



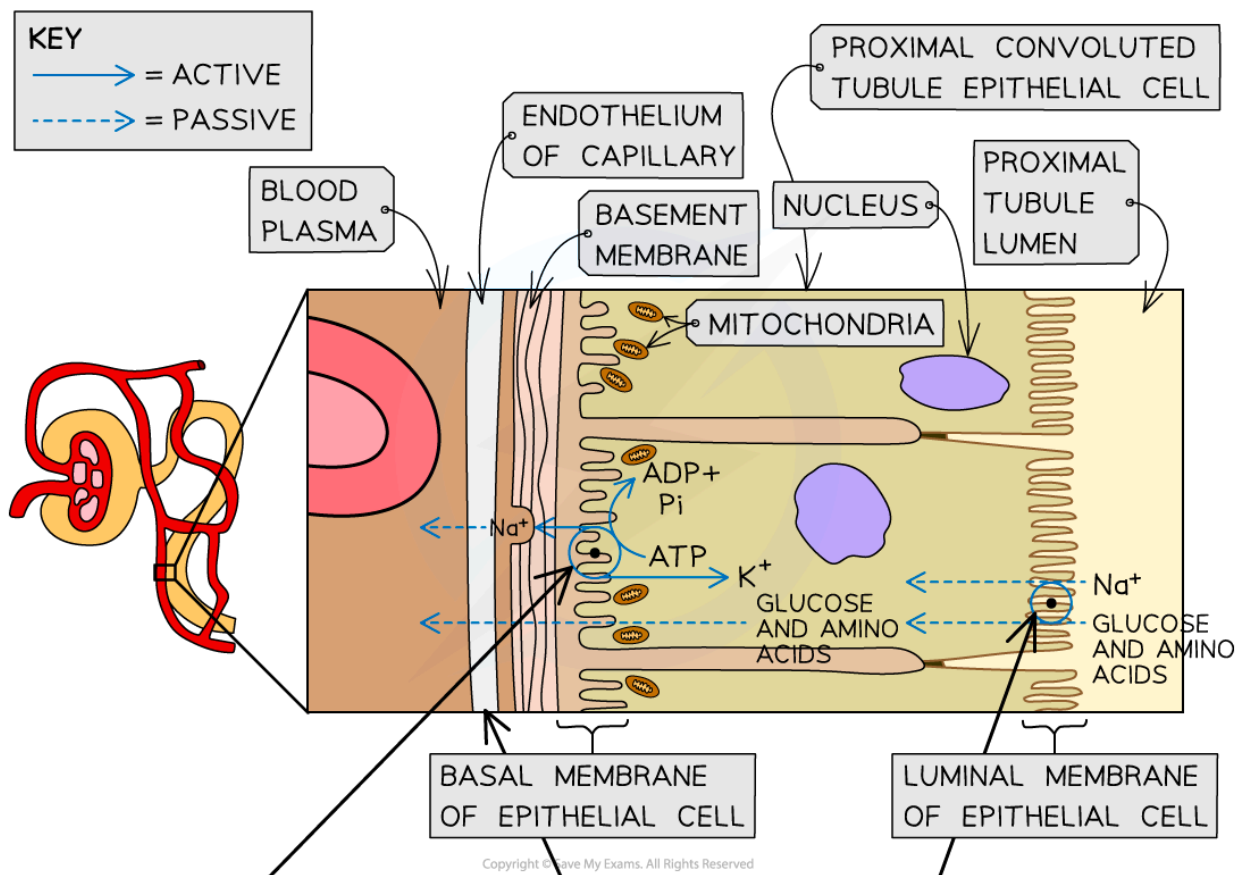
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1. Reabsorption at PCT:

- ❖ $\text{Na}^+ \text{K}^+$ pumps in the basal membrane, pump 3 Na^+ out to the blood and 2 K^+ in the cell
 - Decreasing the concentration of Na^+ in cell.¹
 - So, Na^+ from the lumen enter PCT cells by facilitated diffusion & Secondary active transport through co-transporter proteins in microvilli that allow glucose and amino acids to diffuse along with the Na^+ .²
 - Glucose concentration in the cell increases & it diffuses into the blood by facilitated diffusion through GLUT proteins in the basal membrane.³



[Save my exams. homeostasis revision notes.](#)



2. Osmoregulation:

To increase the Ψ water potential of blood

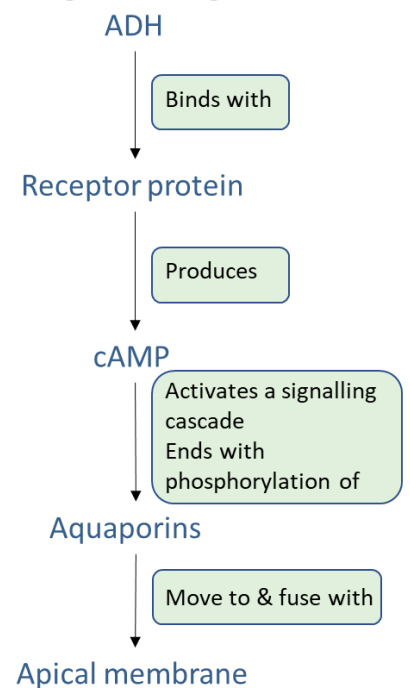
- ❖ Osmo-receptors in the hypothalamus shrink detecting the decrease in Ψ of blood. They send nerve impulses along neurosecretory cells of the posterior pituitary gland, stimulating it to release ADH in blood.

