Unit 5: Energy, Exercise and Coordination

Topics 7 and 8
5.7.1 - Recall the way in which muscles, tendons, the skeleton and ligaments interact to enable movement including antagonistic muscle pairs, extensors and flexors.

- **Cartilage**: a tissue made from collagen, which protects bone ends

- **A muscle**: an organ that produces movement by contraction

- **A joint**: the junction between two bones

- **A tendon**: joins muscle to bone

- **A ligament**: joins bone to bone to stabilise a joint

Muscles work in pairs. One muscle produces the opposite movement from the other muscle, therefore, the pairs are called **antagonistic pairs**.

Muscles which cause a joint to extend are called **extensors**, muscles which cause a limb to retract are called **flexors**.

**A Synovial Joint**
5.7.2 - Explain the contraction of skeletal muscle in terms of the sliding filament theory (including the role of actin, myosin, troponin, tropomyosin, Ca\(^{2+}\), ATP).

Muscles are made from muscle fibres arranged into bundles. Each fibre is made from bundles of **myofibrils**, which are extremely long, cylindrical muscle cells.

The functional unit of contraction is the **sarcomere**. Muscle cells contain many sarcomeres arranged in parallel. The muscle cell takes on a characteristic banded appearance because of the regular arrangement of the sarcomeres. This is called **striation**.

The sarcomere contains overlapping actin and myosin. The myosin is often called the **thick filament** because the myosin heads make it appear thick. The actin is, therefore, the **thin filament**.

The process by which the thin filaments are pulled in towards each other by the myosin is called **cross-bridge cycling**. It is how muscles contract.
1. A nerve impulse arrives at the neuromuscular junction.
2. The muscle cell is depolarised.
3. \( \text{Ca}^{2+} \) is released from the sarcoplasmic reticulum inside muscle cells.
4. \( \text{Ca}^{2+} \) bids to Troponin protein in the thin filament.
5. Troponin protein and Tropomyosin protein move position in the thin filament.
6. Myosin binding sites are exposed on the thin filament.
7. Myosin heads of the thick filament stick to actin.
8. ATP (already bound to the myosin head) is hydrolysed causing the myosin head to pivot forwards in the powerstroke.
9. As the head pivots the thick filament moves across the thin filament – muscle contraction occurs.
10. ADP diffuses away from the myosin head leaving the ATP-binding site empty.
11. New ATP binds & the myosin head & causes the myosin head to detach from the actin.
12. The myosin head re-cocks.
13. The head rebinds further up the myosin.
14. Repeat stages 7 to 13 until the \([\text{Ca}^{2+}]\) falls too low, when contraction stops.

Key Point: ATP is required to release myosin from actin. If ATP levels drop (assuming \( \text{Ca}^{2+} \) is present) the myosin stays attached to the actin and the muscle stays permanently contracted. This is what causes rigor mortis.
5.7.3 - Explain how phosphorylation of ATP requires energy and how dephosphorylation of ATP provides an immediate supply of energy for biological processes

Adenosine TriPhosphate (ATP) is made from three components;

- Ribose (the same sugar that forms the basis of DNA).
- A base (a group consisting of linked rings of carbon and nitrogen atoms); in this case the base is adenine.
- Up to 3 phosphate groups. These phosphates are the key to the activity of ATP.

The energy used in all cellular reactions comes from ATP. By breaking the 3rd phosphate from the ATP molecule energy is released, which can be used to power intracellular reactions. The ATP is then regenerated by recombining the phosphate and ADP in respiration (or another process e.g. photosynthesis).

The recycling of ATP is crucial for life. For example a runner uses ~84kg of ATP in a marathon (more than their total body weight), yet there are only 50g of ATP in the
ATP = one adenosine molecule with 3 phosphate groups attached.

entire body! This means each that each molecule of ATP has been recycled 1676 times during the race!

Less energy rich bond (13.8kJ/mol).

“Energy rich bond” (30.6kJ/mol).

“Energy rich bond” (30.6kJ/mol).

HOW THE ENERGY IN ATP IS LIBERATED:

ATP + H₂O → ADP + Pᵢ

Energy

ADP + H₂O → AMP + Pᵢ

Energy

AMP + H₂O → ADENOSINE + Pᵢ

Normally, as soon as ATP has been converted into ADP + Pᵢ, it is converted back into ATP using energy from respiration. However, during exercise ADP may be converted into AMP or even Adenosine to provide energy.
**Respiration**

Respiration: a process in which the chemical bond energy in glucose molecules is used to convert 38 ADP molecules into 38 ATP molecules. Oxygen is required and Carbon Dioxide and Water are produced as waste products.

