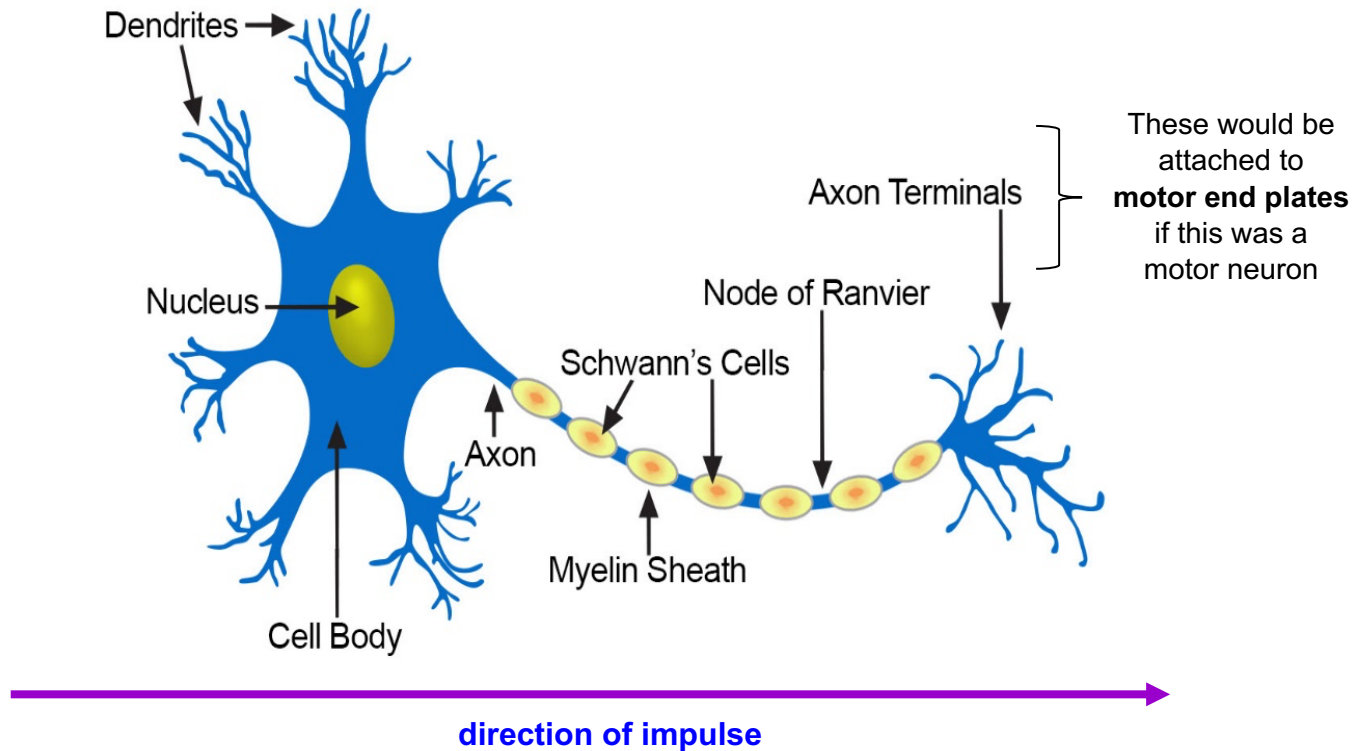


A. STRUCTURE OF A NEURON

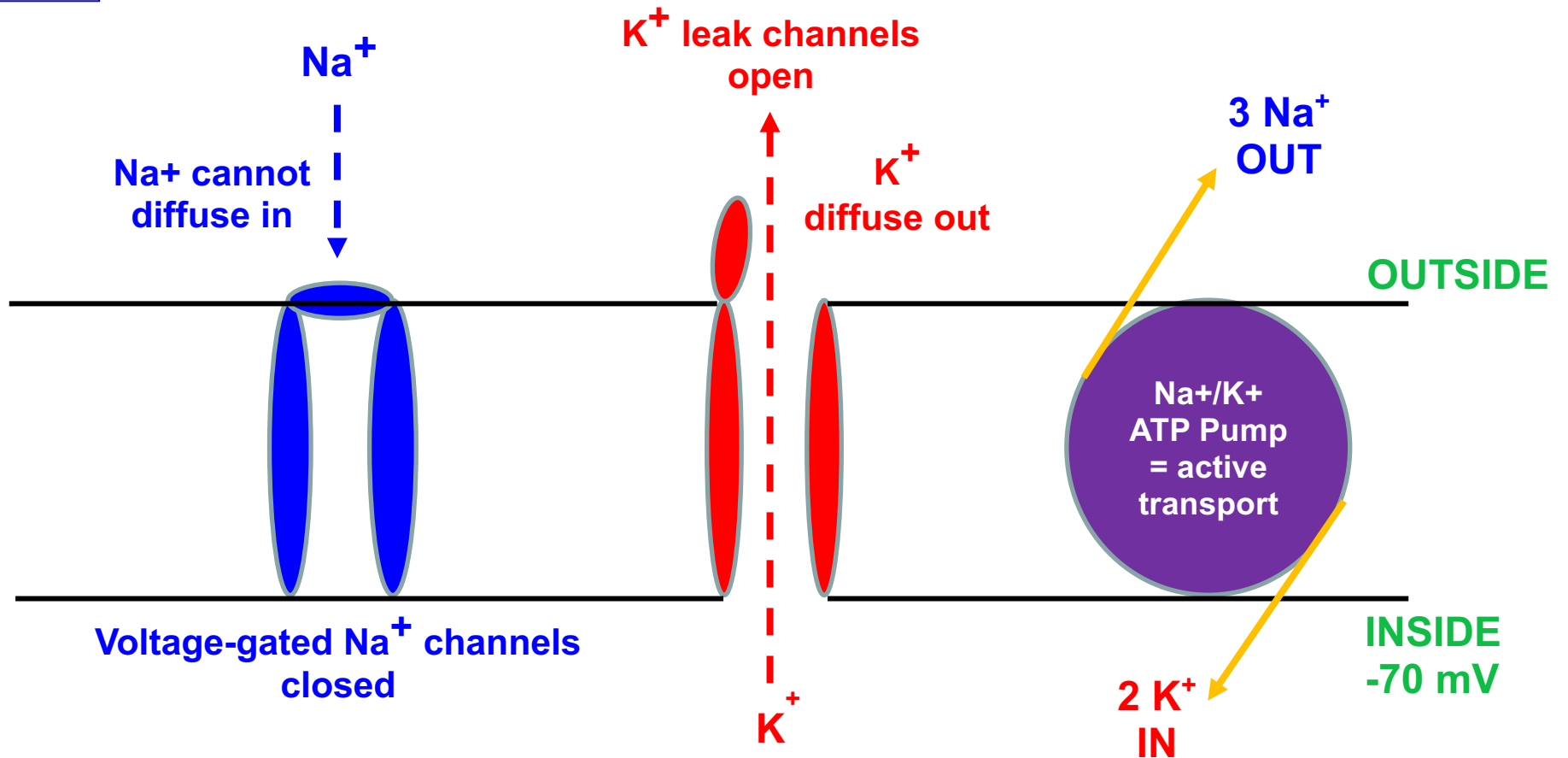


- The **axon terminals** can end at **another neuron**, a **muscle** (motor end plate) or a **gland**.

B. RESTING NEURONES AND FIRING NEURONES

- A **neuron** is **prevented** from **firing** an **impulse** by **keeping the inside negative** relative to the outside by **70 mV**.
- So, when neurons are **resting** they have a **resting membrane potential** of **-70 mV**.
- To **make** a neuron **fire** an **impulse**, this value must be made **more positive** so that it reaches at least **-55 mV**.
- If we "**zoom in**" on the **membrane** of a **neuron**, you will see how both are achieved:

AT REST

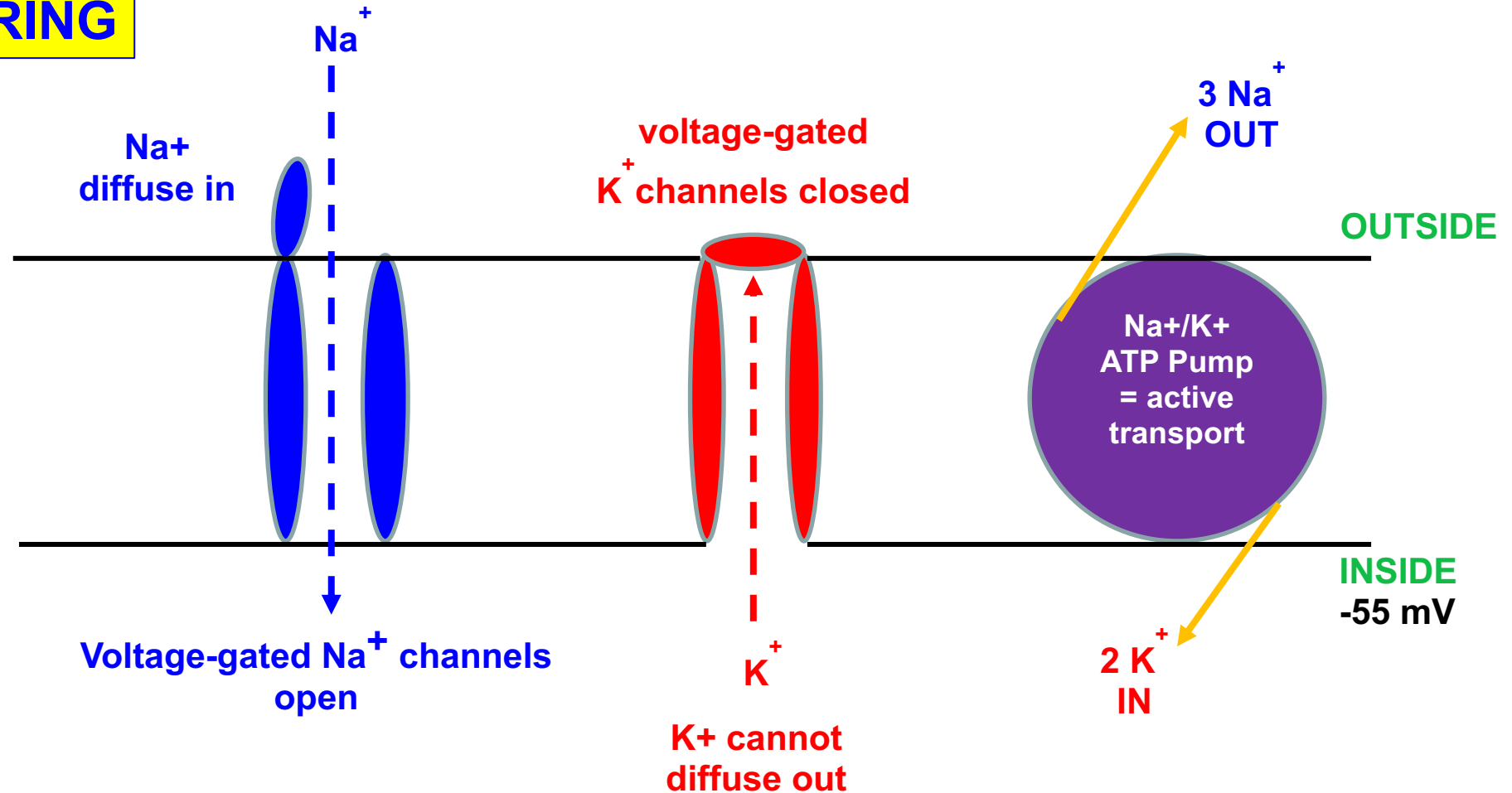


The **inside** of the **neurone membrane** is **negatively charged** compared to the **outside** by **70 mV**. This **prevents** a neurone from **firing an impulse**.

- Voltage-gated Na⁺ channels are closed so Na⁺ cannot diffuse in.
- K⁺ leak channels are open so K⁺ diffuses out.
- The Na⁺/K⁺ ATP Pump moves 3Na⁺ out for every 2K⁺ moved in by active transport.

