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4.5.1

A particular species can be identified using the features (the **phenotype**) which are characteristic to only that species. Species with similar phenotypes are likely to be related to each other. A **key** of characteristics is used to identify the species. (see p10 – 11)

Organisms are classified into families according to similarity of features. The families start off large, but rapidly become smaller. This is the basis of a **hierarchical** classification system.

**Kingdom**

**Phylum**

**Class**

**Order**

**Family**

**Genus**

**Species**

**Taxonomy**: the science of classifying living things.

**Biodiversity**: the variety of life on our planet, measurable as the variety within species, between species, and the variety of ecosystems.

If two organisms can interbreed to produce fertile offspring they are the same species. If not, they are different species.

**Binomial System**: *Felix catus*  
**Italics in print**  
(2 names)

**Genus**: tells you what group the species is from  
(has a capital letter)

**species**: tells you the exact species  
(small case letter)

**DISTINGUISHING CHARACTERISTICS OF THE KINGDOMS**

**PROKARYOTES**

- Microscopic prokaryotic cells (2 - 5μm long rather than 10-100μm)
- Lack of a nucleus (DNA in cytoplasm) and possibly plasmids
- Lack of membrane-bound organelles
- Presence of 70s ribosomes
- No cytoskeleton

**PROTOCTISTS**
- Eukaryotic cell structure
- Simple body form, either unicellular, filamentous (chains), colonial (ball) or macroscopic (large and visible)

The Proctotist’s kingdom tends to be full of organisms that do not fit into any other Kingdom e.g. algae and yeast

**FUNGI**
- Heterotrophic nutrition (get food from eating, unlike plants)
- Made of a network of **Hyphae**, which form a 3D structure called a **Mycelium**. (look up Module 1 notes)
- Call walls containing chitin

**PLANTS**
- Multicellular with eukaryotic structure
- Cell walls containing cellulose
- Complex body form
- Photoautotrophic nutrition (make food themselves through P/S)
- Presence of photosynthetic cells with chloroplasts
- 2 stages in the life cycle: a **diploid** spore-producing stage and a **haploid** gamete-producing stage.

**ANIMALS**
- Multicellular with eukaryotic cell structure
- Cells without cell walls
- Heterotrophic nutrition
• Highly organised organs and tissues including nervous co-ordination

• The only haploid cells they have are gametes

### 4.5.2 GENETIC DIVERSITY

Individuals in the same species look different (have different phenotypes). This is called variation.

Variation is caused by;

1. The genotype of the individual (i.e. which alleles they have).
2. The environment.

**Genetic diversity** describes the range of different genotypes within a species. If there are few genotypes the genetic diversity is small. If there are lots of genotypes the genetic diversity is large.

<table>
<thead>
<tr>
<th>Advantages of little genetic diversity</th>
<th>Advantages of wide genetic diversity</th>
</tr>
</thead>
<tbody>
<tr>
<td>• All individuals have a preferential phenotype</td>
<td>• Less chance of genetic disease</td>
</tr>
<tr>
<td></td>
<td>• Less chance of extinction when faced with disease (i.e. some individuals will have a phenotype that allows them to survive)</td>
</tr>
<tr>
<td></td>
<td>• Environment has less effect on phenotype</td>
</tr>
<tr>
<td></td>
<td>• Species more likely to survive environment change</td>
</tr>
<tr>
<td></td>
<td>• Species more likely to colonise</td>
</tr>
<tr>
<td></td>
<td>• Allows access to more niches, therefore less interspecific competition</td>
</tr>
</tbody>
</table>

**CAUSES OF GENETIC DIVERSITY:**

1. Independent Assortment
2. Mutation
3. Random fusion
4. Crossing Over

**Independent Assortment** = which allele of each pair goes into which gamete. This is caused by the orientation of homologous pairs of chromosomes during metaphase 2 of meiosis.
Changes in the sequence of bases in codons (mutation) cause genetic variation. This usually occurs by DNA being improperly copied or damaged. Chemicals (mutagens) and radiation can do this.

Each gamete is different. Therefore, by combining different gametes new variation occurs (random fusion).

During meiosis sections of DNA are swapped between homologous chromosomes (pairs of chromosomes). This creates more variation by creating new combinations of alleles (crossing over). (See fig. 5.18 on p18).

Figure 5.19 – crossing over can result in a great deal of genetic variation.
**4.5.3 DIHYBRID CROSS**

Dihybrid Crosses are for crosses involving two different genes (2 loci).

A = Purple stem,  a = Green Stem,  D = Big Leaves,  d = little leaves

<table>
<thead>
<tr>
<th>Male</th>
<th>Female</th>
</tr>
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<tbody>
<tr>
<td><strong>Parent’s Phenotype:</strong></td>
<td>Parent’s Phenotype:</td>
</tr>
<tr>
<td>Purple stem &amp;</td>
<td>Purple Stem &amp;</td>
</tr>
<tr>
<td>Big Leaves</td>
<td>Big Leaves</td>
</tr>
<tr>
<td><strong>Parent’s Genotype:</strong></td>
<td><strong>Parent’s Genotype:</strong></td>
</tr>
<tr>
<td>AaDd</td>
<td>AaDd</td>
</tr>
<tr>
<td><strong>Gametes:</strong></td>
<td><strong>Gametes:</strong></td>
</tr>
<tr>
<td>AD</td>
<td>AD</td>
</tr>
<tr>
<td>Ad</td>
<td>Ad</td>
</tr>
<tr>
<td>aD</td>
<td>aD</td>
</tr>
<tr>
<td>ad</td>
<td>ad</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>F1 Genotype:</strong></th>
<th>9 : 3 : 3 : 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>AD</td>
<td>A A D D</td>
</tr>
<tr>
<td>Ad</td>
<td>A A d d</td>
</tr>
<tr>
<td>aD</td>
<td>A a D D</td>
</tr>
<tr>
<td>ad</td>
<td>A a d d</td>
</tr>
</tbody>
</table>

F1 Phenotype:  
A_B_ : A_bb : aaB_ : aabb  
Purple & Big : Purple & Little : Green & Big : Green & Little
4.5.4 ECOLOGICAL SAMPLING TECHNIQUES

**Biotic Factor**: A living variable within the ecosystem, which affects the survival of organisms. Examples include predation, competition, and pollution from excreted waste.

