Cambridge Assessment International Education

A2 Level Chapter 15

CONTROL AND CO-ORDINATION

Introduction

Q: How are messages sent from the receptor to the coordinating centre, and then to the effector?

A: Through **nerve impulses** and/or **hormones**!

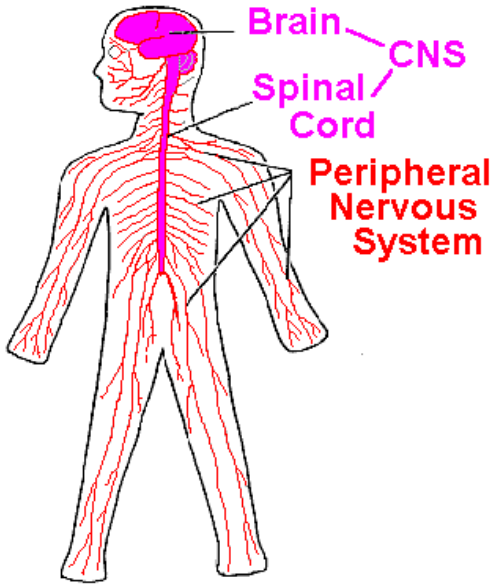
The nervous system and **the endocrine system** work together to monitor external/internal changes and coordinate responses.

Nervous System

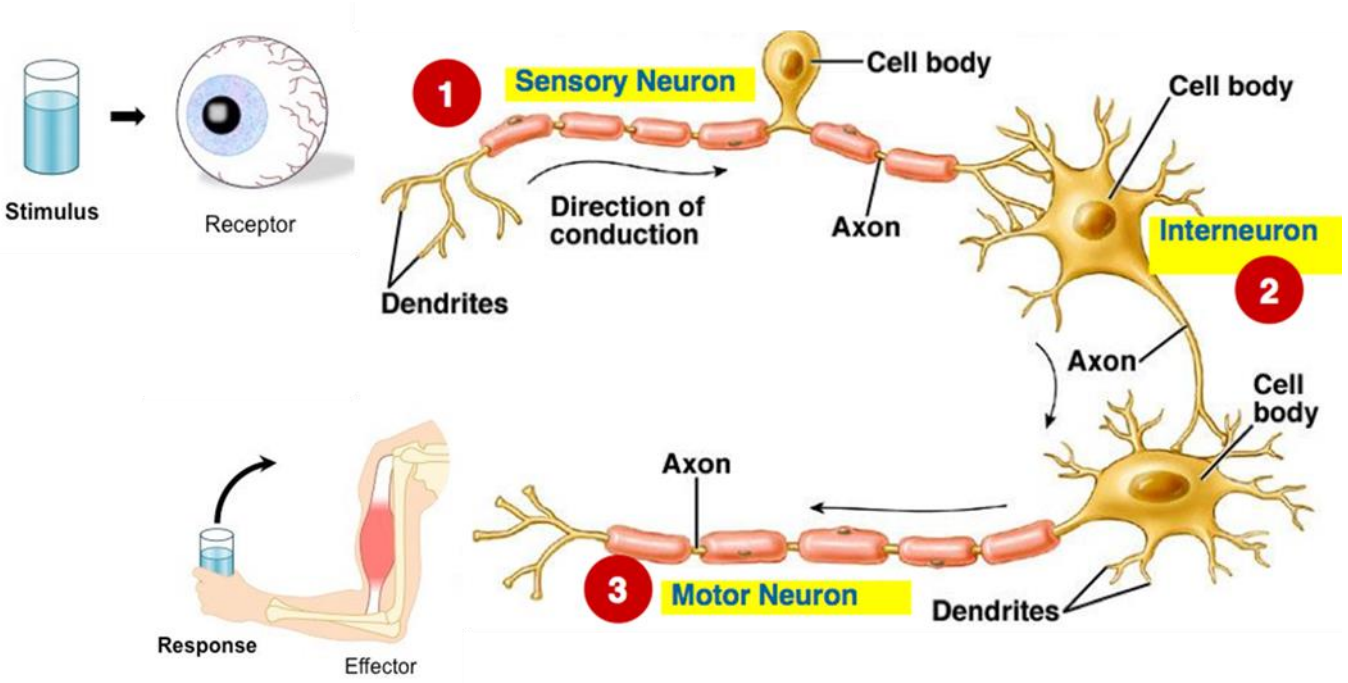
Two parts:

1) Central Nervous System (CNS) → Brain & spinal cord

2) Peripheral Nervous System (PNS) → Neurones



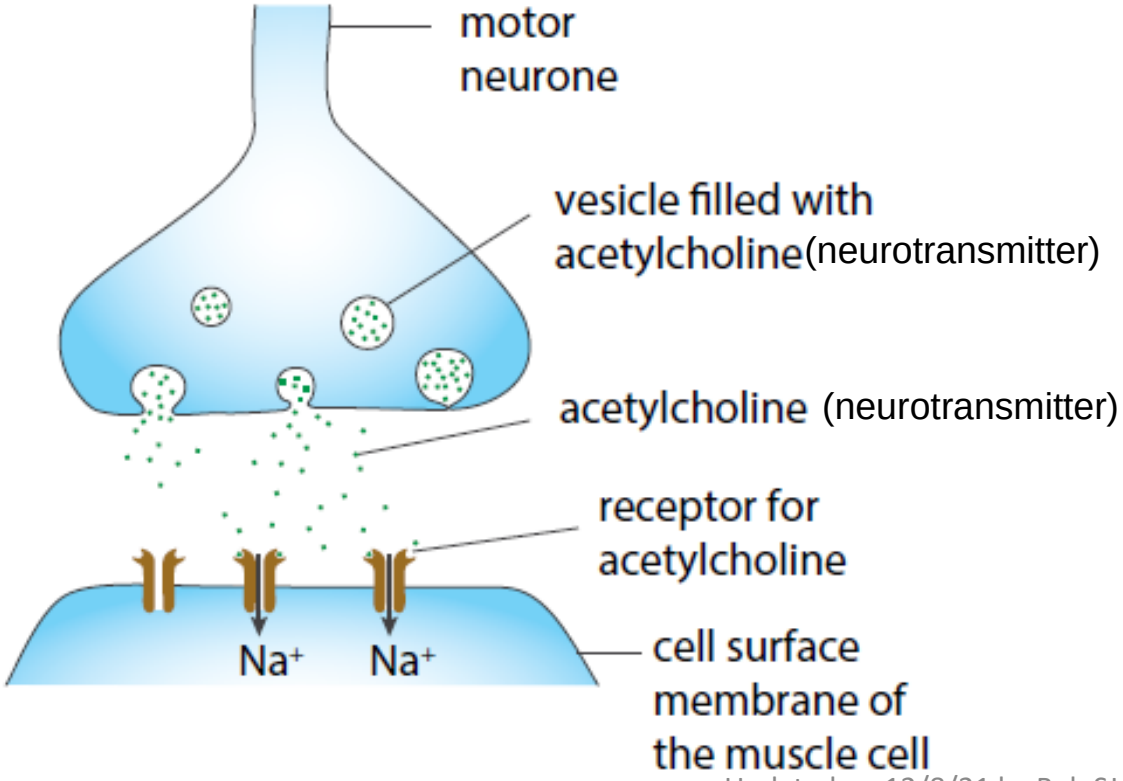
- Messages travel via nerve **impulses / action potentials**
- Along **neurones** / nerve fibres



Nervous System

- Impulse is passed from neurones to target cells via a **synapse**
- Using **neurotransmitters**

E.g. cholinergic synapse at the neuromuscular junction

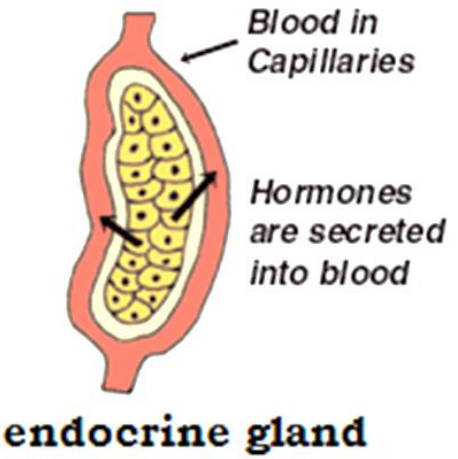


Recap from Chap 14

Endocrine vs Exocrine Glands

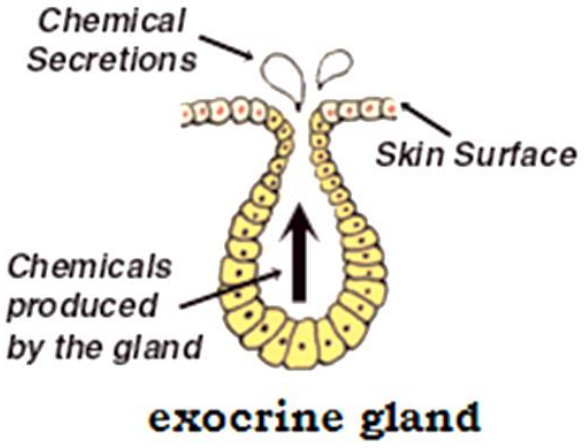
1) Endocrine glands

- Secretory cells
- Releases secretions directly into **blood** capillaries in the glands
- Secretions: **Hormones**
- E.g. pituitary glands, thyroid, adrenal, ovary, testes, ***pancreas***



2) Exocrine glands

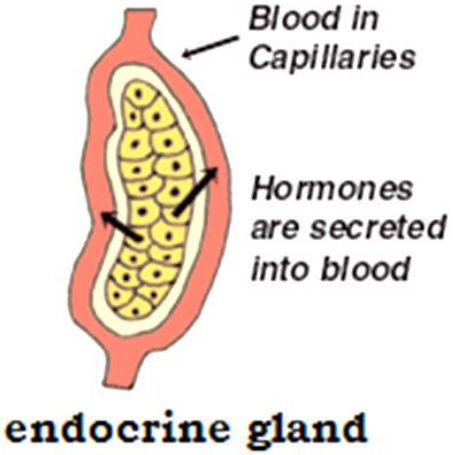
- Secretory cells
- Releases secretion into **ducts/tubes** (not blood capillaries)
- Secretions: **Not hormones**
- E.g. stomach, salivary glands, ***pancreas***



Hormones

- Secreted by **endocrine glands**
- Hormones can be **globular proteins OR steroids**
- E.g. **Insulin – protein hormone**

  Testosterone – steroid hormone



Characteristics:

1. Small molecules, chemical messengers
2. Needed in small quantities
3. Secreted quickly upon receiving a stimulus
4. Short life span, quickly broken down by enzymes/excreted via urine
5. Transported in the **blood stream** to target cells
6. Specific – bind to **receptors** on target cells

   Receptors can be **on cell surface membrane** OR **inside cell**

Nervous System vs Endocrine System

Both involve:

- Cell signalling
- Signal molecule binding to receptor
- Both involve chemicals

Features	Nervous System	Endocrine System
Communication via	Nerve impulses / action potentials	Hormones
Nature of communication	Electrical and chemical	Chemical
Mode of transmission	Neurones	Blood
Effector / Destination	Muscle / gland	Target organ / tissue / cell
Receptor Location	On cell surface membrane	On cell surface membrane OR within cell
Transmission Speed	Faster	Slower
Effects	Localised (Travels to specific targeted parts of body)	Widespread (Travel to all parts of the body, multiple target organs can respond)
Response Speed	Faster	Slower
Duration of Effect	Short-term Effect	Long-lasting Effect

Chapter Outline

<u>3 Parts!</u>

1. Nervous Communication
2. Striated Muscle Contraction
3. *Venus fly trap*

+ More Plant Hormones

Nervous system

Endocrine system

Chapter Outline (Part 1 of 3)

Nervous Communication

- Nervous vs Endocrine systems
- Structure of Neurones
- The Reflex Arc

How do nerve impulses work?

- Roles of **Sensory Receptor Cells**
- **Transmission of Nerve Impulses**
  - Resting Potential
  - Depolarisation / Action Potential
  - Repolarisation
  - Hyperpolarisation / Refractory Period
  - Saltatory Conduction
  - Threshold Potential
- Roles of **Synapses**
  - E.g. Cholinergic Synapse

Chapter Outline (Part 2 of 3)

Striated Muscle Contraction

- Structure
  - Muscle fibre
  - Myofibrils
  - Sarcomere
  - Actin and Myofilaments

- **Muscle Contraction**
  - Roles of neuromuscular junctions, transverse system tubules (T-tubules) and sarcoplasmic reticulum
  - Roles of troponin, tropomyosin, calcium ions and ATP
  - **Sliding filament model**

Chapter Outline (Part 3 of 3)
To Be Continued…

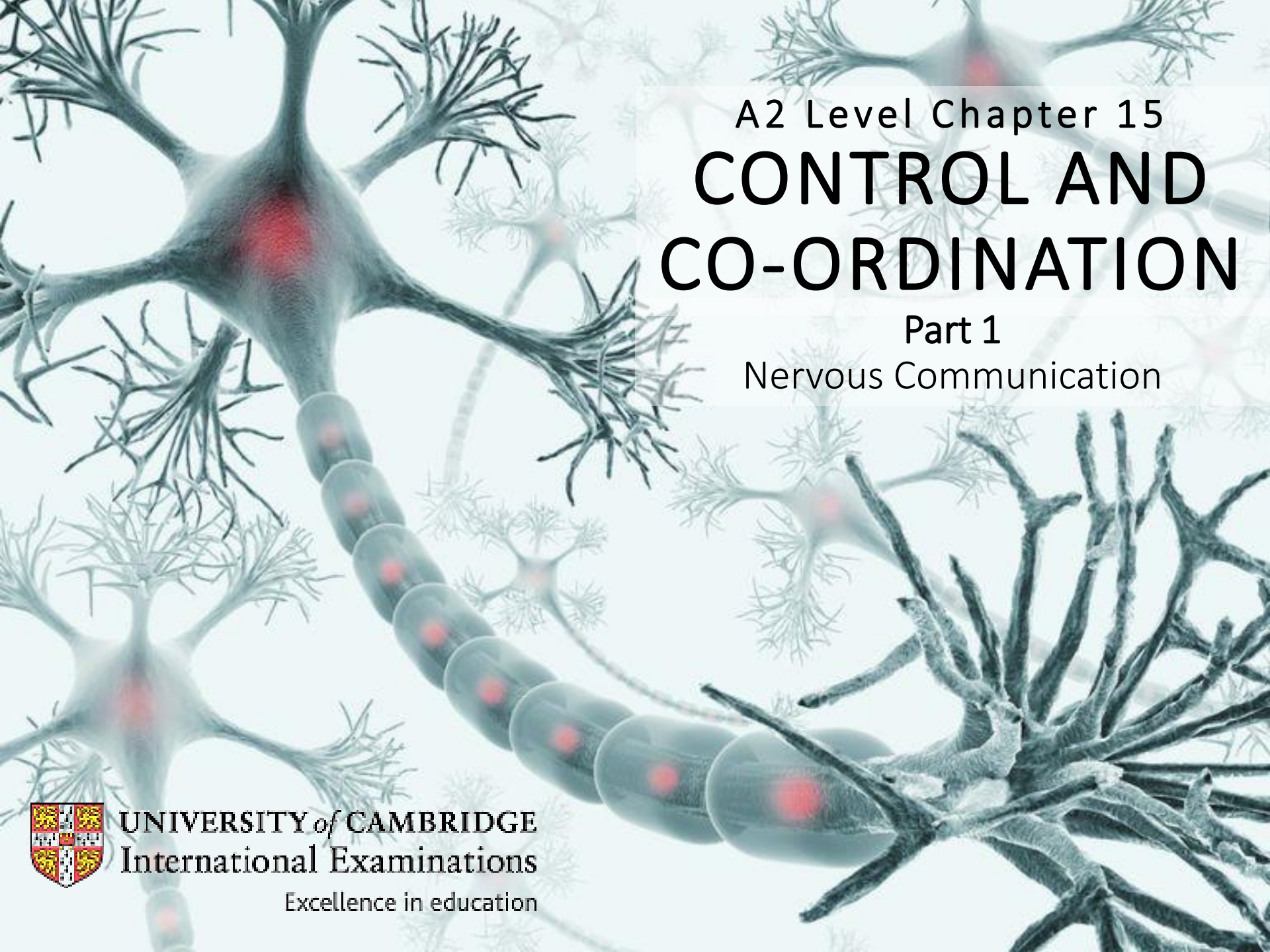
Control and Communication in Plants

Electrical communication in plants

- **Venus fly trap**

Chemical communication in plants

- Role of **auxin** in elongation growth
- Role of **gibberellin** in germination of wheat/barley
- Role of gibberellin in stem elongation (incl. role of dominant allele, *Le*)



A2 Level Chapter 15

CONTROL AND CO-ORDINATION

Part 1

Nervous Communication

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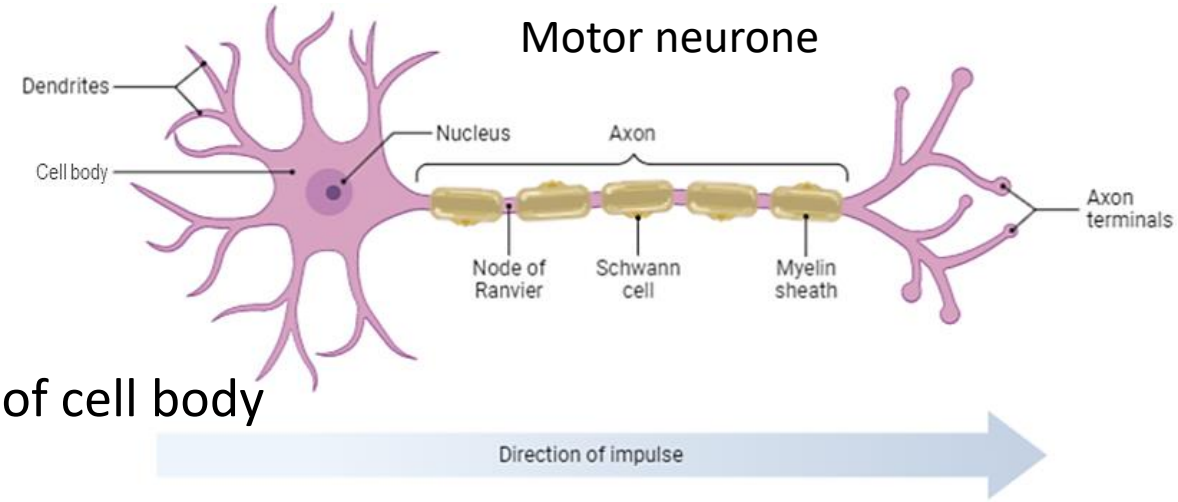
Structure of Neurones

a) **Cell body**

- Has a nucleus and cytoplasm
- Cytoplasm: Many mitochondria, ribosomes, RER, Golgi



b) Cytoplasmic processes

- Thin, cytoplasmic extension of cell body

1. Dendrites

- Carry impulses **towards** the cell body

2. Axons

- Carry impulses **away** from the cell body
- Some enclosed with **myelin sheath**

