

Cells (Interaction and interdependence) revision notes

Cells interact by sending and receiving signals. This group covers chemical signalling (HL only): ligands, receptors and the signal transduction pathways they start. It also covers neural signalling: resting and action potentials, the speed of nerve impulses and the synapses that link neurons to each other and to effector cells.

For IB Diploma Biology, Standard Level and Higher Level (first assessment 2025). **HL only** is additional higher level content, for HL students only.

1 Chemical signalling **Spec C2.1** **HL only**

Receptors, ligands and quorum sensing **Spec C2.1.1, C2.1.2**

Cells receive many different chemical signals. Each signal is detected by a **receptor**: a protein with a binding site for one specific signalling chemical. The IB term for the signalling chemical is the **ligand**.

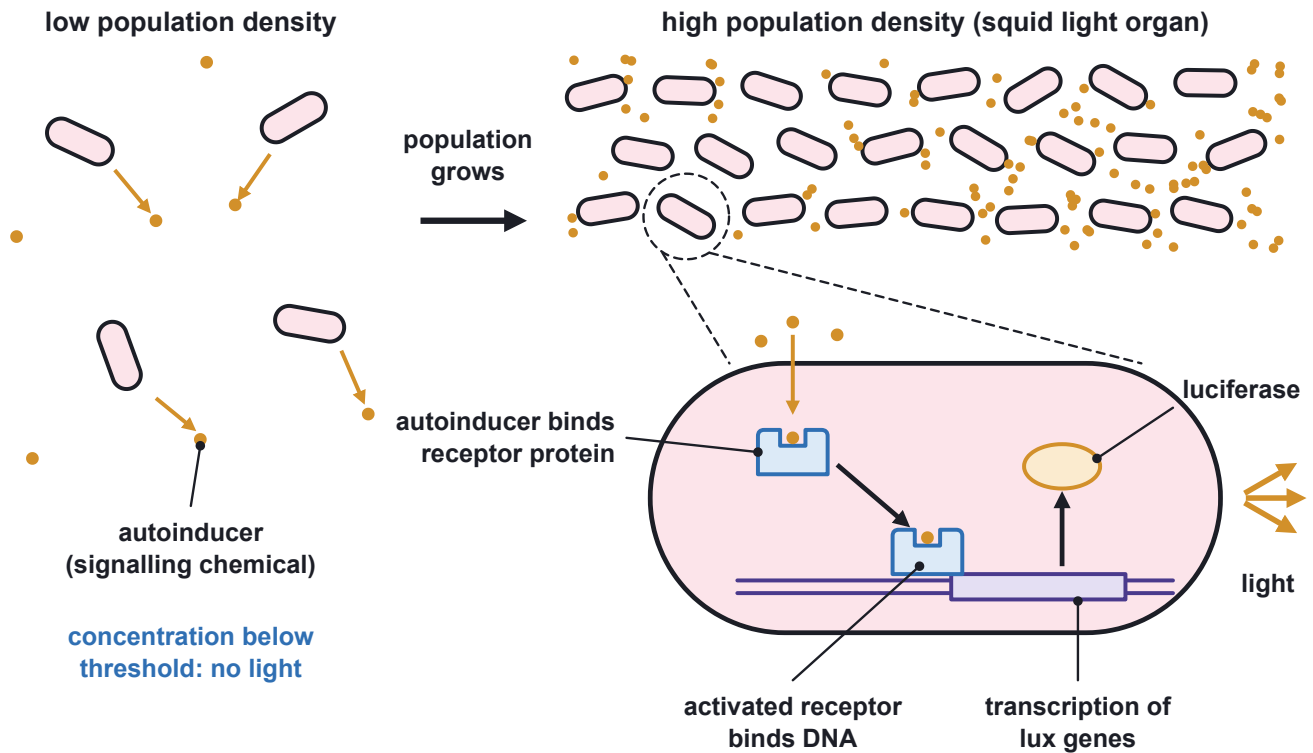
- The binding site has a shape and chemical properties (charge, polarity) that are complementary to the ligand, so only that ligand binds.
- Binding is reversible: the ligand binds, causes a change in the receptor, then leaves.
- When the ligand binds, the receptor changes conformation (shape). This change is what passes the signal on.
- A cell responds only to the ligands it has receptors for. This is how cells distinguish between the many signals they receive.

Quorum sensing in *Vibrio fischeri*

Bacteria also signal to each other. **Quorum sensing** is cell signalling in which bacteria detect the density of their own population and respond only when it is high. The marine bacterium *Vibrio fischeri* lives freely in sea water and also at very high density in the light organ of some squid and fish. It produces light (**bioluminescence**) only at high density.

1. Each *V. fischeri* cell makes a small signalling chemical (an autoinducer) and releases it. It diffuses freely across the bacterial membranes.
2. At low population density the autoinducer diffuses away, so its concentration stays low.
3. At high density, as in a light organ, the autoinducer accumulates and binds to receptor proteins inside the cells.
4. The activated receptor promotes transcription of the genes for bioluminescence, including the enzyme luciferase, so the whole population glows together.
5. The same genes include one for making more autoinducer, so the response reinforces itself (positive feedback).

Light production uses a lot of energy. A single cell's light would be useless, so it only pays when many cells glow together. The squid host benefits from the light; the bacteria gain nutrients and a place to live.



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Quorum sensing in *Vibrio fischeri*: light is produced only when the **autoinducer** concentration is high enough.

Exam tip: Always call the signalling chemical a ligand and say it binds to a specific binding site on the receptor. Avoid 'active site', which is for enzymes and substrates.

Types of signalling chemicals Spec C2.1.3, C2.1.4, C2.1.5

Functional categories of signalling chemicals in animals			
Category	Released by	How it reaches target cells	Effect
Hormones	Endocrine glands, secreted into the blood	Transported by the blood around the whole body	Distant, widespread, slower and longer lasting
Neurotransmitters	Neurons, at a presynaptic membrane	Diffuse across a synaptic gap (about 20 nm)	Localized to one postsynaptic cell, rapid and brief
Cytokines	Many cell types, especially cells of the immune system	Diffuse to nearby cells in tissue fluid	Mainly local, e.g. regulate immune responses and inflammation
Calcium ions (Ca²⁺)	Released inside a cell from stores or entering through channels	Act inside the cell that receives the first signal	Trigger responses such as exocytosis of neurotransmitter or muscle contraction

The difference between **localized and distant effects** is clearest when hormones are compared with neurotransmitters. A hormone reaches every cell the blood supplies, but only cells with the matching receptor respond. A neurotransmitter only crosses the narrow gap at one synapse and is removed within milliseconds.

Chemical diversity

Chemical groups of hormones and neurotransmitters	
Group	Examples
Amine hormones	Epinephrine (adrenaline), norepinephrine (noradrenaline)
Protein (peptide) hormones	Insulin, glucagon
Steroid hormones	Oestradiol, progesterone, testosterone
Amino acid neurotransmitters	Glutamate, glycine, GABA
Peptide neurotransmitters	Endorphins
Amine neurotransmitters	Dopamine, serotonin, norepinephrine (noradrenaline)
Gas	Nitric oxide (NO)

- **Many distinct signals are needed.** Each needs its own ligand and receptor, so cells can tell signals apart.
- **Different properties suit different routes.** Hydrophilic proteins and amines stay outside the cell and bind receptors on the plasma membrane. Hydrophobic steroids pass through the membrane to receptors inside.
- **Speed and duration vary.** Small molecules can be made and broken down quickly; a gas such as NO diffuses freely through membranes and acts briefly near where it is made.
- **One molecule can have more than one role.** Norepinephrine is a hormone when the adrenal gland secretes it into the blood and a neurotransmitter when a neuron releases it at a synapse.

Exam tip: The IB guide's list of neurotransmitters says 'nitrous oxide'. It means the gas that acts as a neurotransmitter, which chemists call nitric oxide (NO). Nitrous oxide (N₂O, 'laughing gas') is a different molecule. Write 'nitric oxide (NO)' in answers. If a question says 'nitrous oxide', it means this same gaseous neurotransmitter.

