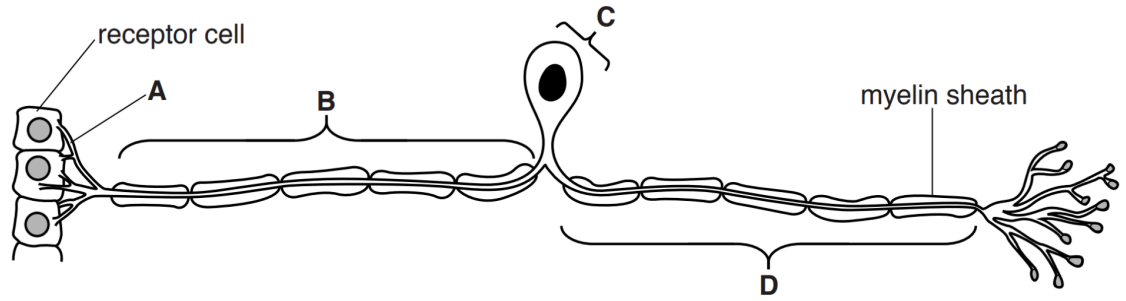
15.1 Control and Coordination in Mammals

Neurones

	Sensory neurone	Motor neurone
Structure	Cell body in the middle of neurone/axon.	Cell body at end of neurone/axon.
	Cell body in dorsal root ganglion.	Cell body in CNS/brain/spinal cord.
	Short axon OR axon and dendron.	Long axon OR only axon.
	Dendrites attached to dendron.	Dendrites attached to cell body.
Function	Transmits impulses/action potentials from receptors/sense organs	Transmits impulses/action potentials from CNS/spinal cord OR relay/intermediate neurone OR sensory neurone
	to relay/intermediate neurone OR CNS/spinal cord OR motor neurone.	to effector/muscle/gland

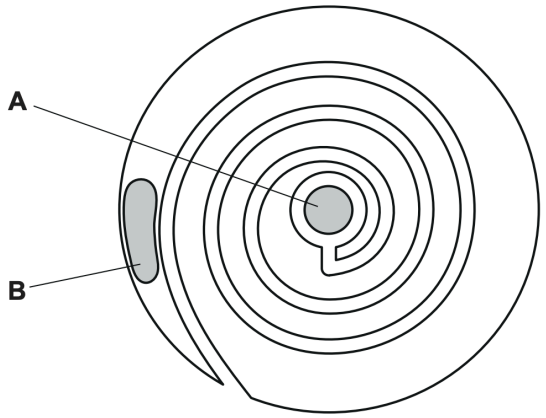
Sensory neurone



A	dendrite
B	dendron / sensory axon
C	cell body / soma
D	axon / terminal axon

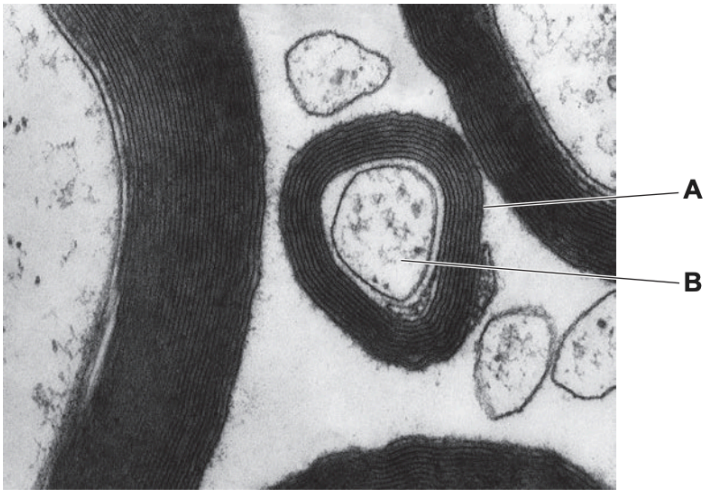
Myelinated neurones

Diagram of cross section through myelinated neurone



A	Axon / axoplasm
B	Schwann cell nucleus

Electron micrograph showing a cross-section of a myelinated neurone



A	Schwann cell / myelin sheath
B	Axon / axoplasm

Reflex Arc

Features of a reflex arc

- Fast
- Automatic / involuntary / no conscious thought / no brain involvement.
- Response is always the same / stereotypic.

Sensory Receptor Cells

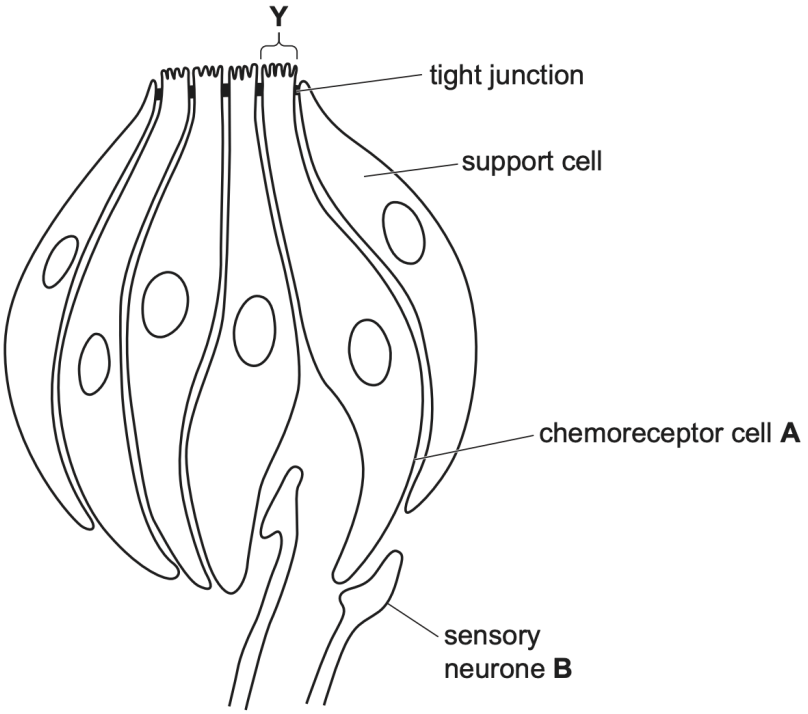
Outline the roles of sensory receptor cells in the mammalian nervous system.

- Detect / respond to changes in stimuli.
- Examples of stimuli: light / sound / heat / touch / pressure / pain / chemicals / taste / smell / tension.
- Act as transducers: convert stimulus energy to electrical energy.
- Produce action potentials.
- Passes impulses to/along sensory neurones.

Sequence of Events Resulting in an Action Potential

Sequence of events resulting in action potentials in sensory neurones

Diagram of chemoreceptor cells in taste buds



Y = microvilli

Describe the function of microvilli in chemoreceptor cells

- They increase surface area
- So more sodium channels are present / so more Na+ enters.

Suggest a reason for the tight junctions between the chemoreceptor cells.

To prevent movement of chemicals / substances / ions between the chemoreceptor cells, to ensure the chemicals pass through the cells rather than between them.

Chemoreceptor cell A responds to sodium ions in salt. Describe how the contact of cell A with Na+ can result in an action potential in sensory neurone B.

- Na+ ions enter chemoreceptor cell A through microvilli
- Cell surface membrane is depolarised
- Receptor potential is generated
- If receptor potential reaches threshold potential
- Voltage-gated Ca2+ channels open
- Ca2+ ions enter cytoplasm / cell / presynaptic knob
- Vesicles of neurotransmitter move towards / fuse with cell surface membrane / presynaptic membrane
- Exocytosis occurs: secretion of neurotransmitter into synaptic cleft
- Neurotransmitter binds to receptor on postsynaptic membrane / sensory neurone B
- Sodium channels open / Na+ enter sensory neurone B
- Postsynaptic membrane / sensory neurone B membrane is depolarised
- Action potential is generated in sensory neurone B when threshold is reached

When a sugar molecule binds to a receptor protein on the cell surface membrane of cell A, calcium ions are released into the cytoplasm of the cell by the endoplasmic reticulum. Explain how the release of calcium ions will lead to an action potential being generated in sensory neurone B.

- Vesicles of neurotransmitter are stimulated to move
- Vesicles fuse with cell surface membrane / presynaptic membrane
- Exocytosis occurs: secretion of neurotransmitter
- Neurotransmitter diffuses across synaptic cleft / gap between cells A and B
- Neurotransmitter binds to receptors
- On postsynaptic membrane / sensory neurone B
- Sodium channels open / Na+ enter sensory neurone B
- Postsynaptic membrane / sensory neurone B membrane is depolarised
- Action potential is generated in sensory neurone B when threshold potential is reached

Describe how olfactory receptor cells in the nasal cavity respond to chemicals to generate an action potential.

- Chemicals stimulate/ enter/ bind to olfactory receptor cells.
- Na^+ channels open and Na^+ enters the receptor cells via microvilli.
- Membrane is depolarised due to influx of Na^+.
- If receptor potential reaches threshold potential, action potential is generated.
- Voltage-gated Ca^{2+} channels open; Ca^{2+} ions enter cytoplasm of receptor cell.
- Vesicles of neurotransmitter move towards/ fuse with presynaptic membrane.
- Exocytosis of neurotransmitter into synaptic cleft.