Structure of Neurones

c) **Axon terminal / presynaptic knob**

- Many mitochondria, synaptic vesicles containing neurotransmitters, voltage-gated Ca^{2+} channels

- Part of a **synapse** = junction between neurones / muscles

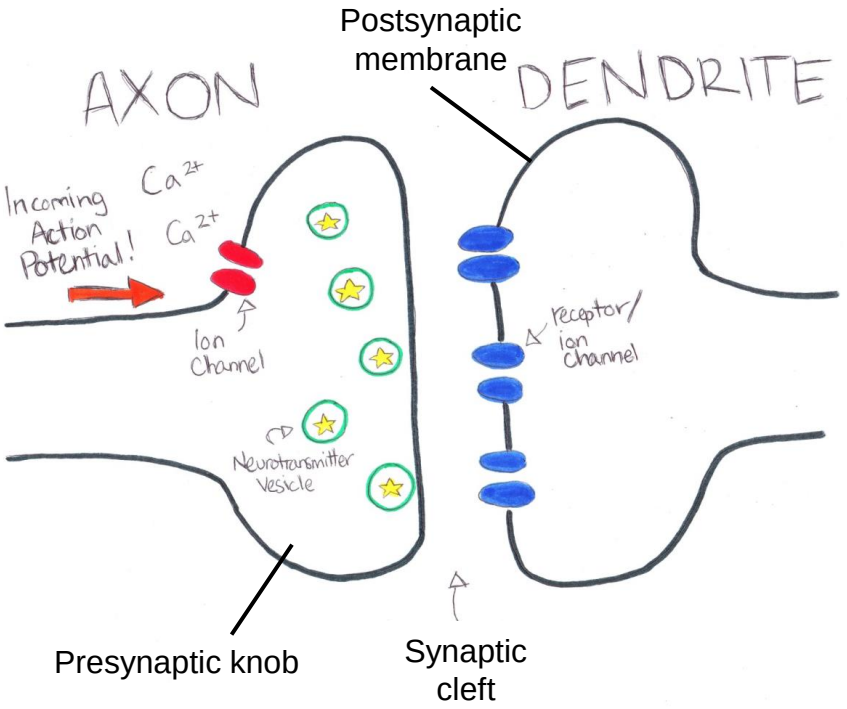
A synapse also includes:

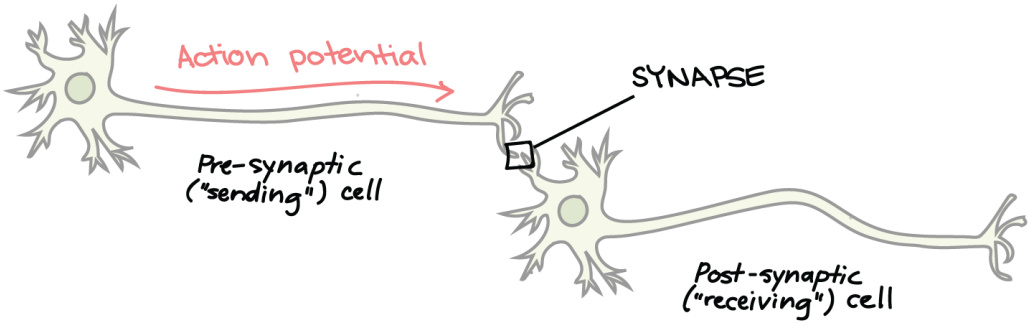
- **Synaptic cleft** = gap

→ has enzymes to breakdown neurotransmitters

- **Postsynaptic membrane**

→ has receptor proteins for neurotransmitters





Structure of a Neurone

d) **Myelin sheath**

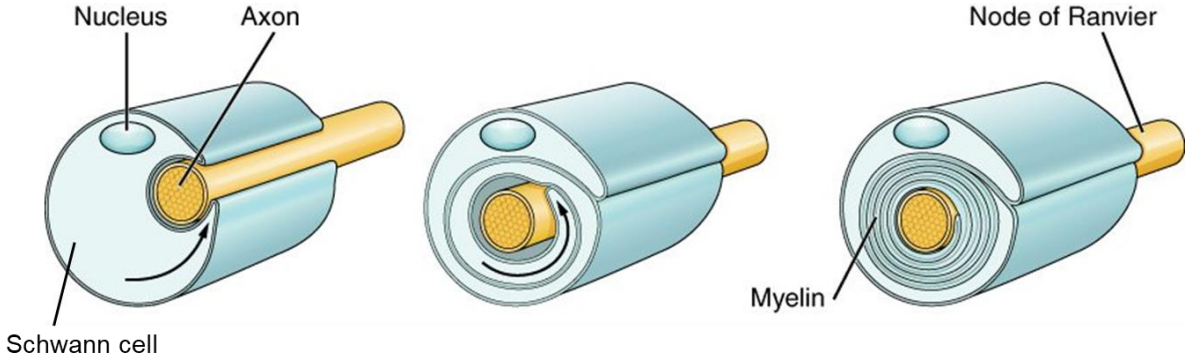
- **Insulates axons** of many neurones
- Function: **Speeds up conduction of nerve impulses**
- Made of Schwann cells

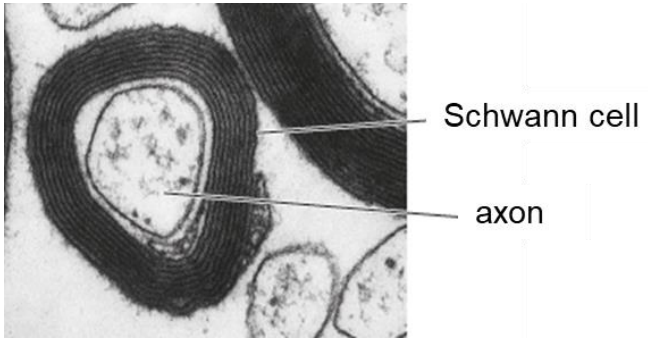
→ Has nucleus

→ Layers of cytoplasm and plasma membrane spirals around the axon

e) **Nodes of Ranvier**

- Between Schwann cells, no myelin



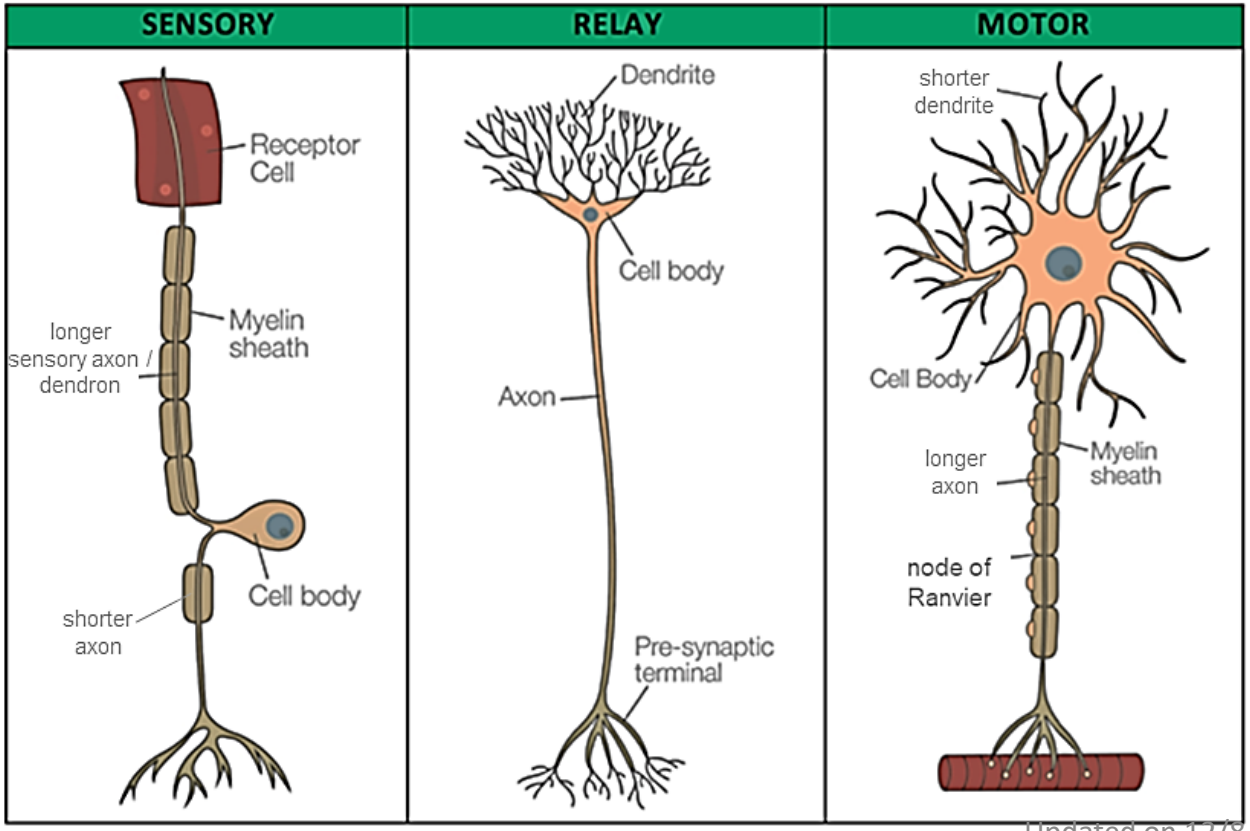


3 Types of Neurones

1. **Sensory neurone** (afferent)
   - Longer sensory axon / dendron
   - Shorter axon

2. **Intermediate / relay neurone**

3. **Motor neurone** (efferent)
   - Shorter dendrites
   - Much longer axon

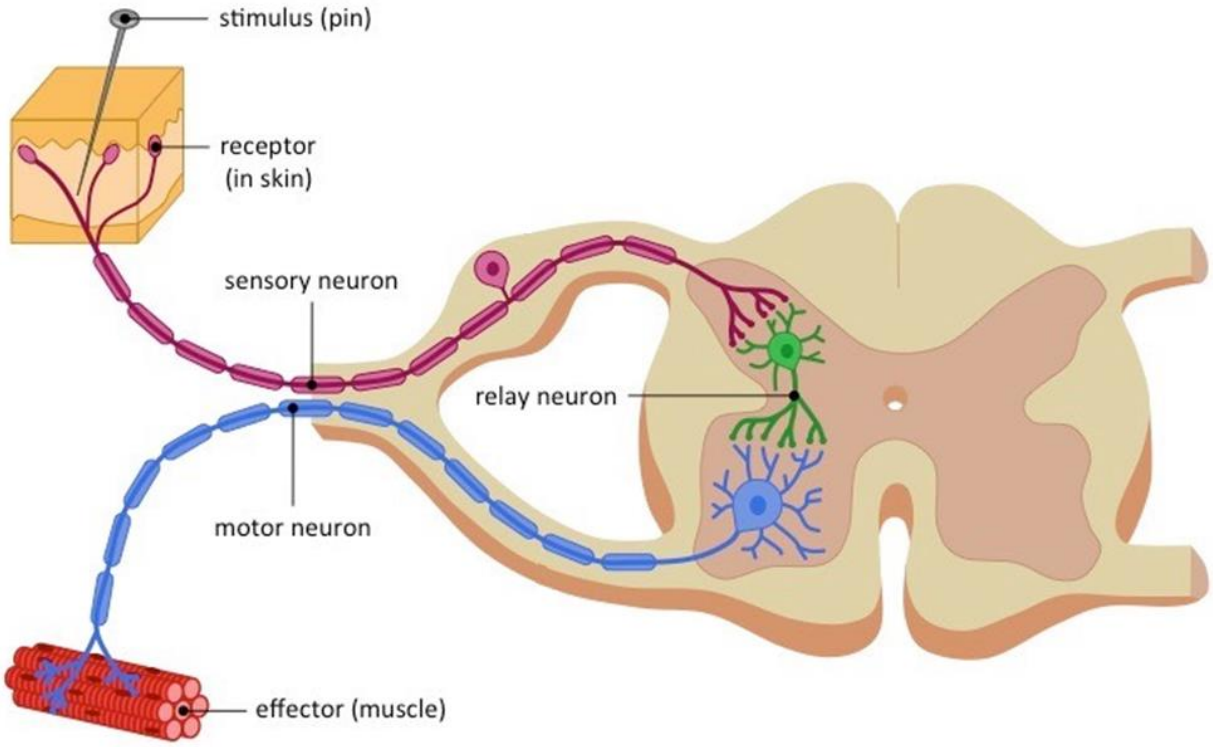


The Reflex Arc

Very strong stimulus detected by receptor (E.g. light, sound heat)

Action potential generated in **sensory neurones**

Sensory neurone connected to spinal cord



Synapse with **relay neuron** → action potential passed to **relay neurone**

Synapse with **motor neurones** → action potential passed to **motor neurone**

Cholinergic synapse of neuromuscular junction → Effector/muscle

Effector/muscle carries out response

The Reflex Arc

- Pathway where impulses are carried along during a reflex action
- E.g. knee jerk reflex, sneezing

Advantages:

- **Fast**
- **Automatic, involuntary,** without conscious thought
- **Innate / instinctive**, response is always the same
- **Protects from harm**

How do nerve impulses work?

- What are **impulses**?

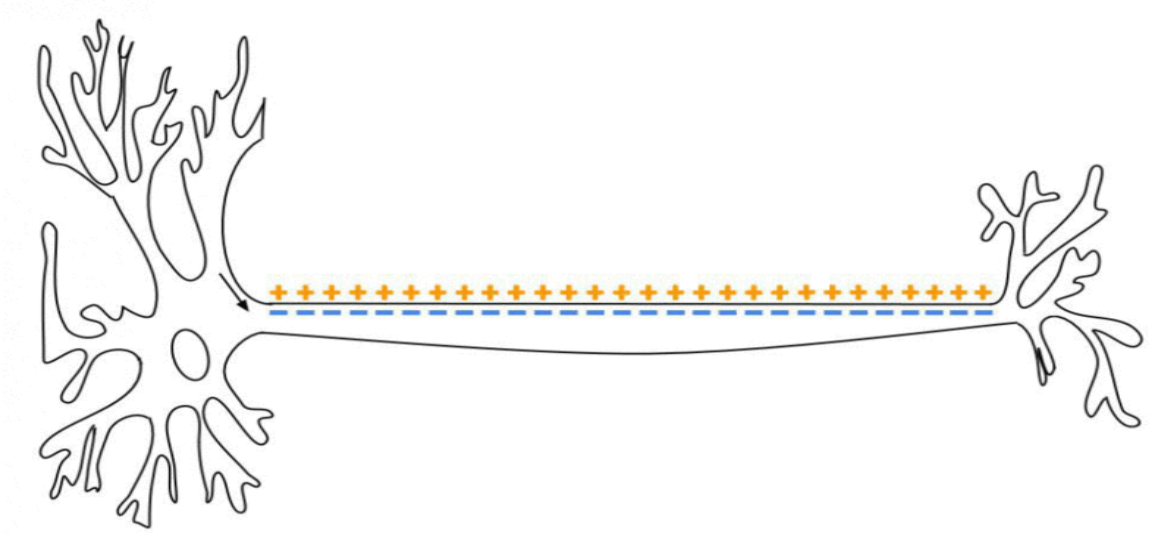
→**Brief changes** to the distribution of electrical charge across membrane (aka **membrane potential**)

At rest: more **negative**ly charged on inside than outside

- **Resting potential** = -70mV

When impulses are formed: more **positive** on inside than outside

- **Action potential / depolarization** = +30mV



Sensory Receptor Cells

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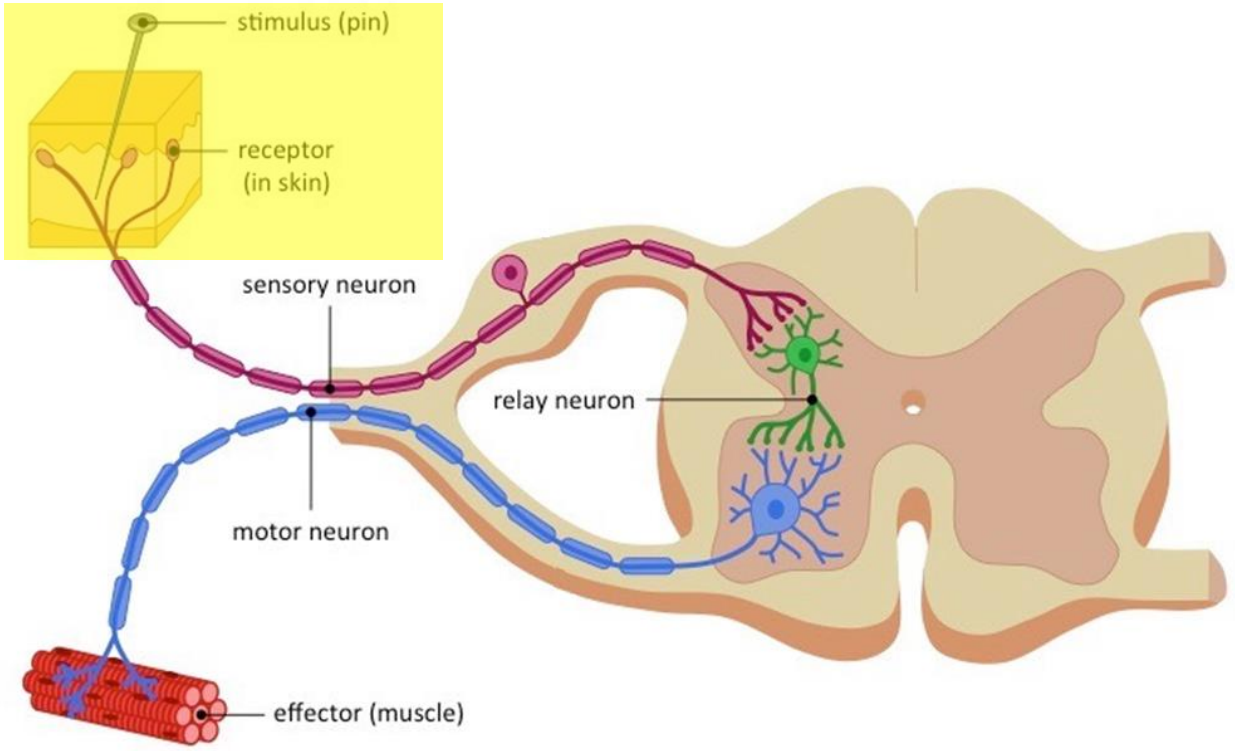