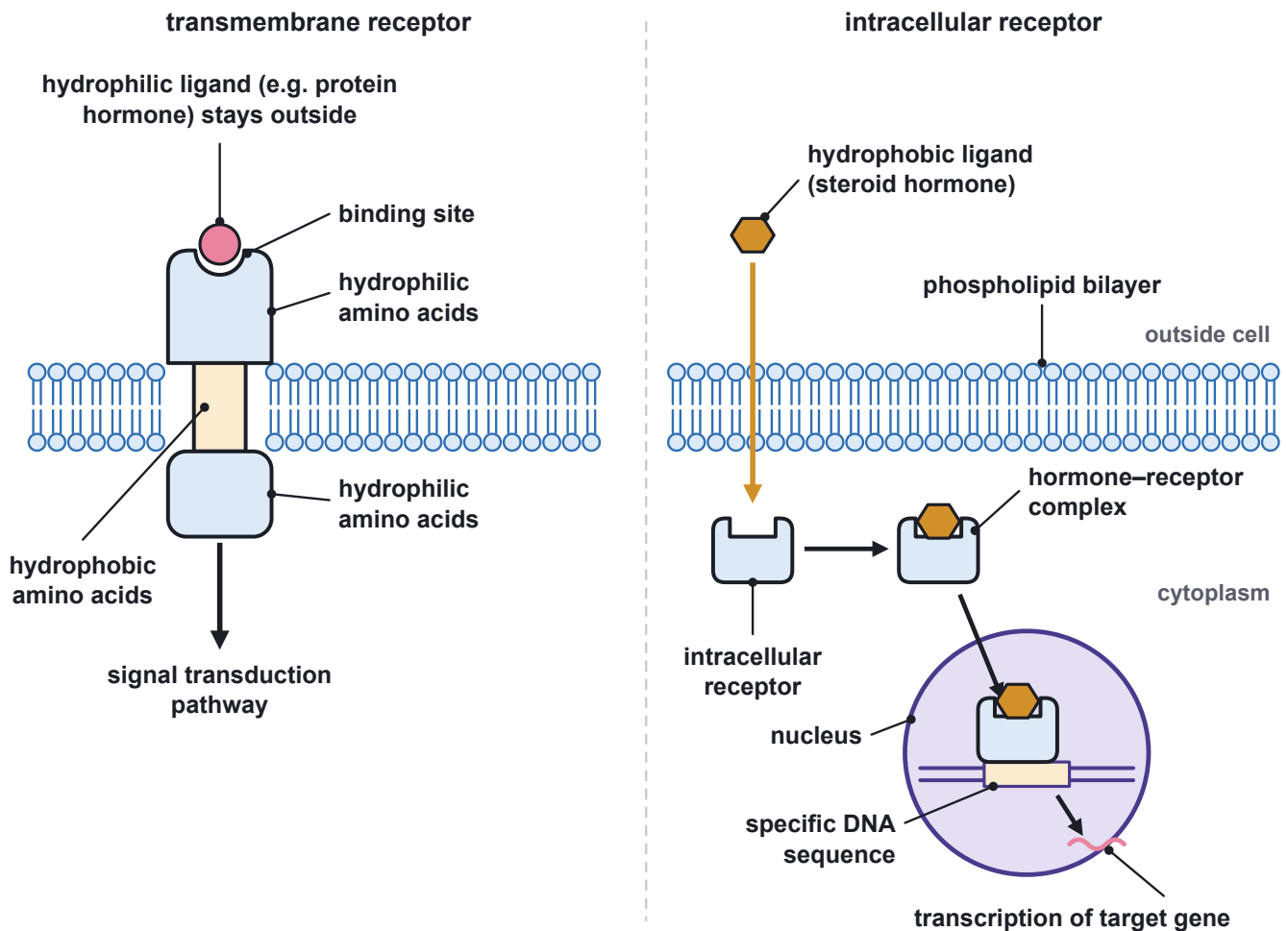
Transmembrane and intracellular receptors Spec C2.1.6, C2.1.7

Comparing the two types of receptor		
Feature	Transmembrane receptor	Intracellular receptor
Position	Spans the plasma membrane	In the cytoplasm or nucleus
Amino acids	Hydrophobic amino acids in the region within the phospholipid bilayer; hydrophilic amino acids in the parts facing the aqueous cytoplasm and extracellular fluid	A soluble protein with hydrophilic amino acids on its surface and no hydrophobic membrane-spanning region; hydrophobic amino acids line the binding site that holds the hydrophobic ligand
Ligand	Hydrophilic or large: proteins, peptides, amines, neurotransmitters	Hydrophobic: steroid hormones
Does the ligand enter the cell?	No, it binds on the outside and remains outside	Yes, it diffuses through the phospholipid bilayer
Typical response	Signal passed in by ion channels, G proteins or enzymes	Activated receptor binds DNA and changes gene expression

Signal transduction pathways

Binding of the ligand is only the start. It causes a conformational change in the receptor that sets off a **signal transduction pathway**: a sequence of responses within the cell that converts the outside signal into a change inside.

- The steps can involve ion movement, relay proteins, second messengers (such as cAMP) and enzymes that add phosphate groups to other proteins.
- Each activated molecule can activate many molecules in the next step, so a few ligand molecules can produce a large response (amplification).
- The final response may be a change in membrane potential, in enzyme activity, in the movement of vesicles or in gene expression.



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Transmembrane receptors bind ligands that stay outside; **intracellular receptors** bind hydrophobic ligands that enter the cell.

Exam tip: When asked to distinguish the receptor types, give paired contrasts: position, distribution of hydrophilic and hydrophobic amino acids, and whether the ligand enters the cell.

Ion channel receptors and G protein-coupled receptors Spec C2.1.8, C2.1.9, C2.1.10

Receptors for neurotransmitters: the acetylcholine receptor

1. Acetylcholine binds to binding sites on the acetylcholine receptor in the postsynaptic membrane.
2. The receptor changes shape and opens an **ion channel** within the receptor itself.
3. Positively charged ions, mainly Na^+ , diffuse into the cell down their concentration gradient.
4. The inside becomes less negative: the voltage across the plasma membrane (the membrane potential) changes. This depolarization may cause other changes, such as an action potential in a

neuron or contraction of a muscle fibre.

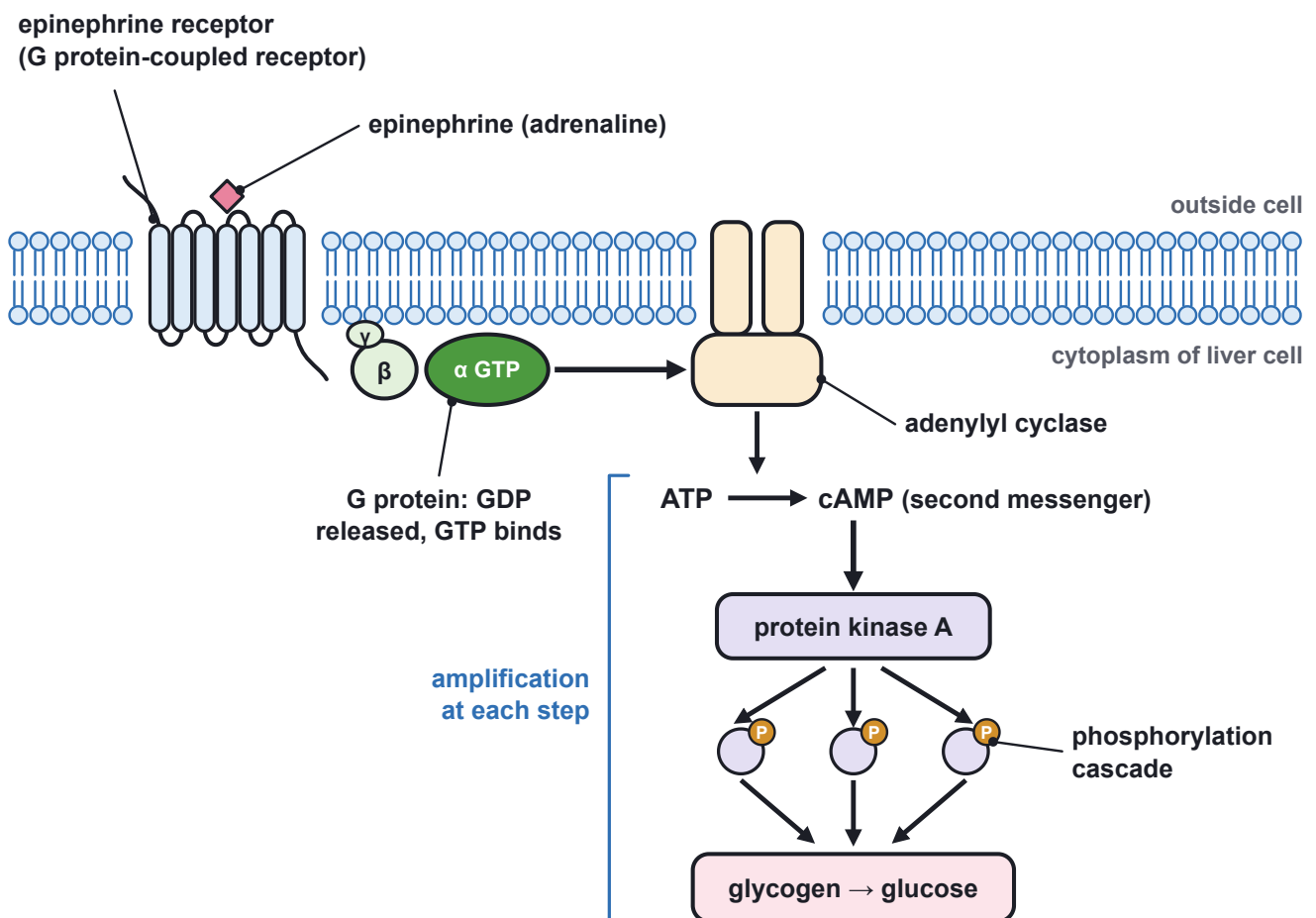
G protein-coupled receptors

A **G protein-coupled receptor** (GPCR) is a transmembrane receptor linked on the inside of the membrane to a G protein. Humans have many different GPCRs (hundreds of genes), detecting ligands such as hormones, neurotransmitters, odours and light.