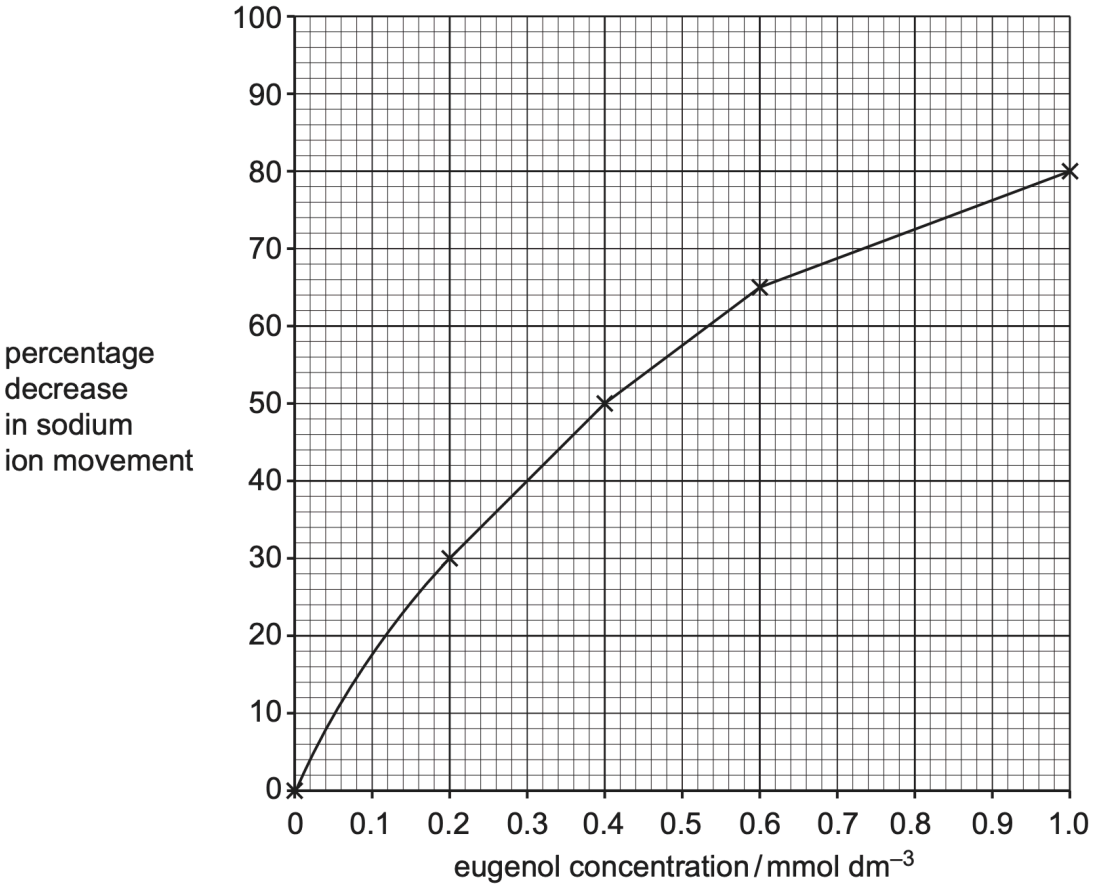
- Neurotransmitter binds to receptors on postsynaptic membrane, allowing signal to be transmitted to the brain.
- Olfactory receptor cell acts as a transducer: converts chemical stimulus to electrical impulse.

Movement of ions

Ion	Direction of movement of ion	Action resulting from movement of ion
Na^+	Into presynaptic knob	Depolarisation of presynaptic membrane
Ca^{2+}	Into presynaptic knob	Exocytosis of acetylcholine
Ca^{2+}	From sarcoplasmic reticulum to cytoplasm of muscle fibre	Binds to troponin and changes its shape / movement of tropomyosin / exposes actin-myosin binding site on actin / myosin head binds to actin / allows power stroke / sarcomere contacts

Graph analysis question

Effect of eugenol concentration on percentage decrease in sodium ion movement:



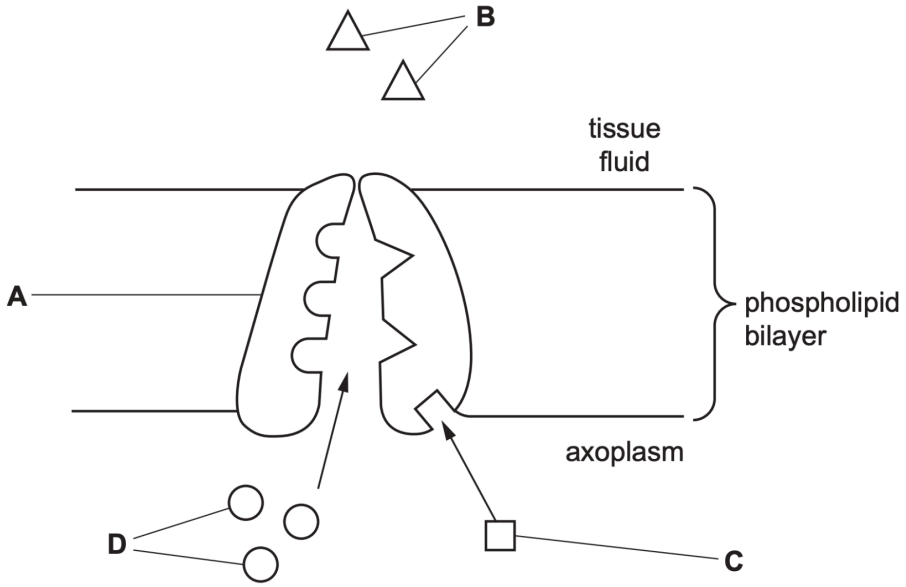
Eugenol is used to relieve toothache. Explain how action of eugenol can relieve pain.

- Eugenol prevents opening of Na^+ channels in membrane of sensory neurones.
- This causes reduced entry of Na^+ into sensory neurone.
- Thus, no / reduced depolarisation of sensory neurone <u>membrane</u> occurs.
- Receptor potential fails to reach threshold potential.
- Thus, no / fewer action potentials / impulses are generated and transmitted along sensory neurone.
- Action potentials / impulses don't reach the brain, so sensation of pain is reduced or blocked.
- Eugenol may affect sodium-potassium pump.
- Which results in resting potential not being restored.

Transmission of Nerve Impulses

Resting potentials

Diagram of part of a neurone membrane at resting potential



A	sodium potassium pump
B	potassium ions
D	sodium ions

Action potentials

Explain what is meant by a voltage-gated channel

- Controls transport of specific ions across cell membrane.
- Each voltage-gated channel is specific to a particular ion.

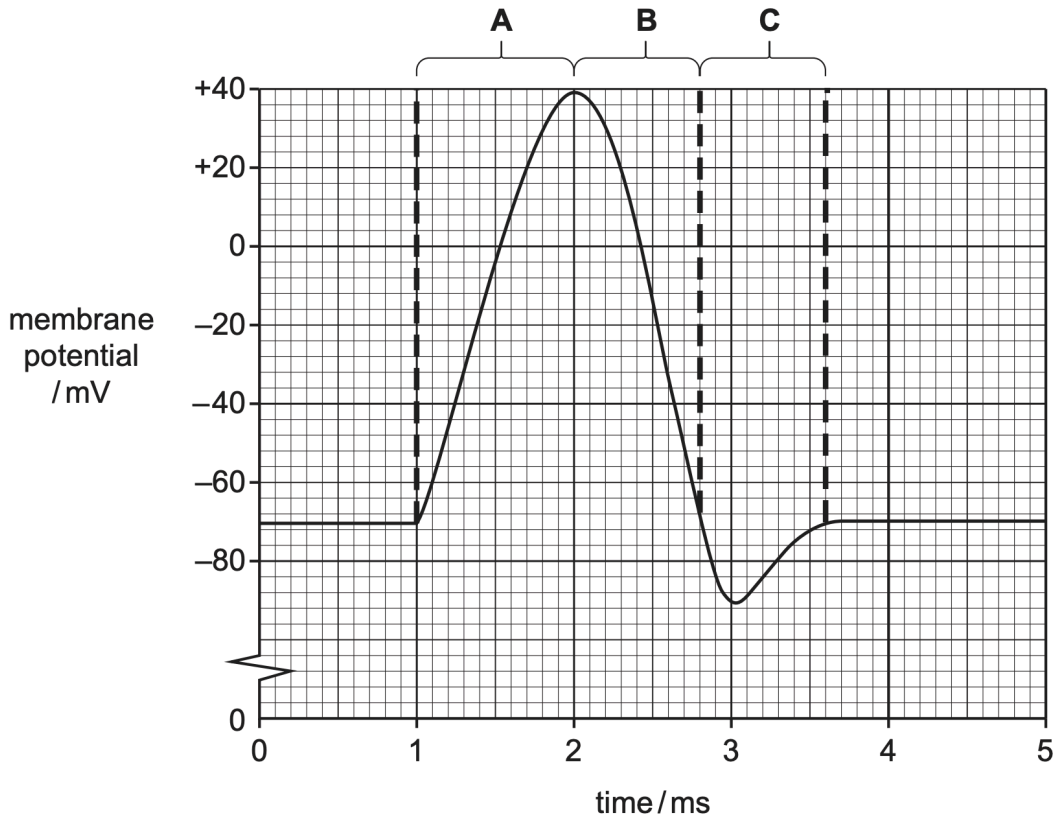
- Opens / closes when when voltage / charge / electrical potential / depolarisation changes.
- Is a transmembrane protein.
- Contains a hydrophilic pore through which ions can pass.

Suggest and explain why preventing the free movement of mitochondria within the cytoplasm of the axon affects the transmission of action potentials along the axon membrane.

- no/less ATP is produced for the sodium-potassium pump / active transport of Na+ and K+
- This reduces / prevents re-establishment of resting potential
- This reduces transmission of action potential along the axon

Action potential graphs

Graph of action potential in mammalian neurone:



Suggest why no further action potential can occur during A and B.

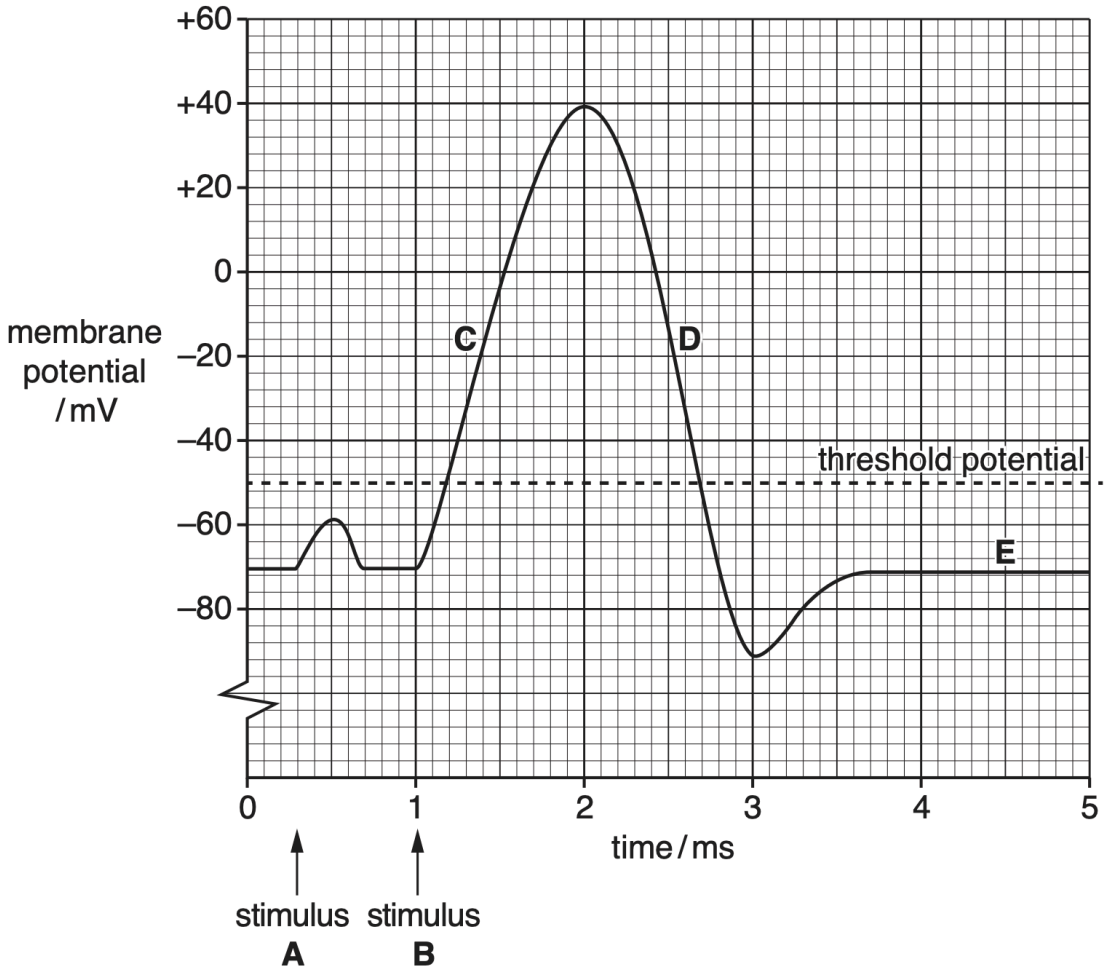
- During A (any one)
    - Na^+ channels already open
    - Na^+ has already entered neurone
    - No more Na^+ channels to open
    - Less Na^+ outside to diffuse in
    - Less steep Na^+ concentration gradient.

