# Topic 6 – Organisms respond to changes in their internal and external environments

## Key words

**Tropism** – a growth response to a stimulus

**Gravitropism** – a growth response to gravity.

**Phototropism** – a growth response to light.

**Taxis** – the directional movement of an organism in response to a stimulus

**Kinesis** – the undirected movement of an organism in response to a stimulus

**Myelin** **sheath** – non conductive tissue that surrounds the neuron. It is made up of Schwann cells

**Visual** **acuity** – the ability to distinguish between to separate sources of light.

**Homeostasis** – maintenance of an internal environment within restricted limits

**Negative** **feedback** – restores the system back to the original levels.

**Glycogenesis** – the formation of glycogen from glucose.

**Glycogenolysis** – the break down of glycogen into glucose.

**Gluconeogenesis** – the formation of glucose from glycerol and amino acids.

**Osmoregulation** – is the control of the water potential of the blood

1. In **shoots indoleacetic acid** (IAA) diffuses to the shady side, this causes cells to elongate which causes phototropism.
2. In **roots indoleacetic acid** (IAA) diffuses to the cells at the bottom on the root, this causes cells to not elongate which causes gravitropism.
3. Simple **reflexes** keep complex organisms safe by quickly responding to stimulus. Simple reflexes are made up of 3 neurones: sensory neuron, relay neurone (within the spine) and a motor neurone.
4. The **Pacinian corpuscle** is a receptor that detects pressure on the skin. It has layers of lamellae with stretch mediated Na+ channels. Inside it has a sensory neurone.
5. When pressure is applied the layers of lamellae become deformed. This causes the stretch mediated Na+ Channels to open and allow Na+ to diffuse into the neurone. This causes a depolarisation of the neurone called a generator potential. If threshold is reached, then an action potential will be initiated.
6. The **retina** is at the back of the eye and contains photoreceptors called rods and cones.
   1. **Rod** cells
      1. Can not distinguish between different wavelengths so will only produce an image in black and white.
      2. Many rod cells are attached to one sensory neurone – this is called special summation.
      3. Because of special summation rod cells are respond to low light intensity. This is because many cells can work together to produce a generator potential to reach threshold to initiate an action potential
      4. However, there is a low visual acuity
      5. Rod cells contain rhodopsin which is broken down into opsin ins the presence of light.
   2. **Cone** cells
      1. There are three different types of cone cells all responding to different wavelengths of light allowing cones to see in colour.
      2. Each cone is connected to one sensory neurone.
      3. Cone cells respond to high light intensity because one cell needs to create a generator potential that needs to reach a threshold to initiate an action potential.
      4. Because cone cells are connected to one sensory neurone cone cells give a high visual acuity
      5. Cone cells contain iodopsin
      6. There is a higher quantity of cone cells at the fovea than rod cells.
   3. There are no rods and cones situated on the optic nerve.
7. Heart rate is **myogenic** – this means individual nerve impulses do not control contractions of the heart muscle and is maintained by waves of electrical activity.
8. The Sino-atrial node (**SAN**) is situated in the right atrium.
9. The Atrioventricular node (**AVN**) is situated in the septum between the ventricles and atria.
10. The bundle of His goes down the centre of the heart and the Purkinje fibres go up the outside of the ventricles.
    1. The SAN sends and electrical impulse around the atrium to the non-conductive tissues and to the AVN. This causes the atria to contract.
    2. The AVN causes a pause in electrical impulse. This leaves enough time for the blood to leave the atria and enter the ventricles.
    3. The AVN then sends electrical impulses down the bundle of His and up the Purkinje fibres causing the ventricles to contract.
11. **Chemoreceptors** in the carotid arteries and the aorta detect pressure changed
    1. Decrease in pH
       1. If the pH of the blood decreases this is because of an increase in CO1 form respiration
       2. The chemoreceptors send an impulse via the sensory neurone to the medulla in the brain.
       3. The sympathetic nerve sends an impulse to the SAN which increased the number of impulses from the SAN to the AVN.
       4. This is because noradrenaline – an excitatory neurotransmitter is released.
    2. Increase in pH
       1. If the pH of the blood increases.
       2. The chemoreceptors send an impulse via the sensory neurone to the medulla in the brain.
       3. The parasympathetic nerve sends an impulse to the SAN which decrease the number of impulses from the SAN to the AVN.
       4. This is because acetylcholine – an inhibitory neurotransmitter is released
12. **Pressure** receptors are located in carotid artery and the aorta
    1. Decrease in blood pressure
       1. The pressure receptor sends an impulse via the sensory neurone to the medulla in the brain.