1. The ligand binds to the receptor on the outside of the plasma membrane.
2. The receptor changes conformation and activates a G protein on the cytoplasmic side.
3. The G protein releases GDP and binds GTP. Part of it (the α subunit) separates and activates or inhibits a target protein, such as an enzyme or an ion channel.
4. The G protein switches itself off by hydrolysing GTP to GDP, so the signal stops when ligand binding stops.

Epinephrine (adrenaline) receptors

1. Epinephrine is carried in the blood and binds to its receptor (a GPCR) on a target cell such as a liver cell. It does not enter the cell.
2. The receptor activates a G protein, which binds GTP.
3. The activated G protein activates the enzyme adenylyl cyclase in the membrane.
4. Adenylyl cyclase converts ATP into **cyclic AMP (cAMP)**, the **second messenger**.
5. cAMP activates protein kinase A, which phosphorylates other enzymes in a cascade. In liver cells this activates glycogen breakdown, releasing glucose into the blood.



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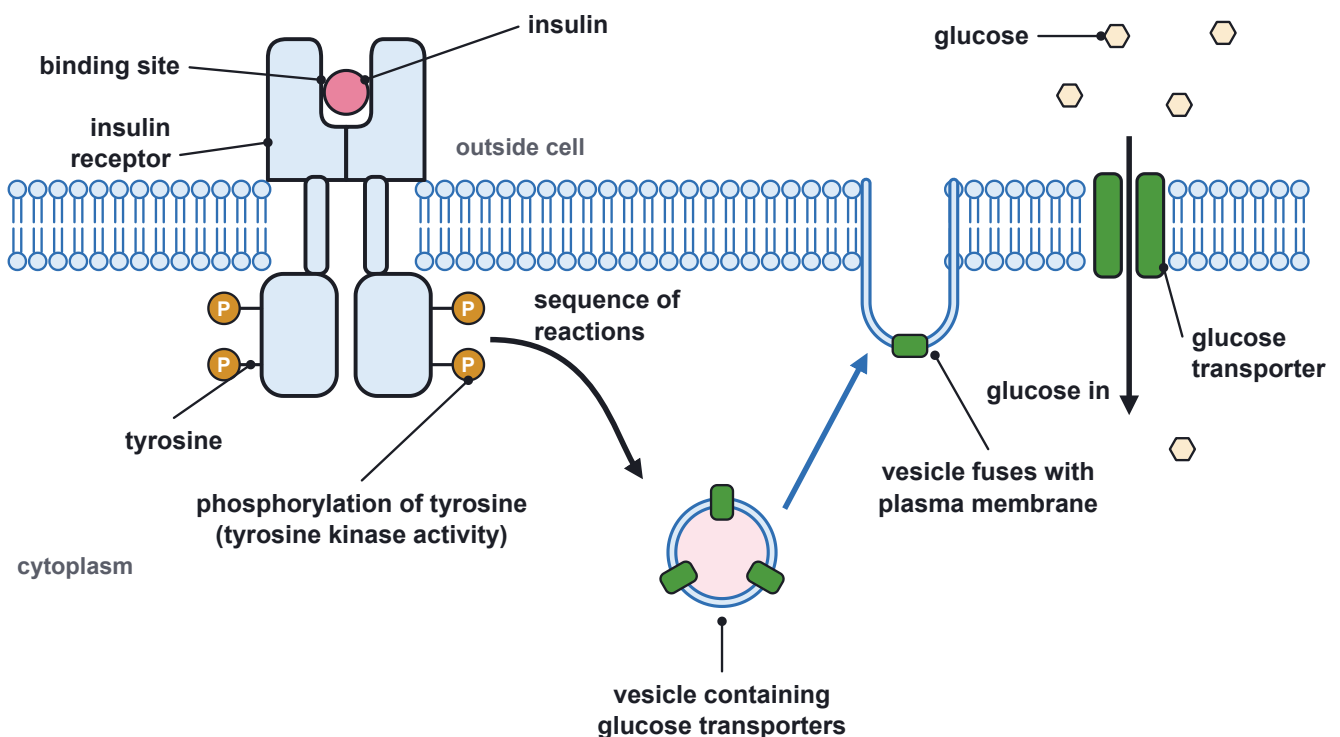
Epinephrine acts through a G protein and the second messenger **cAMP**; each step amplifies the signal.

Exam tip: Epinephrine and adrenaline are the same hormone. Both names come from its production by the adrenal gland, above the kidney; an example of naming conventions that persist side by side in international science.

Tyrosine kinase receptors: insulin Spec C2.1.11

Insulin is a protein hormone, so it cannot cross the plasma membrane. It binds to a transmembrane receptor whose cytoplasmic part acts as an enzyme: a **tyrosine kinase**, which adds phosphate groups to the amino acid tyrosine.

1. Insulin binds to the binding site of the insulin receptor on the outside of the plasma membrane.
2. The receptor changes conformation and its tyrosine kinase activity is switched on.
3. Phosphate groups (from ATP) are added to tyrosine residues on the part of the receptor inside the cell: **phosphorylation of tyrosine**.
4. Relay proteins recognise the phosphorylated tyrosines and start a sequence of reactions in the cytoplasm.
5. This ends with **vesicles containing glucose transporters** moving to the plasma membrane and fusing with it.
6. With more glucose transporters in the membrane, glucose enters the cell faster by facilitated diffusion, lowering blood glucose.



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Insulin binding causes **phosphorylation of tyrosine**, which leads to more glucose transporters in the plasma membrane.

Common mistake: Insulin does not enter the cell, and it does not carry glucose. It changes how many glucose transporters are in the plasma membrane.

Intracellular receptors and steroid hormones Spec C2.1.12, C2.1.13

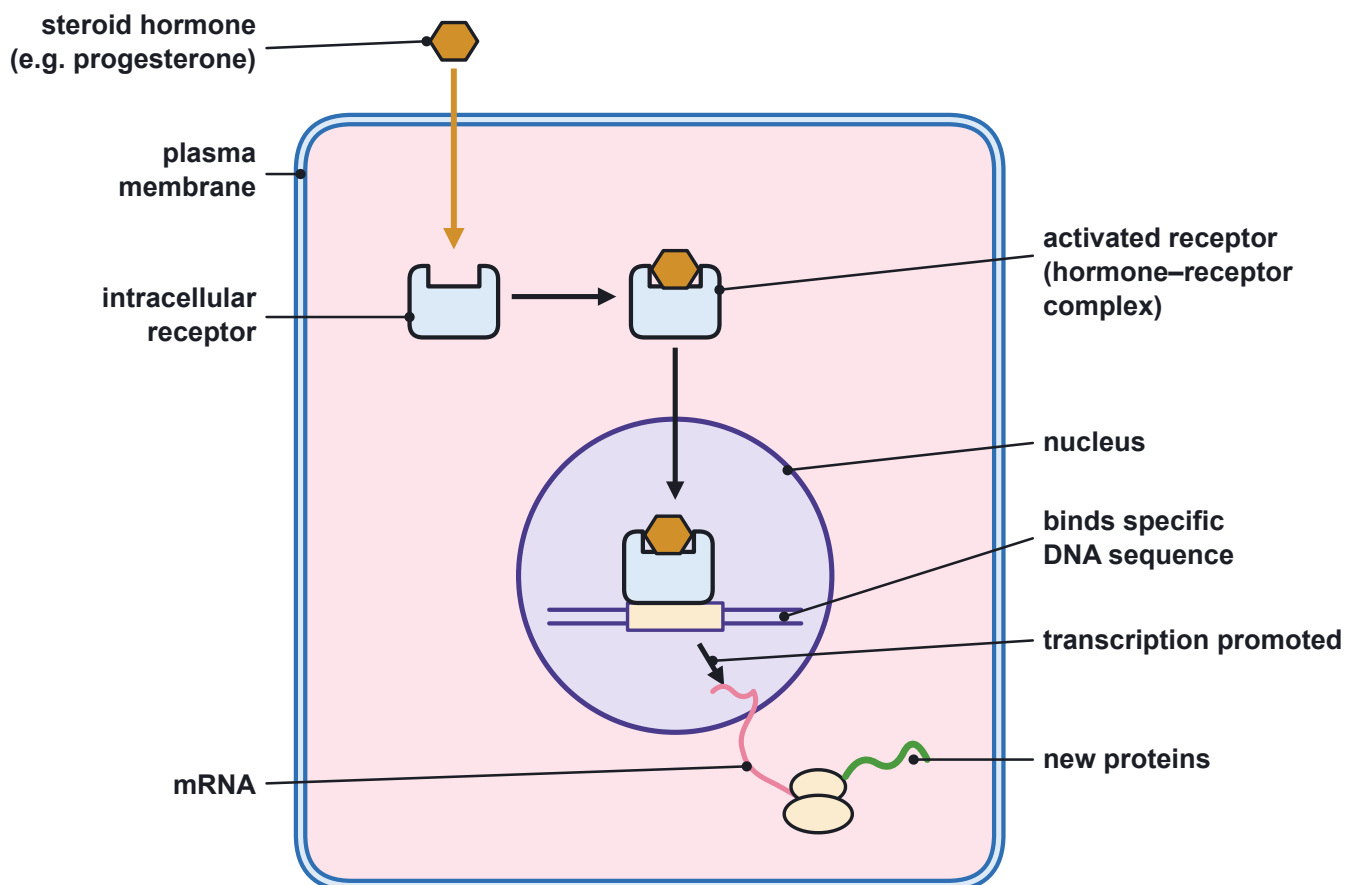
1. A steroid hormone diffuses through the phospholipid bilayer of the target cell.