**Abiotic Factor**: A non-living variable within the ecosystem, which affects the survival of organisms. Examples include temperature, light, and water.

**Random Sampling** (quadrats placed at randomly generated intervals)
- Used where habitat is uniform
- Removes observer bias
- Used in a large area
- Used if time is limited

**Systematic Sampling** (quadrats placed at regular intervals)
- Used to show zonation
- Used where there is continuous variation
- Used to sample linear habitats (e.g. a roadside)

2 types of systematic sampling technique;

**Line Transect**:
- Used where time is limited
- Used to visually illustrate how species change along a line

**Belt Transect**:
- Produces more data, gives detail about species abundance down the line as well as range
- Shows species dominance down the line

**What interval should be used?**

Transects can either be continuous with the whole length of the line being sampled, or samples can be taken at particular points along the line

For both line and belt transects, the interval at which samples are taken will depend on the individual habitat, as well as on the time and effort which can be allocated to the survey.

- Too great an interval may mean that many species actually present are not noted, as well as obscuring zonation patterns for lack of observations.
Too small an interval can make the sampling time consuming, as well as yielding more data than is needed.

4.5.7 LIGHT DEPENDANT STEP OF PHOTOSYNTHEIS

Figure 5.33 – in the light dependant reactions of photosynthesis, the energy from the sun excites chlorophyll molecules. High energy electrons leave the chlorophyll and pass along a series of carrier proteins in the electron transport chain. The ionised chlorophyll causes the photolysis of water; hydrogen is pulled off water molecules raising the local hydrogen ion concentration within the thylakoid. ATP is also formed as a phosphate group is added to the ADP.
LIGHT DEPENDENT STEP:

1. Chlorophyll absorbs light (remember chlorophyll is the trap in the bottom of the photosystem)
2. Chlorophyll emits electrons
3. Electrons are received by electron carrier proteins in the thylakoid membrane (electron transport chain)
4. Electron transport chain uses high energy electrons to power the following conversions; \[ \text{ADP} + \text{Pi} \rightarrow \text{ATP} \text{ and } \text{NADP} + \text{H}^+ + \text{e}^- \rightarrow \text{NADPH} \]
5. Water is split (photolysis) to produce replacement electrons for the photosystems, \( \text{H}^+ \) for the reduction of NADP and \( \text{O}_2 \) which is excreted.

The purpose of the light dependent step is to produce ATP and NADPH. ATP provides the energy for converting \( \text{CO}_2 \) into glucose and NADPH provides the \( \text{H}^+ \) for glucose.

4.5.8 LIGHT INDEPENDENT STEP OF PHOTOSYNTHESIS

There are three steps in the Calvin Cycle;

1. **Carboxylation**: RuBP **fixes** \( \text{CO}_2 \) to form GP. This reaction is catalysed by the enzyme **Rubisco**
2. **Reduction**: In a series of reactions GP reacts with ATP and NADPH reduced GP to form GALP (by reducing GP the NADPH itself is oxidised, reverting to NADP)
3. **Regeneration**: Some GALP is converted back into RuBP so the Calvin Cycle can continue. The rest of GALP is converted into glucose in a series of reactions.

A glucose molecule is generated every 6 turns of the Calvin Cycle.
4.5.9 ANATOMY OF THE PLANT CELL PROTOPLAST

Thylakoid membrane = location of photosystems & electron transport chain

Stroma = site of Calvin Cycle & photolysis of water

Grana provide large surface area for absorption of light

4.5.10 GROSS PRIMARY PRODUCTIVITY, NET PRIMARY PRODUCTIVITY AND PLANT RESPIRATION

NPP = GPP – R

NPP = Net Primary Productivity (amount of stored chemical energy the plant has to use for growth. This is directly proportional to biomass)
GPP = Gross Primary Productivity (amount of stored chemical energy the plant earns through photosynthesis)

R = Respiration (amount of energy lost through respiration, i.e. heat, lost as CO₂ etc)

Best analogy is a salary. GPP is the amount of stored chemical energy the plant earns through photosynthesis. R is like income tax. The plant has to pay “respiration tax” because it can’t photosynthesis at night & not all parts of the plant are capable of photosynthesis.

NPP = disposable income: what the plant has to spend after paying tax.

4.5.11 THE EFFICIENCY OF ENERGY TRANSFERS BETWEEN TROPHIC LEVELS

Energy is lost between trophic levels. Energy is lost in the following ways; in respiration (mostly lost through heat), energy still present in egested food, through movement, through digestion, energy still present in excreted materials etc.

Of the 100% sunlight energy that reaches plants, ~5% is converted into NPP. Energy is lost in the following ways; reflected light, light of wavelengths not useful to plants, passes through leaves, lost in respiration, lost as heat etc.

4.5.12 HOW NATURAL SELECTION CAN LEAD TO EVOLUTION

Evolution: the idea that one species changes into another over time

Natural Selection: Darwin’s suggestion for the process by which evolution might occur

Evolution by Natural Selection (Darwinian Evolution)

1. There is variation in a species
2. More individuals are born than the environment can sustain, so some individuals must die.
3. The individuals that survive tend to be those that have alleles which give them a **selective advantage** in their environment (i.e. they are the best adapted to their environment, e.g. camouflaged). These are the “fittest”

4. The fittest survive long enough to reproduce and pass their alleles onto the next generation.

5. Over a few generations the frequency of “fit” alleles increases and the frequency of “unfit” alleles decreases.

6. Soon all / most individuals have the “fit” phenotype and the “unfit” phenotype is eradicated.

7. This process continues over **many generations**

8. Over this time new mutations occur, which give new even better alleles.

9. Over time the mutations accumulate in the phenotype until the organism is unable to reproduce (i.e. produce fertile offspring) with the original organisms. At this point a new species has been produced (**speciation**)

This process is speeded up by isolation (see 4.5.14) because this stops the influx of alleles from outside and allows new mutations to accumulate in the genotype more quickly.