- During B (any one)
    - Sodium channels are inactive/ unresponsive
    - Potassium channels are open
    - Membrane is impermeable/ less permeable to Na^+
    - Membrane is more permeable to K^+

Suggest why it is difficult for a further action potential to occur during C.

- Harder to reach threshold
- Potassium channels are still open
- Sodium-potassium pumps need to restore the resting potential
- <u>Hyperpolarisation</u> at C.

Changes in potential difference (p.d.) across the membrane of a receptor cell over a period of time. The membrane was stimulated at time A and at time B with stimuli of different intensities:



The Na+ / K+ pump is operating	C or D AND E
The voltage-gated Na+ channels are open	C

The voltage-gated K+ channels are open	D

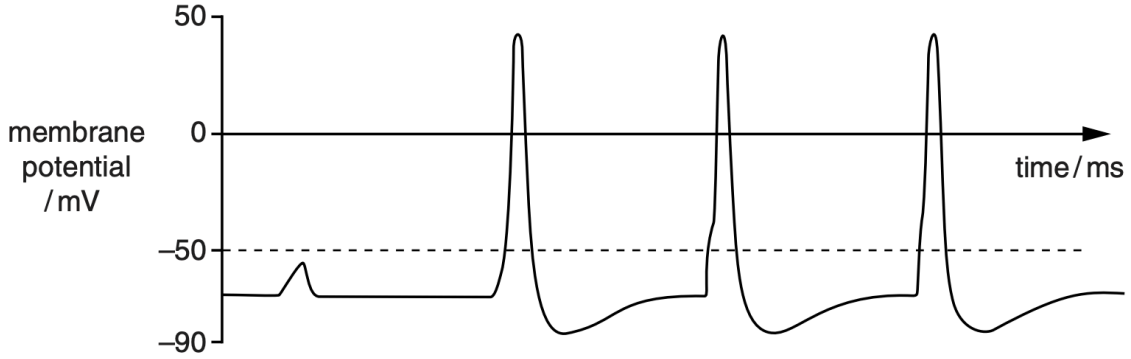
Explain why stimulus A did not result in action potential but stimulus B did

- Both stimuli caused <u>generator / receptor potential</u>
- For stimulus A, generator potential did not reach / exceed / cross the <u>threshold potential</u>

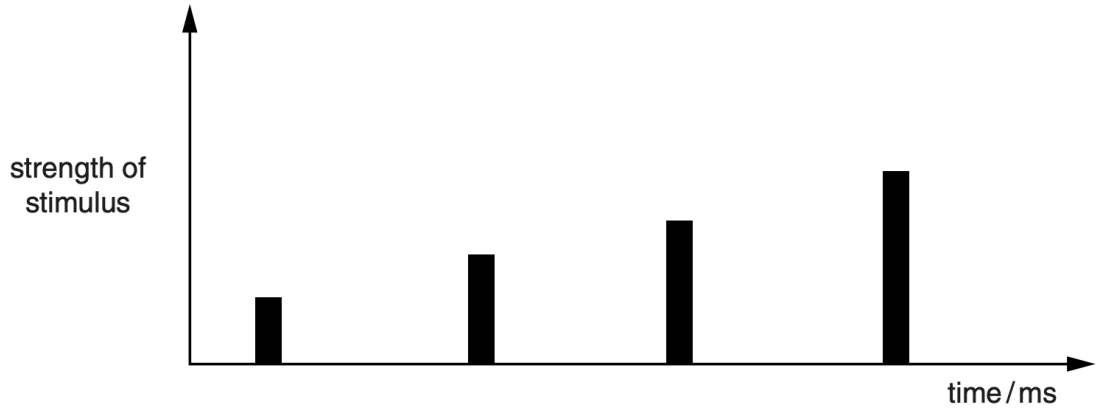
C is required to make A function. Name C.

- ATP

Changes in membrane potential of a sensory neurone when receptor cells are stimulated:



Strength of the stimuli applied to these receptor cells:



With reference to these figures, describe the relationship between the strength of the stimulus and the resulting action potential.

- only stimulus / depolarisation / receptor potential that reaches threshold produces an action potential: –50mV for threshold.
- the action potential is the same size no matter how strong the stimulus.

Speed of Conduction of Impulses

Outline the importance of myelin sheath in transmission of nerve impulses

- Insulates axon/ prevents movement of ions.
- Increases speed of impulses.
- Enables saltatory conduction.

Explain the importance of myelin sheath in transmission of nerve impulses // Explain how the myelin sheath increases the speed of conduction of nerve impulses

- It insulates axon; ions cannot pass through it.
- Depolarisation / action potentials can only occur at nodes of Ranvier.
- Long local circuits/currents are set up between nodes.
- Saltatory conduction occurs: action potentials 'jump' from node to node.
- Transmission / conduction is much faster.

Explain the fast transmission of impulses along a motor neurone

- Schwann cells wrap around the axon.
- To form myelin sheath; the axon is myelinated.
- It insulates axon; ions cannot pass through it.
- Depolarisation / action potentials can only occur at nodes of Ranvier.
- Long local circuits/currents are set up between nodes.
- Saltatory conduction occurs: action potentials 'jump' from node to node.

Describe how action potentials are transmitted along a myelinated axon.

- Myelin sheath insulates axon; ions cannot pass through it.
- Action potentials can only occur at nodes of Ranvier.
- When action potential occurs at one node, Na^+ ions enter axon, making that region temporarily more positive.
- Local circuit: movement of positive ions from positive to negative regions.
- Causes opening of Na+ channels at next node of Ranvier.
- Causing new action potential / depolarisation at the next node.
- Saltatory conduction occurs: action potentials 'jump' from node to node.
- One-way transmission of impulses occurs.

Explain what is meant by saltatory conduction and describe its effect on transmission of nerve impulse.

- Action potential/ impulse jumps from node to node.
- Local circuits are set up between nodes.
- This increases the speed of transmission of nerve impulses.

Explain how speed of transmission of impulses can be increased

- Wider / thicker / larger diameter of axon increases speed of transmission.

- Larger membrane surface area so more ion movement.
- Presence of myelin increases the speed of transmission.
- Myelin sheath enables saltatory conduction of impulses.

Explain how one-way transmission of impulses is maintained

- Neurotransmitters are only released from the presynaptic neurone.
- Neurotransmitter diffusion only occurs in one direction (from high to low concentration).
- Receptors for neurotransmitters are only found on the postsynaptic membrane.
- Synaptic vesicles are absent in the postsynaptic neurone.
- Enzymes in the synaptic cleft quickly remove neurotransmitters, preventing reverse activation.
- Action potentials cannot travel backward along a neurone due to the refractory period, which prevents immediate re-firing of the same neurone.

With reference to voltage-gated sodium ion channels, explain the difference in speed of transmission of an action potential along a myelinated neurone and a non-myelinated neurone.

- Myelinated neurone has faster speed of transmission of action potential: 100 ms^{-1} vs. 2 ms^{-1}

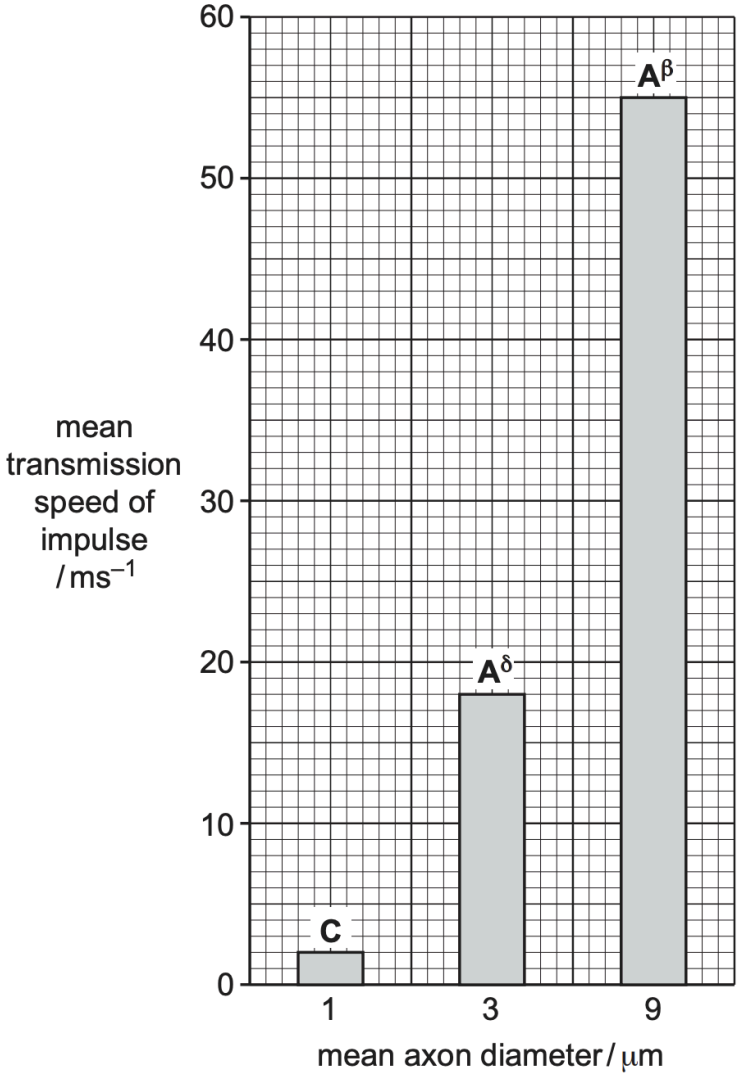
Myelinated	**Non-myelinated**
Na+ channels only occur at nodes of Ranvier.	Na+ channels occur along length of neurone.
Depolarisation only occurs at nodes of Ranvier.	Depolarisation occurs along length of neurone.
Long local circuits	Short local circuits
Saltatory conduction occurs	No saltatory conduction occurs

Walking requires nervous control to coordinate movements.
Giant axonal neuropathy (GAN) = autosomal recessive disease, which affects neurones. One of the first signs of GAN is having problems with walking.
Speed of transmission of nerve impulses is slower in people with GAN. Explain how this can affect walking.