All work together to maintain the resting membrane potential of -70 mV

FIRING

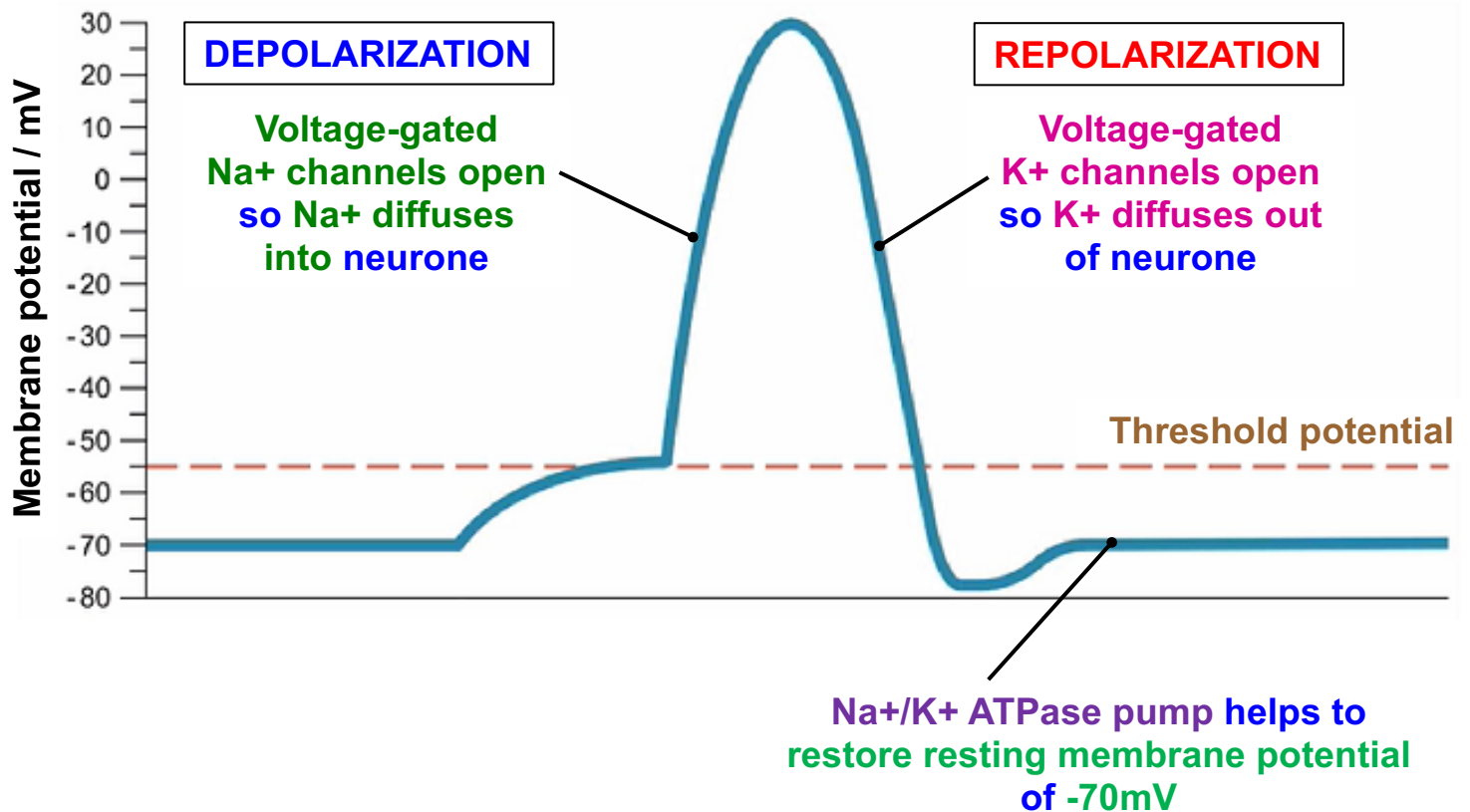


MEMBRANE POTENTIAL **INCREASES**: BECOMES MORE + (DEPOLARISATION)

At **-55 mV**, the neurone will **FIRE** an **IMPULSE** (= 'ACTION POTENTIAL')

C. AN OSCILLOSCOPE SHOWING AN ACTION POTENTIAL

- You **are** expected to know that the equipment used to show an action potential is called an **oscilloscope**.



D. FIRING AN ACTION POTENTIAL IS AN 'ALL-OR-NOTHING' RESPONSE

- The **threshold potential** of -55 mV must be **reached** for an **action potential** to be **fired** by a neuron.
- In other words, **enough Na^+** must **enter** to cause **enough depolarization** to **trigger firing** an **action potential**.
- If the **threshold potential** of -55 mV is **not** reached, the neuron stays at **rest**.