2. It binds to a binding site on its specific receptor in the cytoplasm or nucleus, activating the receptor.
3. The activated hormone–receptor complex binds to **specific DNA sequences** next to target genes.
4. This **promotes transcription** of those genes, so new proteins are made and the cell's activity changes.

Examples are **oestradiol**, **progesterone** and **testosterone**. Because the response needs transcription and translation, steroid effects are slower to start but longer lasting than effects through membrane receptors.

Effects on target cells

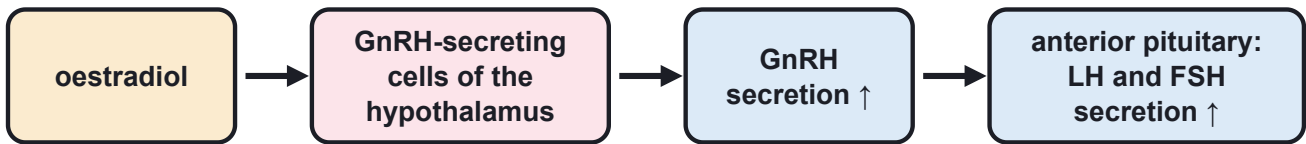
Two steroid hormones and their target cells		
Hormone	Target cells	Effect
Oestradiol	Cells in the hypothalamus that secrete gonadotropin-releasing hormone (GnRH)	Changes how much GnRH these cells secrete. At low or moderate concentrations, oestradiol inhibits GnRH secretion (negative feedback). When oestradiol stays high late in the follicular phase, it increases GnRH secretion (positive feedback). More GnRH raises LH and FSH secretion by the anterior pituitary, giving the LH surge that triggers ovulation.
Progesterone	Cells in the endometrium (uterus lining)	Maintains the thickened endometrium and promotes its further development, including glands and blood supply, ready for implantation. When progesterone falls, the endometrium breaks down (menstruation).



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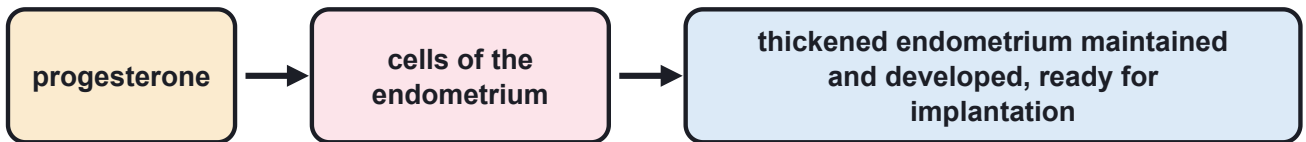
A steroid hormone binds an intracellular receptor, which then binds DNA and **promotes transcription**.

Oestradiol (high, late in the follicular phase)



At low or moderate concentrations, oestradiol inhibits GnRH secretion instead (negative feedback).

Progesterone



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Oestradiol acts on **GnRH-secreting cells of the hypothalamus**; progesterone acts on **cells of the endometrium**.

Exam tip: For C2.1.13 the guide limits you to two targets: oestradiol on GnRH-secreting hypothalamus cells and progesterone on endometrium cells. Answer about those, not the whole menstrual cycle, which is in D3.1.

Positive and negative feedback in cell signalling Spec C2.1.14

Two forms of regulation		
Feature	Negative feedback	Positive feedback
Effect of the response on the stimulus	Reduces it	Increases it
Result	Signal is damped; conditions return towards a set point	Signal is amplified; a rapid, large change
How it ends	Continuous, keeps things stable	Needs an outside event to stop the cycle

Negative feedback example: insulin

A rise in blood glucose stimulates insulin secretion. Insulin signalling makes cells take up glucose, so blood glucose falls. Lower glucose reduces insulin secretion, so the signal switches itself off.

Positive feedback example: oestradiol and GnRH

Late in the follicular phase, the growing follicle keeps oestradiol concentration high. Oestradiol now increases GnRH secretion by the hypothalamus, instead of inhibiting it as it does at lower concentrations. More GnRH raises FSH and LH, which stimulate the follicle to secrete even more oestradiol. The cycle builds to the LH surge and ends with ovulation.

Exam tip: Linking question: in what ways is negative feedback evident at all levels of biological organization? Here it acts on a signalling pathway; in C3.1 it controls heart rate and ventilation rate, and in D3.3 it keeps body temperature and blood glucose near their set points.

Quick check

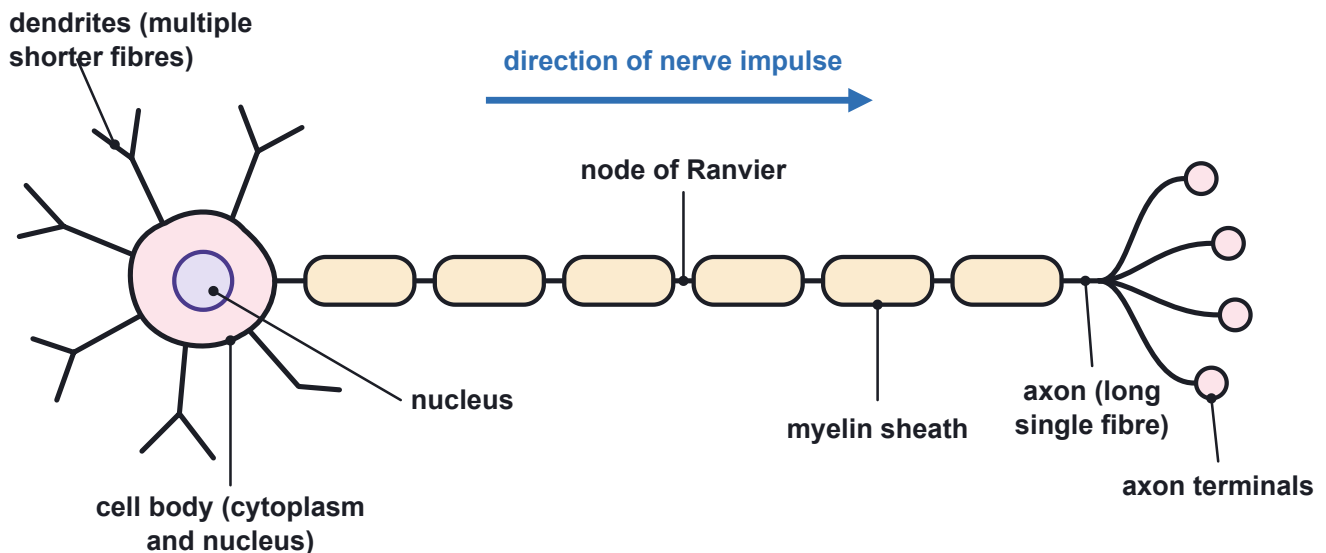
1. **HL only** What term does the IB use for a signalling chemical that binds to a receptor?
2. **HL only** Which marine bacterium is the example of quorum sensing?
3. **HL only** What is the second messenger in the epinephrine pathway?
4. **HL only** Which amino acid in the insulin receptor is phosphorylated?
5. **HL only** Where are the receptors for steroid hormones?

2 Neural signalling Spec C2.2

Neurons and the resting potential Spec C2.2.1, C2.2.2

Neurons are cells within the nervous system that carry electrical impulses. The **cell body** contains the cytoplasm and nucleus. Elongated nerve fibres of varying length project from it.

- **Axon:** a long single fibre that carries impulses away from the cell body. Some are over a metre long.
- **Dendrites:** multiple shorter fibres that receive signals from other neurons.
- Electrical impulses are conducted along these fibres.