- It takes longer for impulses to reach neuromuscular junction / muscles.
- Fewer / slower muscle contractions take place.
- This causes problems in walking, eg. reduced muscle control, slower walking, stumbling, tripping.

- This also results in slower reflexes which affects walking.

Mean transmission speed of impulses for three types of sensory neurone:



- Impulses from pain receptors in the skin are sent along sensory neurones C and Aδ.
- Sensory neurones C and Aδ synapse in the spinal cord with relay neurones known as projection neurones.
- Projection neurones send impulses to the part of brain that perceives pain.
- Impulses from touch receptors in the skin pass along sensory neurones Aβ, which can also synapse with the projection neurones in the spinal cord.

Gently rubbing a damaged area of skin can reduce the perception of pain. Suggest an explanation for this.

- Impulses for touch in Aβ neurones travel faster.
- Aβ neurones inhibit spinal cord/relay/projection neurones.

Importance of the Refractory Period

Describe the importance of the refractory period in the transmission of action potentials.

- It limits/ controls maximum frequency of action potentials.
- It ensures action potentials/ impulses travel in one direction.

Cholinergic Synapses

Outline the roles of synapses in the nervous system.

- Ensures one-way transmission of impulses.
- Allows communication between many neurones / interconnection of nerve pathways
- Allows integration of impulses and filters out less frequent impulses / low level stimuli
- Involved in memory / learning

Describe the events that occur at a synapse that lead to the release of acetylcholine.

- Action potential / depolarisation at presynaptic membrane.
- Ca^{2+} channels open / presynaptic membrane becomes more permeable to Ca^{2+}.
- Ca^{2+} enter presynaptic neurone/knob.
- By facilitated diffusion, down the concentration gradient.
- This causes vesicles of ACh neurotransmitter to move towards/ fuse with the presynaptic membrane.
- Exocytosis occurs: ACh is secreted and enters into the synaptic cleft.

Describe the role of calcium ions in a cholinergic synapse

- Ca^{2+} enters/diffuses into synaptic knob
- Through voltage-gated calcium ion channels
- Vesicles containing acetylcholine
- Move to/ fuse with presynaptic membrane
- Exocytosis of acetylcholine

Blocking calcium ion channels in the presynaptic knob of a cholinergic synapse results in no action potentials in the postsynaptic neurone. Explain why.

- No calcium ions enter the presynaptic knob
- So vesicles do not move to/ fuse with the presynaptic membrane
- No exocytosis/ release of acetylcholine (ACh)
- ACh does not diffuse across synaptic cleft
- No binding with receptor protein on postsynaptic membrane
- Sodium ion channels do not open

- Sodium ions do not enter postsynaptic neurone
- No depolarisation of postsynaptic membrane

Explain the role of acetylcholinesterase in a synapse.

- Breaks down acetylcholine
- So that it leaves the binding site/ receptor
- Hence depolarisation stops in <u>postsynaptic membrane</u>
- Which stops continuous action potentials in postsynaptic membrane
- It enables ACh to be recycled

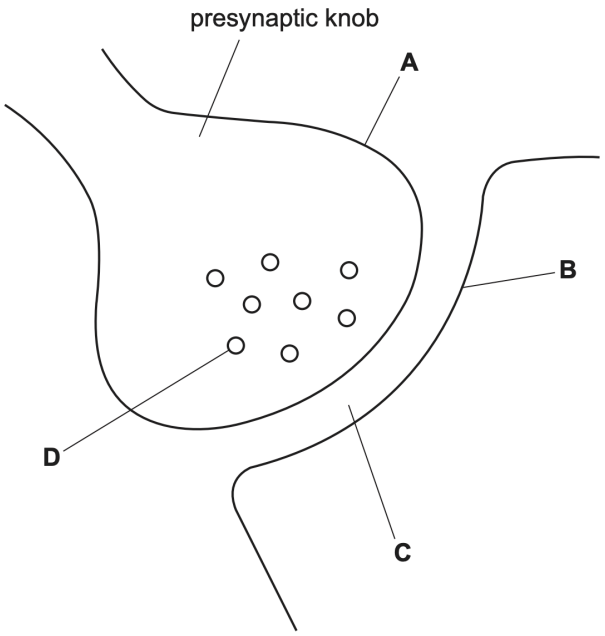
Some insecticides have a similar structure to acetylcholine. Suggest how these insecticides may affect the functioning of acetylcholinesterase.

- acts as a competitive inhibitor
- complementary shape to active site
- binds with / blocks active site
- ACh is not broken down / hydrolysed

Explain the effect on postsynaptic neurone when voltage gated Na^+ channels in presynaptic membrane of synapse are blocked.

- No Na^+ enter postsynaptic neurone.
- No depolarisation of postsynaptic membrane occurs.
- No action potentials/ impuleses are transmitted.

Diagram of a cholinergic synapse



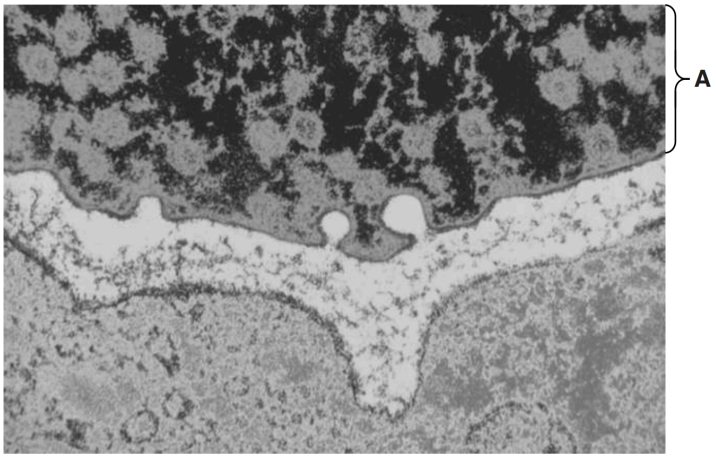
Locations of compounds and structures associated with cholinergic synapse:

acetylcholine	C and D
voltage-gated channel	A
receptor protein	B
acetylcholinesterase	C

Precise locations in cholinergic synapse:

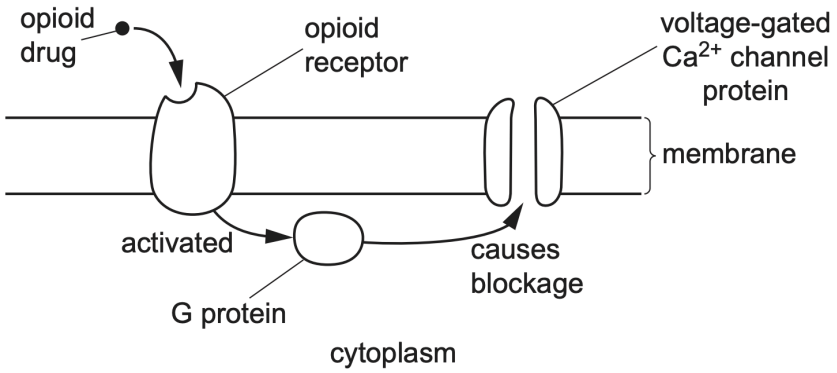
A region that contains many mitochondria.	Presynaptic knob
A region where exocytosis of ACh occurs.	Presynaptic membrane
A region that contains voltage-gated channel proteins.	Presynaptic membrane
A region that contains ligand-gated channel proteins	Postsynaptic membrane

Electron micrograph of part of cholinergic synapse



A structure that indicates that A is a presynaptic neurone	Invagination of the membrane
An area that contains many voltage-gated Na+ channels	Post-synaptic membrane
An area that contains both acetylcholine and acetylcholinesterase	Synaptic cleft

Action of an opioid drug on the presynaptic membrane:



Opioid drugs can be taken to relieve the pain. Opioid receptors are located in the presynaptic membrane of a cholinergic synapse.

Explain how the action of an opioid drug on the presynaptic membrane can prevent the generation of pain impulses in the postsynaptic neurone.

- Opioid binds to receptor.
- This activates G protein.
- This blocks Ca^{2+} channels OR no/less Ca^{2+} enter presynaptic knob.
- Vesicles do not move towards and fuse with the presynaptic membrane OR fewer vesicles move towards and fuse with the presynaptic membrane.
- No/less ACh or acetylcholinesterase is released
  OR no/less exocytosis of ACh.
- No/less ACh or acetylcholinesterase binds to receptors on postsynaptic membrane.
- No/smaller depolarisation of postsynaptic membrane.

Some drugs and their actions:

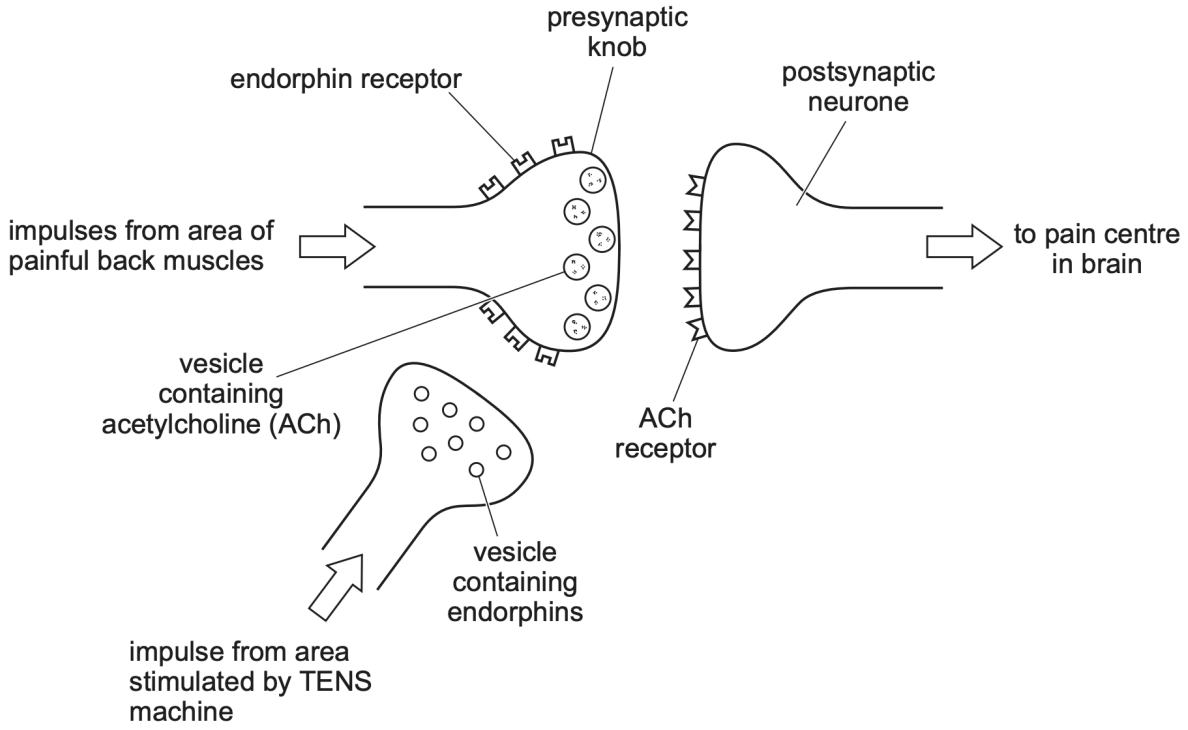
drug	action of drug
curare	blocks muscle cell membrane receptors at neuromuscular junctions
nerve gas	inhibits acetylcholinesterase function in synapses
alcohol	inhibits exocytosis of neurotransmitters in synapses