       2. The sympathetic nerve sends an impulse to the SAN which increased the number of impulses from the SAN to the AVN.
       3. This is because noradrenaline – an excitatory neurotransmitter is released.
    2. Increase in blood pressure
       1. The pressure receptor sends an impulse via the sensory neurone to the medulla in the brain.
       2. The parasympathetic nerve sends an impulse to the SAN which decrease the number of impulses from the SAN to the AVN.
       3. This is because acetylcholine – an inhibitory neurotransmitter is released
13. **Resting potential** of a neuron is created by the movement of Na+ and K+.
    1. The sodium/potassium pump uses the hydrolysis of ATP to move 3Na+ out of the axon and 2K+ ions into the axon.
    2. The axon is more permeable to K+ ions so they diffuse across the membrane.
    3. This maintains the resting membrane potential – normally around -70mV.
14. **Depolarisations** are cause when Na+ ions diffuse into the axon.
15. A **generator** **potential** need to reach a threshold for an action potential to be reached. This means enough Na+ move into the axon to create a positive axon.
    1. When a threshold is reached, voltage gated Na+ ion channels open causing the axon to be more permeable to Na+ allowing Na+ to move down an electrochemical gradient into the axon.
    2. This causes a depolarisation.
    3. Voltage gated Na+ ion channels close
    4. Voltage gated K+ ion channels open causing the axon to be more permeable to K+ so K+ ions diffuse out of the axon down their electrochemical gradient.
    5. This causes the axon to become more negative this is called repolarisation.
    6. The K+ ion channels are slow to close so this causes a hyperpolarisation
    7. The Na+/K+ pump will cause the resting potential to be reached
16. The **refractory** period is the period the immediately follows the action potential when the axon is hyperpolarised.
    1. During this time another action potential cannot be created ensuring that the action potentials are discreate.
    2. It also insures the action potential will only travel in one direction.
17. An action potential is an ‘**all or nothing response’**. It either will or won’t occur – the action potential doesn’t change in size.
18. **Myelination** causes saltatory conduction. This is where the depolarisation only occurs in the nodes of Ranvier. This speeds up conduction of an action potential because a depolarisation doesn’t occur across the entire length of the axon.
19. At the end of a neurone there is a **synapse** or a **neuromuscular** junction.
20. At a synapse
    1. Voltage-gated Ca+ ion channels open and diffuse into the pre-synaptic neurone.
    2. This causes vesicles containing neurotransmitter to fuse to the pre-synaptic membrane
    3. This causes the neurotransmitter to be released to across the synaptic cleft
    4. The neurotransmitter will bind to receptors on the postsynaptic neurone.
    5. This will cause the protein channels to open.
21. Synapses could be **excitatory** or **inhibitory**.
    1. Excitatory causes Na+ to diffuse into the post-synaptic neurone causing a depolarisation
    2. Inhibitory causes Cl- to diffuse into the post-synaptic neurone casing a hyperpolarisation.
22. **Synapses** are **unidirectional** – synapses only occur in one direction
    1. Receptors are only on post-synaptic neurones
    2. Neurotransmitter is released from vesicles in the pre-synaptic neurones
23. **Temporal summation** – there is only one presynaptic neurone, there may need to be a sequence of impulses needed to reach threshold in the post-synaptic neurone to cause a depolarisation
24. **Spatial summation** – when there are multiple pre-synaptic neurones all releasing neurotransmitter to cause reach a depolarisation in the post-synaptic neurone.
25. At a **neuromuscular** junction
    1. Voltage-gated Ca+ ion channels open and diffuse into the pre-synaptic neurone.
    2. This causes vesicles containing neurotransmitter (acetylcholine) to fuse to the pre-synaptic membrane
    3. The neurotransmitter (acetylcholine) binds to acetylcholine receptors on the post-synaptic membrane.
    4. This causes voltage-gated Na+ ion channels to open.
    5. This causes Na+ to diffuse in to the sarcoplasm causing depolarisation.
    6. The depolarisation spreads across the sarcoplasm by T-tubules.
    7. The depolarisation causes the sarcoplasmic reticulum to release Ca2+ ions intothe sarcoplasm.
26. Muscles act in **antagonistic** pairs
27. **Myofibrils** run throughout muscle fibres and are made out up of sarcomeres
    1. Z-disc – is at either end
    2. I-band – is made up of just the actin filaments
    3. A-band – is made up of the actin and the myosin.
    4. H-zone – is the myosin
    5. M-line – is the line in the middle of the sarcomere
28. The sliding filament theory of muscle movement.
    1. Calcium ions are released from the **sarcoplasmic reticulum** in to the **sarcoplasm.**
    2. This binds to **troponin** which moves **tropomyosin** out of the way of myosin binding sites on the actin.
    3. This allows myosin to bind to actin and forms an a**ctin myosin cross-bridge** to form.
    4. Hydrolysis of ATP (attached to the heads of myosin molecules) to ADP allows the cross-bridge to from.