□ ADH then goes to the kidney, its target organ, and specifically the collecting duct cells' basal membranes.

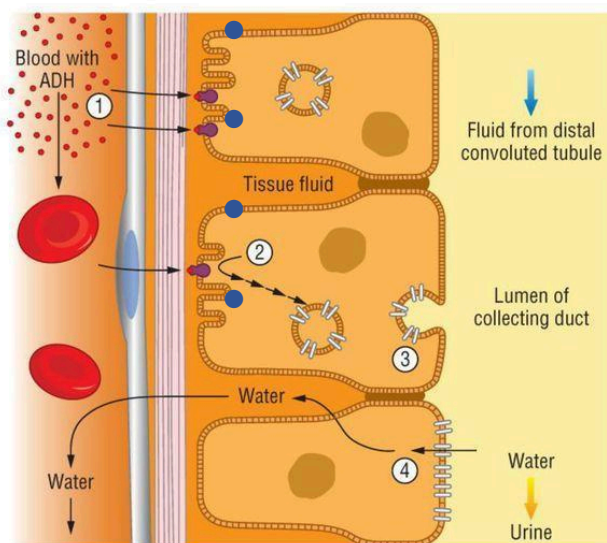
- It binds to receptors on it which are complimentary in shape.¹
Starting in a signalling cascade
- that ends with the production of active phosphorylase enzyme that activates aquaporin molecules,² causing the vesicles to move to and fuse with the luminal membrane.³
- With the increase of aquaporins in the luminal membrane, its permeability to water increases.
- More water moves by osmosis down a Ψ gradient into the cell⁴ then into the blood. As a result, urine concentration increases and its volume decreases

↓ Ψ : No ADH, aquaporins move back into the cytoplasm. Membrane permeability to water decreases. Urine concentration decreases, and its volume increases

The signalling cascade goes as follows:



[slideplayer.14979361](#)



1. ADH detected by cell surface receptors
2. Enzyme-controlled reactions
3. Vesicles containing water-permeable channels (aquaporins) fuse to membrane
4. More water can be reabsorbed

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3. Insulin:

How insulin decreases the blood glucose concentration:

Insulin is secreted by **β cells** of islets of Langerhans when they detect an increase in b.g.c.

Insulin goes to the muscles through the blood, where it binds to specific receptors on the sarcolemma initiating an enzyme cascade.

Which end up stimulating vesicles containing **GLUT4** in their membrane to move to & fuse with the cell surface membrane, increasing its permeability to glucose, allowing it to enter by facilitated diffusion.

- Insulin also stimulates **glucokinase** which is the phosphorylation of glucose to trap it into the cells.
- It inhibits **glycogenolysis**.
- It stimulates *glycogen synthase* to combine glucose molecules by glycosidic bonds, this is called **glycogenesis**.



4. Glucagon:

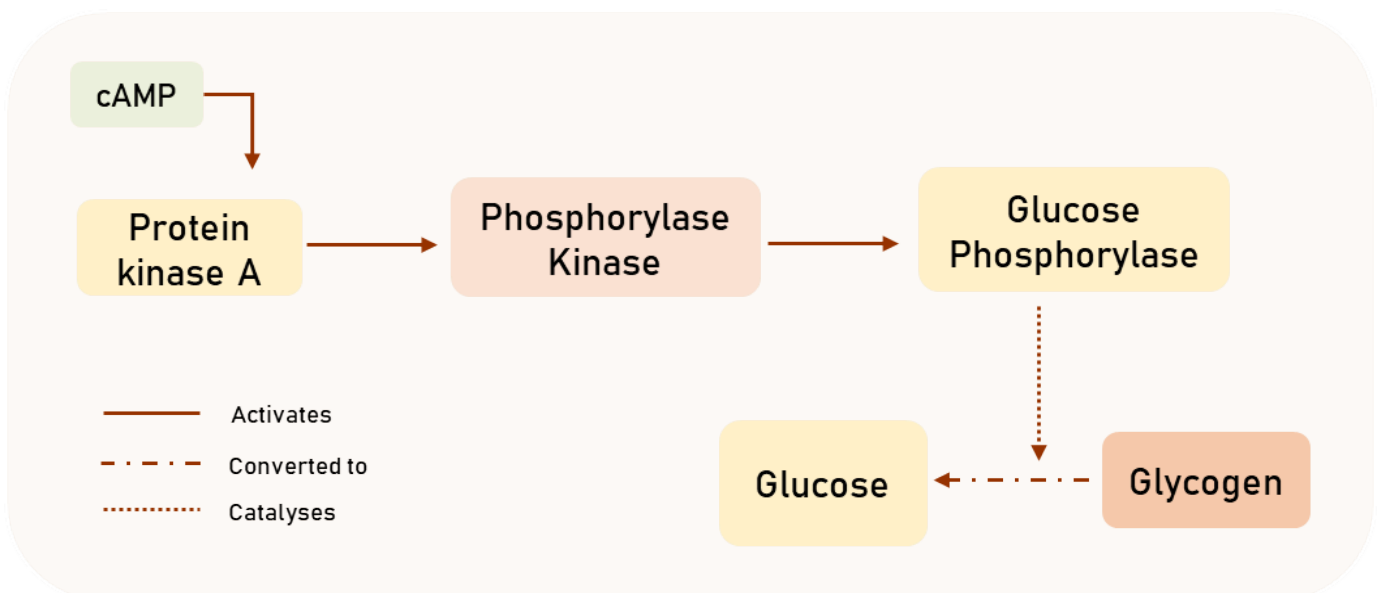
How glucagon increases the blood glucose concentration:

Glucagon is secreted by α cells of islets of Langerhans when they detect a decrease in b.g.c,

Glucagon goes to liver cells through the blood and binds to receptors on their cell surface membranes.

