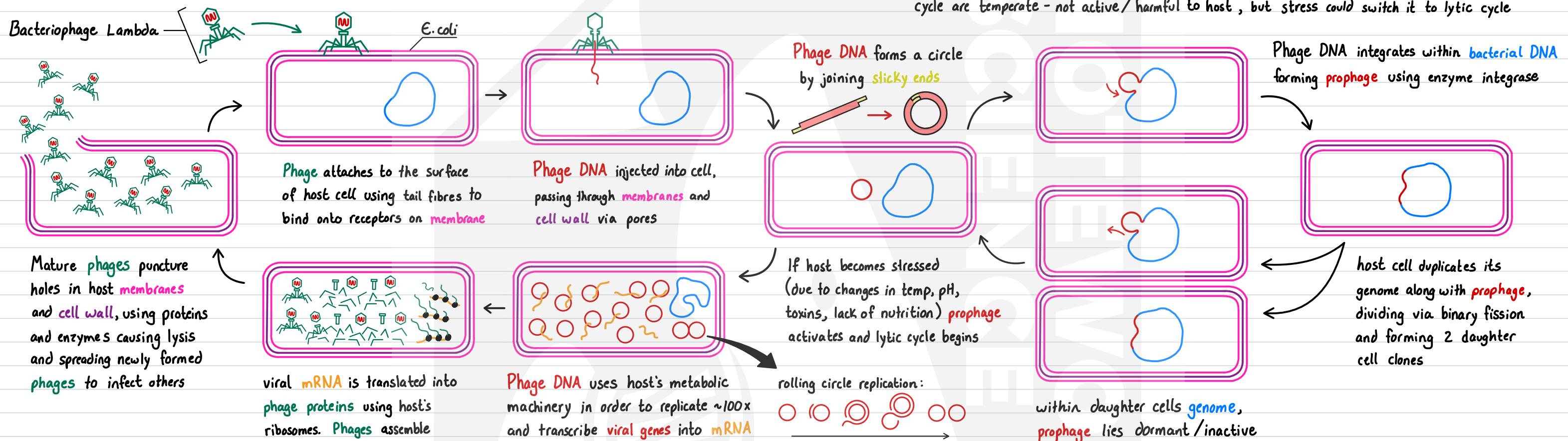
viruses may also be **enveloped** in their host's cell membrane during exocytosis (along with viral surface glycoproteins) allowing more protection and easier cell infection via binding



	SARS-CoV-2 (Severe Acute Respiratory Syndrome Corona Virus 2)	HIV (Human Immunodeficiency Virus)	Bacteriophage lambda (λ)
Genetic material	1 copy of linear + ssRNA genome of ~30000 base pairs	2 copies of linear + ssRNA genome of ~9700 base pairs	linear dsDNA genome of ~48000 base pairs with ss sticky ends
Structural features	50 nm	50 nm	50 nm
Envelopment	Enveloped	Enveloped	Non-Enveloped
Host	Mammalian (human) epithelium cells in respiratory system	human white blood cells (helper T, macrophages, dendritic cells)	E. coli (gram-negative bacterium)
Associated disease	COVID-19 (COroNaVirus Disease of 2019)	AIDS (Acquired ImmunoDeficiency Syndrome)	

Lytic cycle: virus reproduces within the host cell and the many new copies burst out, killing the host and spreading new viruses. Viruses in lytic cycle are virulent as they cause disease

Lysogenic cycle: virus integrates its genetic material into the host bacterium's genome. Each time host replicates its DNA it will also replicate viral DNA, thus passing it onto daughter cells. Viruses in lysogenic cycle are temperate - not active/harmful to host, but stress could switch it to lytic cycle



✗ viruses are extremely diverse in structure, complexity, and genome suggesting different potential origins:

Virus-first hypothesis: viruses evolved from proteins and nucleic acid (RNA), which gave rise to first cells

✗ RNA is enzymatic, allowing it to self-replicate. *least likely

✗ viruses require a cellular host to replicate ✗ viruses share more protein structures in common with cells than each other

Progressive hypothesis: viruses developed from genetic components (DNA, RNA) which 'escaped' from genes of a cell

✗ Retroviruses like HIV convert RNA to DNA using reverse transcriptase which then integrates within host genome.