Respiration occurs in 4 distinct steps;

<table>
<thead>
<tr>
<th>Step</th>
<th>Reactants</th>
<th>Products</th>
<th>Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Glycolysis (cytoplasm)</td>
<td>1 x Glucose 2 x ATP</td>
<td>2 x Pyruvate 4 x ATP 2 x NADH</td>
<td>A 6C glucose molecule is split into two 3C pyruvate molecules. Some ATP is used to split the glucose molecule in the first part of glycolysis.</td>
</tr>
<tr>
<td>2. Link Reaction (mitochondria matrix)</td>
<td>1 x Pyruvate 1 x CoA</td>
<td>1 x Acetyl CoA 1 x CO(_2) 1 x NADH</td>
<td>3C Pyruvate is split into a 2C molecule, which is attached to a CoA enzyme to form Acetyl CoA. The remaining carbon atom is used to form CO(_2).</td>
</tr>
<tr>
<td>3. Krebs’ Cycle (mitochondria matrix)</td>
<td>1 x Acetyl CoA</td>
<td>1 x CoA 1 x ATP 2 x CO(_2) 3 x NADH 1 x FADH(_2)</td>
<td>CoA enzyme gives its 2C atoms to a 4C molecule to form a temporary 6C molecule. In a series of steps the 6C molecule releases the two C atoms as CO(_2) eventually re-forming the starting 4C compound. The cycle is then ready to repeat itself. As the cycle turns ATP, NADH &amp; FADH(_2) are formed.</td>
</tr>
<tr>
<td>4. Oxidative Phosphorylation (mitochondria christae)</td>
<td>10 x NADH 2 x FADH(_2) 6 x O(_2)</td>
<td>34 x ATP 6 x H(_2)O</td>
<td>The electron transport chain uses the NADH and FADH(_2) made in previous steps to make lots of ATP.</td>
</tr>
</tbody>
</table>
**Respiration: Step 1 – Glycolysis**

In **Glycolysis** a Glucose molecule (6C) is split into 2 molecules of Glyceraldehyde Phosphate (3C). 2ATPs are required for this to happen.

Then, each 3C Glyceraldehyde Phosphate molecule is converted into a 3C Pyruvate molecule. In the process of converting one Glyceraldehyde Phosphate to one Pyruvate, enough energy is released to convert one NAD molecules into one NADH molecules and also to make two ATP molecules.

Overall; 4ATP are made, 2NADH are made and 2ATPs are used.

**Net gain: 2ATP and 2NADH**
In **anaerobic conditions** \([H^+]\) rises in the mitochondria as there are no available oxygen molecules to mop it up with and form water. This leads to saturation of the electron transport chain and a build-up of NADH and FADH2. This means [NAD] falls, which stops the Krebs’ Cycle. Acetyl CoA levels build-up, [CoA] falls and the Link Reaction stops. Pyruvate levels start to rise...

Muscle cells turn pyruvate into lactate to stop rising [pyruvate] from stopping Glycolysis (remember, enzyme controlled reactions are reversible and depend on [reactants] and [products]).

\[
\begin{align*}
\text{NADH} & \quad \text{NAD} \\
\text{Pyruvate} & \quad \rightleftharpoons \quad \text{Lactate}
\end{align*}
\]

In the **liver** the lactate is converted back into pyruvate. This requires oxygen, which is the basis of the “Oxygen Debt”

**RESPIRATION: STEP 2 – LINK REACTION**

1 NADH is **made** (2 overall)

1 CO₂ is **made** (2 overall)

**Link Reaction takes place in the matrix of the mitochondria**

In the **Link Reaction** a Pyruvate molecule (3C) is split into a 2C molecule and a CO₂. The 2C molecule is attached to a CoA enzyme, forming Acteyl CoA.

Remember, **two** molecules of Pyruvate were made at the end of Glycolysis, therefore the Link Reaction happens **twice**.

Overall; **2NADH and 2 CO₂** are made.  
**Net gain: 2NADH**
RESPIRATION: STEP 3 – KREBS’ CYCLE

In the Krebs’ Cycle the Acetyl CoA gives its 2C atoms to a 4C molecule (Oxaloacetate) forming an unstable 6C molecule (Citric Acid). The 6C molecule breaks down into a 4C compound (Succinyl – CoA) releasing enough energy to make one NADH. The two spare C atoms are released as two CO₂ molecules.

Succinyl – CoA is converted back into Oxaloacetate and this releases enough energy to make one NADH, one FADH₂ and one ATP. The Oxaloacetate can then be used in the cycle again.

Remember, two molecules of Acetyl CoA were made at the end of the Link Reaction, therefore the Krebs’ Cycle happens twice.

Overall; 4NADH, 2FADH₂, 2CO₂ and 2ATP are made.