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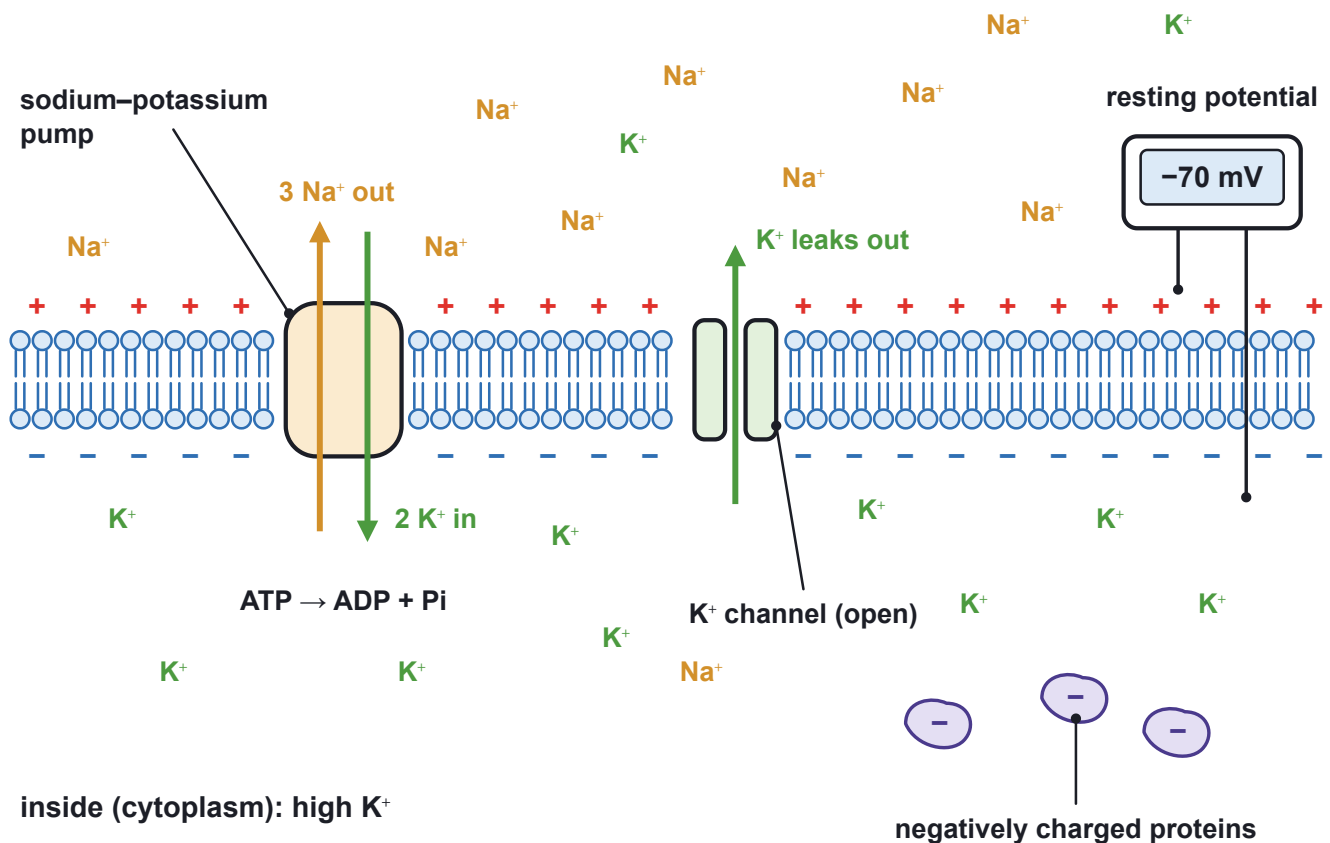
A motor neuron: impulses travel from the **dendrites** and cell body along the **axon** to the axon terminals.

The resting potential

A **membrane potential** is the voltage across a membrane. A neuron that is not conducting an impulse is **polarized**: there is a charge difference across its plasma membrane, with the inside negative. This is the **resting potential**, typically about -70 mV.

1. Sodium-potassium pumps in the plasma membrane use energy from ATP.
 2. Each cycle pumps three Na^+ out of the neuron and two K^+ in: the two ions move in opposite directions.
 3. This establishes and maintains concentration gradients: Na^+ is more concentrated outside, K^+ more concentrated inside.
- More positive charge is pumped out than in (3 Na^+ out for 2 K^+ in).
 - The membrane is much more permeable to K^+ than to Na^+ , so K^+ leaks back out through open channels faster than Na^+ leaks in.
 - The cytoplasm contains many negatively charged proteins and other organic ions that cannot leave.
 - Together these make the inside negative relative to the outside.

outside: high Na^+



inside (cytoplasm): high K^+

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The **resting potential** (about -70 mV): pumps set up the Na^+ and K^+ gradients and K^+ leaks out faster than Na^+ leaks in.

Nerve impulses as action potentials Spec C2.2.3

A **nerve impulse** is an **action potential** that travels along a nerve fibre. At any one point on the fibre the action potential is a brief, rapid change in membrane potential.

- **Depolarization:** Na^+ diffuses into the fibre, so the inside becomes positive.
- **Repolarization:** K^+ diffuses out, so the inside becomes negative again.
- Each part of the fibre that depolarizes causes the next part to depolarize, so the action potential is **propagated** along the fibre.

A nerve impulse is **electrical** because it involves the movement of positively charged ions across the plasma membrane. Electrons do not flow along the axon as they do in a wire.

Common mistake: Do not write that an impulse is 'electricity flowing down the nerve'. Write that an action potential involves movement of Na^+ and K^+ ions across the membrane.

Depolarization, repolarization and oscilloscope traces Spec C2.2.8, C2.2.9, C2.2.10 HL only

Depolarization and repolarization

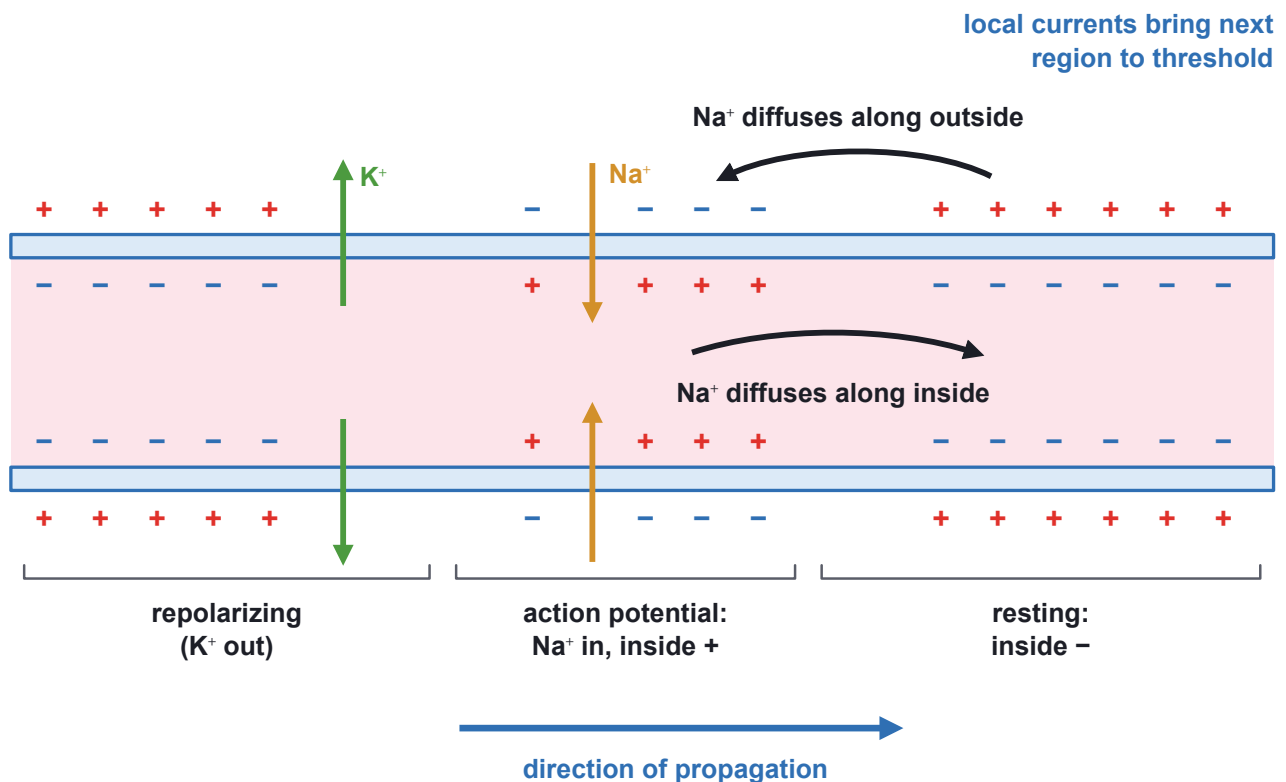
1. A stimulus or local current makes the membrane potential less negative. If it reaches the **threshold potential** (about -55 mV), **voltage-gated sodium channels** open. Below threshold they stay closed and no action potential occurs.
2. Na^+ diffuses in down its concentration gradient. The entry of Na^+ depolarizes the membrane further, opening more sodium channels, until the inside reaches about $+30 \text{ mV}$ (depolarization).
3. The sodium channels close, and **voltage-gated potassium channels** open.