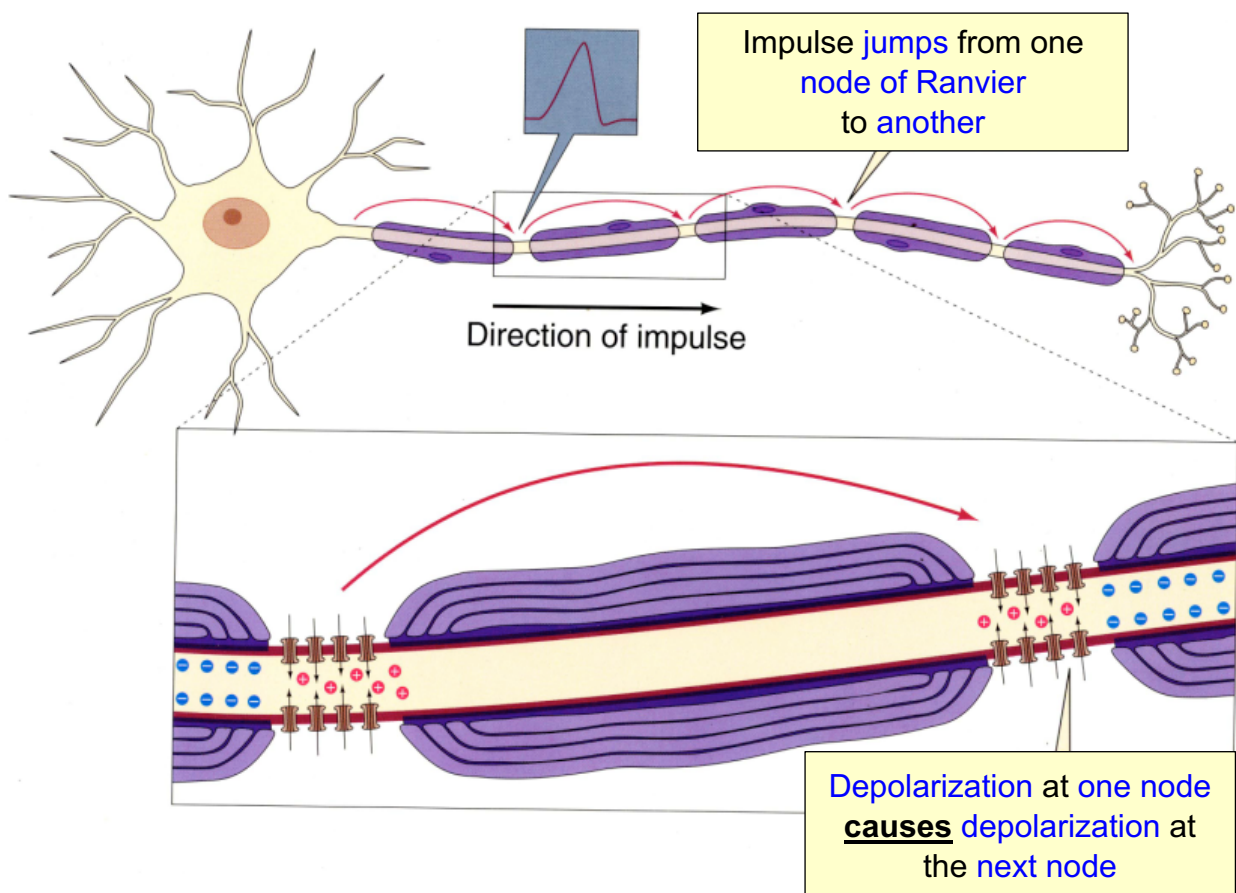
The **HEIGHT** of an **ACTION POTENTIAL** does **NOT CHANGE**.

The **STRENGTH** of a **STIMULUS** is conveyed by the **NUMBER OF ACTION POTENTIALS** produced in a **FIXED TIME**.

Accidentally hitting your thumb **hard** with a hammer will produce **more action potentials** in a fixed time than hitting it with a much **lower pressure**