RESPIRATION: STEP 4 – OXIDATIVE PHOSPHORYLATION

Oxidative Phosphorylation uses the NADH and FADH₂ produced in the previous steps of respiration to make ATP. Each NADH makes 3ATP and each FADH₂ makes 2 ATP.
Hydrogen atoms from the NADH and the reduced FADH₂ are passed onto 2 the first 2 enzymes of the Electron Transport Chain. These enzymes are Hydrogen Carriers and they accept the H atoms from the NADH and the FADH₂.

Electrons, which made up the chemical bond between the hydrogen atoms and the NADH / FADH₂ are passed onto 3 Electron Carrier enzymes further down the Electron Transport Chain.

At the end of the Electron Transport Chain, the electrons are recombined with the H⁺ atoms and oxygen, to form water. This is the only, but crucial, part of respiration to involve oxygen.

NADH starts at the first Hydrogen Carrier and has enough energy to phosphorylate 3ADP. FADH₂ has less energy and starts at the second Hydrogen Carrier, it generates 2 ATPs

Where does the 38 ATP come from?

Glycolysis produces; 2ATP 2NADH
Link Reaction produces; 2ATP 2NADH
Kreb’s Cycle produces; 2ATP 6NADH 2 FADH₂
Total 4 ATP 10NADH 2 FADH₂
Each NADH produces 3ATP. ∴ total production is 30ATP from NADH

Each FADH$_2$ produces 2ATP. ∴ total production is 4ATP from FADH$_2$

\[
\text{Grand Total} \quad 4\text{ATP} + 30\text{ATP} + 4\text{ATP} = 38\text{ATP}
\]

\[\text{Chemiosmosis}\] of H$^+$ ions from the mitochondrial envelope into the matrix through ATP Synthetase proteins is what actually generates the ATP in respiration.

The electron transport chain uses the process of chemiosmosis (the diffusion of ions across a membrane). H$^+$ ions are actively pumped into the mitochondrial envelope. This is done by the proteins in the electron transport chain, using the energy stored in NADH and FADH$_2$.

The [H$^+$] builds up to very high levels in the envelope. However, H$^+$ cannot escape because it is charged (hydrophilic) and therefore cannot move through the phospholipid bilayer in the envelope membranes.

Special proteins called ATP Synthetase do allow H$^+$ to pass through them and escape into the mitochondrial matrix. Whenever an H$^+$ ion moves through the ATP Synthetase protein an ADP is phosphorylated by the ATP Synthetase.

In summary;

1. NADH and FADH$_2$ contain stored chemical energy.
2. The energy is used to pump H$^+$ into the mitochondrial membrane against the concentration gradient.
3. H$^+$ trapped in one place represents a store of potential energy.
4. H$^+$ ions leave the envelope through ATP Synthetase proteins.
5. The potential energy of the H$^+$ is used to phosphorylate ATP as the H$^+$ moves out of the envelope.
5.7.7 - The fate of lactate after a period of anaerobic respiration

In anaerobic respiration lactate is taken via the blood to the liver, where it is broken down into pyruvate using oxygen and NADH.

5.7.8 - How variations in ventilation and cardiac output enable efficient delivery of oxygen to tissues and removal of carbon dioxide from them, how the heart rate and ventilation rate are controlled and the roles of the cardiovascular control centre and the ventilation centre
5.7.9 - How to investigate the effects of exercise on tidal volume and breathing rate

A spirometer is used to plot breathing patterns

**Vital Capacity:** The maximum amount of air a person can exhale after inhaling the maximum possible volume of air

**Tidal Volume:** The volume of air inhaled & exhaled in one breath

**Basal Metabolic Rate:** The rate of respiration

The spirometer can be used to plot VC and TV directly. BMR can be worked out if a CO₂ scrubber is used. The spirometer has fixed volume and is filled with 100% O₂ before the experiment begins. As the person respires, O₂ is replaced proportionally with CO₂. The total volume should stay constant. However, if CO₂ is removed, the total volume will slowly fall as O₂ is used. The rate at which the volume decreases is proportionally to BMR.

You are not expected to know how the spirometer works... although its not very difficult to understand.
5.7.10 & 5.7.11 - Why some animals are better at short bursts of high intensity exercise while others are better at long periods of continuous activity, the structural, and the physiological, differences between fast and slow twitch muscle fibres

Sprinters need lots of fast twitch muscle, joggers need slow twitch. Therefore, the muscle type of a cheetah or a gazelle will be predominantly fast twitch, whereas the muscle of a camel or an elephant will be predominantly slow twitch.

Muscle type in humans is predominantly one or the other due to inherited alleles. However, different training programmes can cause the % of either type to change slightly.