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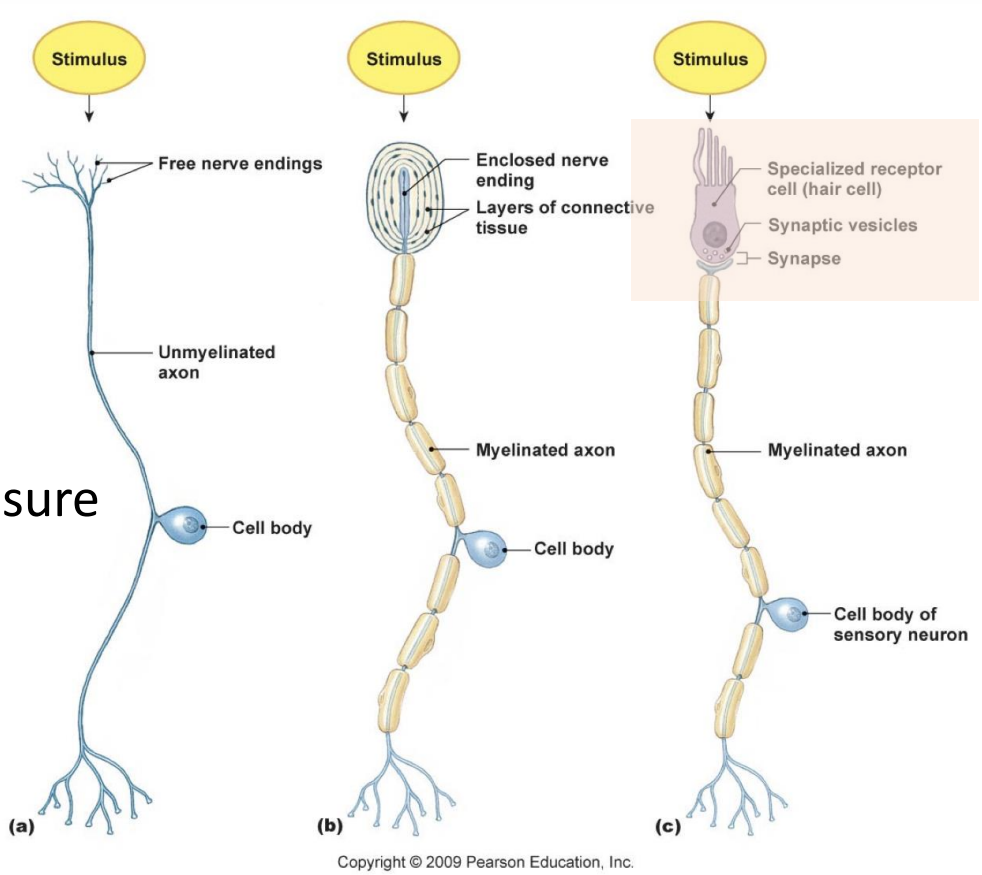
Updated on 12/8/21 by Beh SJ @behlogy

Sensory Receptor Cells

Roles:

1. **Detect stimuli**

- Receptors are specific to one-type of stimulus
- e.g. chemical, light, heat, sound, pressure



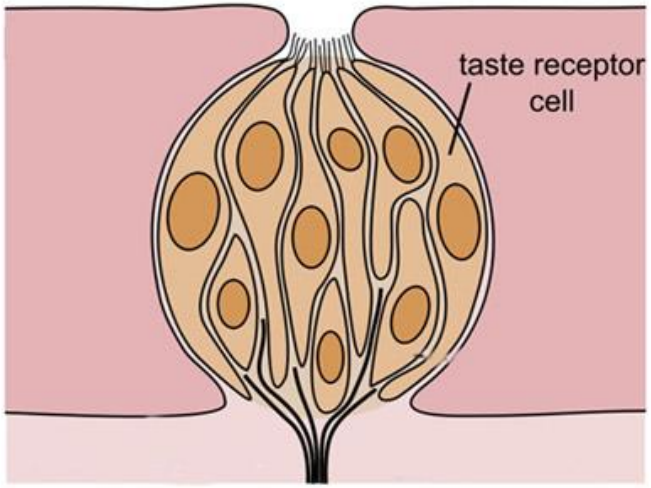
2. Acts as **transducers**

- Converts stimulus energy to electrical energy
- Produce **generator / receptor potential**

→ Pass impulse along **sensory neurone**

E.g. Taste Chemoreceptor Cells

- **Chemicals** act as a **stimulus**
- Diff chemoreceptors are specific for diff chemicals = diff tastes



E.g. salt (NaCl)

1. Na^+ ions diffuse into cell via microvilli
→ Increase in positive charge inside cell

2. Membrane **depolarized**

→ **Receptor / generator potential** generated

E.g. Taste Chemoreceptor Cells

3. Voltage-gated **Ca^{2+} channels** open

→ **Ca^{2+} enter** cell

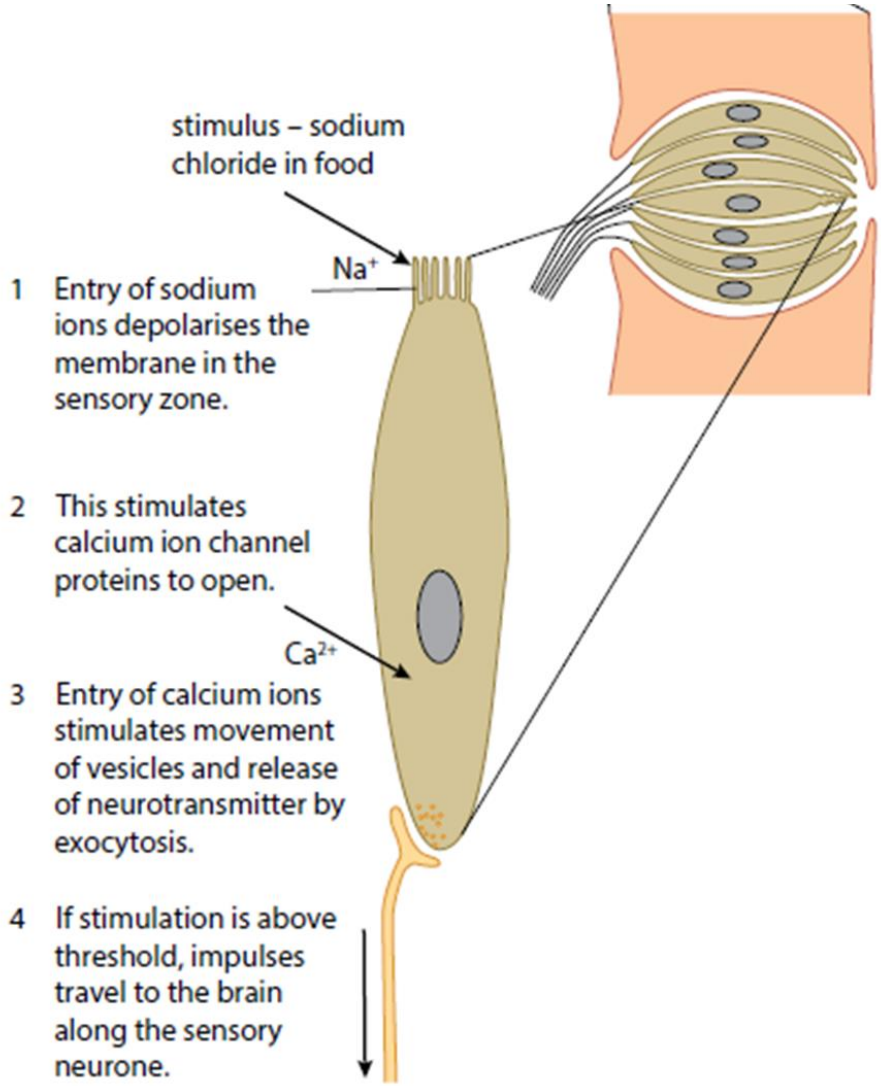
4. Trigger movement of **vesicles containing neurotransmitters**

→ **exocytosis** occurs

→ neurotransmitter released

5. Neurotransmitter stimulate **action potential** / impulse in **sensory neurone**

→ Send impulse to taste centre in brain



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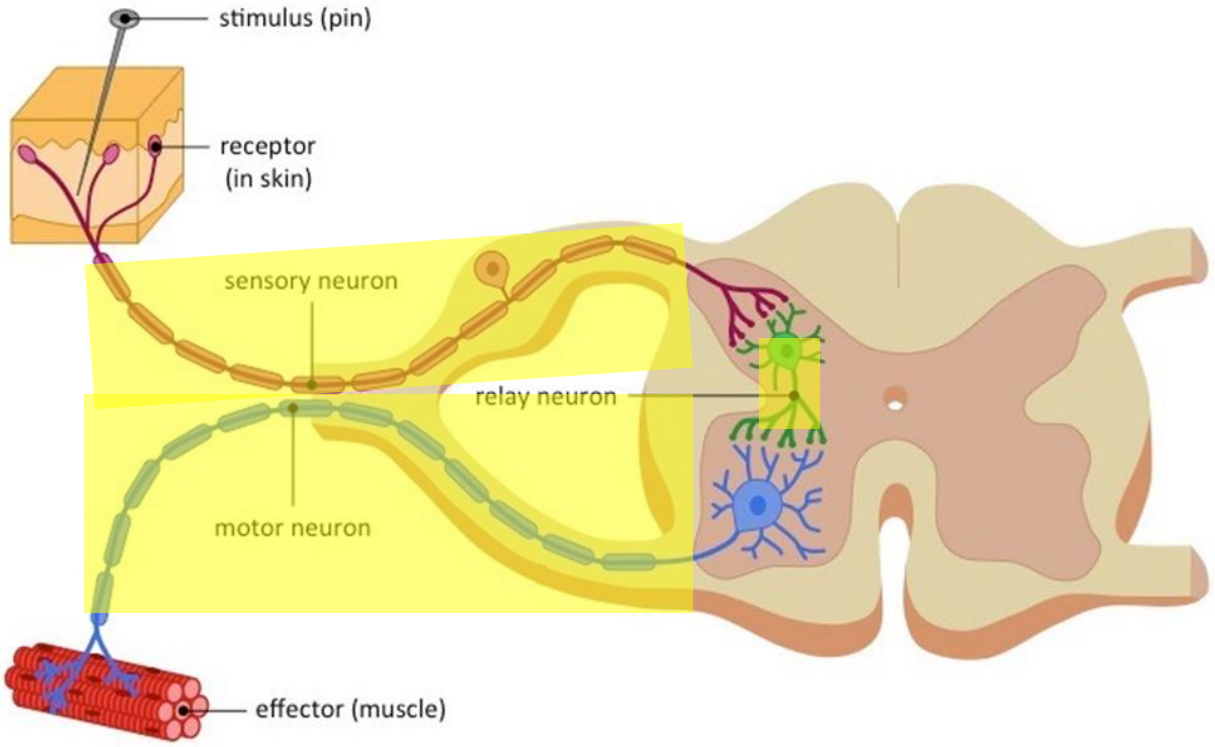

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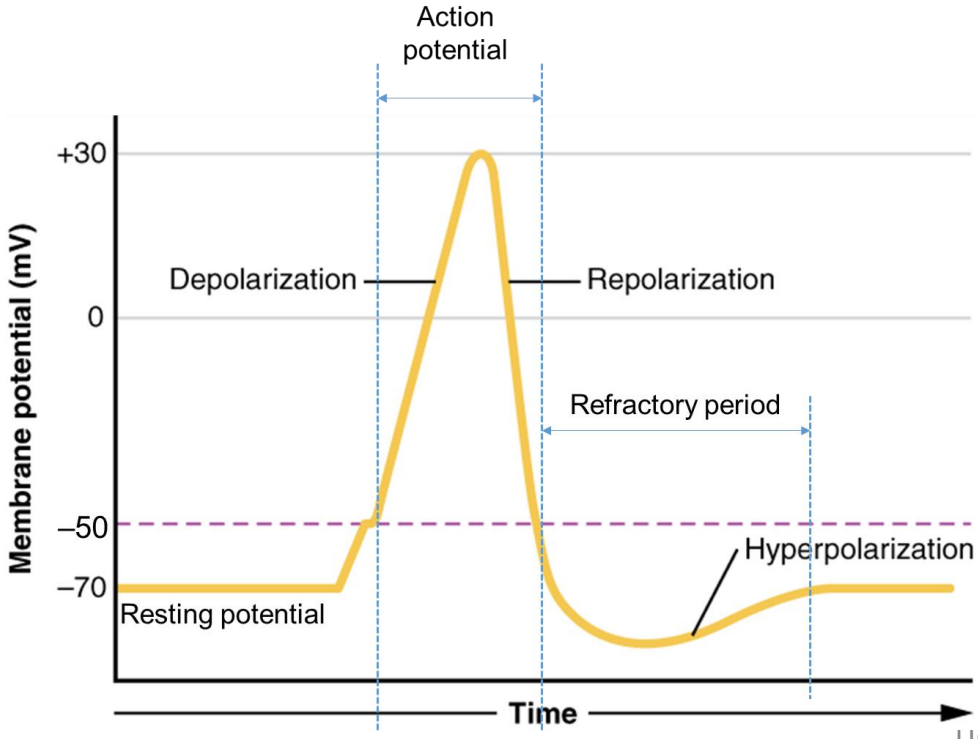
Updated on 12/8/21 by Beh SJ @behlogy

Transmission of Action Potential

1. Resting Potential	-70mV
2. Depolarisation	-70mV → +30mV
3. Repolarisation	+30mV → -70mV
4. Hyperpolarisation / Refractory Period	less than -70mV



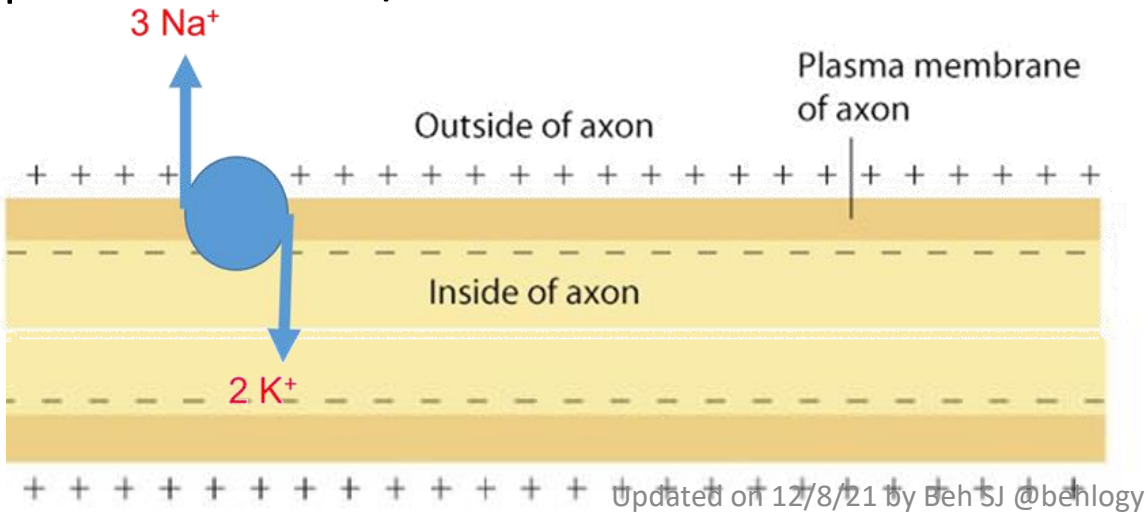
Resting Potential (-70mV)

- At rest = no stimuli, no impulses formed and transmitted
- Inside of axon more **negatively charged** than outside
- Neurone is **polarized** and maintained at -70mV

How is a resting potential maintained?

1. **Na^+/K^+ pump**

- 3 Na^+ pumped out, 2 K^+ pumped in
- ATP needed
- Axon phospholipid bilayer impermeable to K^+ / Na^+

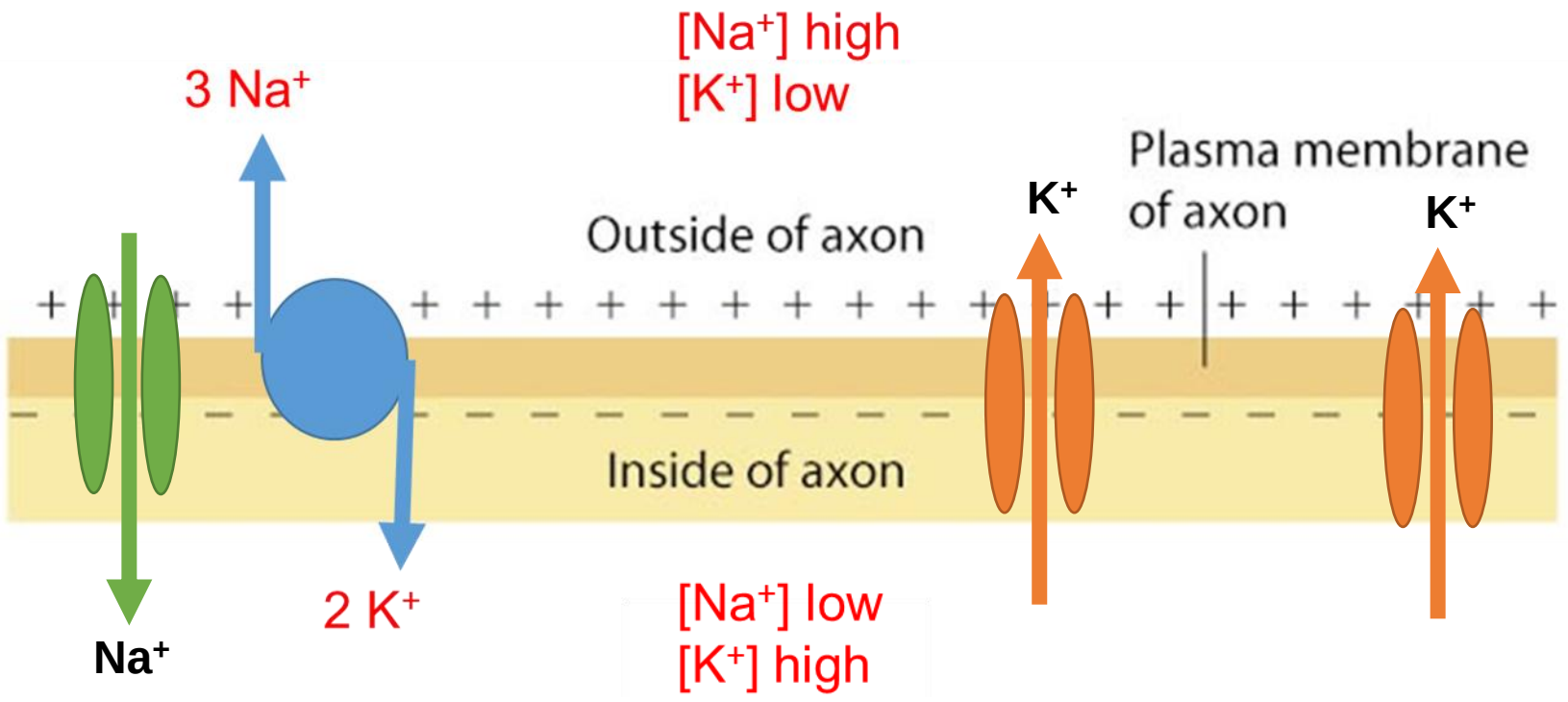


Resting Potential (-70mV)

- **Electrochemical gradient** is set up = difference in both charge and chemical ions across membrane