- The receptor goes through a conformational change that activates a G-protein which in turn activates a cell membrane protein, adenylyl cyclase to produce cAMP, a second messenger, from ATP.
- cAMP binds to and activates protein kinase A.
- The protein kinase A enzymes then activate phosphorylase kinase enzymes by adding a phosphate group to it.
- Phosphorylase kinase enzymes activate glycogen phosphorylase by adding phosphate groups to them.
- Which breaks down glycogen into glucose, this process is called glycogenolysis. Glucose concentration increases and diffuses out the GLUT 4 protein.

– Glucagon also stimulates gluconeogenesis.



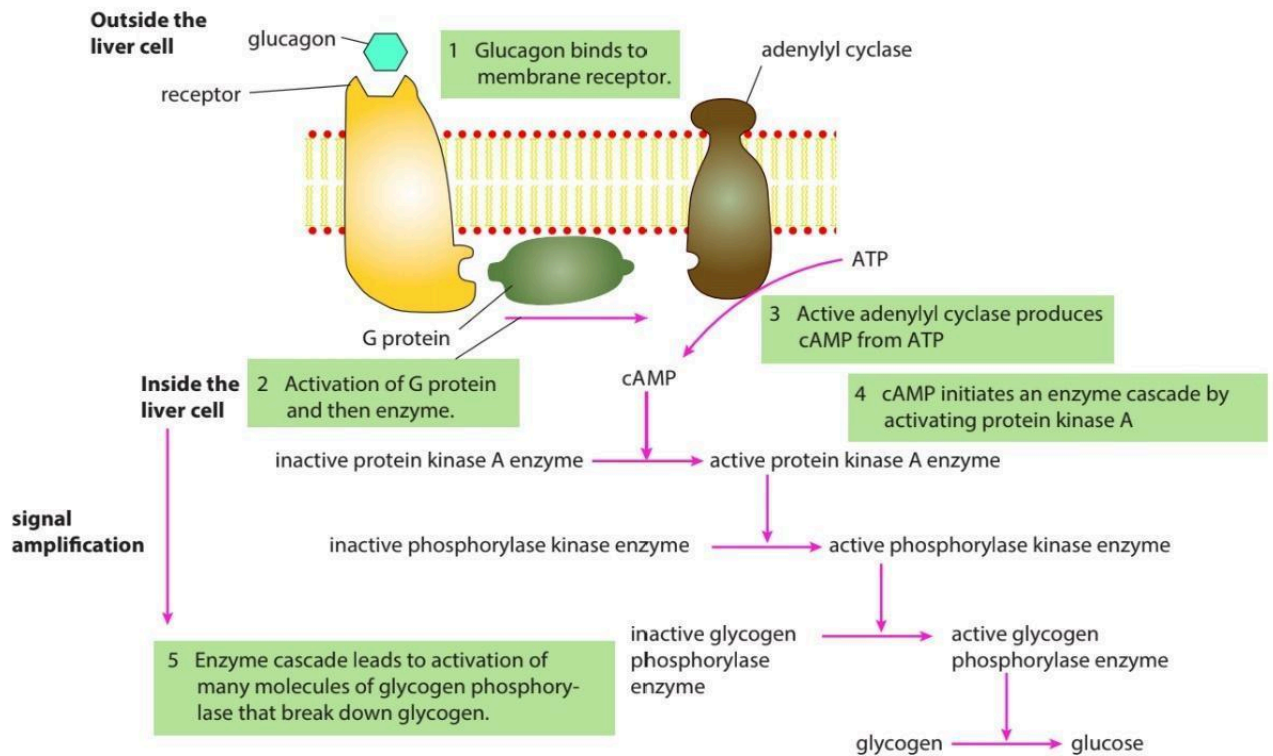


Figure 4, Textbook's page 367

5. How stomata open:

H^+ pumps in the cell surface membrane of guard cells actively pump H^+ out of the cells.

This decreases the H^+ concentration in the cell, which stimulates K^+ channels to open.

K^+ moves in by facilitated diffusion down an electrochemical gradient, decreasing the Ψ and so water moves in by osmosis down a Ψ gradient.

As a result, the volume of the cell increases and it becomes turgid. The thin outer wall expands the most, opening the stomata.

To further decrease the Ψ during opening of stomata and to maintain the electrical balance, Starch stored in chloroplasts is broken down to the negatively charged malate ions, which then enter the vacuoles.



6. How stomata close:

- H^+ pumps stop pumping H^+ out of the cells.
- K^+ leave the guard cells and move to neighbouring cells.
- Malate ions are moved back to the chloroplasts and converted to starch.
- Ψ becomes higher in guard cells than neighbouring ones.
- So, water leaves the guard cells by osmosis, down a Ψ gradient. The guard cells then become flaccid and close the stoma.

7. ABA and stomatal closure during stress:

ABA binds to specific receptors on the cell surface membrane of guard cells.

→ Inhibits H^+ pumps and stimulates the opening of Ca^{2+} channels.

- Ca^{2+} enter the guard cells and act as second messengers, activate negatively charged channel proteins, allowing negative ions to leave the cell.
- This stimulates K^+ channels to open and K^+ leave.
- Ψ increases and water leaves by osmosis.
- Malate ions are also converted back into starch.