4. K^+ diffuses out, making the inside negative again (repolarization). The potential briefly falls below the resting potential before the potassium channels close.
5. Sodium-potassium pumps maintain the concentration gradients, so the neuron can carry further impulses.

Propagation by local currents

Na^+ entering a depolarized region diffuses along the inside of the axon towards the next, still polarized region. Outside the membrane, Na^+ diffuses from the next region towards the depolarized region. These **local currents** make the potential in the next region less negative until the threshold potential is reached and its sodium channels open.

The region just behind the action potential is still repolarizing and its sodium channels cannot reopen yet, so the impulse travels in one direction only.



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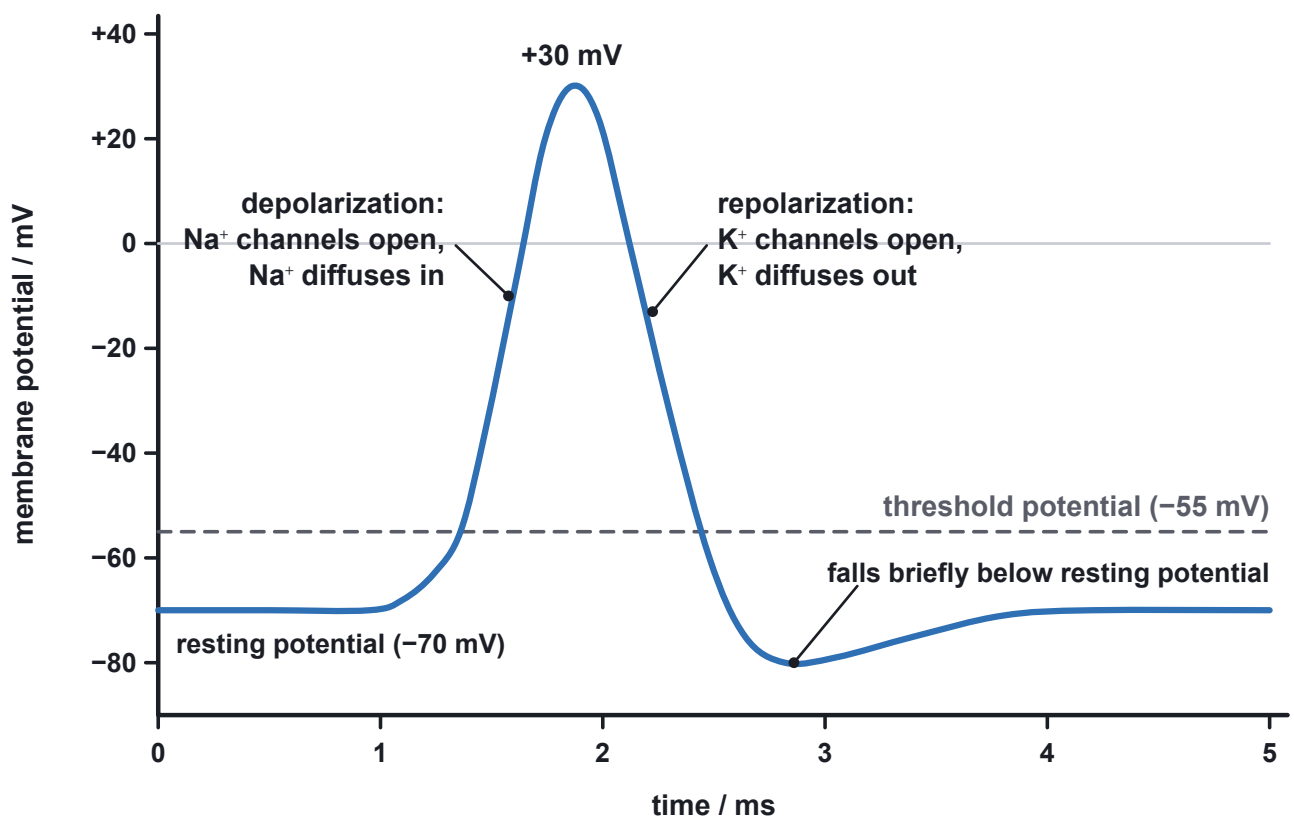
Local currents of Na^+ bring the next region of the axon to the threshold potential.

Oscilloscope traces

An oscilloscope trace plots membrane potential (mV) on the y-axis against time (ms) on the x-axis. You should link each part of the trace to events in the membrane.

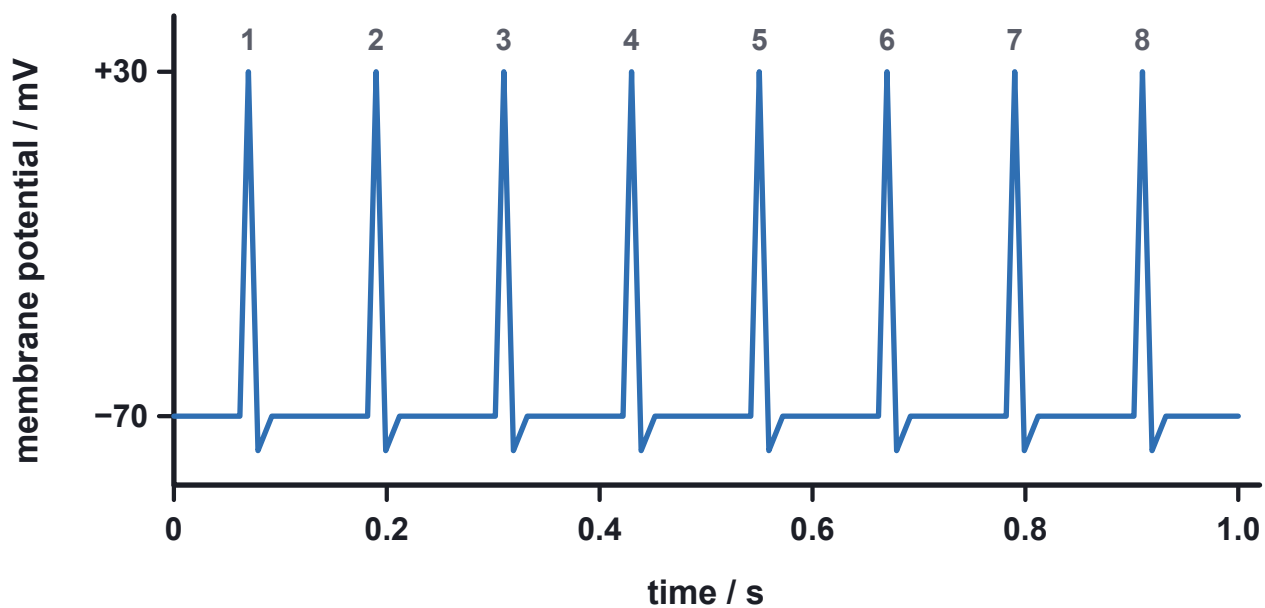
Reading an oscilloscope trace of one action potential

Part of trace	Membrane potential	Cellular event
Flat line before the spike	About -70 mV	Resting potential maintained by pumps and K^+ leak
Small rise to threshold	-70 to -55 mV	Local currents or stimulus depolarize the membrane
Steep rise	-55 to about $+30$ mV	Voltage-gated Na^+ channels open; Na^+ diffuses in
Steep fall	$+30$ mV to below -70 mV	Na^+ channels close; voltage-gated K^+ channels open; K^+ diffuses out
Return to resting level	Back to about -70 mV	K^+ channels close; resting potential restored



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Oscilloscope trace of one action potential (typical values): -70 mV resting, -55 mV threshold, peak about $+30$ mV.



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Counting impulses: eight action potentials in 1.0 s is a frequency of 8 impulses per second (illustrative trace).

Number of impulses per second

An oscilloscope trace shows 18 action potentials in a 0.30 s recording. Calculate the frequency of impulses.

1. frequency = number of impulses $\div$ time

2. = $18 \div 0.30$ s

Answer: 60 impulses per second (60 Hz)

Practical skill: Read the time scale before counting spikes. A stronger stimulus gives more impulses per second, not taller action potentials: each action potential reaches the same peak.

Speed of nerve impulses Spec C2.2.4, C2.2.11

The speed of nerve impulses varies a lot between nerve fibres. Two factors matter most: **axon diameter** and **myelination**.