→ So **K^+ diffuse out, Na^+ diffuse in**

→ via **channel proteins**

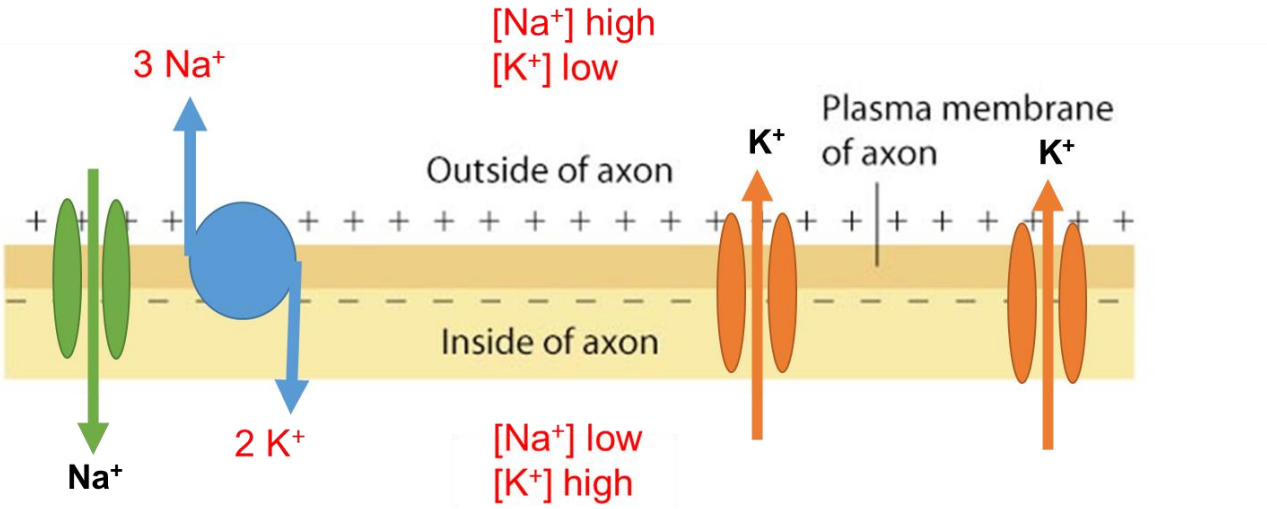


Resting Potential (-70mV)

2. More **K^+ channels** open than **Na^+ channels**

- Membrane more permeable to K^+ than Na^+
- More K^+ leaves than Na^+ enter
- Leaking K^+ is responsible for resting potential

→Inside becomes relatively more negative than outside



P/S: these channel proteins are open all the time. But **voltage-gated K^+ and Na^+ channels are closed.**

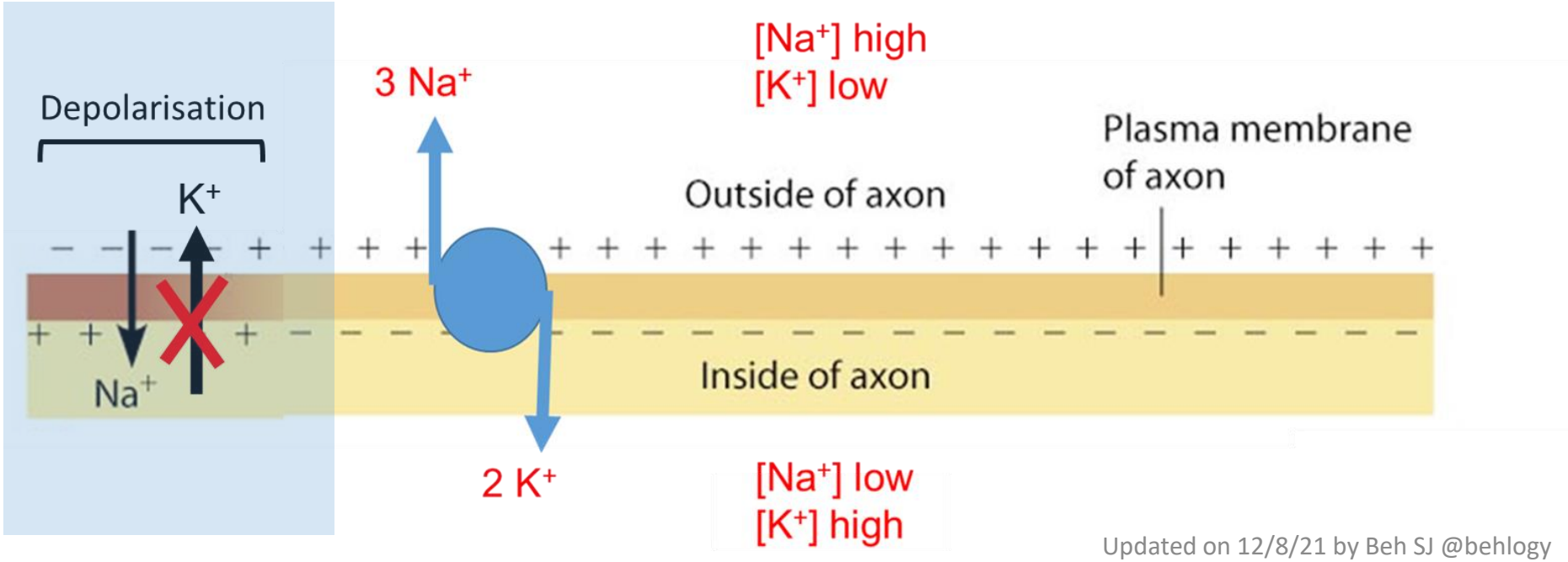
Depolarisation (-70 mV → +30mV)

1. **Voltage-gated K^+ channels remain closed**
2. **Voltage-gated Na^+ channels open**

→ Channels change shape when membrane potential changes when action potential arrives from previous section

- **Na^+ enter cell**
- Membrane becomes less negative / **depolarized** → +30mV

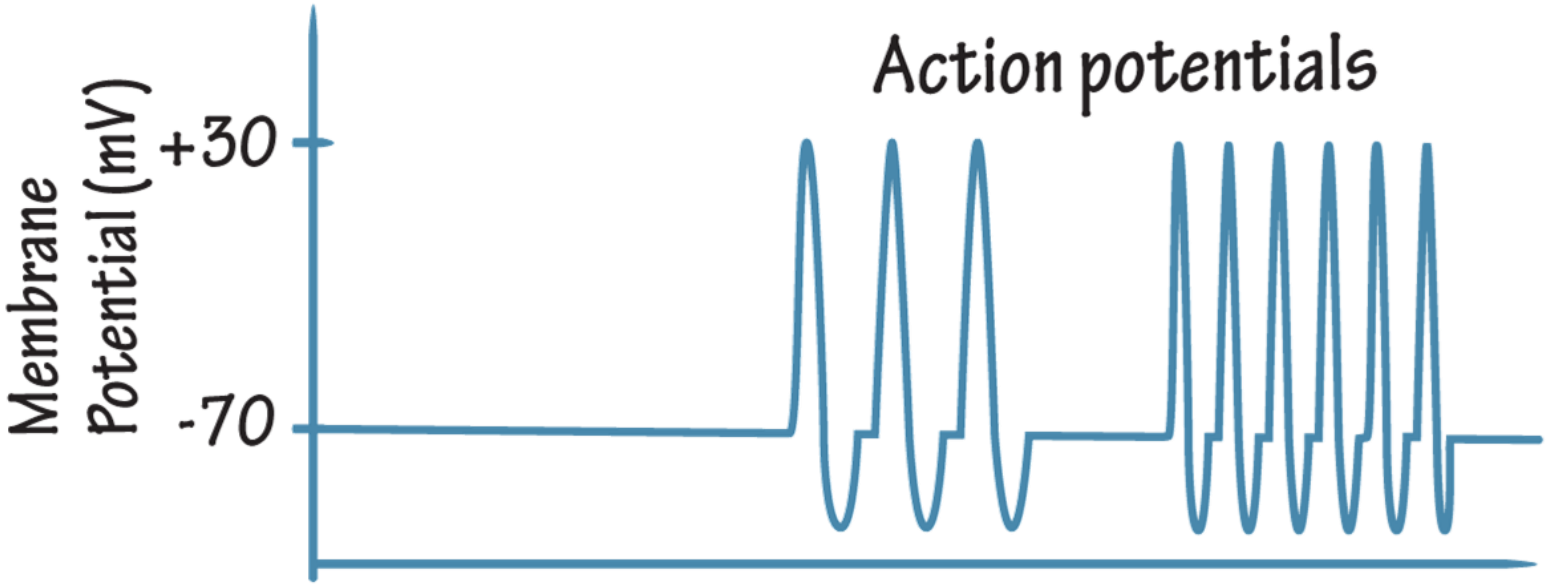
→ **Action potential is generated**



Depolarisation (-70 mV → +30mV)

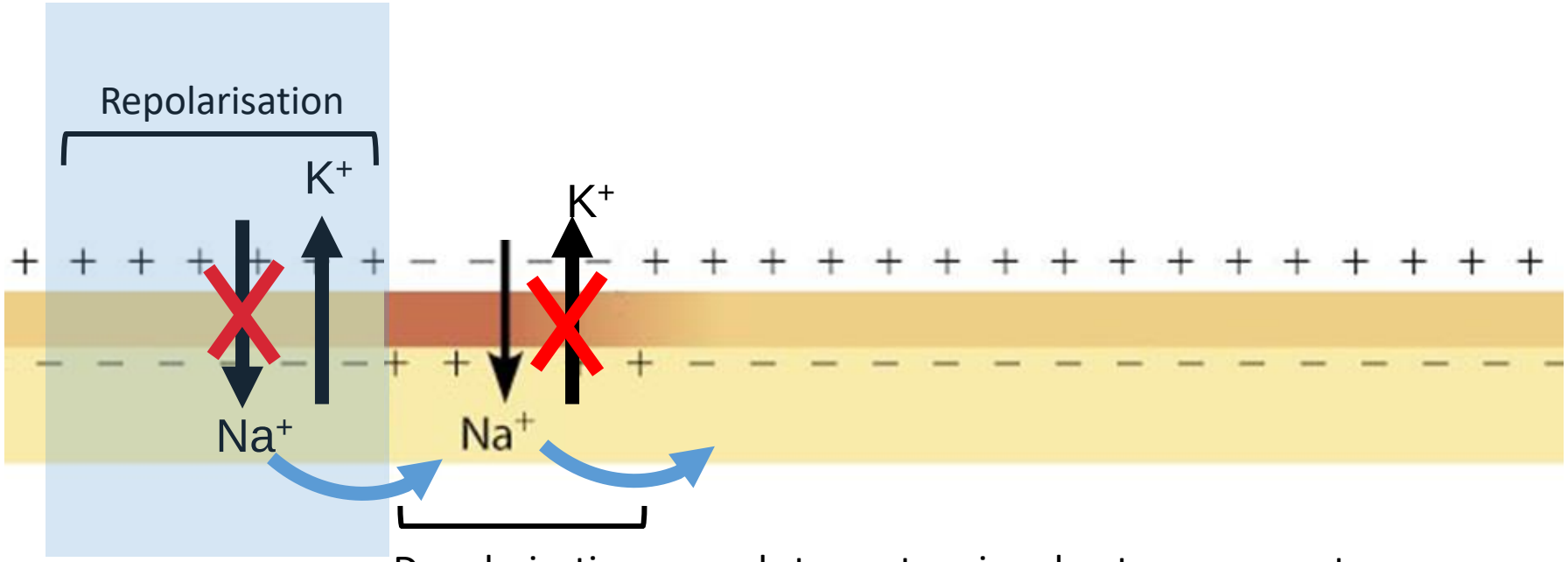
- Size of action potentials **is fixed at +30 mV**

- The higher the strength/ intensity of the stimulus, the **higher the frequency of action potentials**
- Also – the more neurones are depolarised



Repolarisation (+30mV → -70mV)

1. Voltage-gated Na^+ channels **close**
2. **Voltage-gated K^+ channels open**
- **K^+ move out of cell**
- Inside becomes negative / **repolarised** → -70mV

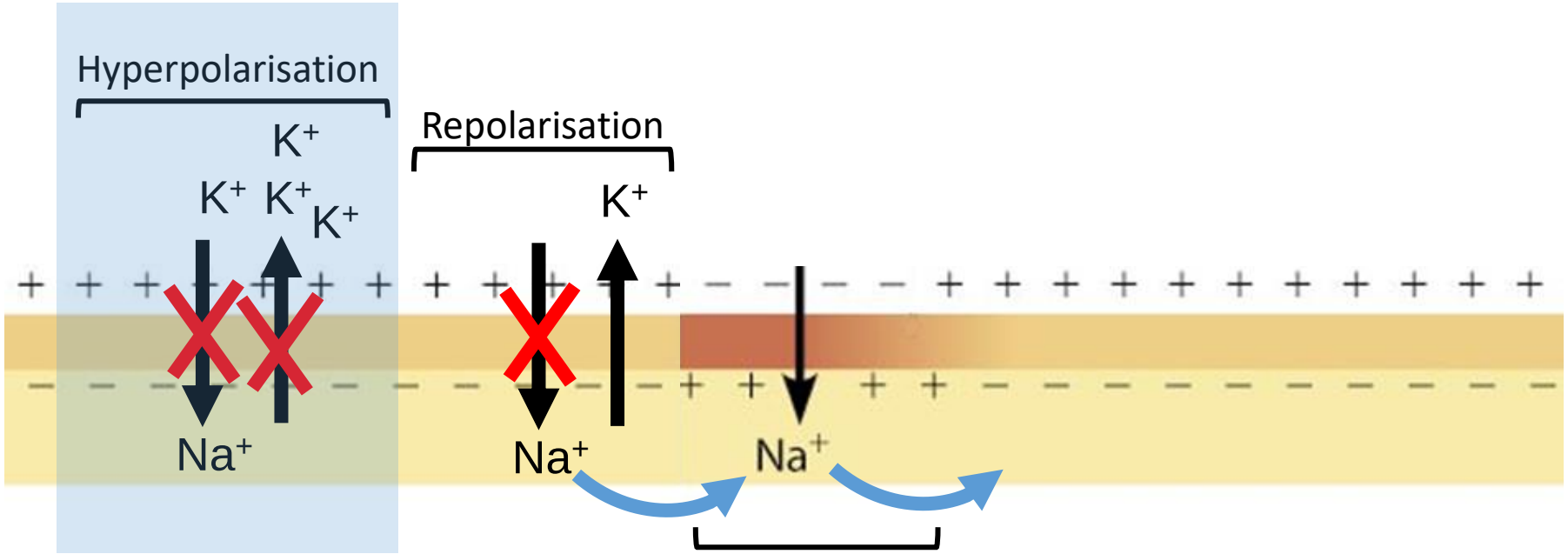


Depolarisation spreads to next region due to movement of +ve ions to -ve regions. A "**local circuit**" is set up.

Hyperpolarisation / Refractory Period (less than -70mV)

1. **Voltage-gated Na^+ channels remain closed**
2. **Voltage-gated K^+ channels close**

- But slight delay so excess K^+ ions have moved out of axon



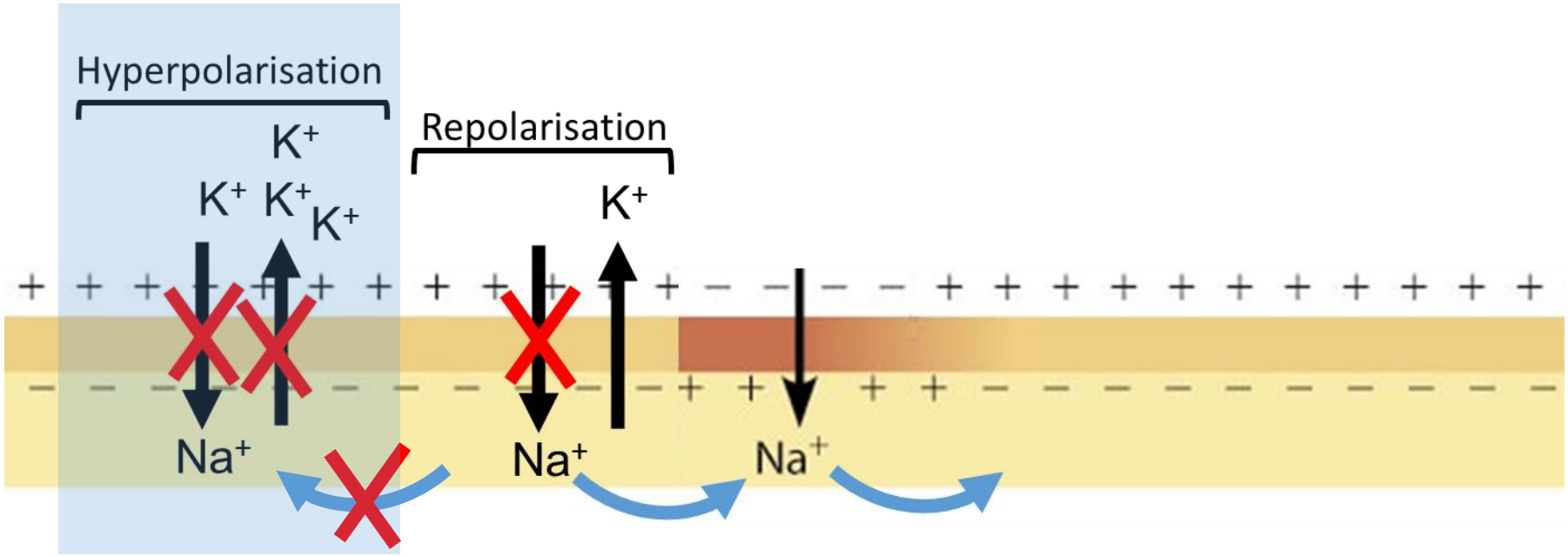
Depolarisation spreads to next region due to movement of +ve ions to -ve regions. A "**local circuit**" is set up.