<table>
<thead>
<tr>
<th>Slow twitch fibres</th>
<th>Fast twitch fibres</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red (lots of myoglobin)</td>
<td>White (little myoglobin)</td>
</tr>
<tr>
<td>Many mitochondria</td>
<td>Few mitochondria</td>
</tr>
<tr>
<td>Little sarcoplasmic reticulum</td>
<td>Lots of sarcoplasmic reticulum</td>
</tr>
<tr>
<td>Low glycogen content</td>
<td>Lots of glycogen</td>
</tr>
<tr>
<td>Numerous capillaries</td>
<td>Few capillaries</td>
</tr>
<tr>
<td>Fatigue resistant</td>
<td>Fatigue quickly</td>
</tr>
</tbody>
</table>

5.7.12 - The concept of homeostasis and its importance in maintaining the body in a state of dynamic equilibrium during exercise as exemplified by thermoregulation, including the role of the heat loss, heat gain centres and mechanisms for controlled body temperature

See 4.6.11 for mechanisms of thermoregulation.

The thermoregulatory process (and most homeostatic systems) are controlled by negative feedback processes. If a system changes, it is detected, a homeostatic response is activated, which aims to return the system to its original level. Negative feedback, therefore, holds systems at a set point, in this case 37.5°C.
5.7.13 - Possible disadvantages of exercising too much (wear and tear on joints, suppression of the immune system) and exercising too little (increased risk of obesity, CHD and diabetes)

A moderate level of exercise improves health & well-being.

However, over-training can result in the opposite effect. This is the phenomenon known as “burn-out”

Positive effects of exercise include:

1. Increased BMR
2. Decreased blood pressure
3. Increased HDL
4. Decreased LDL
5. Maintaining healthy BMI
6. Decreased risk of diabetes
7. Increased bone density
8. Improved well being
9. Decreased adrenaline levels
10. Less stress
11. Decreased risk of CHD
12. Moderate exercise increases levels of Natural Killer cells, which secrete apoptosis-inducing chemicals in response to non-specific viral or cancerous threat

Negative effects of exercise (over-training) include:

1. Decreased levels of Natural Killer Cells, Phagocytes and B & T Cells. This decreases immune response.
2. Increased muscle inflammation
3. Muscle tears and sprains
4. Increased adrenaline levels
5. Increased cortisol levels, which also decreases the immune response
6. Increased stress
7. Damaged cartilage
8. Tendinitis
9. Ligament damage
10. Swollen bursae.

5.7.14 - How medical technology, including the use of key-hole surgery and prostheses, is enabling those with injuries and disabilities to participate in sports

Key-hole surgery is a technique which allows doctors to conduct surgery with the minimum possible damage to the patient. The surgeon makes a small incision (a “key-hole”) and uses a fibre-optic camera to view the damaged area. If required, the surgeon can make a second incision and use a number of small, remote operated tools to repair the damage. Because the incisions are small and only the damaged area is targeted, the patient recovers quickly. There is also less chance of infection.

Unfortunately, the procedure requires a high degree of training, expensive equipment and can only be used on certain types of surgery.

Prosthetics allow people with amputations to participate in many activities, including sports.

5.7.15 - Whether the use by athletes of performance enhancing substances is morally and ethically acceptable.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effect on physiology</th>
<th>Effect on performance</th>
<th>Side-effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythropoietin (EPO)</td>
<td>EPO causes the bone marrow to generate extra red blood cells.</td>
<td>Extra blood cells mean the blood can carry extra oxygen. This increases the level of work the body can sustain through aerobic respiration (aerobic threshold).</td>
<td>Increased haemocrit increases blood viscosity. This causes strain on the heart and can lead to infarction</td>
</tr>
<tr>
<td>Creatine</td>
<td>Creatine combines with phosphate to form Creatine Phosphate (CP). CP can phosphorylate ADP, re-generating ATP.</td>
<td>Because ATP is re-generated without using the respiratory pathways, theoretically it should increase the maximum power of muscles and decrease recovery time</td>
<td>Diarrhoea, vomiting, liver damage and kidney damage.</td>
</tr>
</tbody>
</table>
Why should we allow use of drugs:

- Gives people a chance to be as good as their potential allows
- Removes “unfair” genetic advantages
- Controlled use of drugs is less risky
- People should have the right of choice
- Legalising drugs makes their distribution controllable (no use by underage, infirm etc)

Arguments for not using drugs:

- Dangerous (obviously)
- May be pushed onto athletes by trainers
- Effects are permanent
- Not used under doctor’s supervision
- Often cut with other drugs
- Exposes athletes to criminals (danger of using other drugs)