### 4.5.13 The Historical Development of the Theory of Evolution

**1798 Malthus** publishes paper on population growth. Malthus noticed that the human population was expanding exponentially. He thought that the human population would outgrow its resources and that this would lead to famine and war.

**Darwin** was influenced by this idea, because he noticed that animal populations grow exponentially and then plateau when they reach the limits the environment can sustain (i.e. the population size is determined by the environment).

**1809 Lamarck** publishes a mechanism for evolution based on two laws:

- **Law 1**: Organs / structures grow if they are used. This means that the environment determines the phenotype of an organism.

- **Law 2**: Changes are passed on to the next generation.

So a blacksmith, who uses his muscles all day, will grow bigger muscles. This works! But, will the bigger muscles be passed onto his children? No, so Lamarck’s theory is easy to falsify.

**1859 Darwin** publishes the Origin of species by means of Natural Selection. He publishes with **Wallace** who wrote to Darwin to discuss his own ideas about evolution. They were very similar to Darwin’s and this prompted Darwin to publish.
4.5.14 HOW REPRODUCTIVE ISOLATION CAN LEAD TO SPECIATION

Isolation is important for evolution because it decreases the size of the gene pool. This stops new alleles coming in from breeding with original alleles and speeds the accumulation of new mutations (which is what leads to speciation). The different types of isolation:

<table>
<thead>
<tr>
<th>Method of isolation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ecological isolation</td>
<td>The species occupy different parts of the habitat</td>
</tr>
<tr>
<td>Temporal isolation</td>
<td>The species exist in the same area, but reproduce at different times</td>
</tr>
<tr>
<td>Behavioural isolation</td>
<td>The species exist in the same area, but do not respond to each other’s courtship behaviour</td>
</tr>
<tr>
<td>Physical incompatibility</td>
<td>Species coexist, but there are physical reasons which stop them from copulating</td>
</tr>
<tr>
<td>Hybrid inviability</td>
<td>In some species, hybrids are produces but they do not survive long enough to breed</td>
</tr>
<tr>
<td>Hybrid sterility</td>
<td>Hybrids survive to reproductive age, but cannot reproduce</td>
</tr>
</tbody>
</table>

4.5.15 WHY IS THE THEORY OF EVOLUTION SO CONTREVERTIAL?

Evolution is a theory, not a fact. Many people believe that species were created (creationism). Other people believe in evolution, but by mechanisms other than Natural Selection. You should respect the opinions of other people, even if you do not necessarily agree with them.

4.5.16 THE CONCEPT OF SUCCESSION TO A CLIMAX COMMUNITY

Primary succession is the first stage of the ecological succession of plant life from abiotic land with no soil to fully support plant ecosystems (e.g., a forest). In primary succession, pioneer plants like mosses and lichen, start to "normalize" the habitat, creating rudimentary soil from their dead matter. These pioneer plants create conditions for the start of plant growth and so more complex plants like grasses and shrubs begin to colonise the area.
Over time the grass area is colonised by small woody plants, which give way to small trees and finally, after a few hundred years, large trees take over. The large trees represent the **climax community** because succession stops at this point.

A good example of primary succession takes place after a **volcano** has erupted. The barren land is first colonised by simple pioneer plants which pave the way for more complex plants, such as hardwood trees by creating soils and other necessities. Unlike **secondary succession**, which refers to succession after an environmental disaster (such as a forest fire) primary succession occurs on the geologic timescale, over thousands of years.

### 4.5.17 THE EXTENT TO WHICH ZOOS CAN PLAY A ROLE IN THE CONSERVATION OF ENDANGERED SPECIES

Zoos can play a large role in conserving endangered species by:

1. Conducting research
2. Running captive breeding programmes
3. Reintroducing species into the wild
4. Educating people

**Research** enables scientists to understand the role of a species in an ecosystem. By understanding the niche, food web, reproductive behaviour, habitat, feeding relationships etc scientists can suggest effective methods of conserving species.

**Captive breeding programmes** are used to reintroduce species to the wild, build up population numbers and maintain genetic diversity. In a small population many alleles are lost between generations because an individual only passes on 50% of their alleles. E.g.

\[ R = \text{Red}, \quad r = \text{white} \]

<table>
<thead>
<tr>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parent’s Phenotype: Red</td>
<td>Red</td>
</tr>
<tr>
<td>Parent’s Genotype: Rr</td>
<td>Rr</td>
</tr>
<tr>
<td>Gametes: [R, r]</td>
<td>[R, r]</td>
</tr>
</tbody>
</table>

F1 Phenotype: \[9 : 3 : 3 : 1\]

\[A_B_ : A_bb : aaB_ : aabb\]

Purple & Big : Purple & Little : Green & Big : Green & Little
If the parents only have 2 children and they are both Red (RR) then the r allele has been lost. This is **genetic drift** and is a big cause of the loss of genetic diversity in an endangered species.

To avoid this **studbooks** are kept (basically, a family tree for the captive animals) so that only non-related animals are bred with each other. This decreases the change of genetic drift and also decreases the change of **genetic disease**.

Wild animals are often introduced to captive breeding programmes to avoid these problems.

**Reintroducing species into the wild** has some success, but depends greatly on the species. As a general rule of thumb, the more advanced the species the more difficult reintroduction is. This is because animals need to learn specific behaviours e.g. how to hunt, how to reproduce, how / where to find shelter, group behaviours. Breeding animals in captive environments that mimic the wild has more success because it allows some of these behaviours to be learned in captivity. Feeding the animals in the wild also helps survival rates.

**Educating people** is essential to conservation. Often just doing something slightly differently will have a big impact on conserving a species e.g. building roads with tunnels under them for badgers.