Retrotransposons within eukaryotes also transcribe into RNA and then DNA and integrate into new location in genome

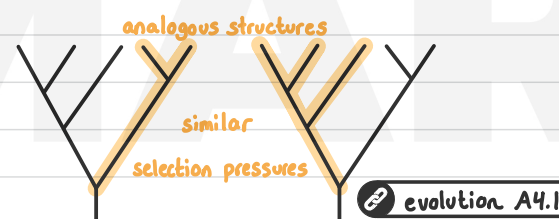
✗ viruses have unique, complex structures not present in cells such as capsids

Regressive hypothesis: viruses developed from parasitic cells in a progressive loss of cellular components, becoming obligate

✗ Viruses such as Mimivirus are very large with a complex structure and large genome, similar to parasitic bacteria

✗ even very small cellular parasites do not resemble viruses

✗ Since all viruses share an extreme form of obligate parasitism, their common structural features (analogous structures) may have arisen independently from **convergent evolution**: when species occupying a similar niche are subject to similar selection pressures and adapt similarly



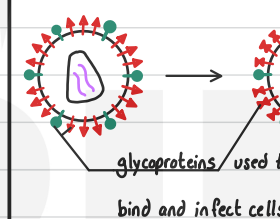
Viruses undergo very rapid rates of **evolution**: change in heritable characteristics of a population evolution A4.1

↳ viruses reproduce very quickly resulting in short generation times - allows more selection over shorter timespan

↳ genetic variation (new alleles) arises from mutations. RNA Viruses have high mutation rates as their enzymes do not proofread during replication, resulting in many errors and new base sequences allowing potential adaptation

↳ natural selection acts on variation in populations resulting in adaptive advantageous features being more successful and selected, driving evolution. As viruses are parasites, hosts actively seek to destroy them leading to a constant, strong selection pressure and rapid evolutionary change natural selection D4.1

Antigenic Drift: genetic variation from accumulation of mutations resulting in new strain

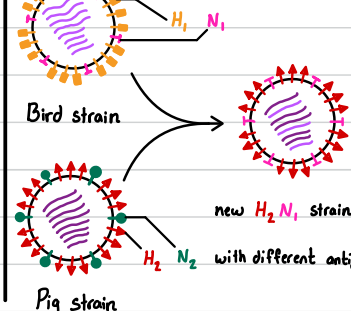


ex: HIV and influenza virus

① during viral RNA replication many errors are made, leading to mutations
② some mutations may lead to new glycoproteins and strains

defence against disease C3.2

Antigenic Shift: recombination of different viral strains resulting in abrupt change



ex: Influenza virus

① Avian and swine flu infect pig cells
② RNA from each mix, forming new strain
③ new strain infects humans

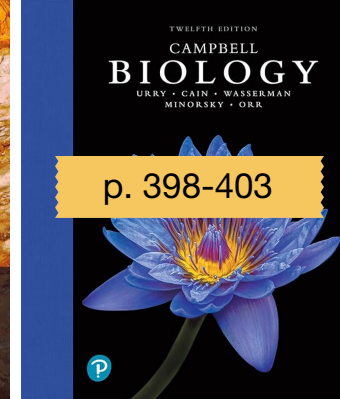
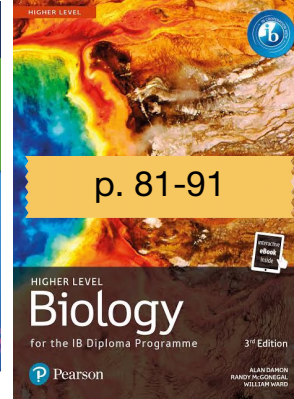
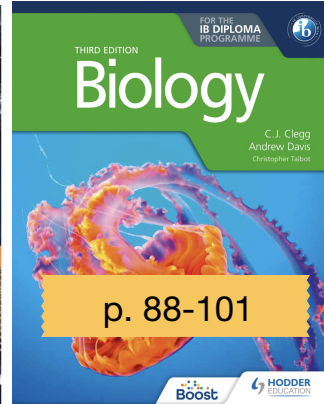
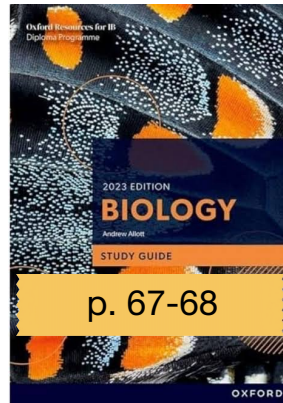
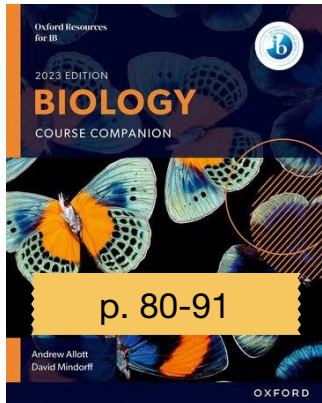
✗ As HIV and influenza's structure keep changing it poses a challenge for the immune system and treatments with drugs