Suggest and describe the immediate consequence of the action of each drug on a neuromuscular junction or cholinergic synapse.

curare	- less / no ACh binds to receptors - fewer / no ligand-gated Na+ channels open OR fewer / no Na+ can enter sarcomere / sarcoplasm OR no / less depolarisation of sarcolemma / muscle cell membrane

nerve gas	- no / less ACh broken down OR ACh remains bound to receptor - ligand gated Na+ channels remain open OR Na+ continue to enter postsynaptic neurone OR permanent depolarisation of postsynaptic membrane
alcohol	no / less binding of neurotransmitter / ACh to receptors

Action of TENS at a cholinergic synapse:



Back pain in humans is reduced by using a TENS machine. TENS machine uses electrical impulses to stimulate nerve endings near the site of the pain.
TENS triggers the release of natural painkillers called endorphins at synapses.

Suggest and explain how endorphins may act to reduce pain.

- Endorphins bind to endorphin receptors.
- This stop calcium ions from entering presynaptic knob.
- No/fewer vesicles move towards/ fuse with the presynaptic membrane.
- No/less acetylcholine is released.
- No/less binding of ACh to postsynaptic receptors occurs.
- No/less depolarisation of postsynaptic membrane occurs.
- No/fewer action potentials/ impulses are sent to pain centre / brain.

Suggest disadvantages of using pharmaceutical drugs for reducing pain compared to using TENS.

- drug dependency / addiction / tolerance
- drug side-effects

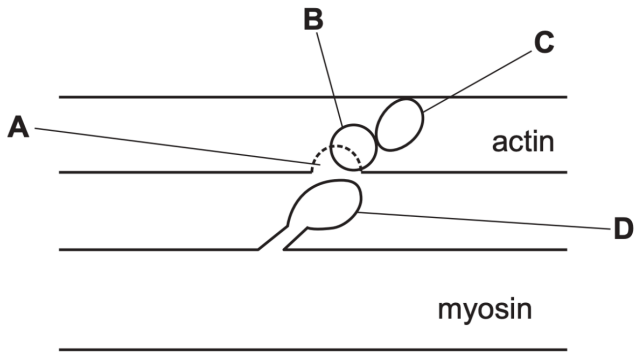
- expensive
- work more slowly

Ultrastructure of Striated Muscle

Structural features of thick filaments and thin filaments in the sarcomere.

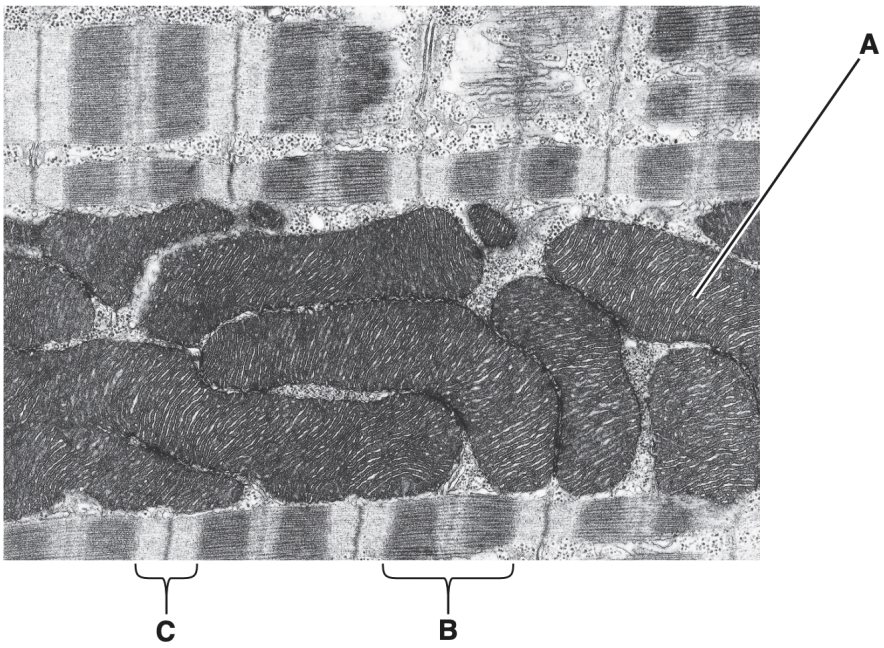
Thick filaments	**Thin filaments**
Myosin	Actin
Fibrous protein	Globular proteins
	Tropomyosin / troponin
Globular heads / ATPase	Binding site for myosin head
15nm diameter	7nm diameter
M lines	Z lines

Part of a sarcomere in striated muscle:



A	binding site
B	tropomyosin
C	troponin
D	myosin <u>head</u> / ATPase

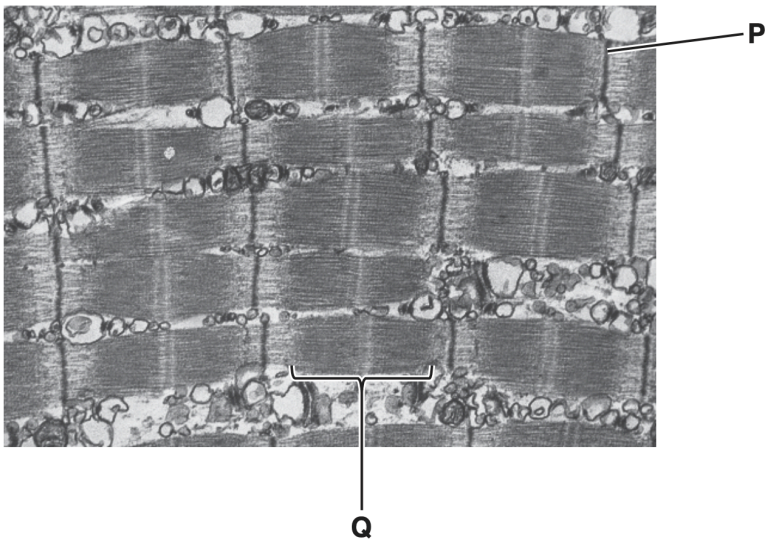
Transmission electron micrograph of a section through striated musc



Location of proteins associated with striated muscle structure.

Myosin and actin	B
Actin alone	C
ATP synthase	A
ATPase	B

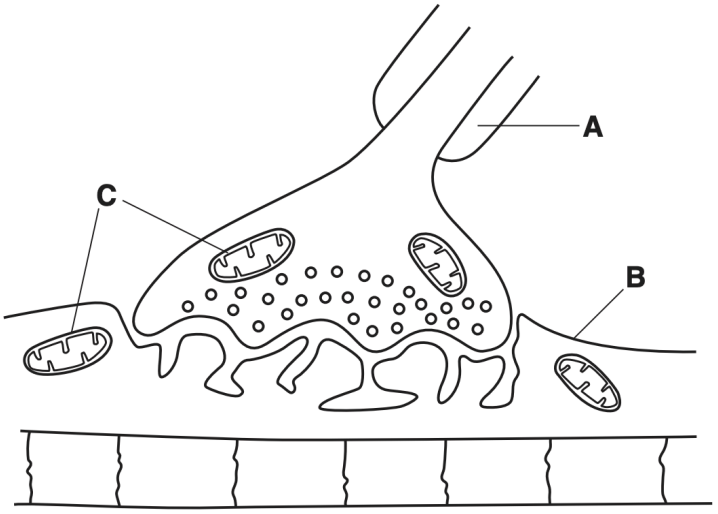
Electron micrograph of a section of striated muscle:



P	Z line
Q	A band

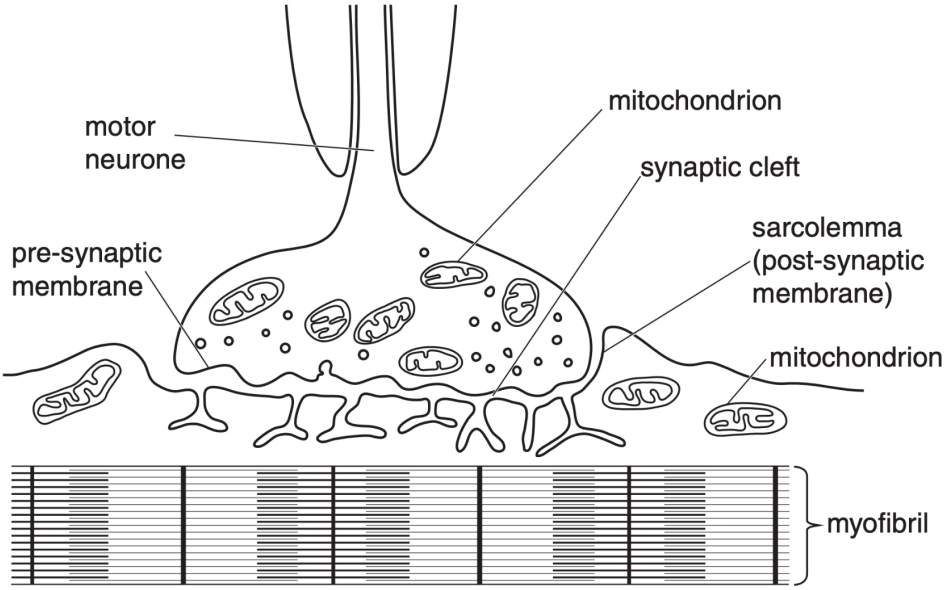
Stimulating Contraction in Striated Muscle

Neuromuscular joint



A	Myelin sheath / Schwann cell
B	Sarcolemma
C	Mitochondria

Mammalian neuromuscular junction



thin filament areas	region containing only actin
overlapping areas	region containing both actin and myosin.