Ca^{2+} stimulates the opening of K^+ channels (that allow K^+ **out** of the cell)
And the closing of K^+ channels (that allow K^+ **into** the cell).

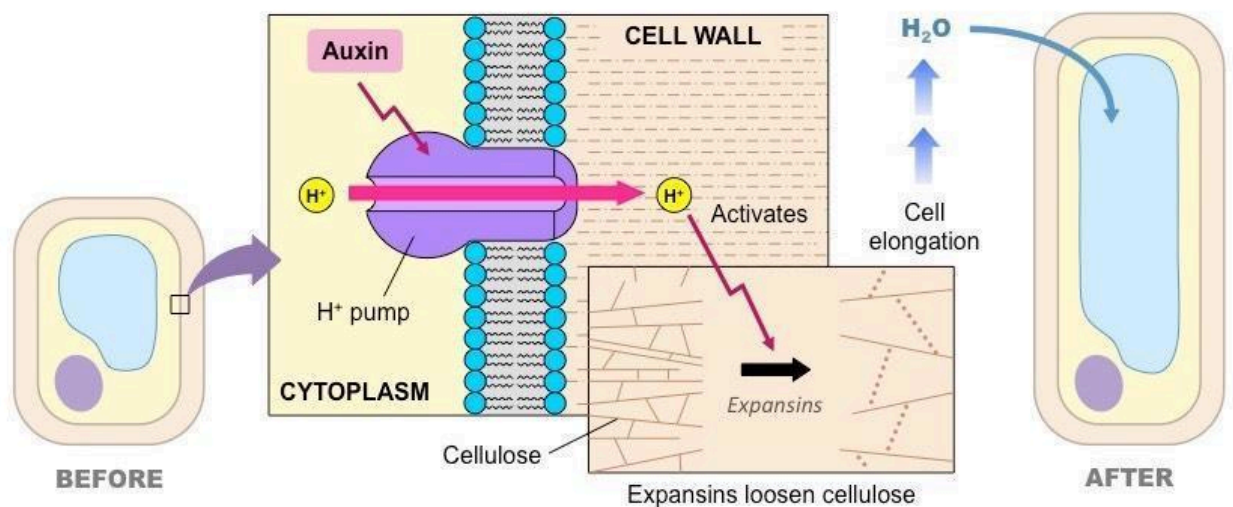


8. Auxins:

Growth by cell elongation / Acid growth

Auxins are synthesised in growing tips/meristems, they work by:

- Binding to receptors
- Stimulating ATPase pumps to pump H^+ into cell walls
- The decrease in cell wall pH activates expansin proteins
- Expansins disrupt hydrogen bonds between cellulose microfibrils & surrounding substances i.e., hemicelluloses.
- Cells absorb water by osmosis
- Cell walls stretch and cell elongates, due to the increase in pressure.



[bioninja auxin](#)



9. Gibberellins:

I. Production of Gibberellin

The gibberellin is produced in an active form (GA_1) by a certain enzyme which is coded by the *Le* gene, the gene which determines the height of a plant.

An allele of this gene is the recessive *le* allele. The mutation of this allele is in 1 nucleotide, which ends up substituting the amino acid Threonine by Alanine, producing a non-functional enzyme.

Thus, no active gibberellin produced, resulting in a dwarf plant, homozygous for *le/le*. But they could be stimulated to increase in height by applying active gibberellin to them.

II. Gibberellin in stem elongation

Gibberellin stimulates enzymes called XET in stem cells' walls.

These XET enzymes break certain bonds between hemicellulose molecules, so that cellulose microfibrils move further apart allowing the cell to expand when water moves in by osmosis.

Refer back to Acid growth and expansin proteins

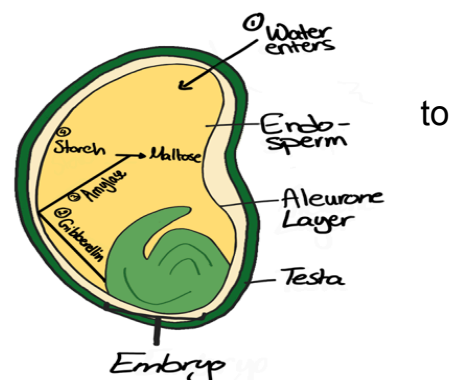
****Note: The DELLA and PIF protein are not only present and prevent the transcription of the Amylase enzyme during seed germination, But also prevent the transcription of other Enzymes/Proteins as a response to Gibberellin, Such as in the stem elongation process.**

'The primary role of DELLA proteins in vascular plants is to repress responses to a group of structurally related plant hormones, gibberellins (GAs), which promote many major developmental responses in plants, such as germination, stem elongation, leaf expansion, and flowering'

For more context check W21_42_Q10_B

III. Gibberellin in seed germination

Absorption of water¹ stimulates the embryo produce gibberellin.



Gibberellin then diffuses to the aleurone layer² and stimulates it to synthesise amylase³, by:

1. binding to receptors on cells' surface membranes.
2. this stimulates an enzyme to destruct DELLA (repressor protein)
 - DELLA prevents PIF (transcription factor) from bonding with promoter on DNA.
3. PIF can now bond with promoter and start transcription.
4. RNA polymerase then starts transcription of the amylase gene.
5. Amylase is then produced.

Starch present in the endosperm is broken down into maltose, by amylase enzyme.⁴

The maltose is then broken down into glucose and used in respiration. To provide energy for growth of the radicle and plumule.