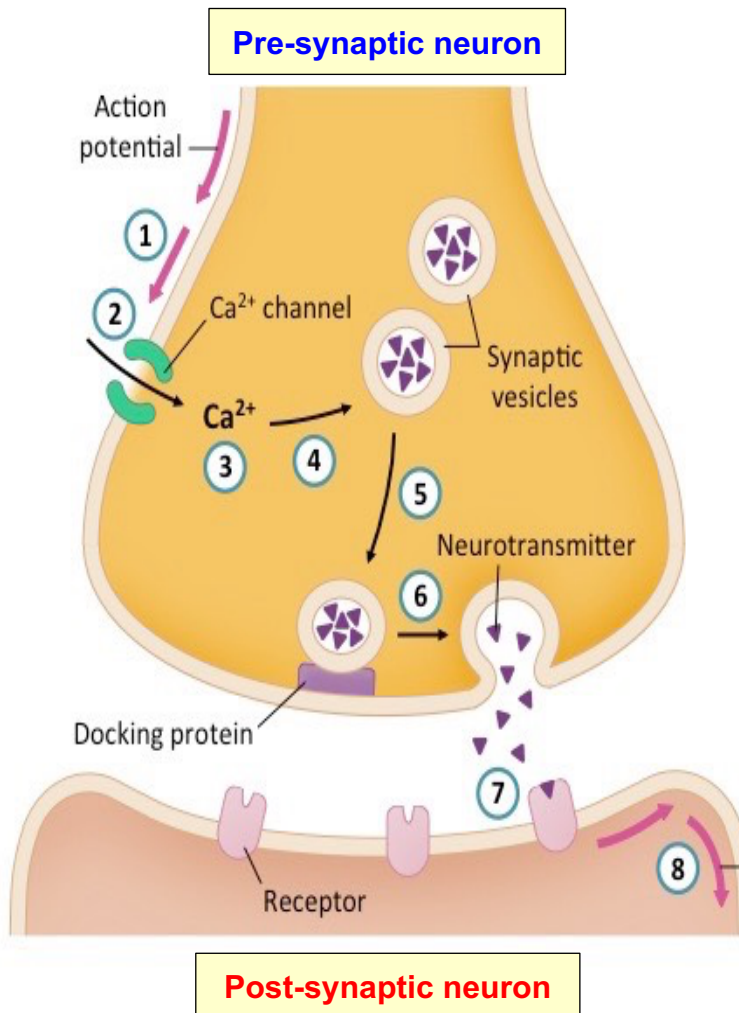
E. PROPAGATION OF AN ACTION POTENTIAL

- Some neurons are **wrapped** in a fatty material called **myelin**.
- In **myelinated** neurons, the **action potentials** 'jump' **between** the **gaps** in the myelin sheath, called the **nodes of Ranvier**.
- This is because depolarisation **only occurs** at the **nodes**.
- This is called **saltatory conduction**: **depolarisation at one node causes depolarisation at next node**.
- This results in an **increase** in the **speed** of **electrical transmission** by up to 100x.



F. HOW AN IMPULSE TRAVELS ACROSS A SYNAPSE

- Synapses are the **gaps** that separate **neurons** from other cells, such as **other neurons**, **muscle** cells or **gland** cells.
- Neurons **transmit information** across synapses by **converting** the **electrical signal** into a **chemical signal**.



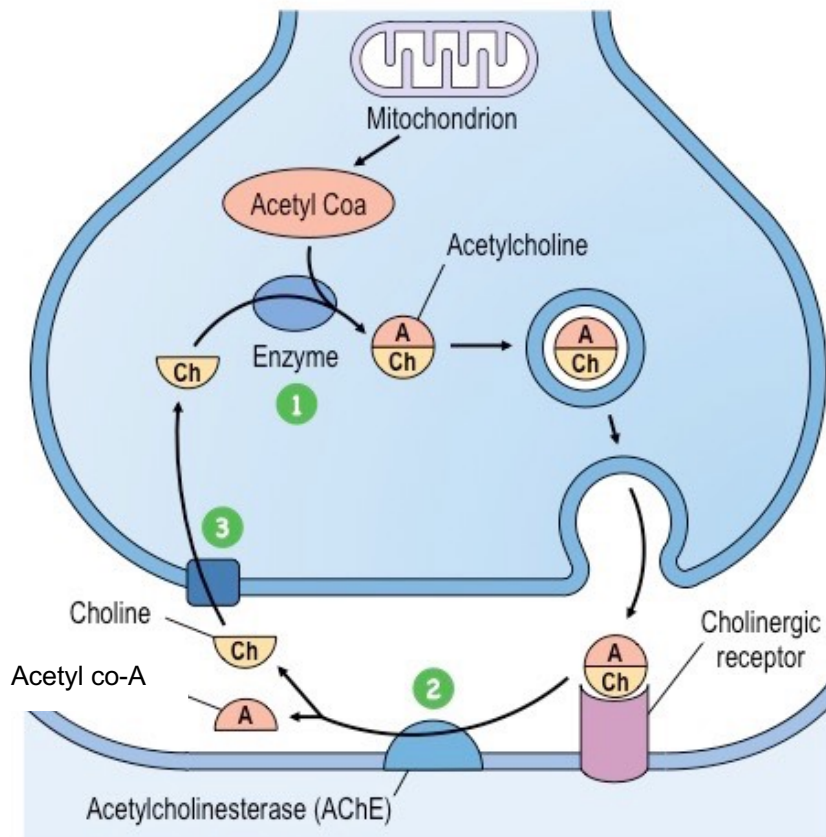
1. **Action potential** arrives at the **pre-synaptic neuron**
2. to 5.
Ca²⁺ channels open so **Ca²⁺ enters** neurone
This causes **vesicles** containing **neurotransmitter** to **move** to and **fuse** with the **presynaptic membrane**.
6. **Vesicles empty** the **neurotransmitter** into the **synapse** by **exocytosis**.
7. **Neurotransmitter diffuses** across the synapse and **attaches** to specific **receptors** on the **post-synaptic neurone**.
8. This causes **voltage-gated Na⁺ channels** to **open** so **Na⁺ enters**, causing **depolarisation**. An **action potential** is then **fired** by the next neuron.