Outline how an action potential arriving at this neuromuscular junction can result in depolarisation of the sarcolemma.

- Ca^{2+} channels open in presynaptic membrane/ presynaptic knob.
- Ca^{2+} enter presynaptic knob/ presynaptic neurone.
- Vesicles contain neurotransmitter/ACh.
- These vesicles move towards / fuse with presynaptic membrane.
- ACh/ neurotransmitter is released (exocytosis) and diffuses across synaptic cleft.
- ACh binds to receptors on sarcolemma / post-synaptic membrane.
- Na+ channels open and Na+ enters muscle fibre / sarcoplasm.

Outline the role of sarcoplasmic reticulum in contraction of striated muscle

- Ca^{2+} channels open in membrane of SR.
- Ca^{2+} diffuses/ is released into sarcoplasm.
- Ca^{2+} binds to troponin.
- Tropomyosin moves/ changes positions.
- Cross bridges form – myosin binds to actin.

Consequences to sarcomere when Ca^{2+} ions from the cytoplasm are not pumped into the sarcoplasmic reticulum when the muscle fibre is no longer stimulated.

- Ca^{2+} stays in the cytoplasm / sarcoplasm.
- Ca^{2+} remains bound to troponin.
- Tropomyosin does not cover myosin binding sites on actin.
- Myosin-actin cross bridges exist/ remain.
- Sarcomere / muscle is contracted / shortened paralysed.

Suggest how enzyme inhibitors in neuromuscular junctions could affect muscle function in humans.

- The inhibitor binds to acetylcholinesterase.
- This blocks active site/ shape of active site changes.
- Thus ACh is not broken down/ stays bound to receptors on sarcolemma.
- Na+ channels stay open.
- Sarcolemma stays depolarised.
- Action potentials continue to be generated and muscles remains stimulated.
- Thus muscles stay contracted.

Explain why mitochondria are present in the neuromuscular junction.

- To synthesis/ produce ATP.
- ATP needed to synthesise / recycle acetylcholine
- ATP needed for movement of vesicles / exocytosis
- ATP needed to transport calcium ions out of presynaptic neurone /into sarcoplasmic reticulum

- ATP needed to synthesise acetylcholinesterase
- ATP needed for sodium-potassium pumps / active transport
- ATP is needed for contraction of sarcomere.

Sliding Filament Model of Muscular Contraction

Events occurring during muscle contraction:

1. sarcolemma depolarised
2. transverse tubules depolarised
3. Ca2+ ions diffuse out of sarcoplasmic reticulum
4. Ca2+ ions bind to troponin
5. troponin changes shape
6. tropomyosin moves
7. binding sites on actin exposed
8. myosin heads bind to actin
9. myosin heads tilt
10. sarcomere shortens

Describe the role of calcium ions in the contraction of striated muscle

- When sarcoplasmic reticulum is depolarised
- Calcium ion channels open
- Calcium ions are released from SR and move into sarcoplasm
- They bind to troponin
- Troponin changes shape
- This causes tropomyosin to move
- This exposes myosin binding sites on the actin
- This allows myosin head to bind to actin, forming cross-bridge
- Myosin head tilts and pulls the actin with it – power stroke

Describe how tropomyosin is involved in the sliding filament model of muscle contraction.

- It covers / uncovers myosin binding sites on actin.
- When calcium ions bind to troponin, it moves/ changes shape.
- This allows myosin to bind to actin and form cross bridges.

Explain the role of ATP in the contraction of striated muscle.

- Myosin head binds to actin to form cross bridge (ADP is still attached to myosin head).
- ADP is then released, which causes motion of myosin head.
- This causes actin to move.
- Power stroke occurs: pulling of actin filament.

- A new ATP molecule binds to myosin head.
- This breaks cross-bridge, causing myosin head to detach from actin.
- Myosin head / ATPase causes hydrolysis of ATP to ADP.
- This releases energy to move myosin head back to original position.
- ATP is needed to pump Ca^{2+} back into sarcoplasmic reticulum.
- This ends contraction by allowing tropomyosin to block actin-binding sites again.

Describe how myosin is involved in the sliding filament model of muscle contraction.

- ATP hydrolysis occurs: ATP → ADP + Pi occurs
- This activates myosin head to pivot/rotate/tilt/stand up.
- Myosin head binds to actin to form cross bridges.
- ADP and Pi detach.
- Myosin head swings back/ returns to previous position.
- Actin is moved/pulled along – power stroke.
- New ATP molecule binds to myosin head.
- Myosin head detaches from actin and cross bridges break.

When a mammal dies, aerobic respiration stops. The striated muscles contract and remain contracted for a few hours after death. Suggest why the muscles remain contracted for a few hours.

- No / little ATP produced / available
- So no breaking of cross bridges / myosin head is not released

15.2 Control and Coordination in Plants

Electrical Communication in the Venus Flytrap

Suggest why venus flytrap needs to capture insects

- Not enough nitrogen / nitrate in the soil OR poor mineral content of soil
- Insects provide mineral ions/ amino acids for growth

Describe how the production of action potentials in the leaf cells of the Venus fly trap can result in the leaves closing and trapping an insect.

- Action potential / depolarisation is generated and reaches lobe of leaf.
- Action potential spreads across lobe of leaf and reaches hinge / midrib cells.
- This triggers H^+ ions to be pumped out of cells, into cell walls of hinge cells.
- Acidic conditions in cell wall causes wall to loosen/ cross links broken.
- Calcium pectate in the middle lamella dissolves, weakening connection between adjacent cells.
- Ca^{2+} ions enter cells, lowering water potential inside cells.
- Water enters by osmosis down the water potential gradient.
- Hinge / midrib cells expand/ become turgid.
- Leaves / lobes snap shut and become concave.

State which part of venus flytrap leaf detects stimulus:
(sensory / trigger / receptor) hair

State which ion moves into the cells at the base of sensory hairs to generate receptor potentials: Calcium ions

Name the mechanism by which enzymes are released from leaf cells to digest the trapped insect: Exocytosis

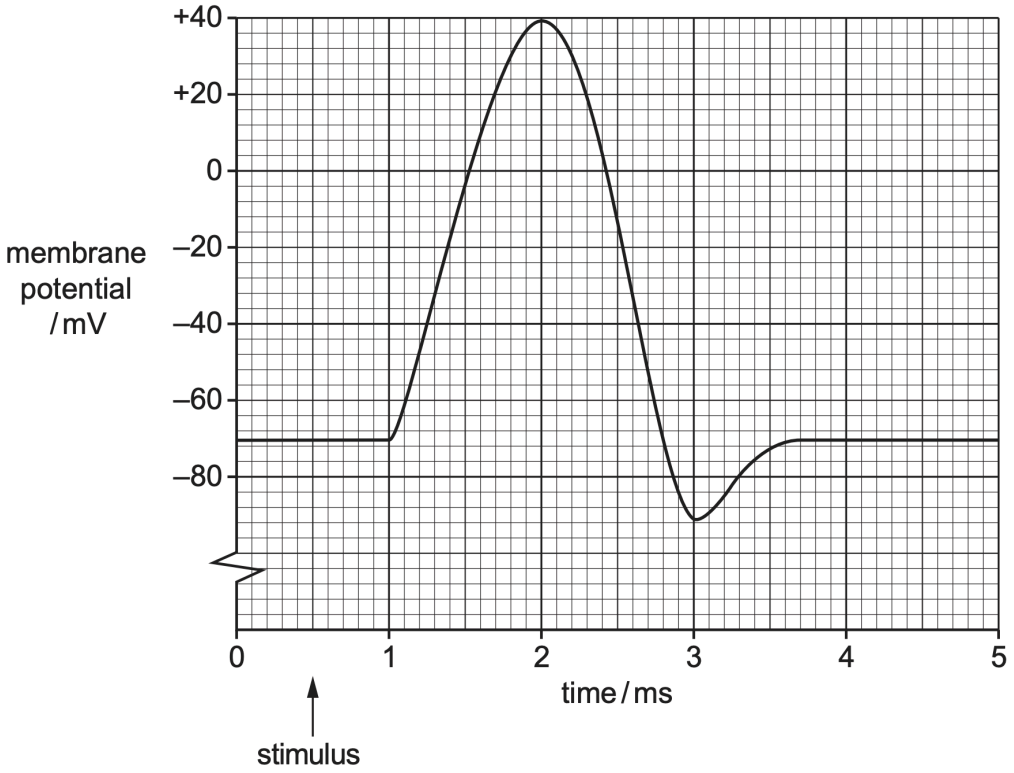
Explain how the plant does not waste energy by closing when it does not need to, for example when a large drop of rain touches the receptor.

- At least 2 hairs must be touched within 35 seconds / short time period / same time OR 1 hair must be touched twice within 35 seconds / short time period.

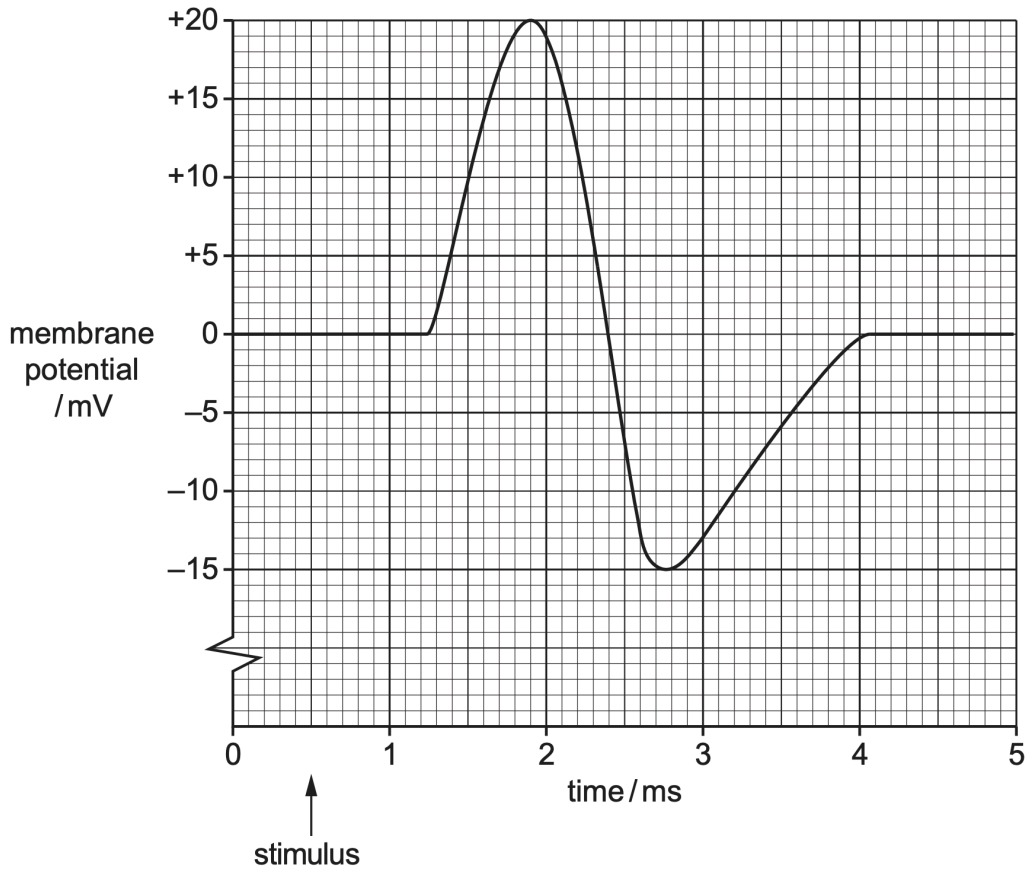
State why it is beneficial to venus flytrap for stimulation of 2 or more hairs to be necessary before the leaf closes

- To avoid wasting energy
- To avoid the leaves closing due to single contact/ by an objects such as water droplet.