10. Rice:

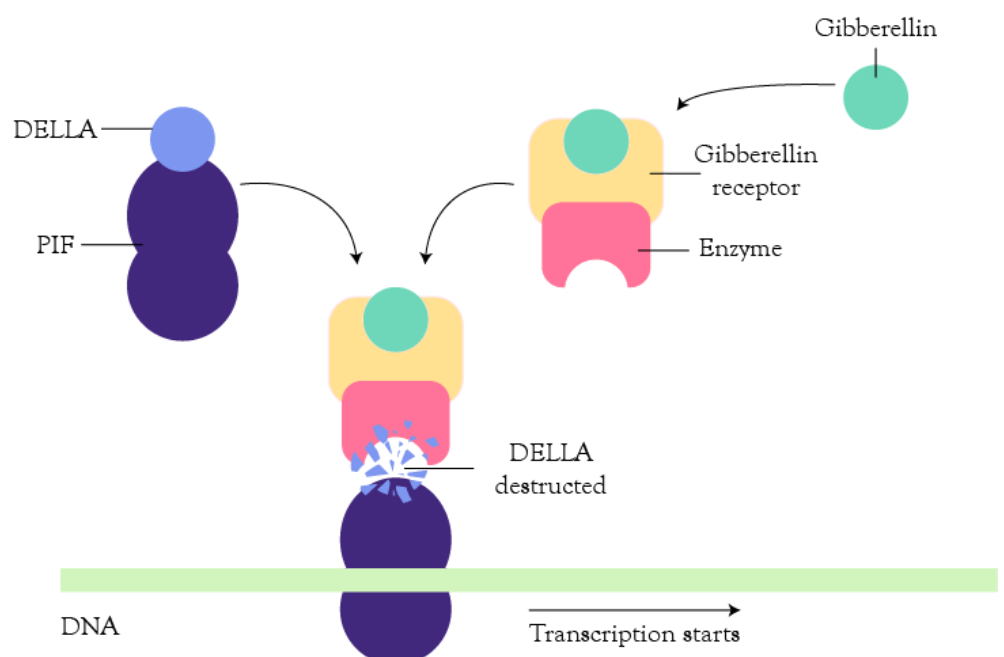
Adaptation to flooding

- Rice grows taller by **gibberellin**, which stimulates cell elongation, as a response to flood. Stimulating leaves and flowers to grow above water level.
- So that the leaves end up above water and be able to **photosynthesize** and carry **aerobic**

respiration.

- Leaves under water have hydrophobic surfaces which **traps air film** on the leaves' surfaces.

- Rice stems and roots also have **aerenchyma** cells, and this allows gases, such as



CO₂ for photosynthesis and O₂ for respiration, to diffuse to the submerged parts/ Shallow roots.

- Cells in submerged roots can also respire by ethanol fermentation when O₂ is not enough. This is possible due to them having **high ethanol tolerance**, as they have high concentrations of **ethanol dehydrogenase**.

11. Venus Flytrap:

If the sensory hairs are deflected at least twice within a 20-35 seconds range, this stimulates Ca^{2+} channels at the base of the hair to open and Ca^{2+} moves in, which in turn stimulates Cl^- channels that allow Cl^- out of the cell to open.

This is how depolarisation occurs in the venus flytrap.

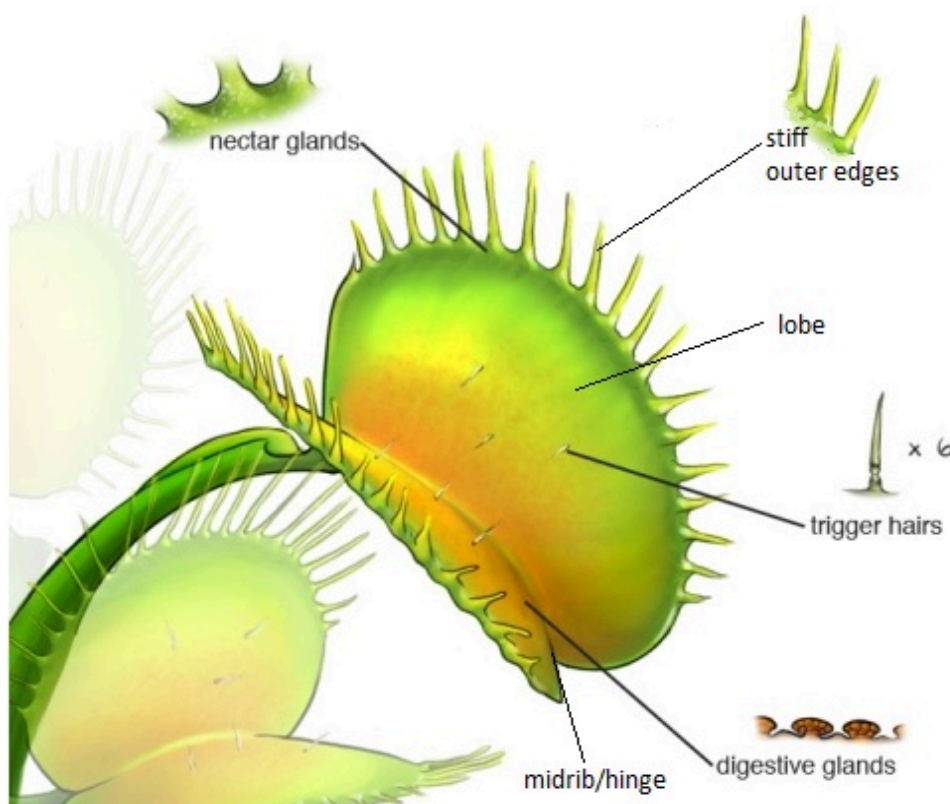
Now the depolarisation starts an action potential throughout the lobes till it reaches the hinge cells at the midrib, by travelling along the cell membranes or through the plasmodesmata from cell to cell.