Approximate conduction speeds (typical textbook values)		
Fibre	Diameter	Approximate speed
Small non-myelinated fibre	About 1 μm	About 1 m s^{-1}
Squid giant axon (non-myelinated)	Up to about 0.5-1 mm	About 25 m s^{-1}
Myelinated vertebrate fibre	About 10-20 μm	Up to about 100 m s^{-1}

- **Squid giant axons vs smaller non-myelinated fibres:** a wider axon has less resistance to the flow of ions inside it, so local currents spread further and faster. The giant axon allows a rapid escape response.
- **Myelinated vs non-myelinated fibres:** myelinated fibres conduct far faster, even when much narrower, so many fast fibres fit into a small nerve.

Correlation and the coefficient of determination

- A **positive correlation**: one variable rises as the other rises, e.g. conduction speed and axon diameter.
- A **negative correlation**: one variable falls as the other rises. The guide's example is conduction speed and animal size. Published data for this vary between groups of animals, so in an exam describe the trend that the data you are given actually show.
- The correlation coefficient, r , runs from -1 to $+1$. Values near ± 1 show a strong correlation; values near 0 show little or none.
- The coefficient of determination, R^2 , is r squared. It gives the proportion of the variation in the dependent variable that is explained by variation in the independent variable.
- Correlation does not by itself show causation.

Using r and R^2

For a set of non-myelinated axons, the correlation coefficient between axon diameter and conduction speed is $r = 0.90$. Calculate R^2 and interpret it.

1. $R^2 = r^2 = 0.90^2$
2. $R^2 = 0.81$
3. $0.81 = 81\%$: 81% of the variation in conduction speed is explained by variation in axon diameter.
4. The other 19% is due to other factors, such as temperature or measurement error.

Answer: $R^2 = 0.81$; a strong positive correlation, with 81% of the variation in speed explained by diameter

Maths skill: State the direction (positive or negative) and strength of a correlation from r . Use R^2 only to say how much of the variation is explained, and never claim it proves cause.

HL only

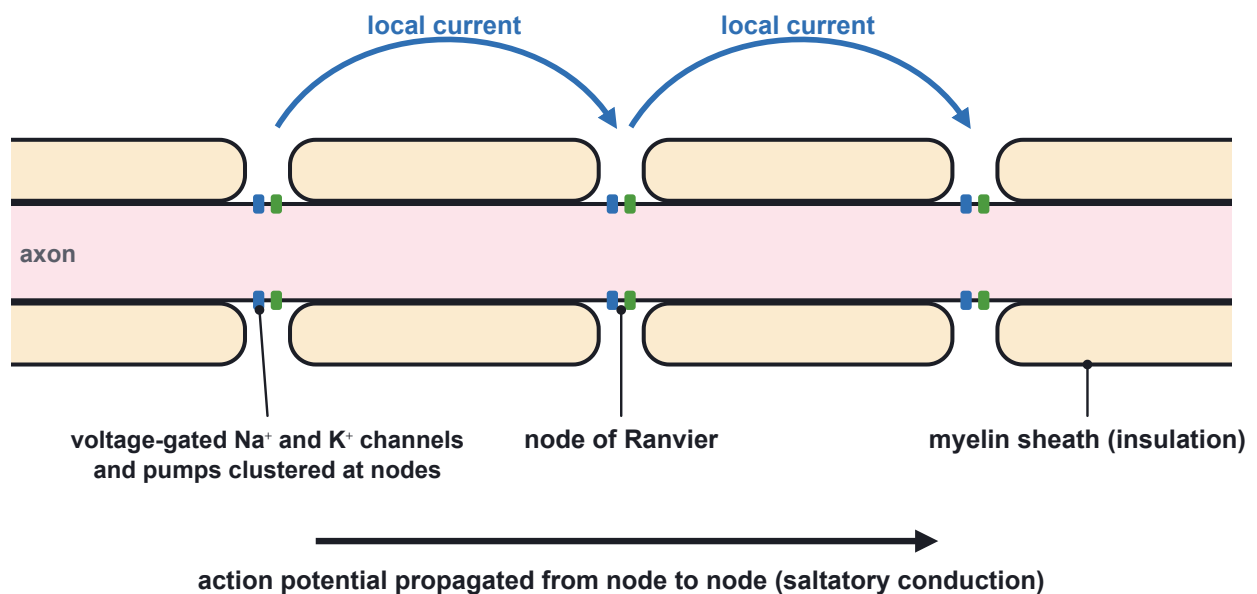
Saltatory conduction

HL only

In myelinated fibres the axon is wrapped in a **myelin sheath** that insulates it, with small gaps called **nodes of Ranvier**. The ion pumps and channels are clustered at the nodes, so an action potential can only occur there.

HL only

1. An action potential occurs at one node.
2. Local currents spread inside the axon, under the myelin, to the next node.
3. The next node reaches threshold, its voltage-gated sodium channels open and an action potential occurs there.
4. The action potential is propagated from node to node. This **saltatory conduction** is much faster than continuous propagation along the whole membrane.



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Saltatory conduction: the action potential jumps from one node of Ranvier to the next.

Synapses and excitatory postsynaptic potentials Spec C2.2.5, C2.2.6, C2.2.7

Synapses are junctions between neurons and between neurons and effector cells, such as muscle fibres at **neuromuscular junctions**. At a chemical synapse (called simply a synapse here) the cells are separated by a narrow **synaptic cleft**.

A signal can only pass in one direction across a typical synapse. Only the presynaptic neuron has vesicles of neurotransmitter, and only the postsynaptic membrane has the receptors.

Release of neurotransmitter from the presynaptic membrane

1. An action potential arrives and depolarizes the presynaptic membrane.
2. Voltage-gated calcium channels open and Ca^{2+} diffuses into the presynaptic neuron (it is more concentrated outside).
3. Inside the neuron, **calcium ions act as a signalling chemical**: they cause vesicles of neurotransmitter to move to the presynaptic membrane and fuse with it.
4. The neurotransmitter is released into the synaptic cleft by exocytosis.

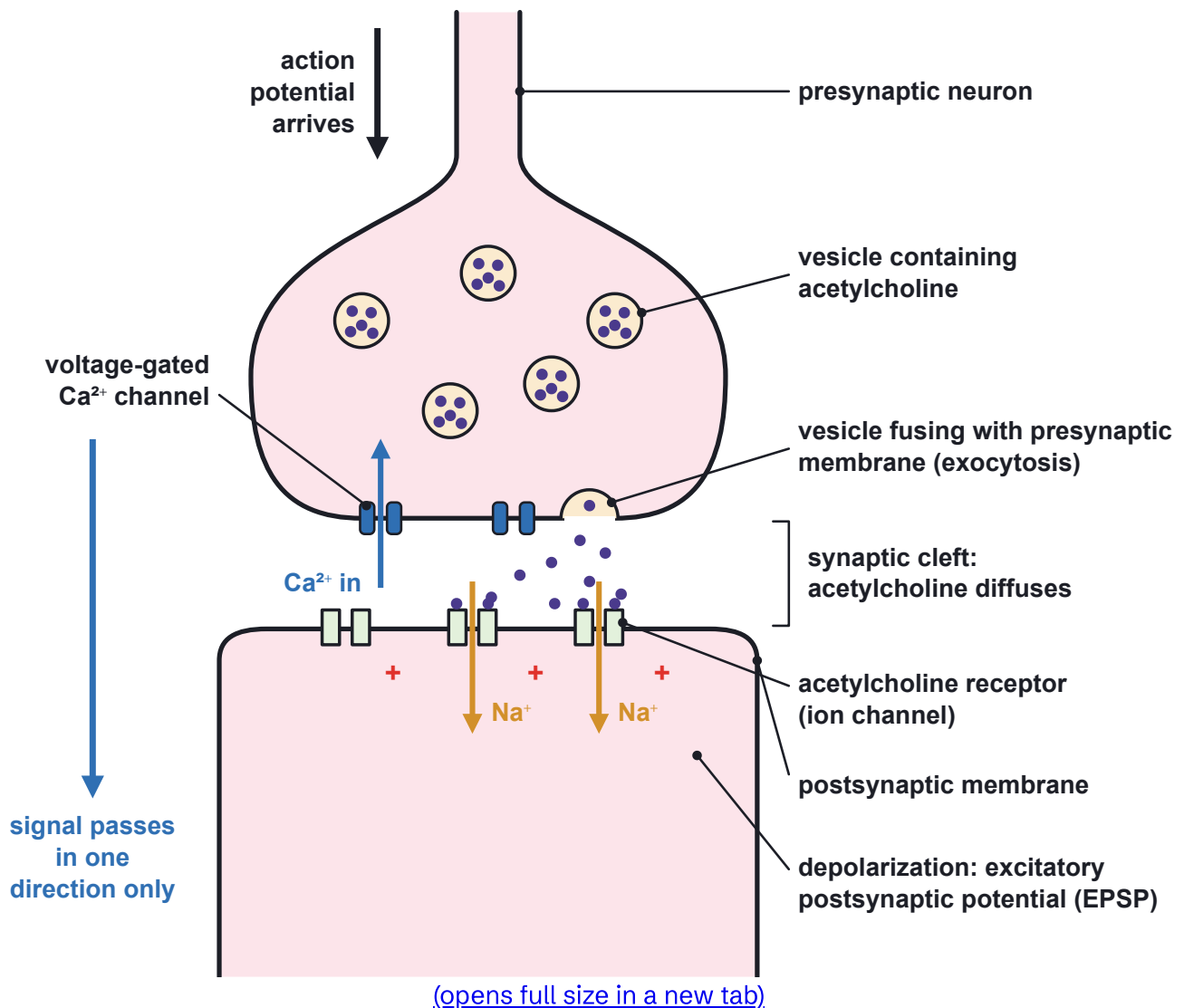
Excitatory postsynaptic potential (EPSP)