    5. The release of the ADP from the myosin head causes the myosin to change shape and create a power stroke to move the actin and myosin over each other,
    6. ATP then binds to the myosin head causing the crossbridge to detach from actin and return the myosin to the high energy state
29. **Phosphocreatine** (PCr) is broken down by a creatine phosphokinase to creatine, in the process the phosphate is released. The phosphate can combine with ADP to form ATP. This can provide ATP form muscle contraction when ATP is needed quickly.
30. **Slow twitch** muscle fibres
    1. Produces slow contractions
    2. Uses ATP from aerobic respiration
    3. Works for a long time without fatigue
    4. Lots of mitochondria and lots of blood vessels to supply muscles with oxygen.
    5. Myoglobin – a red coloured pigment to store oxygen
31. **Fast twitch** fibres
    1. Quick contractions
    2. Fatigues quickly
    3. Energy is released from anaerobic respiration
    4. They use stores of PCr for quick energy release.
    5. Few mitochondria or blood vessels
    6. No myoglobin – which makes them pale
32. It is important to maintain a stable **temperature**, and **pH** to ensure enzymes can still function at their optimum.
33. It is important to maintain a stable blood **glucose** concentration so it can be uses in respiration
34. It is important to maintain a constant **water** **potential** of the blood, so water doesn’t move in or out of cells by osmosis.
35. Control of blood glucose is controlled by hormones.
36. Glucose is stored as **glycogen** in the liver.
37. **Alpha** cells in the pancreas produce glucagon
38. **Beta** cells in the pancreas produce insulin
    1. Blood glucose too high
       1. Insulin is released and travels in the blood.
       2. The insulin attaches to receptors on the surface of target cells.
       3. This causes more channel protein to be inserted into the surface membrane of target cells.
       4. Glucose is able to move into the target cells
       5. Insulin activates enzymes to convert glucose to glycogen.
    2. Blood glucose is too low
       1. Glucagon is released and travels in the blood.
       2. The glucagon attaches to receptors on the surface of target cells.
       3. Glucagon activates enzyme the converts the glycogen into glucose
       4. Glucagon also activates enzymes to convert glycerol and amino acids to glucose.
    3. The release of adrenaline
       1. Attaches onto the receptors on the surface of target cells
       2. Adrenalin activates enzymes to convert glycogen to glucose.
39. Adrenalin and glucagon activate enzymes be a second messenger system
    1. **Adenyl cyclase** is activated
    2. Adenyl cyclase converts ATP to **cyclic AMP** (cAMP)
    3. **cAMP** activates a **protein kinase**
    4. **protein kinases** then active enzymes to convert **glycogen into glucose.**
40. **Type 1** diabetes is caused by the cells inability to produce insulin
    1. It can be controlled by insulin injections and controlling your diet
    2. It general is occurs from an early age
41. **Type 2** diabetes is caused by cells not responding to insulin
    1. It can be controlled by diet and exercise
    2. A risk factor is obesity
42. The **kidney** filters out waste products like urea from the blood
    1. Blood arrives in the afferent arteriole in the **glomerulus**, high hydrostatic pressure is created forcing small molecules out of the glomerulus
    2. Small molecules like urea glucose amino acids and water move out from the glomerulus though the basement membrane into the Bowman’s capsule. This is called ultrafiltration.
    3. In the **proximal convoluted tubule** reabsorption by active transport of all glucose, all amino acids and most of the mineral ions.
    4. The **descending loop of Henle** is permeable to water and impermeable to ions. Water moves out by osmosis.
    5. At the bottom of the loop of Henle ions move out by diffusion.
    6. The **ascending loop of Henle** is impermeable to water and permeable to ions. Ions move out by diffusion then when equilibrium is reached, they then move out by active transport. This maintains a more negative water potential outside the loop of Henle, so water moves from higher water potential inside the loop of Henle to a lower water potential outside the loop of Henle.
    7. The permeability of the **distal convoluted tubule** and the **collecting duct** depends on the presence of ADH.
    8. The collecting duct takes waste to bladder in the form of urine.
43. If there is **too little water** in the blood
    1. The hypothalamus detects water levels within the blood.
    2. The posterior pituitary releases more ADH.
    3. ADH makes the distal convoluted tubule and the collecting duct more permeable to water by including more aquaporins in the membrane
    4. This means more water moves by osmosis out of the nephron back into the blood
    5. Creating a small volume of concentrated urine.
44. If there is **too much water** in the blood
    1. The hypothalamus detects water levels within the blood.
    2. The posterior pituitary releases less ADH.
    3. ADH makes the distal convoluted tubule and the collecting duct less permeable to water by including fewer aquaporins in the membrane
    4. This means less water moves by osmosis out of the nephron back into the blood
    5. Creating a large volume of dilute urine.