Hyperpolarisation / Refractory Period (less than -70mV)

When membrane is hyperpolarized = **refractory period**

- Membrane is insensitive to any depolarisation
- No action potential can be generated

→ Function: **ensure one-way transmission**



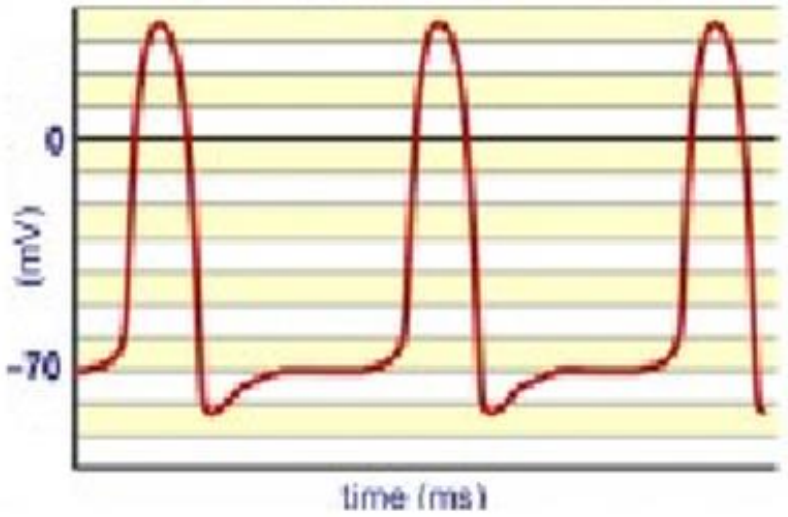
Direction of Transmission

Hyperpolarisation / Refractory Period (less than -70mV)

- Due to the refractory period, action potentials are discrete events / do not merge into one another

→ Function: Length of refractory period **limits maximum frequency of action potentials**

- E.g. longer refractory period = lower maximum frequency

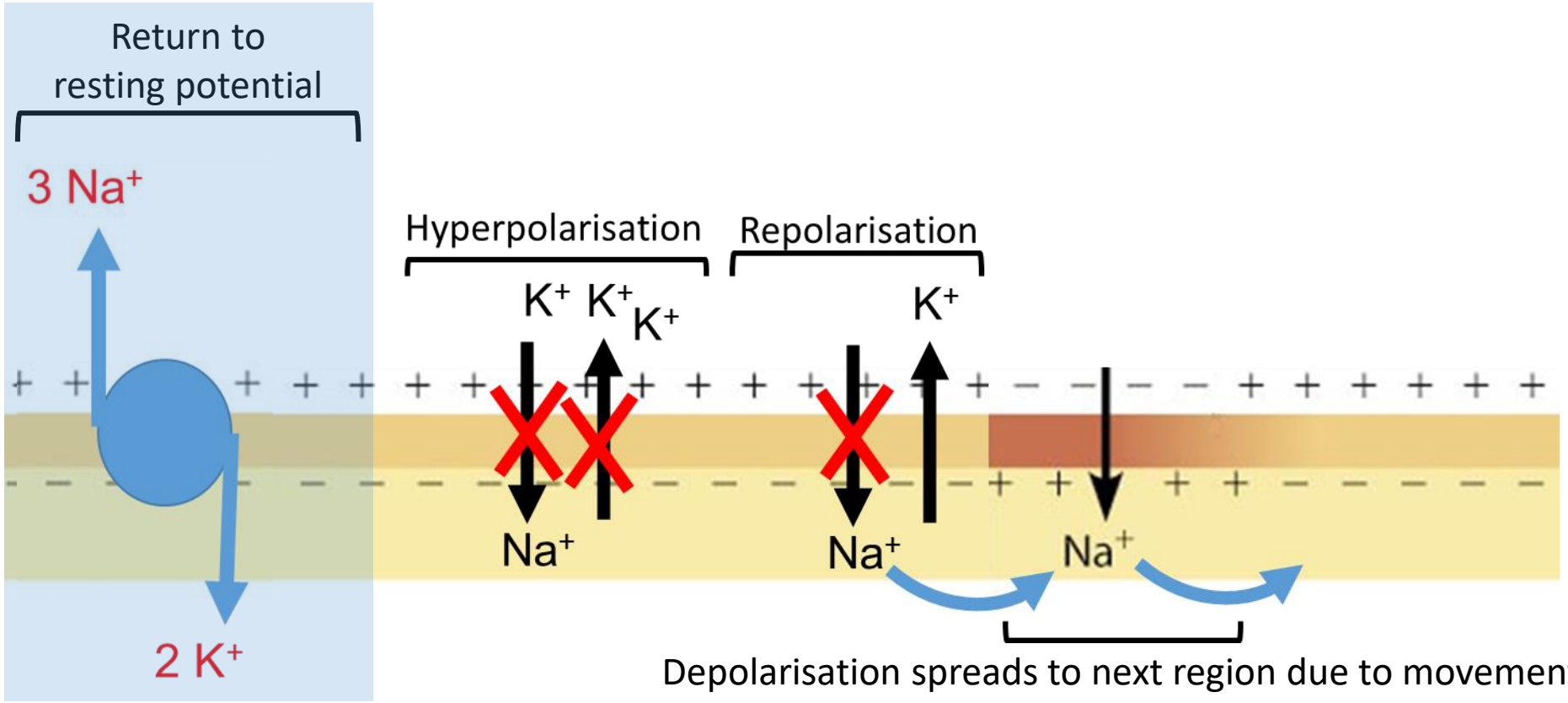


Return to Resting Potential (-70mV)

- **Na^+/K^+ pump** acts again

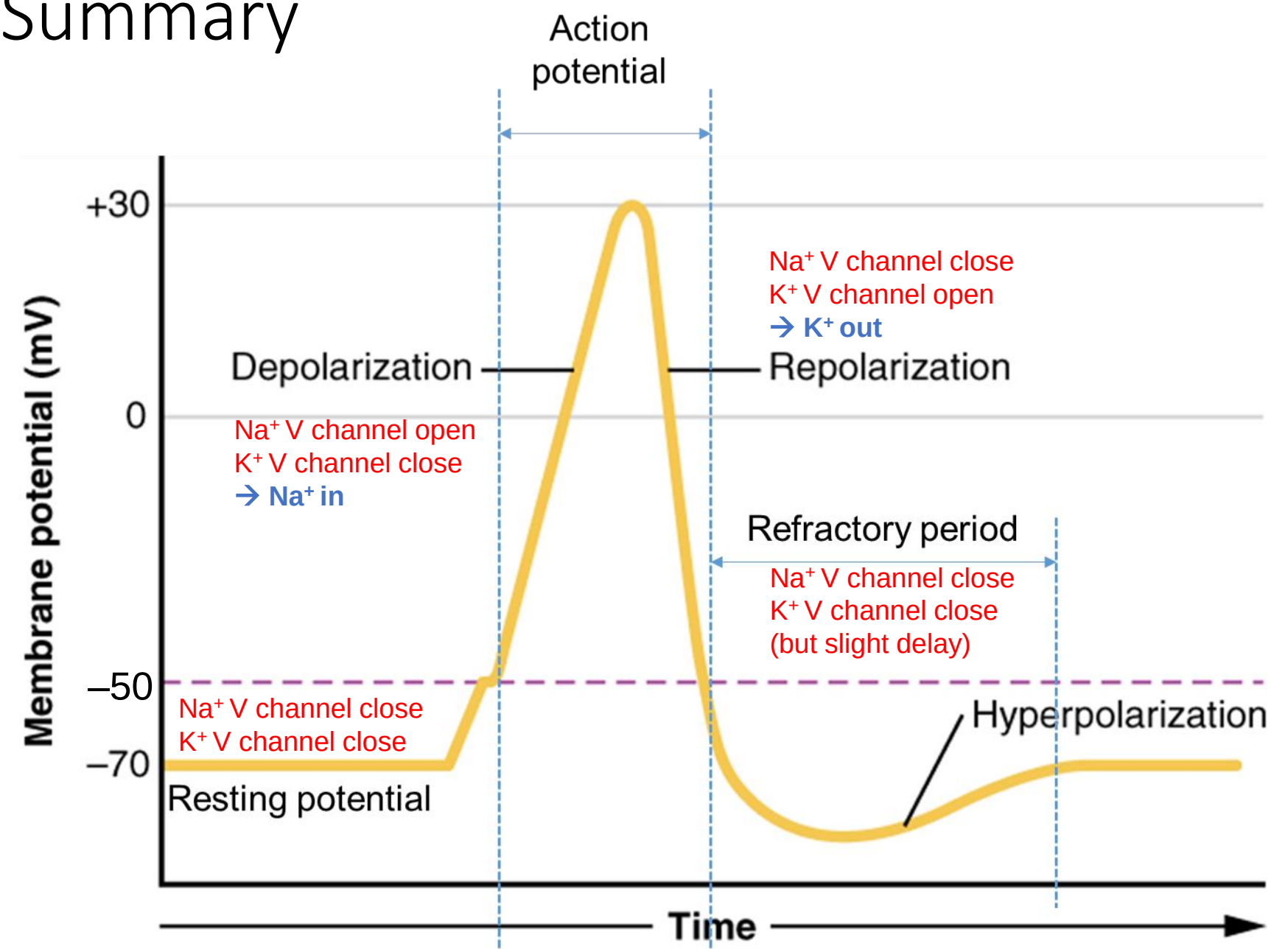
→ Membrane can be depolarized again

→ Action potential can be generated again



Depolarisation spreads to next region due to movement of +ve ions to -ve regions. A "**local circuit**" is set up.

Summary


Action potential
+30
0
–50
–70
Membrane potential (mV)
Depolarization
Repolarization
Na⁺ V channel close
K⁺ V channel open
→ K⁺ out
Na⁺ V channel open
K⁺ V channel close
→ Na⁺ in
Refractory period
Na⁺ V channel close
K⁺ V channel close
(but slight delay)
Na⁺ V channel close
K⁺ V channel close
Resting potential
Hyperpolarization
Time

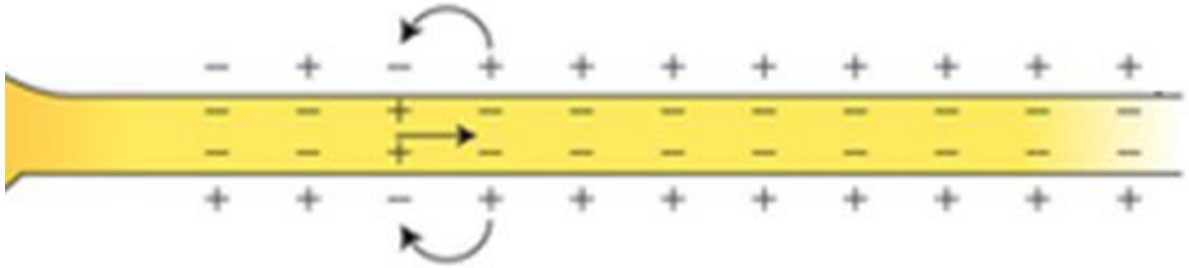
How action potentials are transmitted along a *non*-myelinated axon?

- Depolarisation spreads to next region due to **movement of positive ions to negative regions**

→ A "**local circuit**" is set up

→ This causes voltage-gated Na^+ channels to open in the next region

→ Causing next action potential



Non-myelinated axon

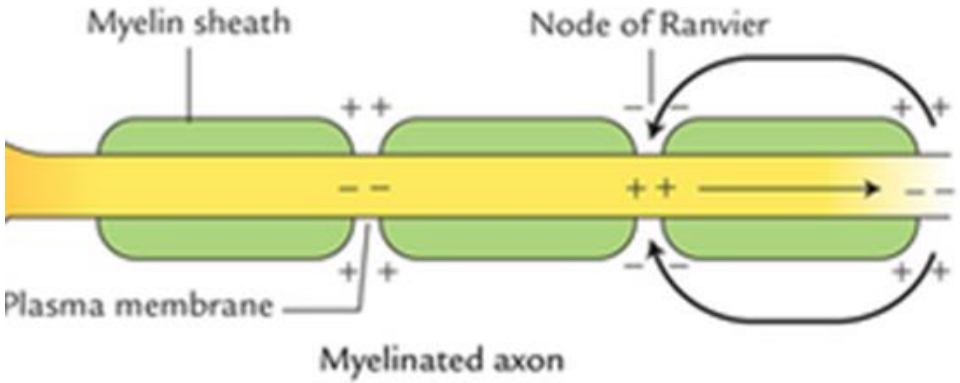
How action potentials are transmitted along a *myelinated* axon?

But with the **MYELIN SHEATH**… there is an increased speed of conduction!

- **Myelin insulates axon**

→ Does not allow movement of ions

→ Lengthens local circuits



- Passage of ions only at **nodes of Ranvier**

→ Action potential / depolarization only at nodes of Ranvier

→ **Local circuit** is set up **between nodes**

→ **Action potential 'jumps'** from node to node

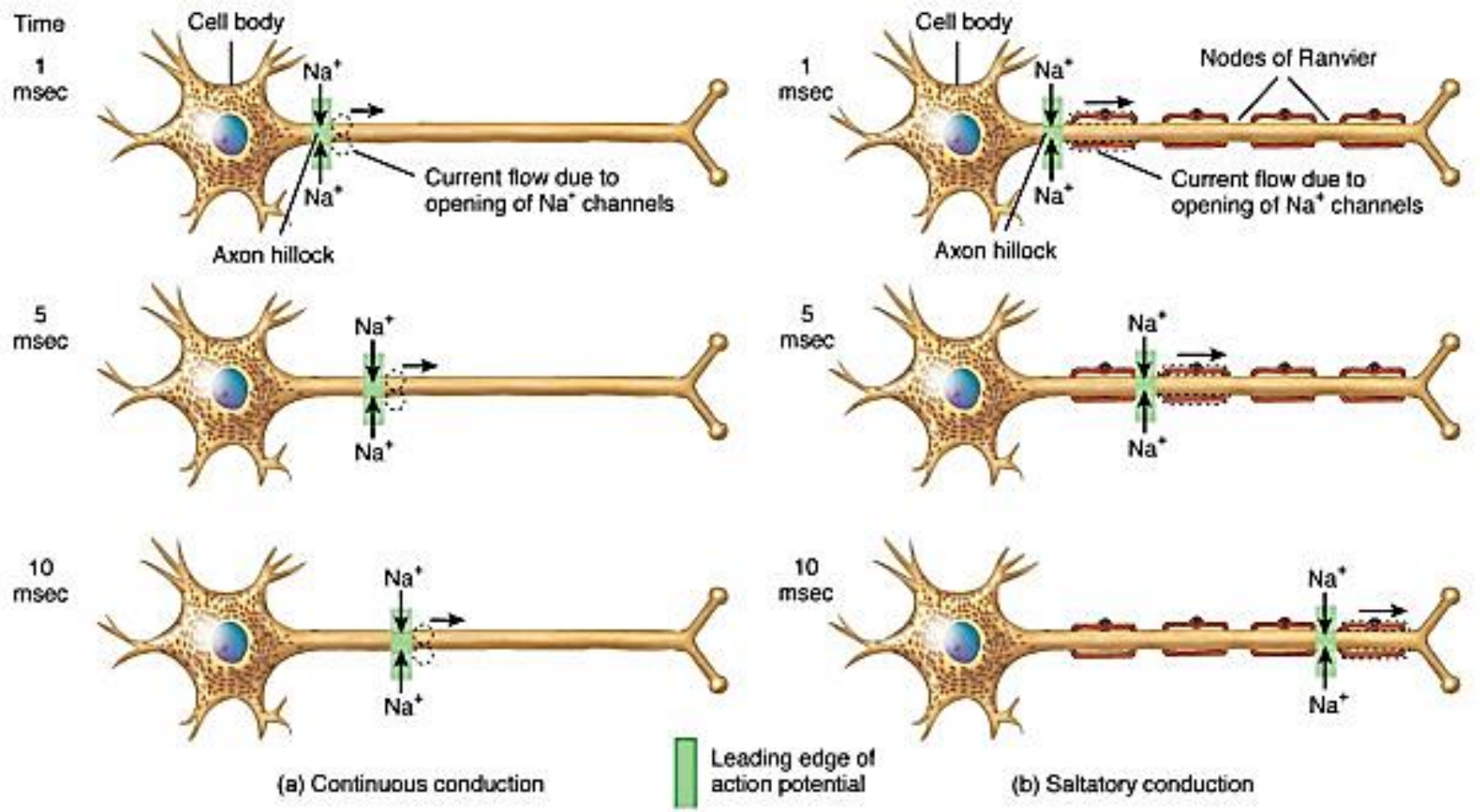
→ This is called **saltatory conduction**

Saltatory Conduction

Faster transmission because **myelin sheath** insulates axons

→ **Local circuit** is set up **between nodes**

→ **Action potential 'jumps'** from node to node



Threshold Potential (-50mV)

- **Minimum potential** needed for **action potential to be generated**

→ Only depolarisation that reaches threshold produces an action potential

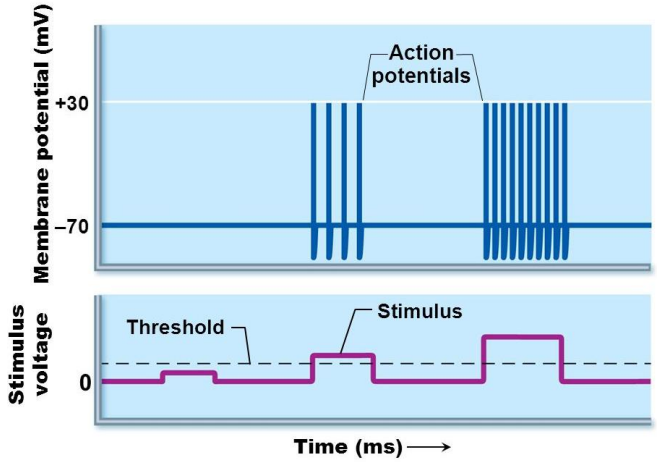
- If depolarisation <-50mV, action potential is not generated