G. NEUROTRANSMITTERS

- **Different synapses** can use **different neurotransmitters**.
- Synapses that use **acetylcholine** as the neurotransmitter are called **cholinergic synapses**.
- Other examples include: dopamine, serotonin, noradrenaline and GABA.
- **Excitatory** neurotransmitters **open Na⁺ channels** to cause **depolarization** and an **action potential** to be **fired**.
- **Inhibitory** neurotransmitters **open Cl⁻ channels** to causes **repolarization** and **prevent** an **action potential** from being **fired**.

H. HOW THIS IS “SWITCHED OFF”

- In synapses that use the neurotransmitter **acetylcholine (ACh)**, an enzyme called **acetylcholinesterase** breaks down acetylcholine, attached to the receptor, into **acetyl-coA** and **choline**.
- **Mitochondria** make **acetyl-coA** in the neuron.
- The **choline** is **reabsorbed** at the **pre-synaptic membrane**, back into the **first** neuron, and used to **make** more **acetylcholine**.



1 Acetylcholine (ACh) is made from choline and acetyl CoA

2 In the synapse, ACh is rapidly broken down by the enzyme **acetylcholinesterase (AChE)**

3 Choline is transported back into the axon terminal and used to make more ACh

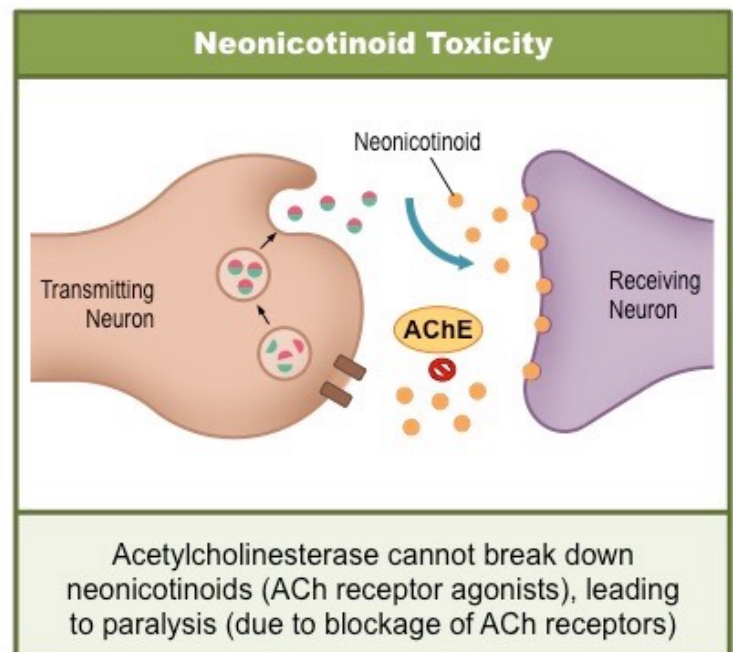
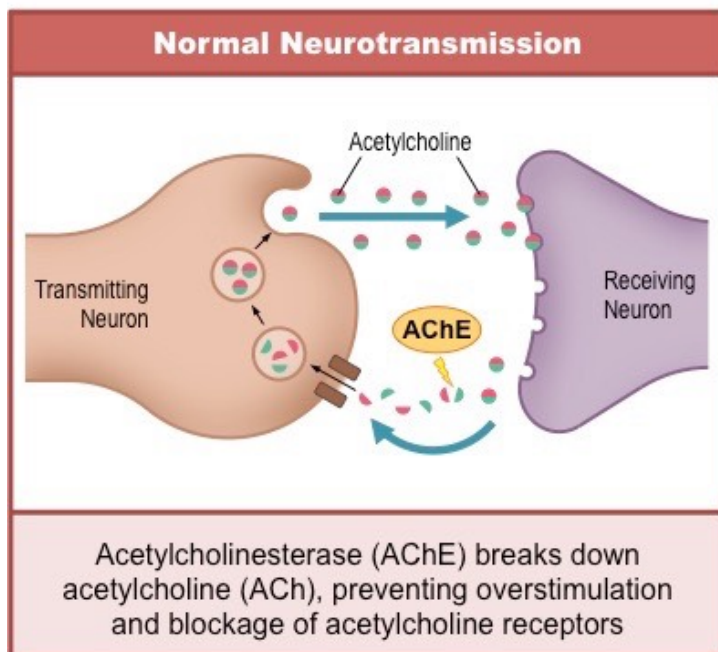
I. NEONICOTINOID PESTICIDES

Overview

- Kill **insects** by **binding** to **acetylcholine receptors**.
- **Human** and **insect** acetylcholine receptors have a **different structure**, so they bind to those of **insects** much **more strongly**.
- They are therefore much **more toxic** to **insects** than to humans.