The list goes on, just think for yourself in the context of the question. You can argue the toss either way, but make sure you can back up your opinion with some sensible, logical arguments.
5.8.1 - Describe the structure and function of sensory, relay and motor neurones including the role of Schwann cells and myelination

**Sensory nerve:** carries electrical message from receptor to spine

**Motor nerve:** carries electrical message from spine to effector.

**Relay nerve:** connects sensory and motor nerves. Also relays message to the brain.

**Schwann cells:** wrap around the axon of the long nerves, creating a thick layer of membrane, which insulates the nerve and allows for much faster conduction speed. The thick layer of membrane has gaps in it between adjacent Schwann cells, these are called **Nodes of Ranvier.**
5.8.2 - How the nervous systems of organisms can cause effectors to respond as exemplified by pupil dilation and contraction

High light intensity
Circular muscles: contracted
Radial muscles: relaxed
Pupil diameter: small

Low light intensity
Circular muscles: relaxed
Radial muscles: contracted
Pupil diameter: large

5.8.3 - The Action Potential


3. The membrane is hyperpolarised. Voltage-dependent K⁺ channels close. K⁺ diffuse back into the axon to recreate the resting potential.
Sequence of events in an action potential;

1. Nerve is at resting membrane potential (-70mV)
2. A stimulus depolarises the nerve to **threshold** (-50mV)
3. **Voltage-gated Na**⁺ **Channels** open
4. Sodium floods into the cell and the membrane potential depolarises to +30mV
5. **Voltage-gated K**⁺ **Channels** open
6. Potassium floods out of the cell and the membrane potential falls to -90mV
7. The nerve is in the refractory period and cannot conduct another action potential.
8. The 3Na⁺/2K⁺ ATPase (**Na⁺/K Pump**) restores the ion concentrations.
9. The nerve is ready to fire again.

As one part of the nerve fires off, Na⁺ diffuses into the next section of the nerve, which depolarises the nerve to threshold. This sequence is repeated like a tiny Mexican wave down the axon of the nerve.

Nodes of Ranvier speed this conduction process up. When one node depolarises it induces the next section of the nerve to depolarise by forming a mini-circuit between nodes. This causes the action potential to “jump” between nodes of ranvier, making conduction speed much faster.

**5.8.4 - The structure and function of synapses including the role of neurotransmitters (including acetylcholine)**

A **synapse** is the junction between two nerves. It is also a verb, i.e. one nerve **synapses** with another (meaning, passes a message to another).

The neurotransmitter on your syllabus is Ach, but over 2000 other transmitters have been discovered.
1. The wave of depolarisation arrives at the synaptic knob. The membrane in the presynaptic neuron is depolarised to −50mv (threshold potential) and the voltage-gated Na⁺ channels open, letting Na⁺ into the cell.

2. The membrane is depolarised to +30mV and voltage-gated K⁺ channels open. The membrane potential falls to −90mV and the cell goes into its refractory period, where the 3Na⁺/2K⁺-ATPase restored the ion concentrations.

3. Unlike axons, presynaptic nerves also contain a Voltage-gated Ca²⁺ channel. As the presynaptic membrane depolarises these channels open and let Ca²⁺ into the cell.

4. The Ca²⁺ causes vesicles in the presynaptic nerve to migrate and fuse with the presynaptic membrane, where they spill neurotransmitter chemical into the synaptic cleft.

5. Neurotransmitter binds with receptors on the postsynaptic membrane. Cation channels open. Sodium ions flow through the channels.

6. The membrane depolarises and initiates an action potential.

7. When released the neurotransmitter will be taken up across the presynaptic membrane (whole or after being broken down), or it can diffuse away and be broken down.
5. The neurotransmitter (Acetyl Choline) diffuses across the cleft and binds to receptors on the postsynaptic membrane.

6. The receptors let a little Na\(^+\) into the postsynaptic neuron, which is enough to initiate another action potential in the postsynaptic nerve.

7. The ACh is broken down by an enzyme called Acetyl Choline Esterase (AchE), which allows the postsynaptic receptors to be freed ready for a second synapse.

In a neuromuscular junction the sequence of events in the synapse is exactly the same. The only difference is that the posysynaptic nerve is a muscle cell and, instead of being flat, the postsynaptic membrane has deep grooves (t tubules) which allow the depolarisation to spread quickly through the muscle so all parts of the muscle contract at the same time.

Some neurotransmitters can hyperpolarise postsynaptic nerves, which essentially switches them off. An example of this type of inhibitory neurotransmitter is GABA.