1. **Acetylcholine** diffuses across the synaptic cleft.
2. It binds to transmembrane receptors in the postsynaptic membrane.
3. The receptors open ion channels and positively charged ions, mainly Na^+ , diffuse into the postsynaptic cell.
4. The postsynaptic membrane is depolarized: an **excitatory postsynaptic potential**. If it reaches the threshold potential, an action potential starts in the postsynaptic cell.
5. The neurotransmitter is quickly removed from the synaptic cleft, so each signal is brief.

Beyond the syllabus: the guide does not name the enzyme that removes acetylcholine. It is acetylcholinesterase, which breaks acetylcholine into acetate and choline; the presynaptic neuron

reabsorbs the choline to make more acetylcholine.

Acetylcholine is used at many types of synapse, including neuromuscular junctions, where it triggers contraction of skeletal muscle fibres.



At a cholinergic synapse, Ca^{2+} entry triggers exocytosis of acetylcholine, which causes an EPSP in the postsynaptic cell.

Exam tip: Linking question: in what ways are biological systems regulated? Synapses are control points: whether a signal passes depends on how much neurotransmitter is released and how many receptors respond.

Exogenous chemicals, inhibition and summation

Spec C2.2.12, C2.2.13, C2.2.14

HL only

Effects of exogenous chemicals

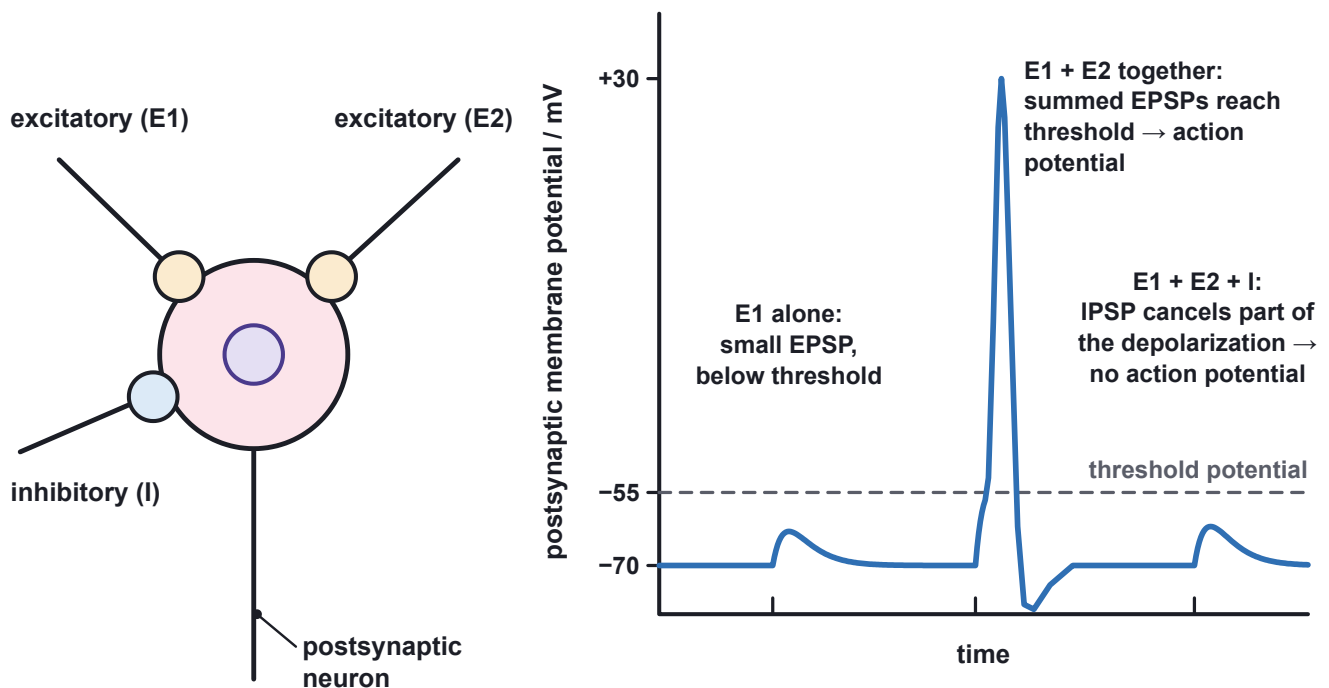
Two exogenous chemicals that affect synapses		
Chemical	Action at the synapse	Consequence
Neonicotinoids (pesticides)	Bind to acetylcholine receptors in the postsynaptic membrane of insect neurons. Unlike acetylcholine, they are not broken down by the enzyme in the synaptic cleft, so they stay bound and block synaptic transmission.	Paralysis and death of insects. They bind far more strongly to insect receptors than to mammalian ones, but they also harm useful insects such as bees.
Cocaine (drug)	Binds to the transporter proteins that take dopamine back into the presynaptic neuron, blocking reuptake of the neurotransmitter.	Dopamine builds up in the cleft and keeps stimulating postsynaptic neurons in the brain's reward pathways, causing euphoria and addiction.

Inhibitory postsynaptic potentials

Inhibitory neurotransmitters, such as GABA, bind to receptors that let negative ions (Cl^-) in or positive ions (K^+) out. The postsynaptic membrane becomes **hyperpolarized**: more negative than the resting potential. This **inhibitory postsynaptic potential** (IPSP) moves the membrane further from threshold, so an action potential is less likely.

Summation

- A postsynaptic neuron may receive synapses from many presynaptic neurons, some excitatory and some inhibitory.
- One EPSP is usually too small to reach threshold on its own.
- The effects add together (**summation**): EPSPs depolarize, IPSPs hyperpolarize, and the net change decides the outcome.
- The result is all-or-nothing: if the combined depolarization reaches the threshold potential, an action potential is generated; if not, there is no action potential.



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Summation: the neuron fires only if the combined EPSPs and IPSPs reach the threshold potential.

Exam tip: For an IPSP the guide's key word is hyperpolarized. Say the inside becomes more negative and the threshold is harder to reach.

Pain perception and consciousness Spec C2.2.15, C2.2.16 HL only

Perception of pain

1. Pain receptors are neurons with **free nerve endings** in the skin.
2. The nerve endings have **channels for positively charged ions**. They open in response to a stimulus such as high temperature, acid, or certain chemicals such as **capsaicin** in chilli peppers.
3. Positively charged ions enter the nerve ending and depolarize it until the threshold potential is reached.
4. Nerve impulses pass along the neurons to the brain.
5. Pain is perceived in the brain, not in the skin.

Capsaicin opens the same channels as high temperature, which is why chilli peppers feel hot even though they do not change the temperature of the mouth.

Consciousness as an emergent property

Consciousness is a property that **emerges** from the interaction of individual neurons in the brain. No single neuron is conscious. The property arises only from huge numbers of neurons (tens of billions in the human brain) interacting through their synapses.

Emergent properties are another example of the consequences of interaction: a whole system has properties that its parts do not have on their own, so they cannot be predicted by studying the parts separately.

Exam tip: Say pain is perceived in the brain. The receptors in the skin only generate nerve impulses.

Quick check

1. What are the two types of fibre that project from the cell body of a neuron?
2. How many sodium and potassium ions does one cycle of the sodium-potassium pump move?
3. Why can a signal only pass one way across a synapse?
4. What ion enters the presynaptic neuron and triggers neurotransmitter release?
5. **HL only** Where are ion pumps and channels clustered in a myelinated fibre?

Quick check answers

Chemical signalling

1. A ligand.
2. *Vibrio fischeri*, which becomes bioluminescent at high population density.
3. Cyclic AMP (cAMP).
4. Tyrosine.
5. Inside the cell, in the cytoplasm or nucleus.

Neural signalling

1. One long axon and multiple shorter dendrites.
2. Three Na⁺ out and two K⁺ in, using one ATP.
3. Only the presynaptic neuron releases neurotransmitter, and only the postsynaptic membrane has receptors for it.
4. Calcium ions (Ca²⁺).
5. At the nodes of Ranvier.