Resource Links

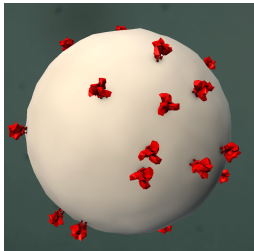
each resource is hyperlinked



Textbooks



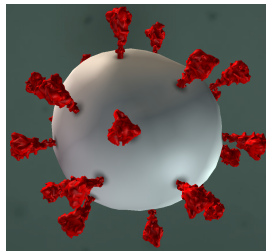
3D models



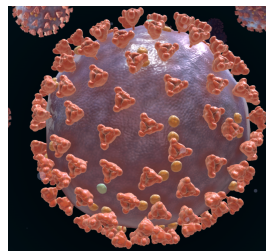
HIV



T7 phage

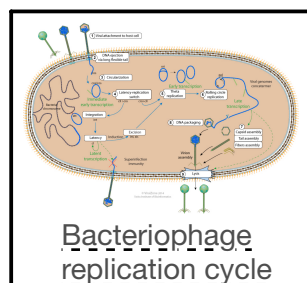
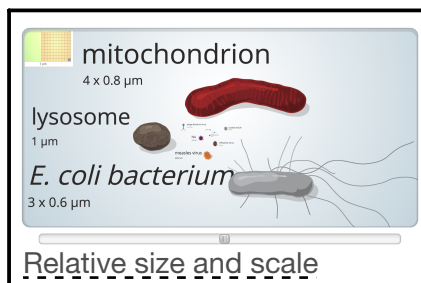


SARS-CoV-2



SARS-CoV-2

Simulators / Interactives



Articles

Carrat, F., & Flahault, A. (2007). Influenza vaccine: the challenge of antigenic drift. *Vaccine*, 25(39-40), 6852-6862. <https://doi.org/10.1016/j.vaccine.2007.07.027>

Koonin, E. V., Krupovic, M., & Agol, V. I. (2021). The Baltimore classification of viruses 50 years later: How does it stand in the light of virus evolution? *Microbiology and Molecular Biology Reviews*, 85(3). <https://doi.org/10.1128/mmbr.00053-21>

Li, F., Hou, C. D., Lokareddy, R. K., Yang, R., Forti, F., Briani, F., & Cingolani, G. (2023). High-resolution cryo-EM structure of the *Pseudomonas* bacteriophage E217. *Nature Communications*, 14(1). <https://doi.org/10.1038/s41467-023-39756-z>

Nasir, A., Kim, K. M., & Caetano-Anollés, G. (2012). Viral evolution. *Mobile Genetic Elements*, 2(5), 247-252. <https://doi.org/10.4161/mge.22797>

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Petrova, V. N., & Russell, C. A. (2017b). The evolution of seasonal influenza viruses. *Nature Reviews Microbiology*, 16(1), 47-60. <https://doi.org/10.1038/nrmicro.2017.118>