### 5.8.5 - How the Nervous Systems of Organisms Can Detect Stimuli

**Visual transduction** is the process by which light initiates a nerve impulse. The structure of a rod cell is:

The detection of light is carried out on the membrane disks in the outer segment. These disks contain thousands of molecules of rhodopsin, the photoreceptor molecule. Rhodopsin consists of a membrane-bound protein called ops in and a covalently-bound prosthetic group called retinal. Retinal is made from vitamin A, and a dietary deficiency in this vitamin causes night-blindness (poor vision in dim light). Retinal is the light-sensitive part, and it can exists in 2 forms: a cis form and a trans form:
In the dark retinal is in the *cis* form, but when it absorbs a photon of light it quickly switches to the *trans* form. This changes its shape and therefore the shape of the opsin protein as well. This process is called **bleaching**. The reverse reaction (*trans* to *cis* retinal) requires an enzyme reaction and is very slow, taking a few minutes. This explains why you are initially blind when you walk from sunlight to a dark room: in the light almost all your retinal was in the *trans* form, and it takes some time to form enough *cis* retinal to respond to the light indoors.

Rod cell membranes contain a special sodium channel that is controlled by rhodopsin. Rhodopsin with *cis* retinal opens it and rhodopsin with *trans* retinal closes it. This means in the dark the channel is open, allowing sodium ions to flow in and causing the rod cell to be depolarised. This in turn means that rod cells release neurotransmitter in the dark!

However the synapse with the bipolar cell is an **inhibitory synapse**, so the neurotransmitter **stops** the bipolar cell making a nerve impulse. In the light everything is reversed, and the bipolar cell is depolarised and forms a nerve impulse, which is passed to the ganglion cell and to the brain.
Summary for light;

1. Photon hits rhodopsin.
2. Bleaching occurs and trans retinal is formed.
3. Trans retinal blocks Na⁺ channels.
4. The rod is hyperpolarised and stops releasing inhibitory neurotransmitter.
5. The bipolar cell is no longer inhibited and depolarises.
6. The ganglion cell is activated, which carries the message to the brain.

Cones work in exactly the same way, except that they contain the pigment iodopsin, which is found in 3 different forms; red-sensitive, blue-sensitive and green-sensitive. This gives us colour vision.

5.8.6 Compare and contrast nervous and hormonal coordination

Homeostasis is the maintenance of the internal environment.

- Nerve reflexes give immediate responses
- Hormone responses give responses over weeks – months

Hormones are released from glands, which release hormone into the blood. The hormone is carried all over the body. It binds to hormone receptors on cell membranes and initiates responses in those cells.

5.8.7 Locate and state the functions of the regions of the human brain

- Corpus callosum – white matter composed mainly of axons, whose white myelin sheaths give it its characteristic appearance.
- It provides connections between the cortex and the brain structures below. It also forms connections between the two hemispheres of the cortex.
**Hindbrain**

**Brainstem** – Uppermost part of the spine, where the spine joins the brain.

**Medulla** - controls vital ‘housekeeping’ functions, such as heartbeat, blood pressure and peristalsis.

**Cerebellum** - controls muscle co-ordination & learns motor programmes (e.g. like how to ride a bike, or write).

**Midbrain:**

**Thalamus** – a relay station that carries sensory information from the sense organs to the correct part of the cortex and hypothalamus. The thalamus contains the Superior Colliculi, which control the initial processing of visual information. The Superior Colliculi control object tracking, spatial position and partial recognition (i.e. whether a stimulus is food or a threat).

**Hypothalamus** – receives sensory information from the thalamus. Contains homeostatic centres, which control factors like body temperature and blood osmolarity. The hypothalamus is connected to the Pituitary gland and therefore the hypothalamus can stimulate the release of a great number of pituitary hormones.

**Forebrain:**

**Cortex** – processes sensory information and controls the body’s voluntary behaviour, i.e. learning, personality and memory. This is the part of the brain that actually “thinks.” The cortex is very large in humans and is folded to increase the surface area further. Other animals have roughly similar size hind- and midbrains. However, their cortex is much, much smaller.
Occipital lobe - processes & interprets information from the eyes

Temporal lobe - processes & interprets information from the ears and processes language and the meaning of words

Parietal lobe – processes and interprets information about touch, taste, pressure, pain, heat and cold. Also initiates motor commands.

Frontal lobe - plans and organises thought, is involved with short term memory and puts speech together.