---

**4.5.18 DISCUSS WAYS IN WHICH CONFLICTS BETWEEN WILDLIFE AND HUMANS CAN BE RECONCILED AND THE SOCIAL AND CULTURAL ISSUES INVOLVED**

There are no set facts to learn.

**4.5.19 DISCUSS HOW CULTURAL ISSUES ARE REFLECTED IN THE LEGISLATION WHICH DRIVES UK AND INTERNATIONAL INITIATIVES THAT USE BIOLOGICAL PRINCIPLES TO MANAGE CONSERVATION AND DEVELOPMENT SUSTAINABLY.**

There are no set facts to learn.
4.6.1 HOW FORENSIC PATHOLOGISTS DETERMINE THE TIME OF DEATH

Time of death can be measured using the following factors:

- Body temperature
- Extent of rigor mortis
- Level of decomposition
- Forensic entomology

BODY TEMPERATURE:

A body cools following an S-shaped (sigmoid) curve. The initial plateau at 37˚C lasts 30 – 60 min, then the body cools quickly to ambient temperature.

After 24hrs a body has usually finished cooling and temperature is no longer useful.

Temperature is measured using a long thermometer with a wide range. Temperature is usually taken rectally or using an abdominal stab.

The rate of cooling depends on the situation the body is found in e.g.

- Clothing – slows cooling
- Found in water – speeds cooling
- Found indoors – slows cooling
- Air movements – speed cooling

EXTENT OF RIGOR MORTIS:

<table>
<thead>
<tr>
<th>Temperature of body</th>
<th>Stiffness of body</th>
<th>Approx time since death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warm</td>
<td>Not stiff</td>
<td>No more than 3 hrs</td>
</tr>
<tr>
<td>Warm</td>
<td>Stiff</td>
<td>3 – 8hrs</td>
</tr>
<tr>
<td>Cold</td>
<td>Stiff</td>
<td>8 – 36hrs</td>
</tr>
</tbody>
</table>
Rigor mortis is the stiffening of joints and muscles. Small muscles stiffen first and unstiffen last.

Muscles stiffen because they run out of ATP, causing the actin and myosin muscle fibres to stick permanently to each other. Muscles unstiffen because the muscle fibres begin to break down.

On page 80 of your textbook is a little more detail about the sequence of events that causes muscles to run out of ATP.

**LEVEL OF DECOMPOSITION:**

**Autolysis** is the break down of body tissues using the body’s own enzymes from the digestive system and from lysosomes.

After this, bacteria from the gut invade tissues and release more enzymes. This tends to happen in anaerobic conditions, which favours the growth of anaerobic bacteria.

Greenish discolouration of abdomen (36hrs)

↓

Spreads across rest of body (36 – 72hrs)

↓

Discolouration darkens to reddish green (36 – 72hrs)

↓

Discolouration darkens to purple-black (72hrs)

↓

Body becomes bloated with gas (one week)

↓

Gas is released, body deflates & shrinks (one week +)

Autolysis is increased by mild heat and slowed by intense heat. Humidity has a big involvement as well – dry conditions slow autolysis and, in some cases (e.g. mummies) stop it completely.

The presence of wounds, the clothing the person was wearing and the combination of gases released during decomposition also have an effect.
The insects found in a dead body can help identify time of death in 3 ways;

1. If the temperature of the body has remained relatively constant the age of the maggots growing in it can be determined by their starting length and the temperature of the part of the body they grew in.

   e.g. a maggot 3mm long found growing at 28°C will be roughly 0.3 days (8 hrs old)

2. Using the life-cycle of the maggot to identify age

3. If maggots are taken from the body, allowed to grow and the time taken to pupate is recorded; it is sometimes possible to work backwards from the pupation date and work out how old the maggots must have been when they were taken from the body. This works because maggots of different species usually take a fixed number of days to pupate.

**4.6.2 HOW FORENSIC PATHOLOGISTS DETERMINE THE IDENTITY OF A DEAD PERSON**

The identity of a dead person can be ascertained by;

1. Identity papers – obvious.
2. Fingerprints
3. Dental records
4. Genetic Fingerprint

**Fingerprints:** The skin on fingers, toes etc is ridged into specific patterns (arches, tented arches, whorls & loops). Sweat and sebum oil is left behind from our fingers on the things we touch. Using aluminium powder or protein stain (e.g. ninhydrin) fingerprints are revealed. Fingerprints are unique and can be used to identify people.
**Dental Records**: Can be used to identify age and to identify a person based on their dentist’s record of their teeth. This is usually used when the body is damaged (e.g. a corpse from a fire)

![Dental Records Example](image)

**Figure 6.7** – On the record CR is a crown and BR is a bridge.

**Genetic Fingerprint**: Used because DNA is unique to individuals (except identical twins and clones grown by mad scientists). Genetic fingerprinting looks for the presence of repeated sequences of bases in the non-coding sections of DNA (introns). The repeated sequences are called **satellites** and can be 2 – 4 bases long (**Micro-satellite**) or 5 – 20 bases long (**Mini-satellite**). The satellites are repeated anything from 5 – 500 times and this produces a unique DNA signature.

Fingerprinting process:

1. A sample of DNA is copied using PCR
2. Sample is cut using a restriction enzyme
3. Sample is run on an electrophoresis gel, often using a DNA sample of known length to act as a standardization.
4. A southern blot is taken
5. DNA is labeled using a DNA probe specific to the satellite
6. An X-ray is taken to reveal the location of the bands of DNA

The fingerprint is the pattern of bands on the electrophoresis gel. Assuming the original DNA sample has not been contaminated (by e.g. a hair from the pathologist) the fingerprint will be exact. **See Fig. 6.10.**

### 4.6.3 Interpret Data on the Typical Stages of Succession on Corpses

The idea that as each organism or group of organisms feeds on a body, it changes the body. This change in turn makes the body attractive to another group of organisms, which changes the body for the next group, and so on until the body has been reduced to a skeleton. This is a predictable process, with different groups of organisms occupying the decomposing body at different times. This technique allows you to tell, by the age and specific species living on a corpse, how old the corpse is.