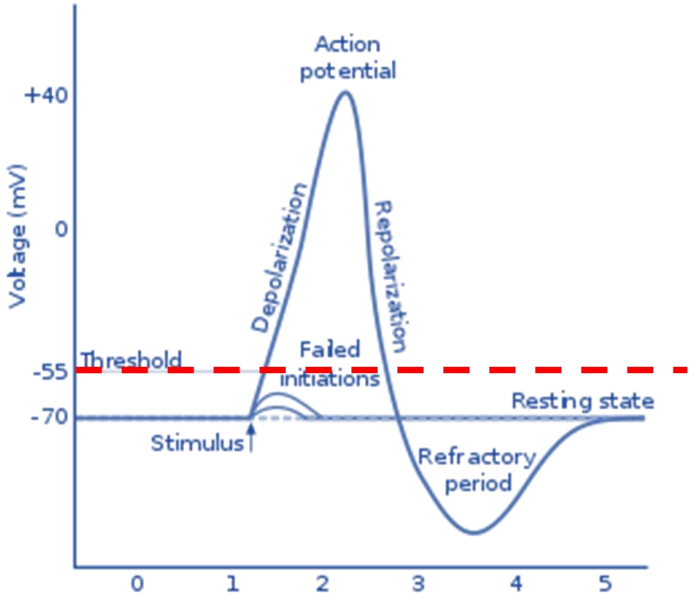
→ only local depolarisation occurs

- Only if depolarisation >= -50mV, action potential is generated

→ Size of action potential is **fixed at +30mV**

→ all-or-nothing law



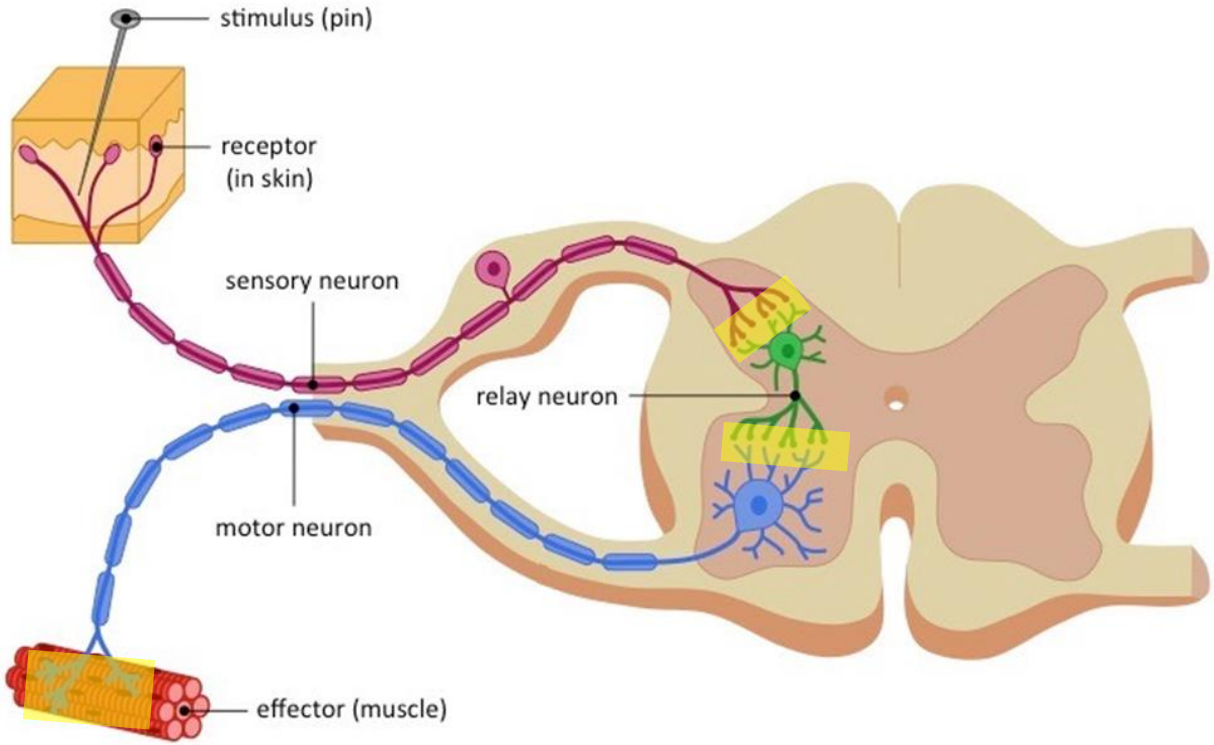


Synapses

Very strong stimulus detected by receptor (E.g. light, sound heat)

Action potential generated in **sensory neurones**

Sensory neurone connected to spinal cord



Synapse with **relay neuron** → action potential passed to **relay neurone**

Synapse with **motor neurones** → action potential passed to **motor neurone**

Cholinergic synapse of neuromuscular junction → Effector/muscle

Effector/muscle carries out response

Structure of Synapse

- **Synapse** = junction between neurones / muscles

A synapse includes:

- **Presynaptic knob**

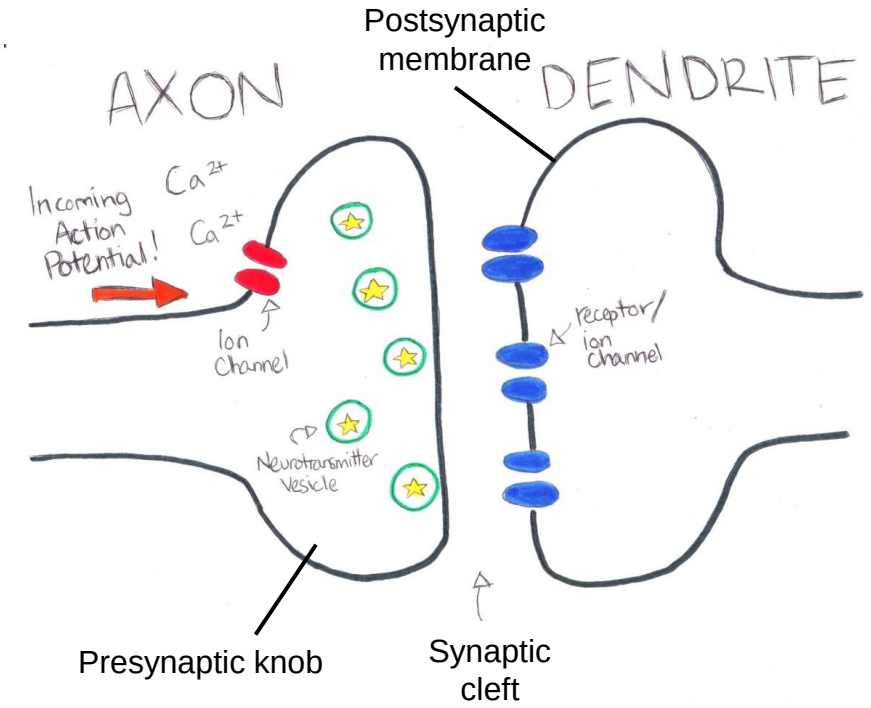
→ Many mitochondria, synaptic vesicles containing neurotransmitters, voltage-gated Ca2+ channels

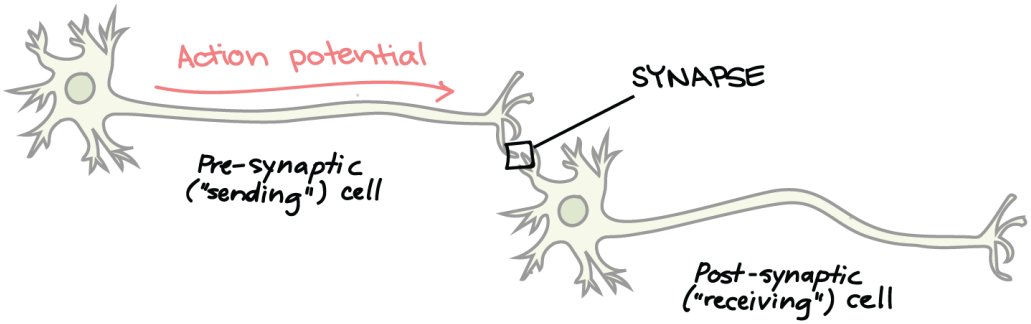
- **Synaptic cleft** = gap

→ has enzymes to breakdown neurotransmitters

- **Postsynaptic membrane**

→ has receptor proteins for neurotransmitters





Roles of Synapses

1. Ensure one-way transmission

- Receptors only on postsynaptic neurone
- Neurotransmitter vesicles only on presynaptic neurone

2. Allow interconnection of nerve pathways

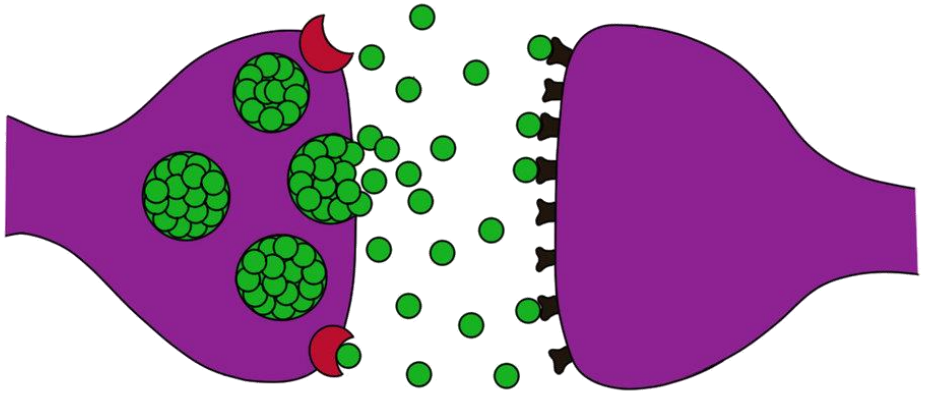
- Nerve impulses can diverge / integrate
- Allow wider range of behaviour / action in response to a stimulus

3. Involved in memory and learning

- Due to new synapses being formed

4. Filter out low-level stimuli

- Weaker stimulus cause release of low quantities of neurotransmitters
- No impulse generated in postsynaptic neurone → brain
- Prevent brain from being overloaded with sensory information



Recap from Chap 11

The Cholinergic Synapse

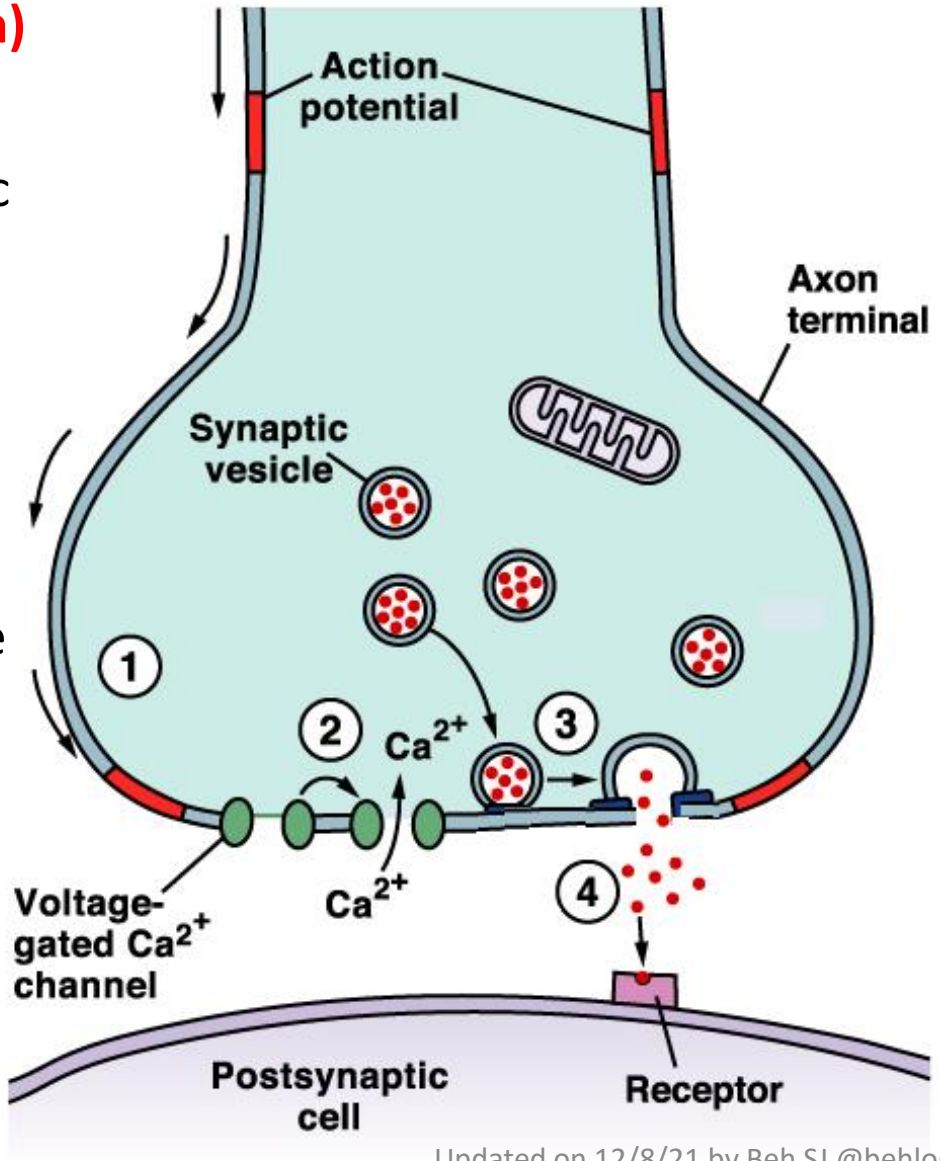
Neurotransmitter = **acetylcholine (ACh)**

1. Action potential reaches presynaptic membrane

2. Voltage-gated **Ca^{2+} channels open**

→ Presynaptic membrane becomes more permeable to Ca^{2+}

→ **Ca^{2+} ions enter** presynaptic neurone



Recap from Chap 11

The Cholinergic Synapse

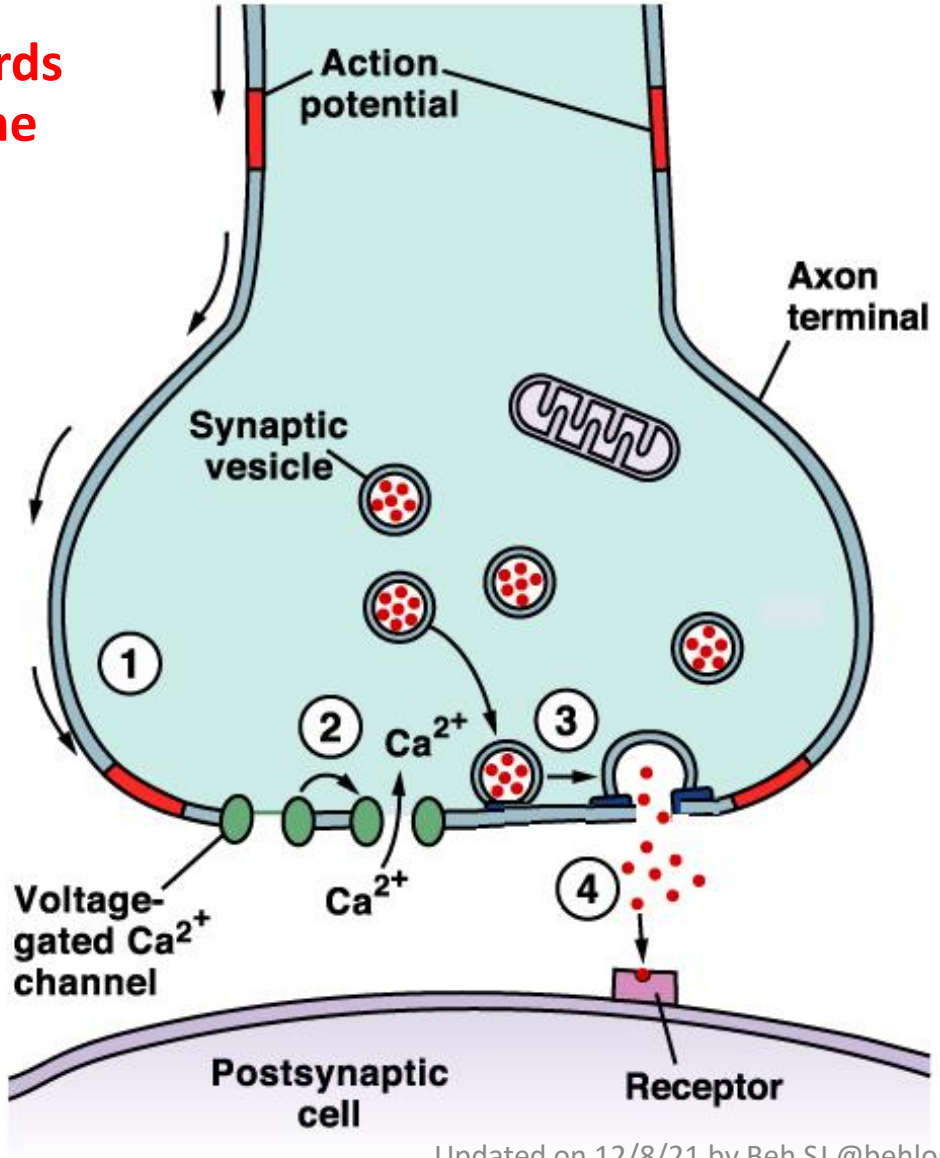
3. **Vesicles** containing ACh **move towards** and **fuse with presynaptic membrane**

→ Exocytosis occurs

→ ACh released into synaptic cleft

4. ACh **diffuse** across synaptic cleft

5. ACh binds with **receptor proteins on postsynaptic membrane**



The Cholinergic Synapse

6. Receptor proteins change shape and **Na^+ channels open**

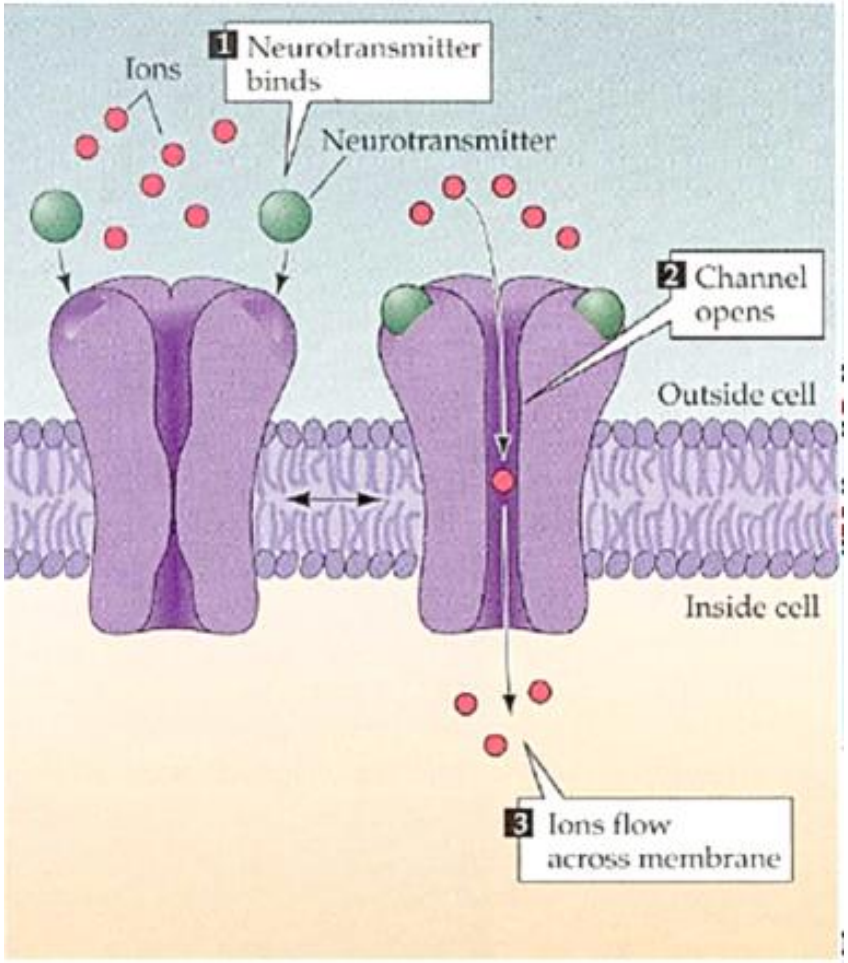
→ **Na^+ enter** postsynaptic neurone

- Postsynaptic neurone **depolarized**
- **Action potential** is generated

- As long as ACh binds with receptors, Na^+ channels will stay open

→ Continuous transmission of action potential

→ Can cause synaptic fatigue / paralysis



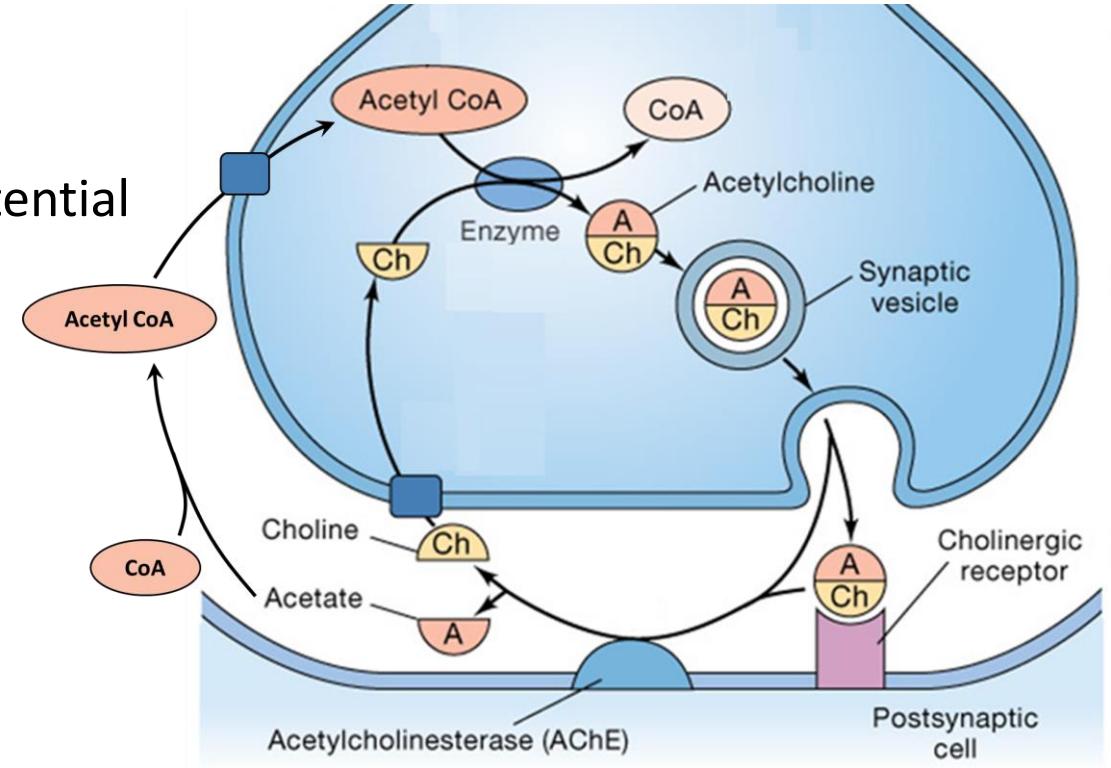
The Cholinergic Synapse

7. ACh breakdown by **acetylcholinesterase** at synaptic cleft

- **ACh → acetate & choline**
- ACh is recycled (ATP needed)

- **Depolarisation stops** in postsynaptic membrane

→ stop continuous action potential



Chapter Outline (Part 1 of 3)

Nervous Communication

- Nervous vs Endocrine systems
- Structure of Neurones
- The Reflex Arc

How do nerve impulses work?