5.8.8 - Explain how images produced by MRI, fMRI and CT scans can be used to investigate brain structure and activity

<table>
<thead>
<tr>
<th>Technique</th>
<th>How it works</th>
<th>What it allows us to see</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery</td>
<td>During brain surgery a local anaesthetic is often used. This allows the surgeon to ask the patient questions as he operates on their brain.</td>
<td>The patient can tell the doctor what he/she is feeling as the doctor stimulates parts of his/her brain. This can tell us a lot about the function of the brain.</td>
</tr>
<tr>
<td>C T Scan</td>
<td>Thousands of narrow-beam X-rays pass through the patient’s head from a rotating source. The rays are collected on the other side of the head and their strength measured. The density of the tissue the X-ray passes through decreases the strength of the signal, and therefore, lets us work out what type of tissue is in the brain.</td>
<td>CT Scans show brain structures, not brain activity. They also only give “frozen” still images. However, they are very useful for picking up diseases, such as cancer, stroke and oedema.</td>
</tr>
<tr>
<td>MRI Scan</td>
<td>Magnetic fields are used to align protons in water molecules in the patient’s brain. When the fields are switched off, the protons give out a little energy, which can be detected.</td>
<td>By recording the energy given out by protons we can build up a sequence of thin pictures of the types of tissues inside the brain. This can be fed into a computer, which uses the picture to build up a 3D image of the inside of the head</td>
</tr>
</tbody>
</table>
5.8.9 - The evidence that there exists a critical ‘window’ within which humans must be exposed to particular stimuli if they are to develop their visual capacities to the full

How to process stimuli correctly must be learned. The cortex is split into column of cells. When we are born, the columns overlap and are tangled. As we learn to process stimuli, the cells organise themselves into discrete columns, which no longer overlap. There is a “critical window” for this to happen (usually before puberty, younger for visual processing). If we miss the window, our brains will become “fixed” with tangled columns and won’t be able to process stimuli properly.

Hubel & Wiesel’s experiments prove this.

5.8.10 - How to investigate visual perception in humans

The Muller-Lyer illusion;

Lines A and B are the same length, yet look different – why? The answer is that you have learned to process this kind of stimuli in a certain way. We live in a “carpentered world” of straight lines and we interpret line B as a corner (therefore larger than it appears, because it must be far away) and line A as a corner (therefore, smaller than it appears, because it must be close).

These optical illusions do not work on Zulus, which proves the illusion is caused by learned visual processing, rather than an innate function of the eye / brain.
5.8.11 - Ways in which animals including humans can learn

**Association (classical conditioning):**

\[ \text{US} \rightarrow \text{UR} \quad (\text{Food} \rightarrow \text{Salivation}) \]

Over time, if a neutral stimulus (CR) is played with the US, it becomes associated with the US and begins to elicit the same response. Eventually, the animal learns

\[ \text{CS} \rightarrow \text{CR} \quad (\text{Bell} \rightarrow \text{Salivation}) \]

Pavlovian conditioning occurs by synapses between nerves growing together. This means that the sensory nerve carrying the message of the CS will always lead to the firing of the motor nerve, which triggers the CR.

**Operant Conditioning:**

This is very similar to classical conditioning except the animal learns by doing something i.e. it learns that an action has a certain outcome

\[ A \rightarrow O \ (\text{pushing a level} \rightarrow \text{food}) \]

**Habituation:**

If the neutral stimulus is continuously present (not just before the US), but all the time, the animal learns to ignore the CS. The animal learns the bell signals nothing and it ignores the CS totally. This is called **habituation**.

If a nerve is frequently stimulated, the amount of \( \text{Ca}^{2+} \) that enters the pre-synaptic nerve gradually diminishes, until it is no longer enough to trigger vesicles to fuse with the pre-synaptic membrane. This means no neurotransmitter is released, which results in no post-synaptic depolarisation. The effect is, essentially, that the stimulus is ignored.

**Insight Learning:**

In the early 1900s, Wolfgang Kohler performed insight experiments on chimpanzees. Kohler showed that the chimpanzees sometimes used insight instead of trial-and-error responses to solve problems. When a banana was placed high out of reach, the animals discovered that they could stack boxes on top of each other to reach it. They also realized that they could use sticks to knock the banana down. In another experiment, a chimp balanced a stick on end under a bunch of bananas suspended from the ceiling, then quickly climbed the stick to obtain the entire bunch intact and unbruised (a better technique than the researchers themselves had in mind). Kohler’s experiments showed that primates can both see and use the relationships involved to reach their goals.
This type of learning is very difficult to explain using the Pavlovian model of conditioning. It is also difficult to explain using neuronal models of learning (i.e. synapses growing together through use) developed through studies on *Aplysia*. How insight learning occurs is unknown at the moment.

**5.8.12 - The role animal models have played in understanding human brain development and function**

**Pavlov’s Dogs**

Pavlov had observed that an *unconditioned stimulus* causes an *unconditioned response*, i.e. food causes salivation. This is not learned and is, therefore, *unconditioned*.