Succession and forensic entomology also show if the body has been moved.
4.6.4 STRUCTURE OF BACTERIA AND VIRUSES

A TYPICAL PROKARYOTE

**Ribosomes.** Same function as eukaryotic cells (protein synthesis), but are smaller (70s rather than 80s).

**Nuclear Zone.** The region of the cytoplasm that contains DNA. There is no nuclear membrane.

**DNA.** Always circular, and not in chromosome form.

**Plasmid.** Very small circles of DNA, containing non-essential genes. Can be exchanged between different bacterial cells.

**Cell membrane.** Made of phospholipids and proteins, like eukaryotic membranes.

**Mesosome.** Tightly-folded region of the cell membrane containing all the proteins required for respiration and photosynthesis.
**Cell Wall.** DIFFERENT from plant cell wall. Made of murein (a protein). There are two kinds of cell wall, which can be distinguished by a Gram stain:

A: **Gram positive** bacteria have a thick cell wall and stain purple

B: **Gram negative** bacteria have a thin cell wall with an outer lipid layer and stain pink.

**Capsule** (or **Slime Layer**). Thick polysaccharide layer outside of the cell wall. Used for:

1. Sticking cells together
2. As a food reserve
3. As protection against desiccation (drying out) and chemicals, and as protection against phagocytosis (being broken down by a white blood cell).

**Flagellum.** A rotating tail used for propulsion.

<table>
<thead>
<tr>
<th>Prokaryotic Cells</th>
<th>Eukaryotic cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small cells (&lt; 5 mm)</td>
<td>Larger cells (&gt; 10 mm)</td>
</tr>
<tr>
<td>Always unicellular</td>
<td>Often multicellular</td>
</tr>
<tr>
<td>No nucleus or any membrane-bound organelles</td>
<td>Always have nucleus and other membrane-bound organelles</td>
</tr>
<tr>
<td>DNA is circular, without proteins</td>
<td>DNA is linear and associated with proteins to form chromatin</td>
</tr>
<tr>
<td>Ribosomes are small (70S)</td>
<td>Ribosomes are large (80S)</td>
</tr>
<tr>
<td>No cytoskeleton</td>
<td>Always has a cytoskeleton</td>
</tr>
<tr>
<td>Cell division is by binary fission</td>
<td>Cell division is by mitosis or meiosis</td>
</tr>
<tr>
<td>Reproduction is always asexual</td>
<td>Reproduction is asexual or sexual</td>
</tr>
</tbody>
</table>

**A TYPICAL VIRUS**

Viruses have a wide range of different structures. Some viruses are about 100nm in diameter, whilst others can range from 20 – 3000nm.
All viruses have a protein coat (the capsid), which contains genetic material. The genetic material is either DNA or RNA, and can be single or double-stranded.

**Figure 6.24. A – The basic structure of a virus. B – Some examples of different viruses.**

The virus genetic material (the viral genome) contains only a few genes, from about 20 in the polio virus to more than 200 in the herpes virus (human genome contains ~80,000 genes). The viral genome codes for the proteins required to manufacture the virus.

The protein capsid is made from identical subunits (called capsomeres). The capsomeres can be arranged into an icosahedral shape (e.g. polio & herpes), or a cylindrical shape (e.g. TMV & rabies) or a loose containment structure (e.g. measles & influenza).

In addition, some viruses also have an outer membrane envelope, which allows the virus to penetrate the host cell membrane by endocytosis. Influenza, HIV and measles virus all have membrane envelopes.
VIRAL DAMAGE – WHAT DO VIRUSES ACTUALLY DO TO US?

Like bacteria, viruses have protein ligands on their capsid that attach to ligand receptors on eukaryotic cells. After a virus ligand attaches to a host cell ligand receptor it becomes anchored to the host cell. The virus attempts to get its viral genome into the host cell, usually through endocytosis using its lipid membrane. Viruses without lipid membranes may have specialised proteins designed to help inject the viral genome into the cell cytoplasm.

(i) Virus RNA enters host cell
(ii) Virus may also inject RNA Polymerase into host cell as well.
(iii) Viral RNA and RNA Polymerase enter host cell nucleus via nuclear pores
(iv) Viral RNA is copied in nucleus
(v) Viral RNA is transcribed using viral RNA Polymerase
(vi) Viral mRNA is translated in the cytoplasm
(vii) New Virus proteins formed
(viii) Viral proteins associate with copied RNA forming new complete viruses
(ix) New viruses leave host cell to infect other cells

Viruses that have a DNA code instead of an RNA code often insert their viral DNA into the host cell’s DNA. Other RNA viruses inject the enzyme Reverse Transcriptase, which makes a cDNA copy of the viral RNA. The cDNA copy is then inserted into the host cell’s DNA. Other viruses (e.g. HIV) also inject the enzyme integrase, which helps insert the viral cDNA into the host’s DNA.

Be sure you can recall what the 3 viral enzymes do;

- DNA Polymerase:
- RNA Transcriptase:
- Integrase:

Some viruses target specific tissues (e.g. Poliomyelitis virus targets motor neurones, HIV targets helper T cells, Influenza targets epithelial cells & rabies virus targets specific brain cells). If lots of new virus is being made, these host cell may lyse (burst) and die.

4.6.5 & 4.6.6 THE COURSES OF BACTERIAL AND VIRAL INFECTIONS

COURSE OF INFECTION FOR TUBERCULOSIS:

Tuberculosis (TB) is caused by the Mycobacterium tuberculosis bacterium.

1. Mycobacterium tuberculosis is inhaled into the lungs in droplets of water & mucus from another person’s lung (droplet infection)
2. TB begins to reproduce in the lungs.

3. The bacteria produce toxins, which damage lung tissue & cause coughing, increasing the transmission of the disease.

4. The body launches an immune response to the TB bacterium.

5. Histamine release and inflammation occur (see 4.6.7)

6. **Macrophages** enter the lungs in large numbers.