- Roles of **Sensory Receptor Cells**
- **Transmission of Nerve Impulses**
  - Resting Potential
  - Depolarisation / Action Potential
  - Repolarisation
  - Hyperpolarisation / Refractory Period
  - Saltatory Conduction
  - Threshold Potential
- Roles of **Synapses**
  - E.g. Cholinergic Synapse

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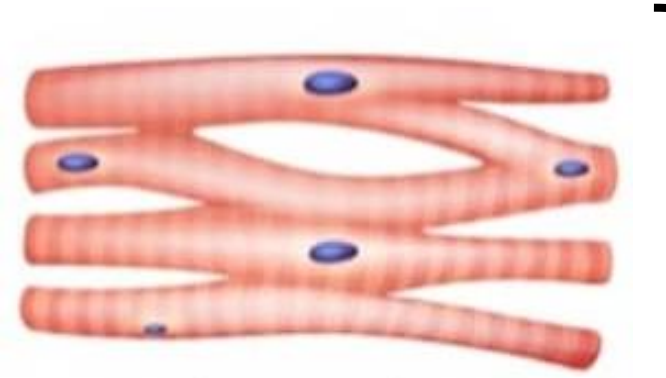
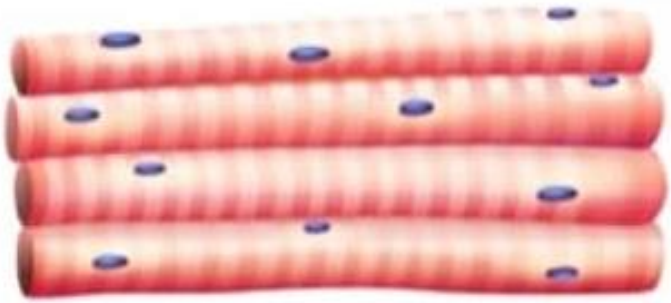
A2 Level Chapter 15

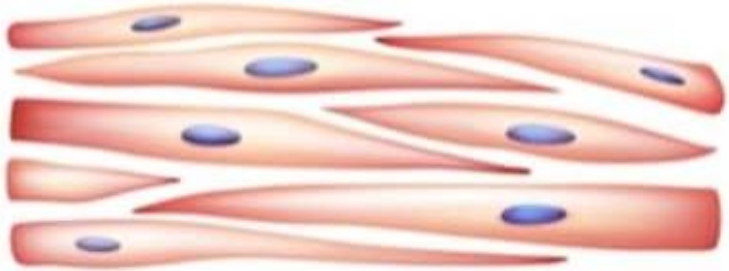
CONTROL AND CO-ORDINATION

Part 2
Striated Muscle Contraction

2 types of Striated Muscle


Cardiac muscle


Skeletal muscle


Smooth muscle

Chapter Outline (Part 2 of 3)

Striated Muscle Contraction

- Structure
  - Muscle fibre
  - Myofibrils
  - Sarcomere
  - Actin and Myofilaments

- **Muscle Contraction**
  - Roles of neuromuscular junctions, transverse system tubules (T-tubules) and sarcoplasmic reticulum
  - Roles of troponin, tropomyosin, calcium ions and ATP
  - **Sliding filament model**

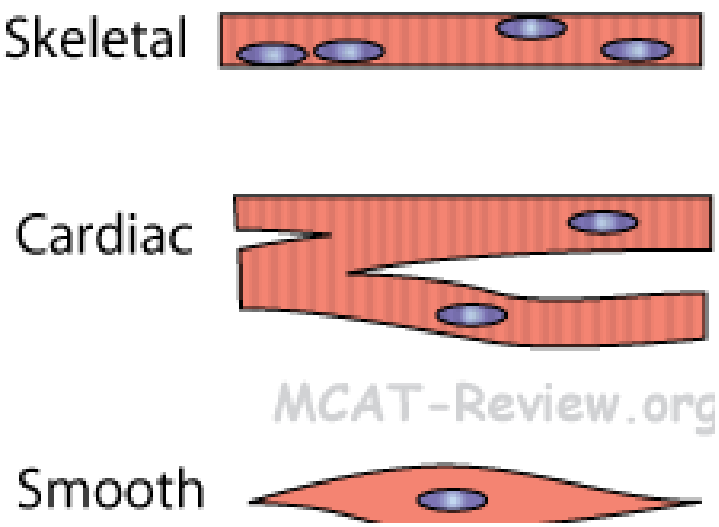
Striated Muscles

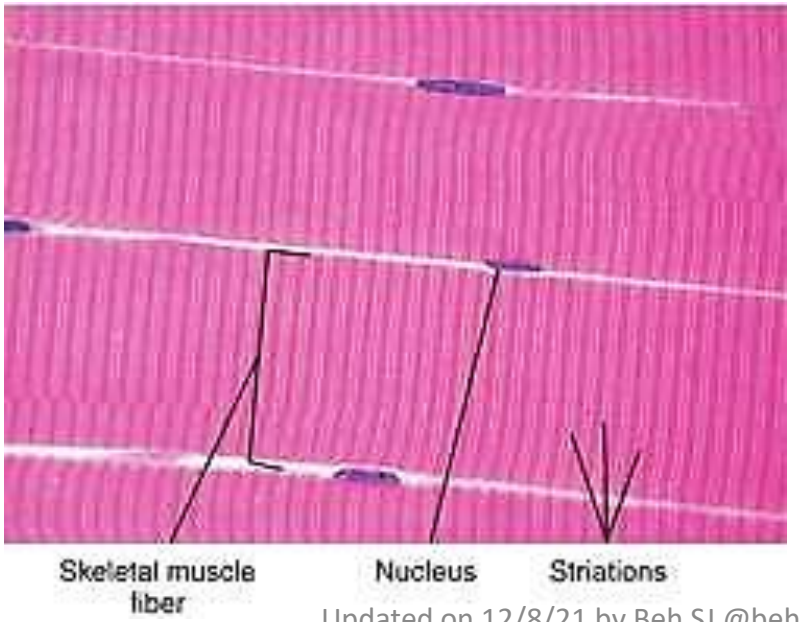
- **Striated** = striped under microscope
- Attached to bones by tendons

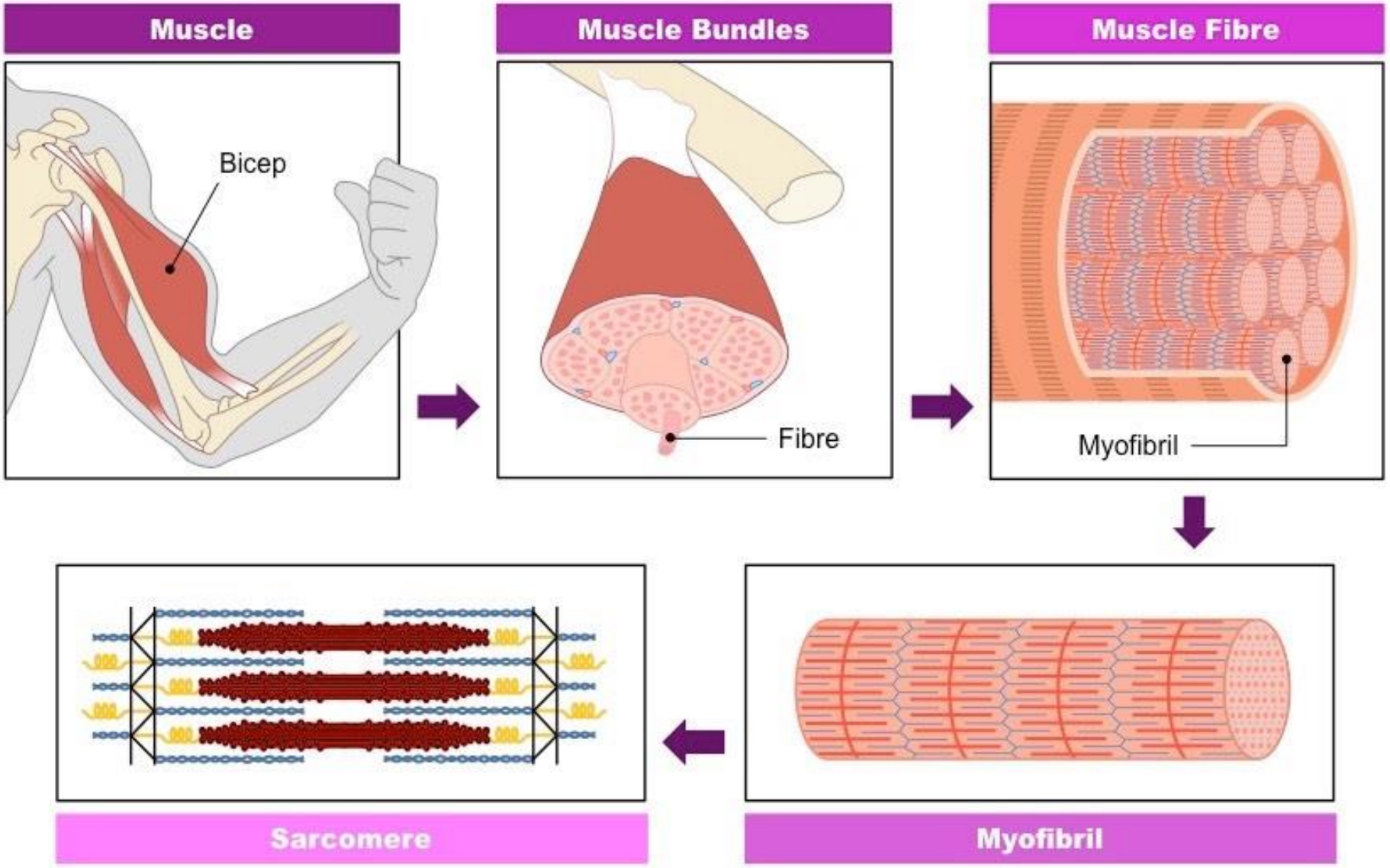
- Many long, cylindrical **muscle fibres**

→ **Multinucleated**

→ Each muscle fibre is made up of **myofibrils**






Muscle
Bicep
Muscle Bundles
Fibre
Muscle Fibre
Myofibril
Myofibril
Sarcomere

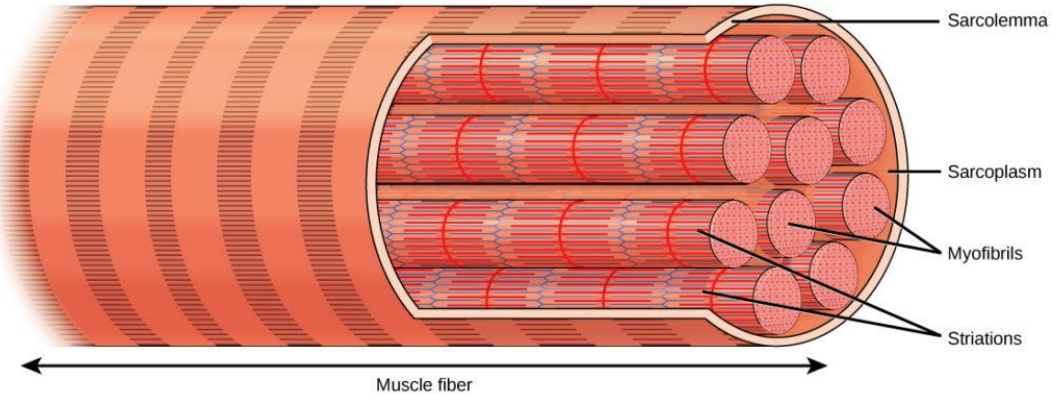
Muscle Fibre

Muscle fibres have:

- Plasma membrane = **sarcolemma**

→ sarcolemma infoldings = transverse system tubules (**T-tubules**)

→ can conduct action potentials



- Cytoplasm = **sarcoplasm**

→ Many parallel **myofibrils**

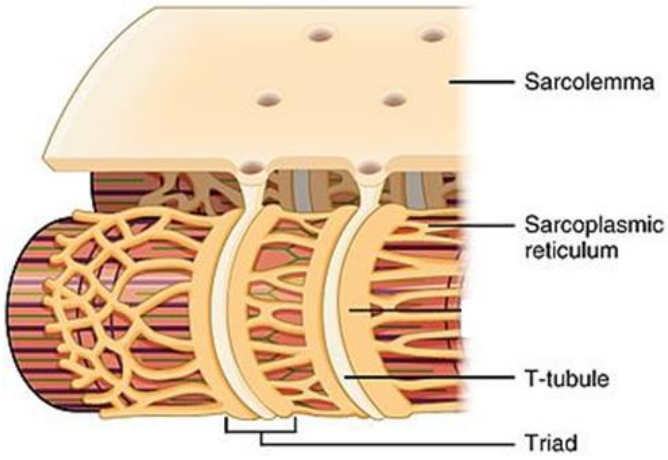
→ Fibres are **multinucleated**

→ Many **mitochondria**

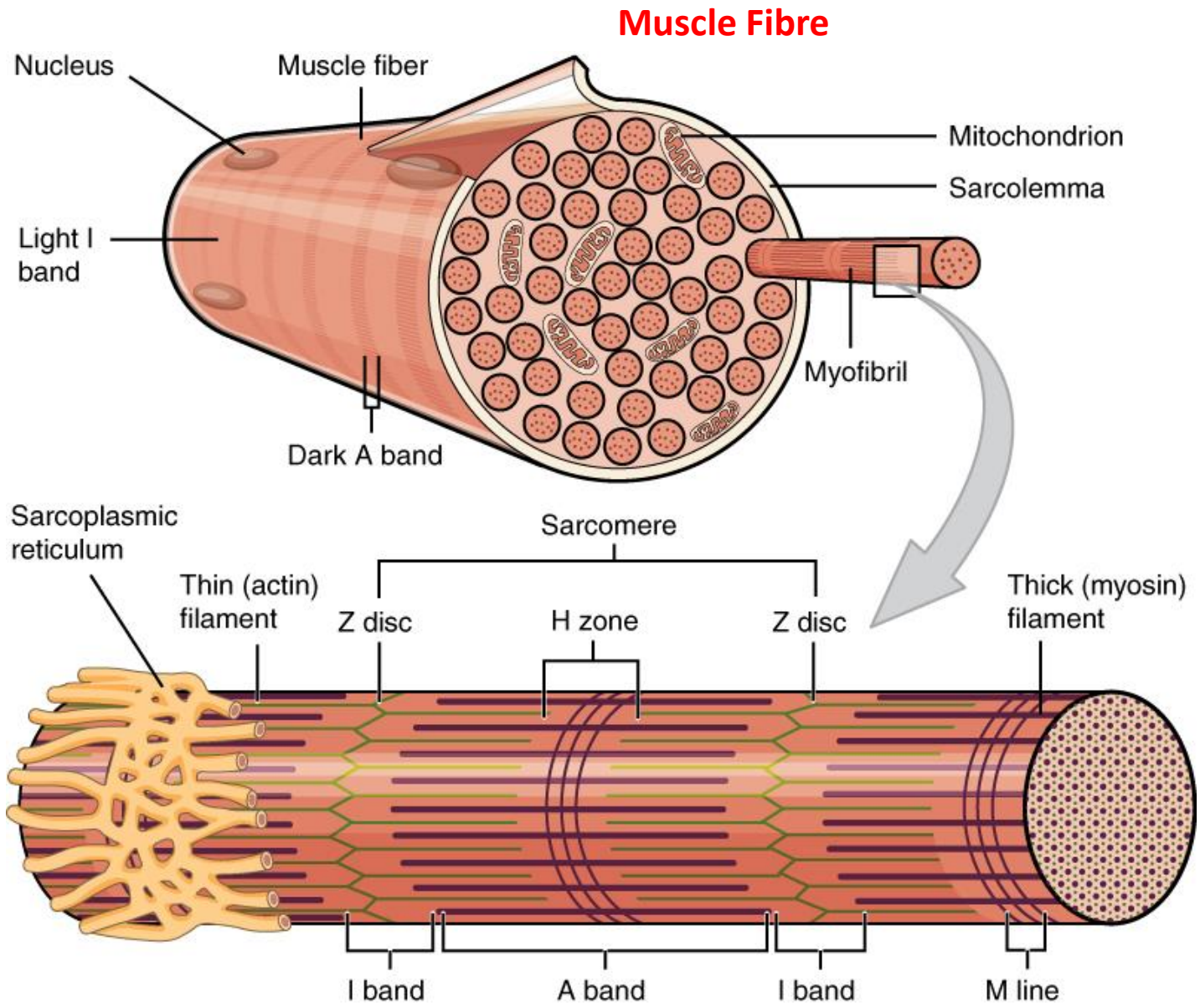
- Specialised ER = **sarcoplasmic reticulum**