What Pavlov discovered was that if a neutral stimulus, such as a bell is rung *just before* the food is given for a few occasions, the dog will salivate every time the bell is rung, even if no food is presented. In this case, the dog has learned that the bell signals food. The food is, therefore, a *conditioned stimulus* and it prompts a *conditioned response*.

\[
\text{US} \rightarrow \text{UR} \\
\text{US} + \text{CS} \rightarrow \text{UR} \\
\text{Eventually, CS} \rightarrow \text{CR}
\]

**Hubel & Wiesel**

- *Hubel & Wiesel* investigated the critical window.
- They used *monkeys* and *kittens* in their studies.
- Their work permanently blinded some animals and can be argued to be *unethical*.

**Hubel & Wiesel’s Method:**

1. Raise monkeys from birth in three groups for *6 months*
2. Group 1 are the control (no blindfold), Group 2 are blindfolded in both eyes, Group 3 are blindfolded in one eye (monocular deprivation)

3. Test the monkeys to see whether they can see using each eye
4. Test the sensitivity of retinal cells
5. Test the activity of nerves in the visual cortex in response to stimuli

The results:

- Monkeys in Group 2 (both eyes blindfolded) had impaired vision
- Monkeys in Group 3 (monocular deprivation) were blind in the deprived eye
- Retinal cells were responsive in all groups
- Cortical activity was reduced in parts of the brain that process information from the deprived eye
- Adults undergoing the same tests showed no difference between groups. All could see.

The Conclusion:

There is a critical window for visual neural development, which requires stimulus from the eye. If this window is missed the monkey is blind, because of events happening in the brain, not the eye.

You need to know about these experiments because they all use animals.

5.8.13 Discuss the moral and ethical issues related to the use of animals in medical research

<table>
<thead>
<tr>
<th>Arguments For</th>
<th>Arguments Against</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Trials Stage 1 involves animals. Without animals we would not be able to discover new drugs.</td>
<td>Why not use computer simulations in Clinical trials instead?</td>
</tr>
<tr>
<td>Animal testing is better than nothing and does, in some cases, avert potential loss of human life</td>
<td>Animal physiology is different to human physiology. Animal testing is, therefore, unhelpful.</td>
</tr>
<tr>
<td><strong>Utilitarian argument:</strong> Animal testing is for the greater good.</td>
<td>Animals have rights too.</td>
</tr>
<tr>
<td>Machines like the MRI were unvested using animals.</td>
<td>Animals have no informed consent.</td>
</tr>
<tr>
<td>Animal testing has advanced our understanding of human physiology.</td>
<td>Testing on animals when the potential side-effects are unknown is immoral.</td>
</tr>
<tr>
<td></td>
<td>Animals can’t tell you when they are suffering.</td>
</tr>
<tr>
<td></td>
<td>Animals are often poorly cared for in labs.</td>
</tr>
</tbody>
</table>
5.8.14 - How imbalances in, naturally occurring brain chemicals can contribute to health consequences and the development of new drugs

In Parkinson’s disease neurons in the brain die. All these neurons secrete dopamine neurotransmitter, which causes difficulty in movement and limb shaking.

In depression neurons in the brain that secrete serotonin neurotransmitter stop working properly and serotonin levels fall.

In both cases treatments that increase the levels of neurotransmitter might prove successful in relieving the symptoms of these diseases.

5.8.15 - The effects of drugs on synaptic transmissions

Drugs that affect synapses can drastically alter the functioning of the brain;

**MDMA:**

Active ingredient in ecstasy. This binds to protein pumps on the pre-synaptic membrane of nerves that secrete serotonin. The pumps would normally take serotonin up after it had been released, therefore reducing firing in post-synaptic nerves. BUT, when these channels are blocked, serotonin builds up in the cleft, giving greater post-synaptic activation and a sense of euphoria.

**L-Dopa:**

This is a precursor of dopamine. When given to Parkinson’s sufferers it is turned into dopamine, which helps alleviate some of the symptoms of the disease.

5.8.16 - Some characteristics are controlled by alleles at many loci and how this can give rise to phenotypes which show continuous variation

**Continuous variation:** there is a wide range of phenotypes (e.g. height)

**Discontinuous variation:** phenotypes fall into discrete categories (e.g. blood type)

Discontinuous variation tends to be coded for by one gene with a few different alleles. However, continuous variation is more complex. This is usually coded for by many genes (**polygenes**), with many alleles, which produces the much greater range of possible phenotypes.

Polygenes can give rise to susceptibility to disease, usually with an environmental trigger. Diseases that are both genetic and environmental are called **multifactorial**.

5.8.17 - The methods used to compare the contributions of nature and nurture to brain development

Brain development is a combination of **nature** and **nurture**.