At the hinge cells, [Acid growth](#) occurs due to the action potential, and thus the lobes change shape from convex to concave as the elastic tension (which kept them open) releases, closing the lobes.

After closing and trapping the insect, the venus flytrap secretes digestive enzymes onto it to digest it. By the entrance of into gland cells which stimulates the exocytosis of vesicles with the enzymes.

Two adaptations of the flytrap's to conserve energy and not close unnecessarily are:

1. The moving of one sensory hair does not trigger closure, a hair has to be moved twice or two hairs moved within a 20-35 seconds range.
2. There are large gaps between the stiff hairs to allow small insects to escape, as they would not provide much energy.



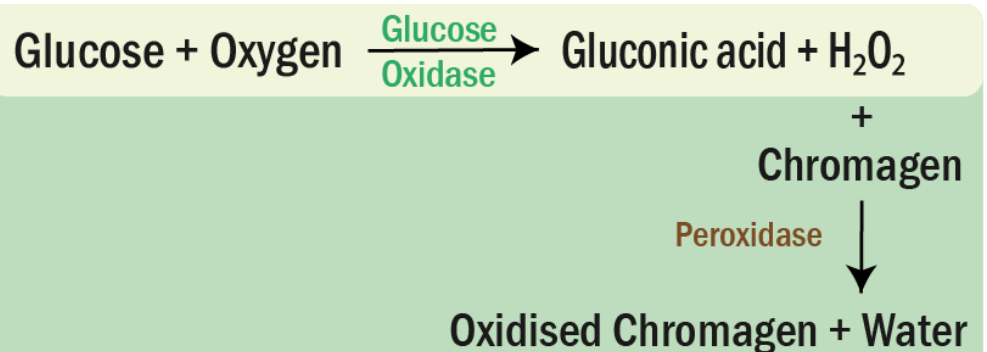
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12. Biosensors:

Biosensors measure the b.g.c using immobilised **glucose oxidase** on a recognition layer by:

- Blood on the strip is inserted into the biosensor.
- Glucose in the blood plasma pass through the membrane and binds to the active site of the **glucose oxidase**.
- where it is then catalysed into **Gluconic acid** and **H₂O₂**.
- The H₂O₂ is oxidised at an electrode where the electron transfer is detected.
- **The electron flow is proportional to the b.g.c.**
- The biosensor **amplifies** this current and it is then read by a meter to produce a **digital reading** for b.g.c.

Biosensor:
Dipstick:



**Only H₂O₂ reacts with chromagen in the dipstick

13. Dipsticks:

- Dipsticks detect the b.g.c using both immobilised **glucose oxidase** and **peroxidase** on a small pad at the end of the stick.
- The pad is covered by a cellulose membrane which only allows small molecules to pass through.

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- The pad is immersed in urine and the same process in biosensor repeats.
- **Peroxidase** then catalyses the reaction between H_2O_2 and **chromagen** (a colourless chemical).
- Producing **oxidised chromagen** (a coloured chemical).
- The stick is then compared with a colour chart.
- The more glucose there is the darker the colour.

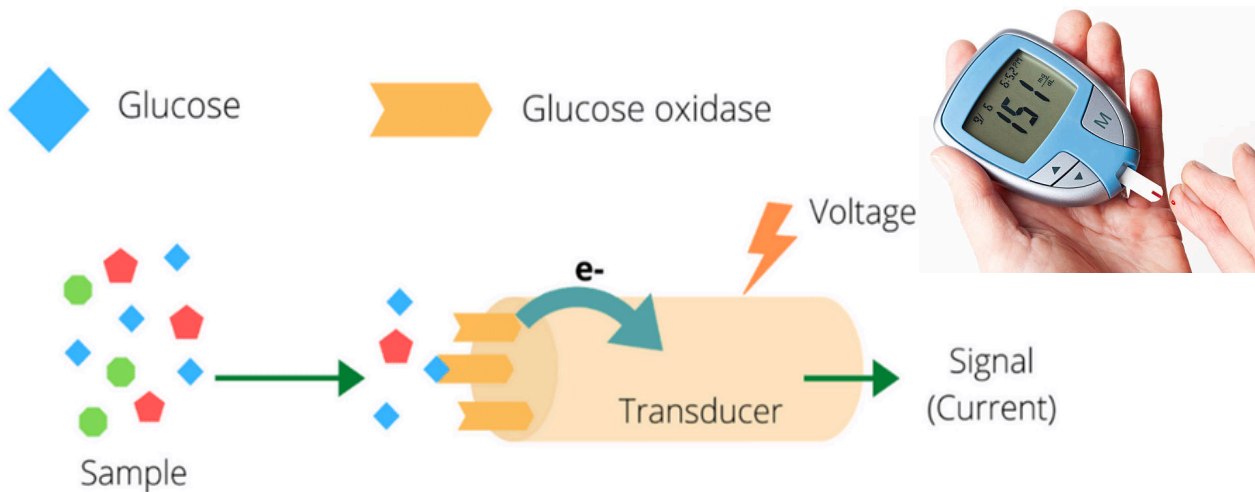
Biosensor VS Dipstick

Advantages	
Biosensor	Dipstick
Immediate	Non invasive
Precise/Quantitative	Painless
Shows current b.g.c	Less risk of infection
Re-usable	Easy to Use
	Cheap



The dipstick colour chart

How the biosensor works:



What's important is that I tried.....