7. The macrophages engulf the TB bacteria in large groups, forming a mass of tissue called a **granuloma**. The inside of the granuloma is starved of oxygen, which kills the bacteria.

8. Once the bacteria are dead, the lung heals

**BUT**

9. TB bacteria can survive inside macrophages as the cell wall of the bacterium is very thick and waxy and is resistant to the macrophage enzymes.

10. The bacterium can survive and reproduce inside the macrophage for many years without causing infection. When the immune system is weakened (by stress, malnutrition, or another disease – HIV is a common cause) the TB bacterium breaks out and re-infects the body.

11. The bacteria reproduce too rapidly for the body to destroy

12. The lungs are progressively damaged, which eventually leads to death.

13. TB can also spread to the lymph nodes in the body, where it reproduces causing the disease **scrofula**

**COURSE OF INFECTION FOR HIV**

**HIV** is the Human immunodeficiency Virus, which eventually leads to Acquired Immunodeficiency Syndrome (**AIDS**).

HIV is spread by **direct contact** i.e. through sexual intercourse, blood-to-blood transfer (tattoos, needle sharing, piercing & cut-to-cut transfer).

Once inside the bloodstream an HIV infection occurs in 3 distinct phases;

1. **The acute phase.** HIV virus has a ligand (GP120), which attaches to a receptor (CD4) on the membrane of a type of white blood cell called a **Helper T cell**. HIV rapidly infects Helper T cells and the virus population increases quickly. At the same time the population of Helper T cells falls rapidly. The acute phase ends when the Killer T cells begin to recognise infected Helper T cells and kill them, which slows the replication of the virus.
2. **The chronic phase.** This can last for many years. The virus continues to replicate, but the Killer T cells keep the numbers in check. However, because the immune system is weakened other bacteria and viruses are more likely to infect the person (TB may reactivate at this point)

3. **The disease phase.** As the numbers of virus increase and the numbers of Helper T cell fall the immune system becomes weaker and weaker. Eventually a second pathogen will infect the person (an **opportunistic infection**) which cannot be fought off. The person will die quickly from the secondary infection and this is the AIDS disease state.

The huge problem with HIV is that it mutates very quickly. Once inside the body the viral antigens change and the (already damaged ) immune system can’t keep pace with the changes. Another problem is that HIV attacks Helper T cells, which are crucial for activating the B cells and also play a role in activating Killer T cells. With low numbers of Helper T cell, the immune system cannot communicate effectively and this increases the ability of HIV to survive in the body.

### 4.6.7 NON-SPECIFIC IMMUNE RESPONSES

**Inflammation:** damaged white blood cells and mast cells release **histamine** at the site of infection. Histamine causes local arterioles to vasodilate, increasing the blood supply to the area. It also causes holes to open between endothelial cells in capillary walls. This causes local oedema (the swelling associated with inflammation). It allows monocytes and neutrophils into the infected area, which engulf and destroy foreign bodies and pathogens. Eventually phagocytes arrive and complete the job. Dead monocytes and pathogen form pus.

**Lysozyme:** an enzyme that breaks down bacterial cell walls, causing them to lyse and die. Lysozyme is made in lysosomes inside phagocytes and is responsible for digesting engulfed bacteria. Lysozyme is also made by the skin, epithelial cells, and is present in tears.

**Interferon:** a protein made by virus-infected cells. It blocks RNA synthesis and therefore stops virus replication.

**Phagocytosis:** the process in which a pathogen is engulfed and destroyed. Macrophages engulf pathogens using pseudopodia ("fake feet"). The bacterium is taken into the macrophage by endocytosis and enters the macrophage inside a vacuole. Lysosomes containing lysozyme fuse with the vacuole and digest the bacterium inside.

### 4.6.8 THE ROLES OF ANTIGENS AND ANTIBODIES IN THE BODY’S IMMUNE RESPONSE

**Pathogens** have proteins on their surface that our immune system has learned to recognise as foreign. These proteins are called **antigens**. T cells, B cells & Macrophages all have the ability to recognise an antigen and once this has happened, they will trigger an immune response.
In addition to this, macrophages have the ability to present foreign antigens to T and B cells. Once a pathogen has been engulfed and destroyed MHC proteins inside the Macrophage stick to the pathogenic antigen. They are then incorporated into the cell membrane of the Macrophage, so it can present the foreign antigen and activate the T and B cells responses.

Antibodies (also called Immunoglobulins) are proteins produced by B cells. They are found in blood plasma, lymph, tissue fluid, tears, mucus and milk.

![Antibody Diagram](image)

**Figure 6.35 – A simplified diagram of an antibody. The antigen with a complementary shape can bind to the antibody’s antigen binding site.**

Each B cell produces a different immunoglobulin molecule which recognises and binds to a specific antigen. There are over a million different B cells in your body, therefore you have the ability to recognise and react to a million different antigens.

The **variable region** of the immunoglobulin protein is what recognises & binds to the antigen. Each variable region is different, hence the name.

There are 5 different families of immunoglobulin molecule in the human body (G, M, A, D & E. IgG - also known as γ-globulin). The families can be distinguished from each other by slight differences in the **constant region** of the protein.

Each antibody molecule contains two pairs of proteins;

- Two heavy chains
- Two light chains

Each pair of chains is held together by disulphide bridges (hydrogen bonds would be too weak).
Each immunoglobulin molecule has 2 antigen binding sites and can, therefore, bind 2 antigens at one time. This means that a single antibody molecule can bind to 2 pathogens at the same time, which causes pathogens to clump together and form the Antibody-Antigen Complex.

The formation of the Antibody-Antigen Complex is important because it;
- Isolates pathogens so they cannot infect other host cells
- Makes it easier for macrophages to engulf & destroy the pathogens.
- Stops the pathogen from entering a host cell
- Makes it easier for T cell activation as more antigens are presented in one area

4.6.9 THE ROLES OF B CELLS AND T CELLS IN THE BODY’S IMMUNE RESPONSE

There are two different types of Immune Response;

A  Cell-mediated Immune Response
B  Antibody-mediated Immune Response.