Action potential in human neurone:



Action potential in leaf cells of venus flytrap:

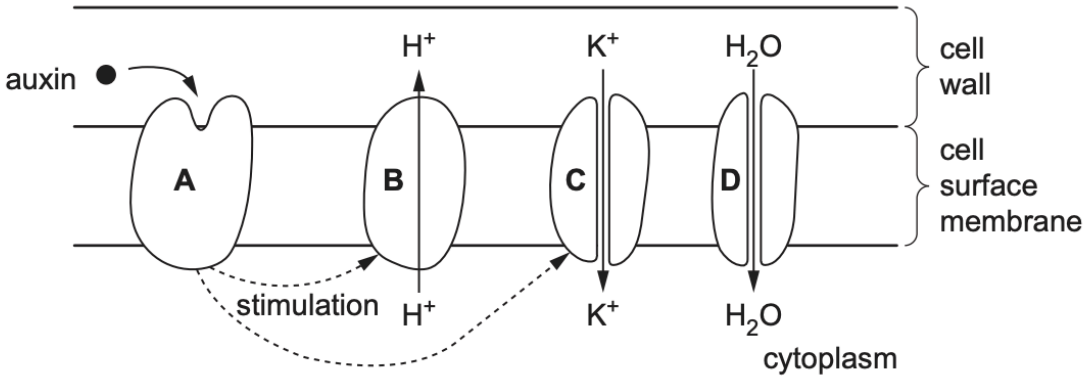


Describe how action potential of venus flytrap differs from that of human
For venus flytrap:

- Smaller change in membrane potential / smaller depolarisation
- Longer duration of action potential / shorter duration of depolarisation
- Shorter duration of repolarisation / longer duration of hyperpolarisation
- Longer refractory period
- Data to support
- Membrane / resting potential is 0mV compared with -70mV for human

The Role of Auxin in Elongation Growth

Part of a cell in growing region of a plant



Identify proteins A,B,C,D

- A: auxin binding protein
- B: proton pump // hydrogen ion (H^+) pump
- C: potassium ion (H^+) channel/ pore
- D: aquaporin

State the type of protein represented by A

- Receptor

Describe the effects on the cell wall of many hydrogen ions moving into the cell wall.

- pH of cell wall decreases/ becomes more acidic
- Expansins are activated by acidic conditions
- They break/ loosen non-covalent bonds/ cross-links
- Between cellulose and hemicellulose OR between cellulose microfibrils
- Cell wall expands/ stretches OR microfibrils move past each other
- Due to turgor pressure on cell wall

Explain the consequences of an influx of potassium ions into the cell.

- Water potential decreases/ becomes more negative
- Water moves into the cell by osmosis, down water potential gradient
- Cell expands/ increases in volume/ elongates

The Role of Gibberellin in Germination of Barley

Describe the sequence of events that lead to the production of amylase during germination of barley seeds.

- The seed embryo absorbs water.
- This stimulates the embryo to produce gibberellin.
- Gibberellin moves/diffuses to the aleurone layer.
- Gibberellin binds to receptor proteins in aleurone cells, which triggers signalling cascade.
- Signalling pathway leads to destruction of DELLA proteins that normally inhibit gene expression.
- Removal of DELLA proteins allow transcription factors to activate.
- The gene coding for amylase is expressed / transcribed, producing mRNA.
- Translation of mRNA occurs to produce amylase.

Outline the role played by gibberellin in the germination of wheat seeds.

- Seed absorbs water, which triggers germination.
- Embryo produces gibberellin.
- Gibberellin diffuses to aleurone layer.
- It stimulates aleurone cells to produce amylase: Gibberellin activates genes or stimulates synthesis of mRNA coding for amylase.
- Amylase hydrolyses/ breaks down starch stored in endosperm
- To maltose / glucose.
- Embryo uses the sugar for respiration / growth.

Outline the role of amylase in seed germination

- Amylase enters endosperm
- Here it hydrolyses / breaks down / digests / converts starch / amylose
- To form maltose / glucose.
- Glucose is needed / used by the embryo
- Glucose is needed for respiration / to release energy / for ATP production
- For growth of the embryo.

State the location of gibberellin synthesis in a barley seed during germination.

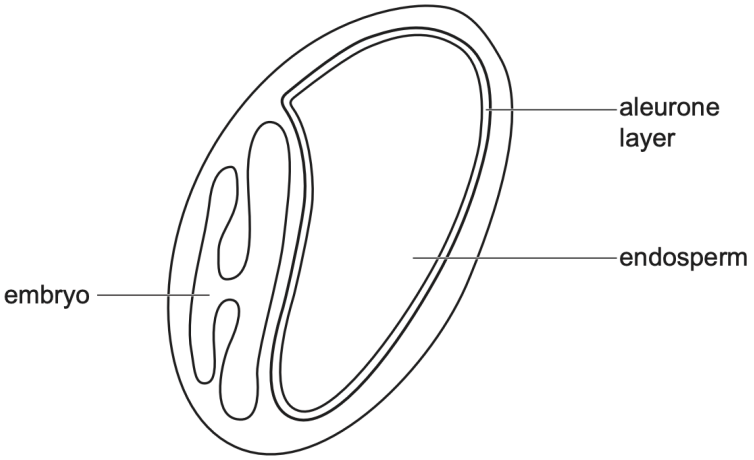
Embryo

During barley seed germination, gibberellin stimulates the synthesis of enzymes. State the name of one of these enzymes and the precise location of its synthesis.

- Name of enzyme: amylase / maltase / protease
- Location of synthesis: aleurone layer

State the precise location of starch reserves in a barley seed.
Endosperm

Section through a barley seed

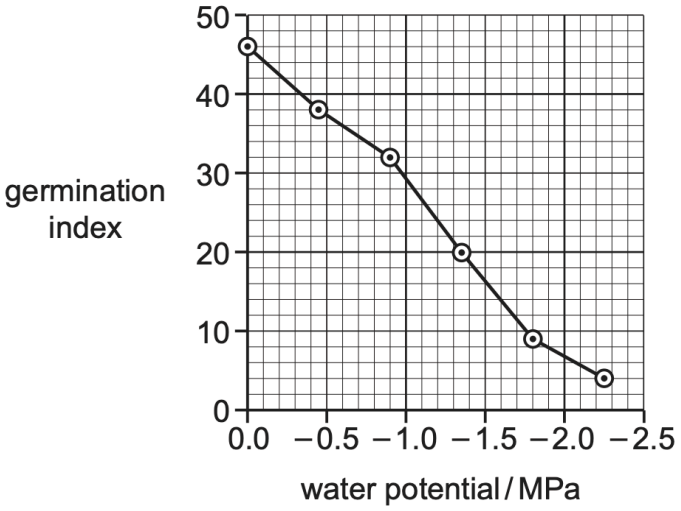


Movement of gibberellin and amylase during germination:

Movement of gibberellin during germination.	embryo to aleurone layer
Movement of amylase during germination.	aleurone layer to endosperm

Graph Analysis

The germination of barley seeds placed on blotting paper soaked in solutions of different water potential was investigated. Results:

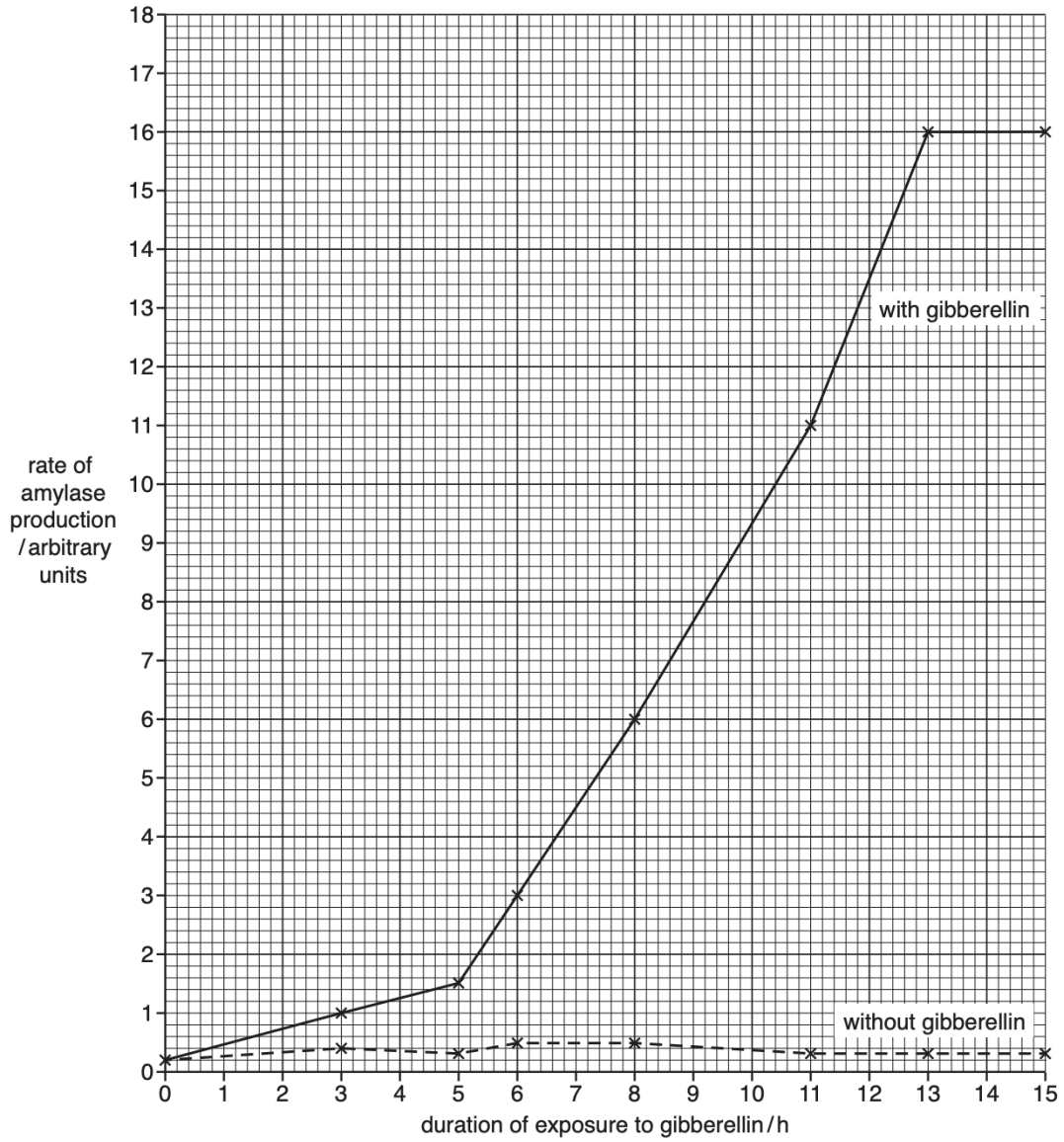


Higher germination index value = more successful germination of barley seeds.