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Diseases

1. Sickle cell anaemia: (Recessive)

A mutation occurs in the HBB gene for the β -globin polypeptide
Where the codon **CTT** is replaced to **CAT**, **CTT** codes for the hydrophilic **Gluconic acid**, while **CAT** codes for the hydrophobic **Valine**.

CTT	→	CAT
Glutamic acid	→	Valine
Hydrophilic	→	Hydrophobic

- Haemoglobin then becomes **less soluble** when in **low** partial pressure of O_2 .
- Which makes the molecules tend to stick together.
- Forming **long fibres** inside the RBC pulling it out of shape into a **sickle** shape.
- The sickled RBCs **cannot transport O_2** and tend to get **stuck in capillaries**.
- Which in turn causes **severe anaemia**, As normal RBCs cannot move through.

2. Haemophilia: (Recessive sex-linked)

- **F8 gene** which codes for the coagulation **factor VIII** in liver cells, secreted into blood plasma where it's needed for blood clotting.
- The F8 gene is found on the non-homologous region of **X chromosome**.
- Mutation in the gene causes **faulty** or **No** Factor VIII.
- So blood clotting is **reduced/prevented**.
- Which results in **excessive bleeding**, bleeding into joints, large bruises and/or internal bleeding.

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3. Huntington's: (Dominant)

HTT gene → Huntingtin protein

Which is important in the development of neurones especially in the brain

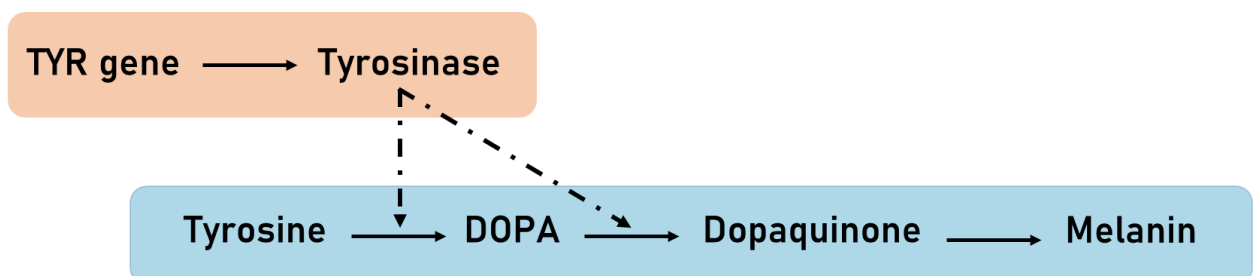
If the codon **CAG** in the gene is repeated over **40 times**, the person has **Huntingtons**. Where development of the neurones is abnormal and brain cells die more rapidly.

If it is repeated between **36-39**, the disease may develop gradually as the person grows older.

Symptoms include:

- Loss of ability to control movements and walk/Involuntary movements.
- Loss of ability to talk or think clearly/mental deterioration.
- Mood swings.
- Death within 15-20 years after the first appearance of symptoms.

4. Albinism: (Recessive)



1. A base substitution OR deletion/insertion → frameshift. happen in the TYR gene, resulting in a mutated gene.
2. Primary structure of protein is changed.
3. 3D structure and active site change.
4. **No/Nonfunctional** Tyrosinase enzyme produced.
5. Tyrosine **not** converted to DOPA/Dopaquinone.
6. No production of melanin.

Symptoms/Phenotype:

- White skin and hair.
- Red or pink eyes.
- Weak eyesight.

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- Eye sensitivity to light.
- Jerky eye movements.
- Increased susceptibility to skin cancer and sunburns.

5. Cystic fibrosis: (Recessive)

CFTR gene → CFTR transporter protein

Controls movement of Chloride ions down an electrochemical gradient.

The transport of Cl⁻ helps in altering the water potential.

In a healthy person:

- Ionocytes pump out Cl⁻ to a high conc. outside the cell.
- Decreasing the Ψ , so water moves outside thru the aquaporins
- making the thick and sticky mucus much thinner so that it could be moved by the cilia or by coughing.

So for a person with CF they have repeated lung infections and continuous cough fits as the pathogens build up in the airway.

•

14. Lac Operon:

Lac Z

β -galactosidase. Hydrolyses lactose into glucose and galactose.

Lac Y

Lactose permease. Moves lactose into cells.

Lac A

Transacetylase.

15. Genetic Markers

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Antibiotic Resistance

Gene transfer

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*Method will be illustrated in the next page.

Disadvantage

Risk of creating pathogenic antibiotic resistant bacteria, As the resistance can be transferred by vertical or horizontal transmission.

3 types of bacteria are needed to be differentiated between:

1. Bacteria that did not take the plasmid
2. Bacteria that took the plasmid **without** GOI
3. Bacteria that took the plasmid **with** GOI

Bacteria 1 will have no resistance

Bacteria 2 will be resistant to both antibiotics A and B

Bacteria 3 will be resistant to antibiotic A only as GOI inactivates antibiotic B's resistance gene

GFP protein

Gene for an enzyme that produces **GFP protein** is inserted, using a plasmid.

Fluoresces bright green under UV light.