NB: ISOLATED VIRUSES DO NOT PRESENT ANTIGENS AND THEREFORE DO NOT TRIGGER EITHER THE CELL- OR ANTIBODY-MEDIATED IMMUNE RESPONSE. HOWEVER, WHEN VIRUSES INVADE HOST CELLS, VIRAL PROTEINS ARE EXPRESSED WHICH BECOME INCORPORATED INTO THE HOST CELL SURFACE MEMBRANE VIA MHC. THESE PROTEINS ARE RECOGNISED AS ANTIGENS.

CELL-MEDIATED IMMUNE RESPONSE:

1. Competent T Cells recognise a specific foreign antigen using its T cell receptor.
2. Activated T Cell undergoes rapid mitosis forming a large number of identical clone T

3. Cloned T Cells differentiate into Killer, Helper, Memory or Suppressor T Cells.

4. Killer and Helper Cells migrate to the site of infection

**Killer T Cells**: attach to the infected / foreign cell and release the enzyme Perforin, which makes holes in the pathogen’s cell membrane causing it to die

**Helper T Cells**: stimulate B cells to start producing antibody and attract macrophages to the site of infection

**Memory T Cells**: remain in the lymph nodes. They will respond rapidly if the same pathogen invades the body again, because they have the right T cell receptor to recognise the pathogen. This means that the body can mount an immune response before infection becomes serious

**Suppressor T Cells**: stop the immune reaction after about a week

**ANTIBODY-MEDIATED IMMUNE RESPONSE**:

1. B cells are recognise a specific foreign antigen using the antibody molecules on their surface. B cells can also be activated by macrophages & Helper T cells. When a macrophage digests a pathogenic cell antigens from the cell membrane get stuck in the macrophage’s membrane; any B Cells which come into contact with the antigen will then be activated

2. The activated B cell undergoes rapid mitosis and lots of clone B cells are produced

3. Cloned B Cells differentiate into either Plasma or Memory cells

**PLASMA CELLS**

a) Plasma cells antibody, which is specific for one antigen only

b) Antibody is transported via the lymph to the site of infection

c) Antibody attaches to the specific antigen

d) An antigen-antibody complex is formed

**MEMORY CELLS**

Memory Cells continue to secrete antibody for many years, so that if the body is infected by the same pathogen the Memory B cells can produce an instant supply of antibody before the infection becomes serious
4.6.10 THE ROLE OF NEGATIVE FEEDBACK IN MAINTAINING SYSTEMS WITHIN NARROW LIMITS

Negative feedback systems aim to keep something (e.g. blood glucose or body temperature) at a constant level.

Negative feedback works as follows;

1. Signal causes action
2. Action has effect
3. Effect removes original

E.g.

1. High glucose in blood causes insulin release
2. insulin stimulates liver to take up glucose & convert it into glycogen stores
3. glucose falls

4.6.11 HOW AN INFECTIOUS DISEASE CAN INTERFERE WITH THE BODY’S NEGATIVE FEEDBACK MECHANISMS FOR THERMOREGULATION

Homeostasis is the maintenance of the body’s internal environment. This is carefully controlled by a series of systems, which aim to keep conditions at a stable controlled level.

BODY TEMPERATURE:

Body temperature is carefully regulated to maintain a steady 37.5°C, which is the optimum temperature for human enzymes. Sensors (thermoreceptors) in the hypothalamus continually monitor blood temperature and activate warming / cooling processes to keep the temperature as stable as possible.
Tuberculosis bacterium (*Mycobacterium tuberculosis*) causes fever.

**HOW DOES FEVER WORK?**

All white blood cells communicate with each other and the rest of the immune system using a class of hormones called **cytokines**. The cytokines have hundreds of different roles and many more are yet to be discovered. One class of cytokine is the hormone **interleukin**, which causes fever.

Fever can be induced by many factors. The general class of hormones that lead to fever are called **pyrogens** (interleukin is a natural pyrogen). However, bacterial toxins, viral proteins and substances produced by necrotic tissue may also trigger fever.

Pyrogens travel in the blood to the **hypothalamus** in the brain. They bind to receptors there and trigger a complex set of reactions that lead to the production of PGE2 hormone, which
elevates the **thermoregulatory set point**, i.e. it re-sets the body’s natural thermostat to a higher temperature.

The hypothalamus now thinks body temperature is too low and triggers a system of responses which aim to generate heat (thermogenesis) and raise body temperature. These mechanisms include; shivering, increased muscle tone, vasoconstriction and the production of thyroxine hormone (which makes respiration less efficient, therefore producing more heat).

### 4.6.12 THE MAJOR ROUTES PATHOGENS MAY TAKE IN ENTERING THE BODY AND THE ROLE OF BARRIERS IN PROTECTING THE BODY FROM INFECTION

Barrier Mechanisms include;

Skin, Stomach Acid, Normal Flora, Epithelial cells.

#### SKIN ADAPTATIONS FOR DEFENCE:

![Skin Diagram](image)

**THE SKIN IS MADE FROM 2 LAYERS;**

- Outer epidermis layer
- Inner dermis layer

The epidermis provides a physical barrier to invading pathogens. There are 2 layers in the epidermis;

A Outer cornified layer, composed of compacted dead dry cells filled with indigestible **keratin** protein (which also forms nails and hair)

B Inner Malpighian layer, site of rapid mitosis and keratinisation.
The skin also has chemical defence mechanisms;

- sweat & sebaceous glands secrete **sebum**, which is an oil with pH 3 – 5. This makes the skin acidic

- sebaceous glands also secrete the enzyme **lysozyme**, which is a natural antibiotic. Lysozyme destroys bacterial cell walls.

**STOMACH ACID:**

Is made from HCl at pH 1 – 2. it is a very effective barrier.

**NORMAL FLORA:**

The skin, respiratory tract and gut are covered with **commensal** bacteria, which are part of the **normal flora** of the body. Commensal bacteria are adapted to live in the environment of the skin and the gut and the and compete with invading pathogens for the limited supply of nutrients.