Describe and explain the relationship between germination index of barley seeds and water potential.

- As water potential increases, the germination index increases.
- Comparative data
- Low / negative water potential decreases water uptake into the seed.
- Water is needed / used to activate embryo / produce gibberellin.
- Water is needed / used for hydrolysis reactions.
- Example of hydrolysis reaction: starch to maltose / maltose to glucose.
- Water is needed / used as a medium for reactions.
- Solutes lowering water potential may be toxic / may inhibit enzymes.

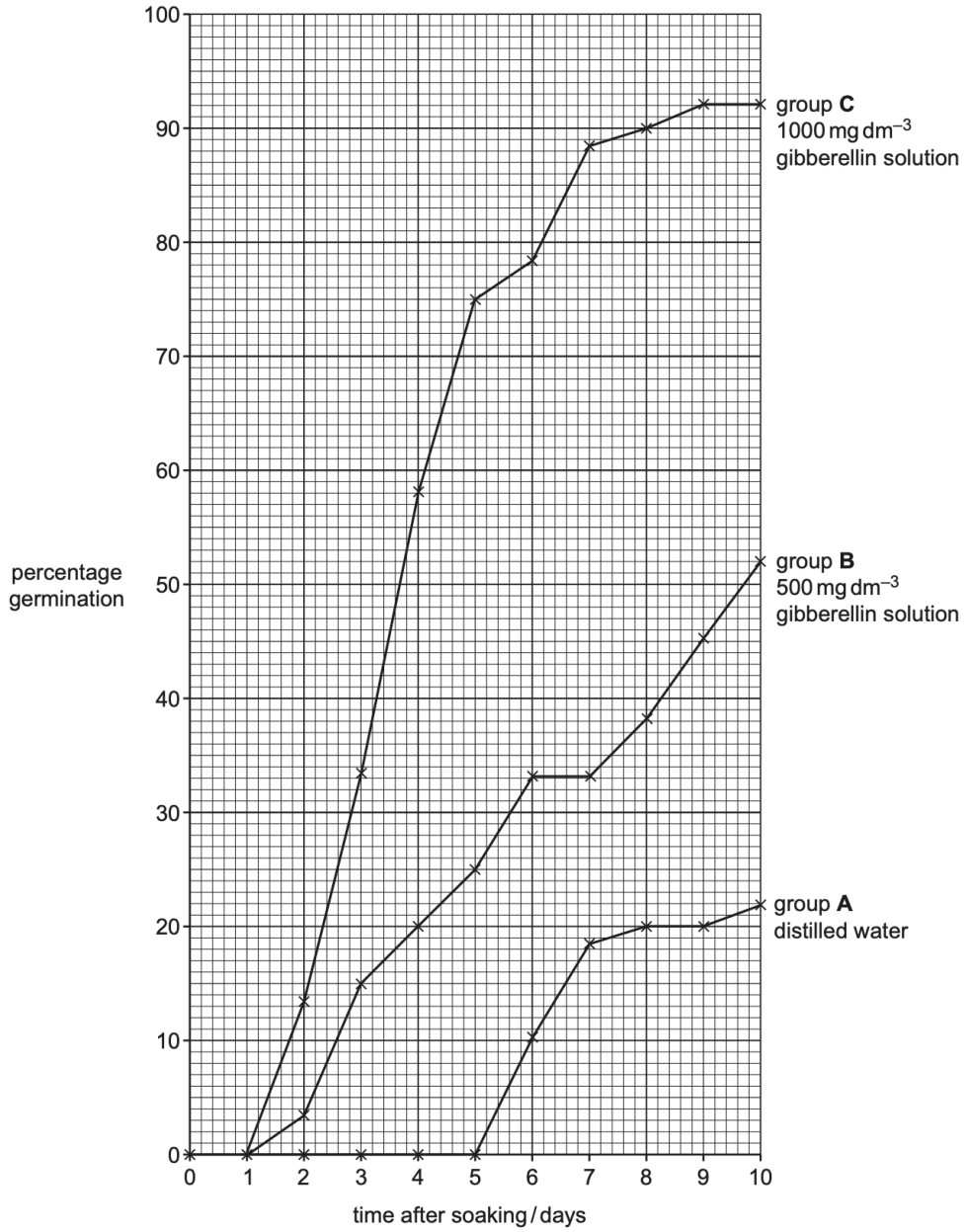
In an investigation, aleurone layers from barley seeds were extracted. One sample was treated with gibberellin and the other sample was given no gibberellin treatment. The rate of production of amylase enzyme by the aleurone layers was measured over 15 hours. Results:



Describe the results shown

- <u>Rate of amylase production</u> is higher with gibberellin.
- Data comparison: with and without gibberellin.
- With gibberellin: rate of amylase production increases/gets faster over time for 13 hours // rate increases more after 5 hours // rate is maximum at 13-15 hours // rate plateaus from 13-15 hours.
- Without gibberellin: rate of amylase production is constant / fluctuates in a narrow range / is low throughout.

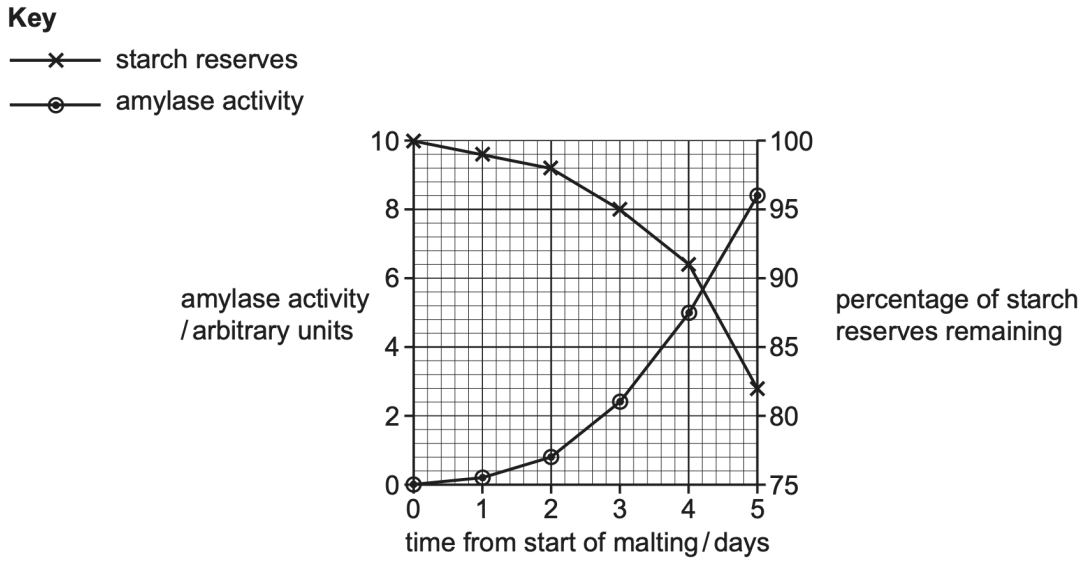
3 groups of seeds were soaked for 24 hours in distilled water or in a solution of gibberellin. They were then sown on filter paper in dishes and kept moist for 10 days. Results:



Describe the results

- seeds of B and C germinated before seeds of A OR
  seeds soaked in gibberellin germinated before
- rate of increase in percentage germination was highest in C OR higher in C than B / A OR higher as the concentration of gibberellin increases
- final / day 10 / maximum percentage germination was highest in C OR higher in C than B / A OR higher as the concentration of gibberellin increases
- data quote between two groups:
  - A by day 6 and B and C by day 2
  - A germinates four days after B and C
  - A – 2.2; B – 5.2; C – 9.2, percentage germination day^{-1}
  - A – 22%; B – 52%; C – 92%,
  - A to C = 70% more, A to B = 30% more, B to C = 40% more
- percentage germination in C levels off after 9 days
- no seeds germinate by day 1

Malting = process involved in production of beer. During malting, barley seed germination is controlled so that the sugars produced during germination can be used in the production of beer.



Graph shows 2 features of a germinating barley seed during first 5 days of malting:
• activity of amylase enzyme
• percentage of starch reserves remaining in the barley seed

Describe and explain the effect of malting on amylase activity and the percentage of starch reserves remaining in the germinated barley seed.

- Amylase activity increases
- As seeds produce more amylase
- Percentage of starch decreases

- As amylase hydrolyses / breaks down starch
- The change occurs at an increasing rate for both
- Data quote:

time / days	amylase activity / au	percentage of starch remaining
0	0.0	100
1	0.2	99
2	0.8	98
3	2.4	95
4	5.0	91
5	8.4	82

In the malting process, germination is stopped before the concentration of sugars in the germinating barley seeds exceeds a concentration that causes shoot or root growth. Drying germinating barley seeds at 50 °C is one method used to stop malting.

Explain how this method stops malting.

- Enzyme get denatured / deactivated / inactivated.
- Changes tertiary structure / 3D shape of amylase / enzyme.
- Changes shape of active site.
- Active site no longer complementary / does not bind to starch / substrate OR no ESC / enzyme-substrate complexes formed.
- Drying removes water from embryo for gibberellin synthesis.
- Drying removes water from endosperm for hydrolytic reactions.

Suggest why malting is stopped before shoot or root growth occurs.

- Stops sugars / glucose from being used for root and shoot growth
- To ensure enough sugars for beer production.