Disadvantage

Only a few molecules produced so may not fluoresce very brightly.

GUS Enzyme

Gene for **GUS enzyme**, taken from E. coli is inserted, using a plasmid.

Turns colourless or non fluorescent substances into coloured or fluorescent.

Advantage

Produces more fluorescent substances as it's an enzyme and can be reused.

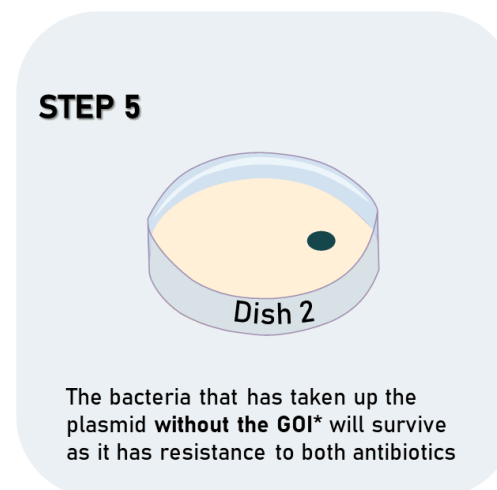
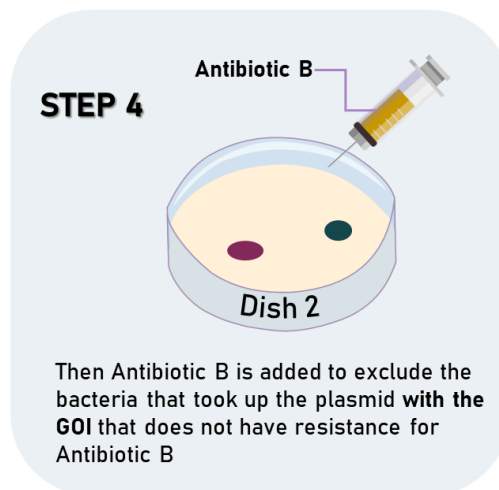
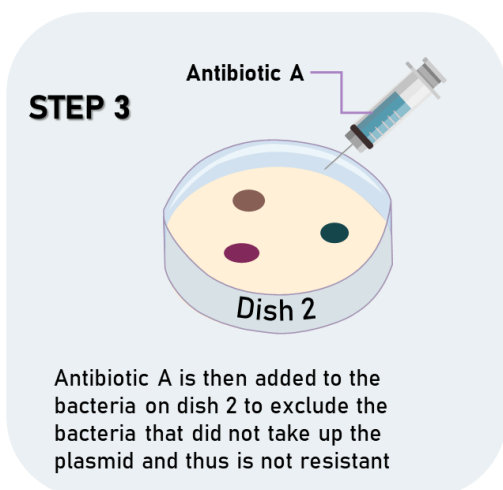
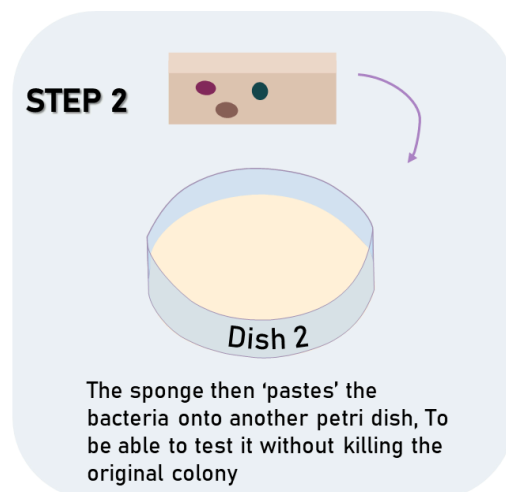
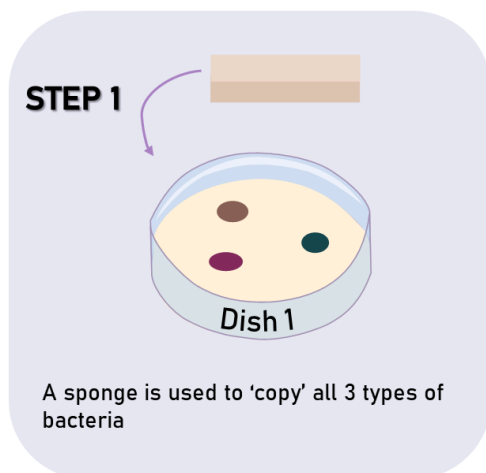
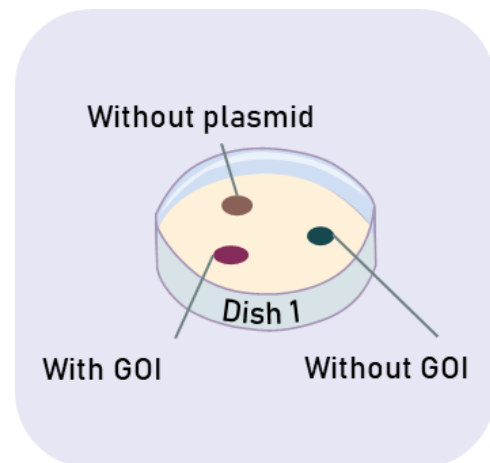
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This bit might be a bit confusing, if it does not make sense to you, try this [video](#) out

First work on excluding the non resistant **Bacteria 1** in step 3

Then find out which bacteria took up the plasmid with the GOI by exposing it to antibiotic B as it has no resistance to it.

We can then go back to the original dish and culture it



*GOI: gene of interest

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