**EPITHELIAL CELL ADAPTATIONS FOR DEFENCE:**

1. Epithelial cells are closely packed & connected by tight junctions forming a continuous impermeable layer
2. Epithelial cells have cilia, which form a direct physical barrier preventing pathogen attachment
3. Cilia ‘beat’ in waves, which helps clear bacteria out of the lungs and into the throat, where they are swallowed. Ingested bacteria are quickly killed by the low stomach pH and digestive proteases. Cilia also beat in the GI tract.
4. Epithelial cells secrete mucus, which is trapped by cilia. Mucus also directly prevents pathogen attachment
5. Mucus contains lysozyme

**4.6.13 HOW INDIVIDUALS MAY DEVELOP IMMUNITY**

Both T and B Cells differentiate into Memory Cells, which remain in our lymph nodes and wait until we are re-exposed to the same pathogen.

When the Memory B cell is activated by the old antigen it makes large quantities on antibody quickly and kills the pathogen **before it can infect us properly**. The memory cells provide **active immunity**.

When we are exposed to a **new** antigen it takes us about a week to be able to make new antibody. However, a second exposure to antigen produces a much faster response, and several orders of magnitude higher levels of antibody are produced.
Passive Immunity is immunity to a pathogen without Memory cells. It can occur through antibody injection or from drinking breast milk (breast milk contains high [antibody]).

Active Natural Immunity – the process above

Passive Natural Immunity – beastfeeding (antibody in milk)

Artificial Active Immunity - vaccination

Artificial Passive Immunity – antibody injection

4.6.14 HOW ‘THE EVOLUTIONARY RACE’ BETWEEN PATHOGENS AND THEIR HOSTS HAS RESULTED IN SOPHISTICATED EVASION MECHANISMS

We have evolved a very effective immune system, consisting of barriers, non-specific defence mechanisms and specific ones. If we’re so good at fighting infections, why do we still get ill?

Answer: pathogens are evolving as well.

So how has TB evolved to beat us?

1. It is spread by droplet infection, which is the most effective method of infection

2. It specifically targets epithelial cells, which means that, when inhaled, it is exactly where it wants to be

3. It does not kill immediately. This means that it has a large window of opportunity to spread to others
4. It has a very thick waxy cell wall, which means it is partially protected against lysozyme.

5. It can survive inside macrophages and lie dormant until the immune system is weakened, when it can re-infect.

So how has HIV evolved to beat us?

1. It weakens the immune system to increase its chance of survival
2. It stays in the body for years, so it can spread
3. It specifically targets Helper T cells
4. It is spread by sexual contact, so it is easily spread

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**4.6.15 BACTERIOSTATIC AND BACTERICIDAL ANTIBIOTICS**

Antibiotics work by targeting prokaryotic features not found in eukaryotic cells, e.g. penicillin targets the cell wall and breaks it down. Penicillin can be taken in large doses by humans because it has no effect on our cells (we have no cell walls).

**Bacteriostatic** antibiotics stop bacteria reproducing, they do not kill bacteria

**Bactericidal** antibiotics kill bacteria

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**4.6.16 HOW TO INVESTIGATE THE EFFECT OF DIFFERENT ANTIBIOTICS ON BACTERIA**

The effectiveness of antibiotics can be measured using a disc diffusion technique.

1. A **bacterial lawn** is grown on an agar plate (either by spreading the bacteria over the plate, or by using a pour plate).

2. A disc of blotting paper is soaked in antibiotic of known concentration and placed in the centre of the plate.

3. A clear circle of dead bacteria will form around the disc.

4. The diameter / radius of the circle of dead bacteria is proportional to the effectiveness of the antibiotic.

5. This can be compared to other antibiotics, as long as the same concentration of antibiotic is used. In addition, one can also compare the effectiveness of an antibiotic with a disinfectant or sanitiser (e.g. Phenol coefficient).

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**4.6.17 WHY ANTIBIOTIC RESISTANCE IN BACTERIA IS AN INCREASING PROBLEM**

Bacteria are becoming resistant to antibiotics. Bacteria develop resistance through mutation. A bacteria can mutate and develop resistance by;
1. Having an enzyme that breaks the antibiotic down
2. Having a protein which pumps antibiotic out of the cell
3. Mutating the structure of the bacterium so that the antibiotic no longer works

This problem is very serious. Bacteria become resistant because;

1. Bacteria mutate very easily. One in every million bacteria contains a mutation. That might sound like a small amount, but consider that one *E. coli* bacterium can reproduce to form a colony of 2 million bacteria in two hours. Over weeks, months and years that’s a lot of mutations, some of which will be beneficial
2. Bacteria reproduce very quickly (they divide every 20min) so a bacterium with a beneficial mutation will spread quickly
3. Bacteria have the ability to pass copies of plasmids from one to another (*conjugation*). So a mutation in one bacterium can quickly be copied to others, even others in different species.
4. The use of antibiotics speeds the rise of immunity. If a bacterial population is continually exposed to antibiotic all bacteria will die. As soon as a bacterium mutates the rest of the bacteria will be killed off by the latest dose of antibiotic; now the field is open for the mutated bacterium to grow without competition.
5. Humans have been reckless with use of antibiotics. They are often given to people who don’t need them (i.e. they have viral infections) or to people who don’t bother to complete the course of antibiotic.

### 4.6.18 HOW AN ‘EVOLUTIONARY RACE’ EXISTS BETWEEN PATHOGENS AND DRUG DEVELOPERS

The evolutionary arms race between bacteria and drug developers is, at the moment, tipped against humans. There are over 100 different types of antibiotic and in the 40 years since their development 4 species of bacterium have developed resistance against all of them. E.g. *Methicillin Resistant Staphylococcus Aureus* (MRSA) has been named the Superbug, because we have do drugs left that can kill it.

Unless drug developers discover another branch of antibiotics we’re not currently using (i.e. another way of targeting prokaryotic structures without damaging eukaryotic ones) there may well be a global pandemic of resistant bacteria.