How they work

- Bind to **acetylcholine (ACh) receptors**
- (But) **cannot be broken down** by the **enzyme acetylcholinesterase**
- (So) **block synaptic transmission** / **Na⁺ channels cannot open** (as it is not ACh)
- (So) **muscles cannot contract**
- (So) death by **paralysis**



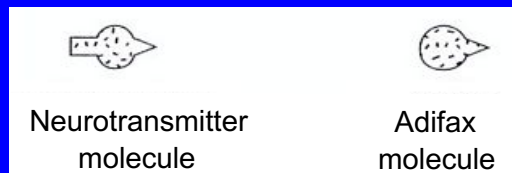
Many **MEDICAL DRUGS** exploit what happens naturally at the **SYNAPSE** to help treat the **SYMPTOMS** of a **DISORDER**

J. APPLYING WHAT YOU KNOW

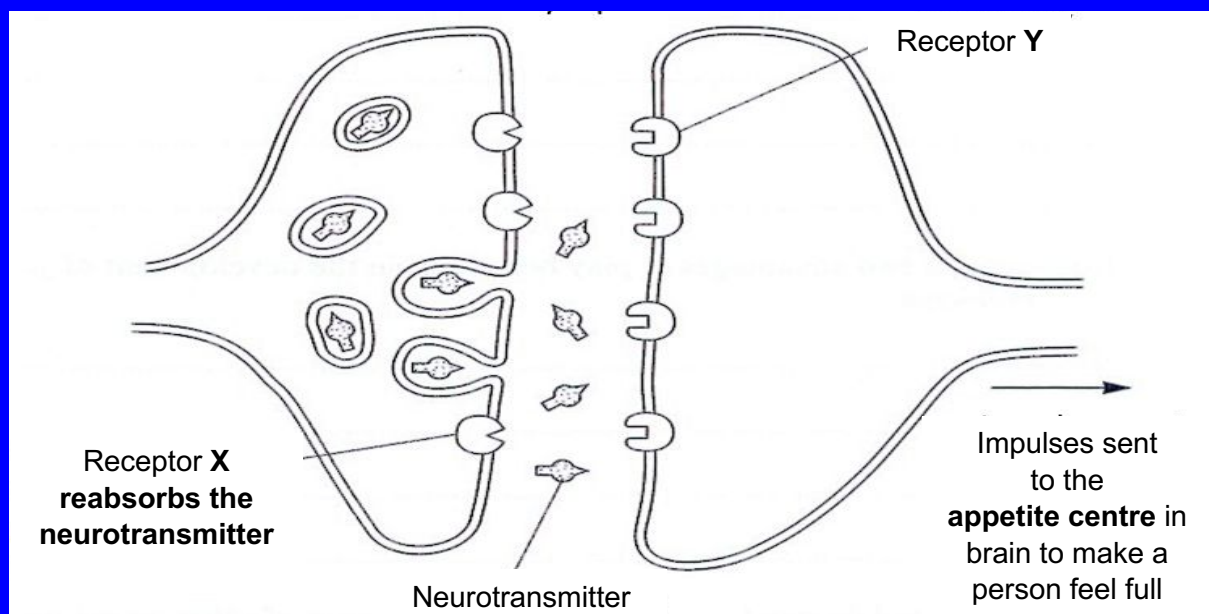
Adifax

Adifax is a drug that can be used to affect people's eating habits.

A neurotransmitter molecule and an adifax molecule are shown below.



The diagram shows how these molecules affect a synapse.



Explain how the drug Adifax would affect a person's eating habits. [6 marks]

- Adifax is a **similar shape/structure** to the **neurotransmitter**
- (So) adifax **binds** to receptor **X**
- (So) neurotransmitter **cannot bind** to receptor **X**
- (So) neurotransmitter **cannot be reabsorbed** / **stays bound** to receptor **Y**
- (So) **continuous impulses** sent to **appetite centre**
- (So) person **always feels full**

(Adifax is a diet pill)

Snake Venom

Venom from a certain species of snake contains molecules that have a similar structure to the neurotransmitter acetylcholine.

Explain how this venom can cause death by respiratory failure.

- (Venom molecules) **bind to acetylcholine receptors**
- (So) acetylcholine **cannot bind to receptors**
- (But) **no depolarization / Na⁺ channels do not open** (as it is not the real neurotransmitter)
- (So) **no action potential**/impulse fired
- (So) **muscles cannot contract**

If the **intercostal muscles** between the **ribs cannot contract (stay relaxed)**, the **ribcage cannot be moved**, so a person will **not be able to carry out ventilation**