→ have **protein pumps**

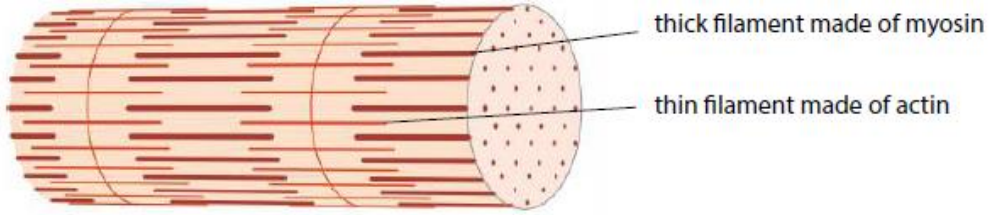
→ have a lot of **Ca^{2+}**



Myofibrils


Muscle Fibre
Nucleus
Muscle fiber
Mitochondrion
Sarcolemma
Light I band
Myofibril
Dark A band
Sarcoplasmic reticulum
Sarcomere
Thin (actin) filament
Z disc
H zone
Z disc
Thick (myosin) filament
Myofibril
I band
A band
I band
M line

Myofibrils



Two types of myofilaments:

- **Thick** filaments = made of **myosin**

→ fibrous protein with globular protein head

→ Attached to M line

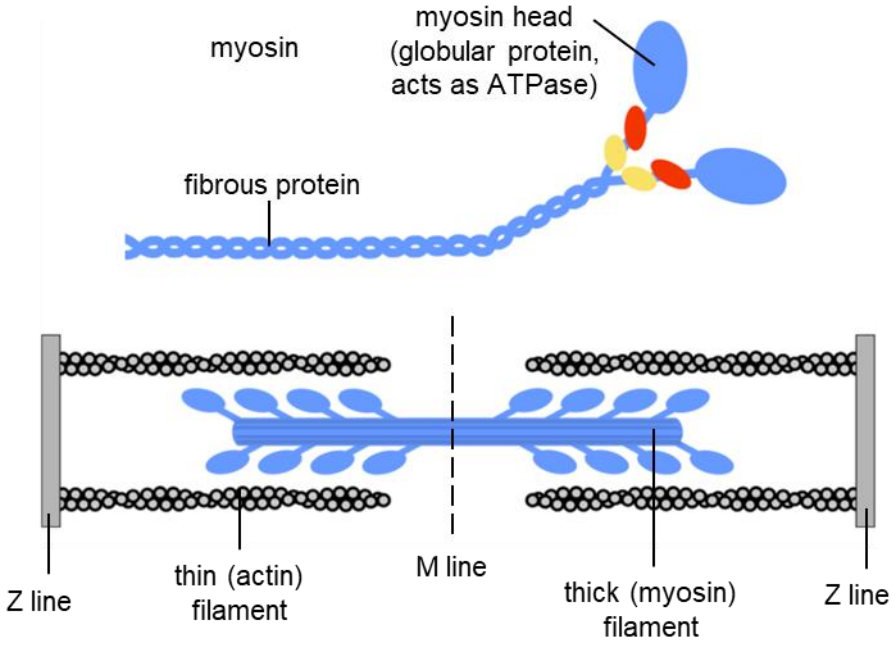
- **Thin** filaments = made of **actin**

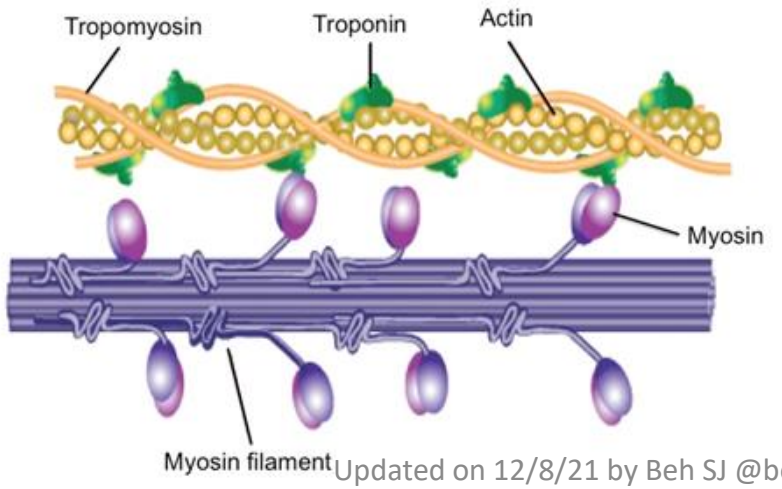
→ chain of globular protein molecules

→ has binding site for myosin

→ troponin and tropomyosin is attached to actin

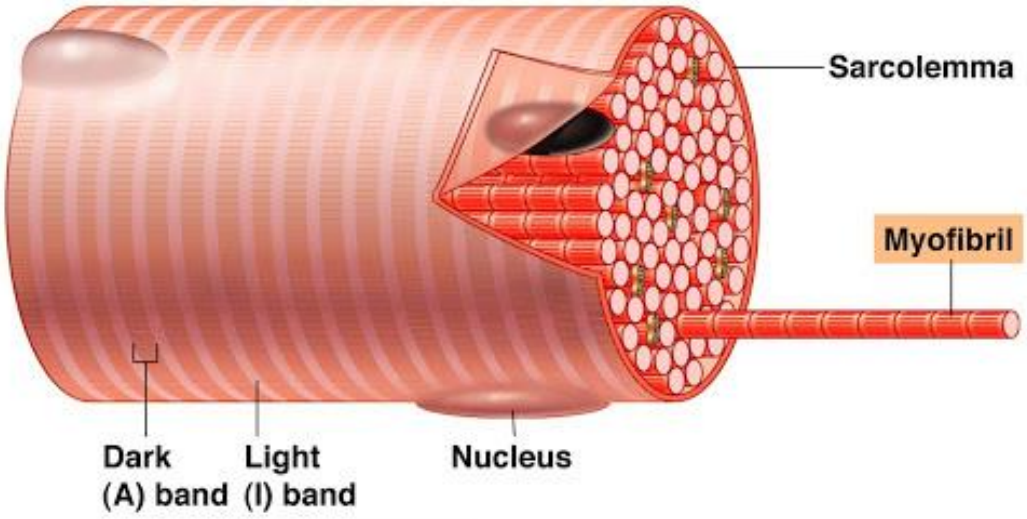
→ Attached to Z line

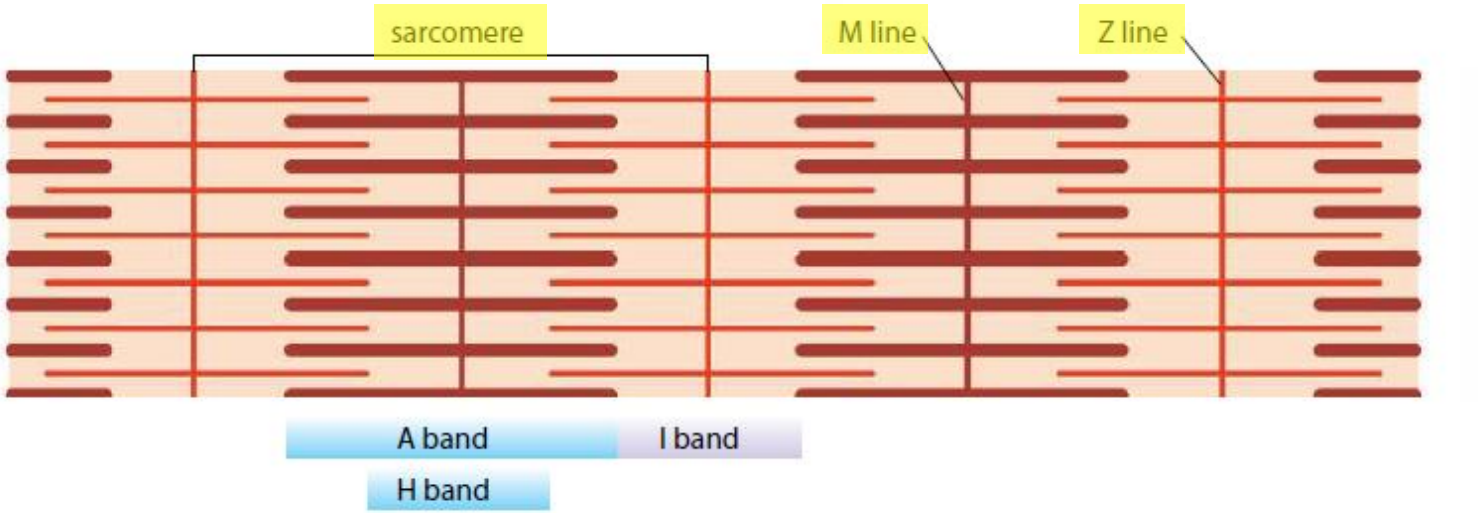




Sarcomere

- Interdigitation of **thick and thin filaments** give striated appearance



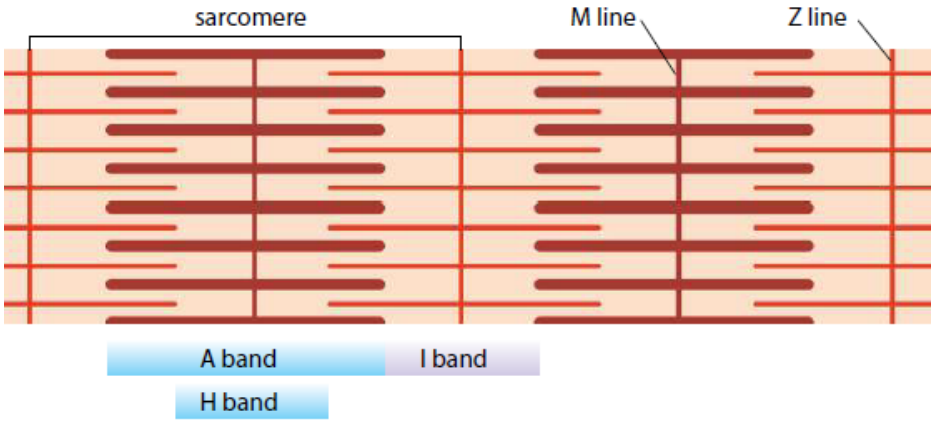


Sarcomere

- Myosin attached to **M line**
- Actin attached to **Z line**

- **Sarcomere** = between 2 Z lines
- Distance between Z line decreases during muscle contraction

- **I band** = light band
- Only thin filaments
- Shortens during muscle contraction

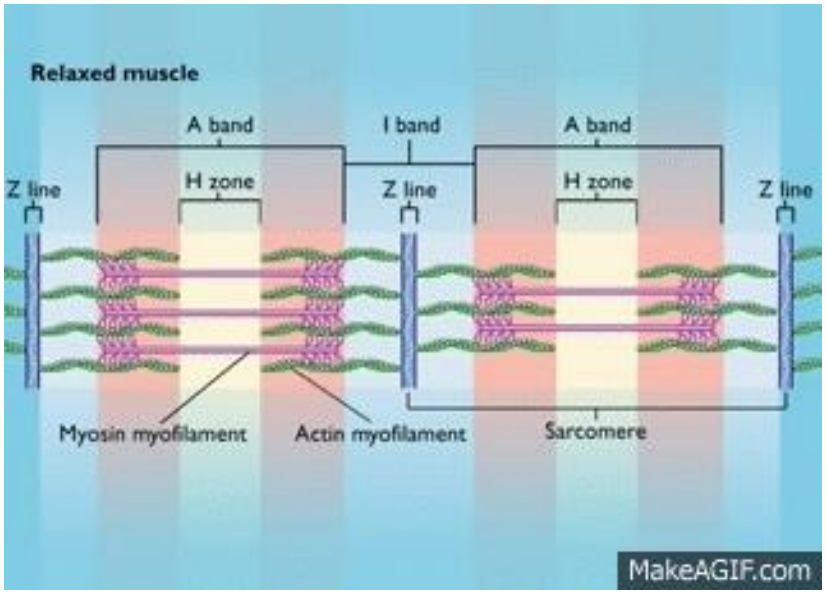


H band = light band at centre of dark band

- Only thick filaments
- Shortens during muscle contraction

A band = dark band

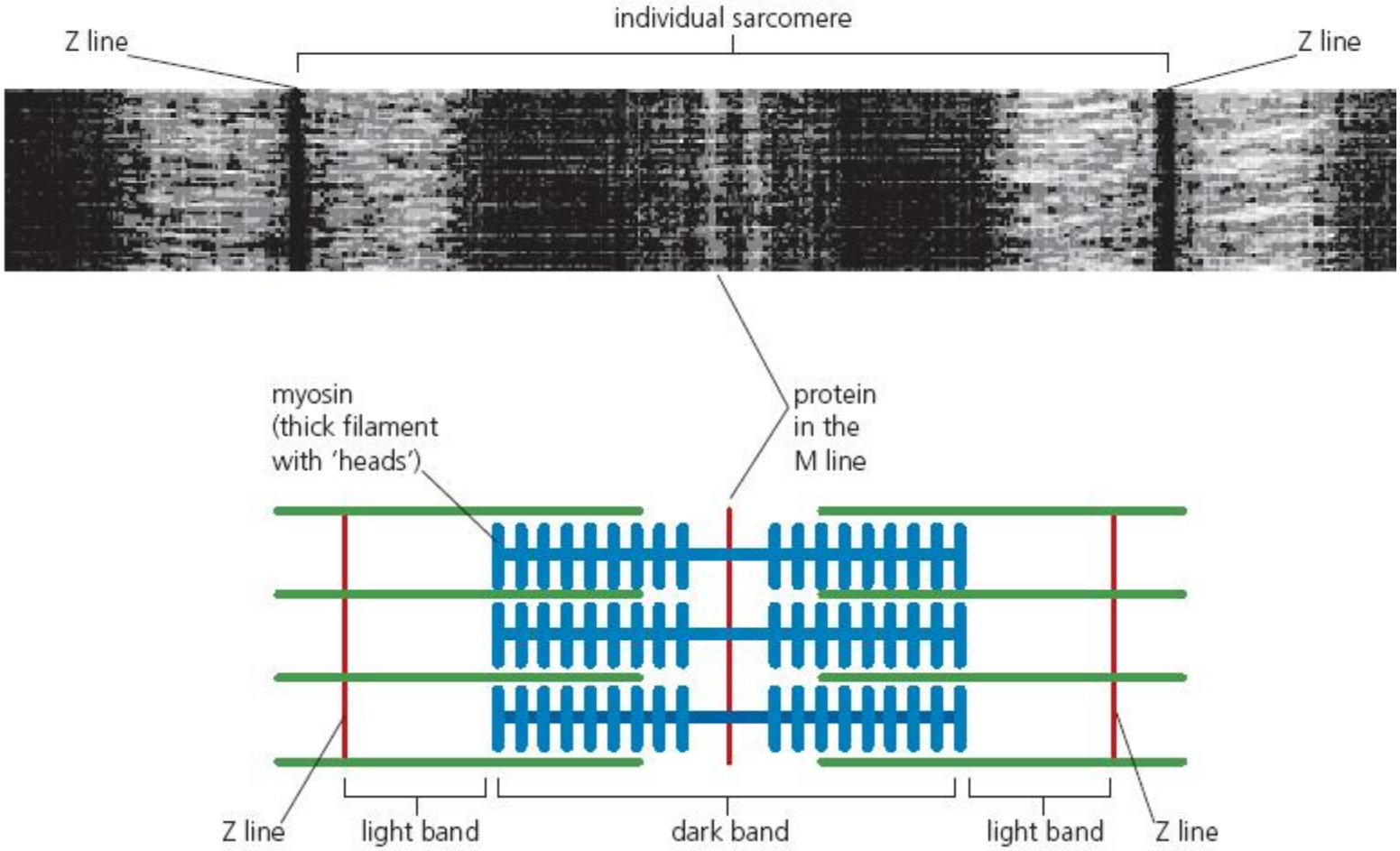
- Overlap of thick and thin filaments
- Stays the same during muscle contract



Sarcomere

Label I band, A band and H band

Electron micrograph of an individual sarcomere (×34 000)

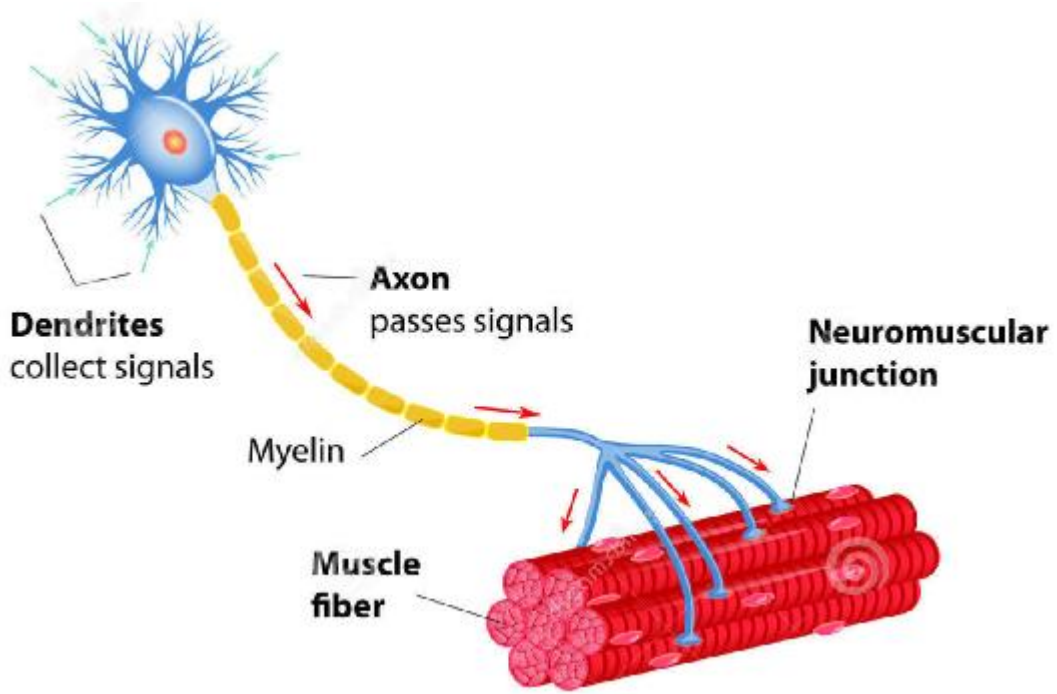


Muscle Contraction

- Begins at **neuromuscular junction**

→ Cholinergic synapse between a motor neurone and a muscle fibre

- Terminal knobs of motor neurone = motor end plate
- Neurotransmitter = **acetylcholine (Ach)**

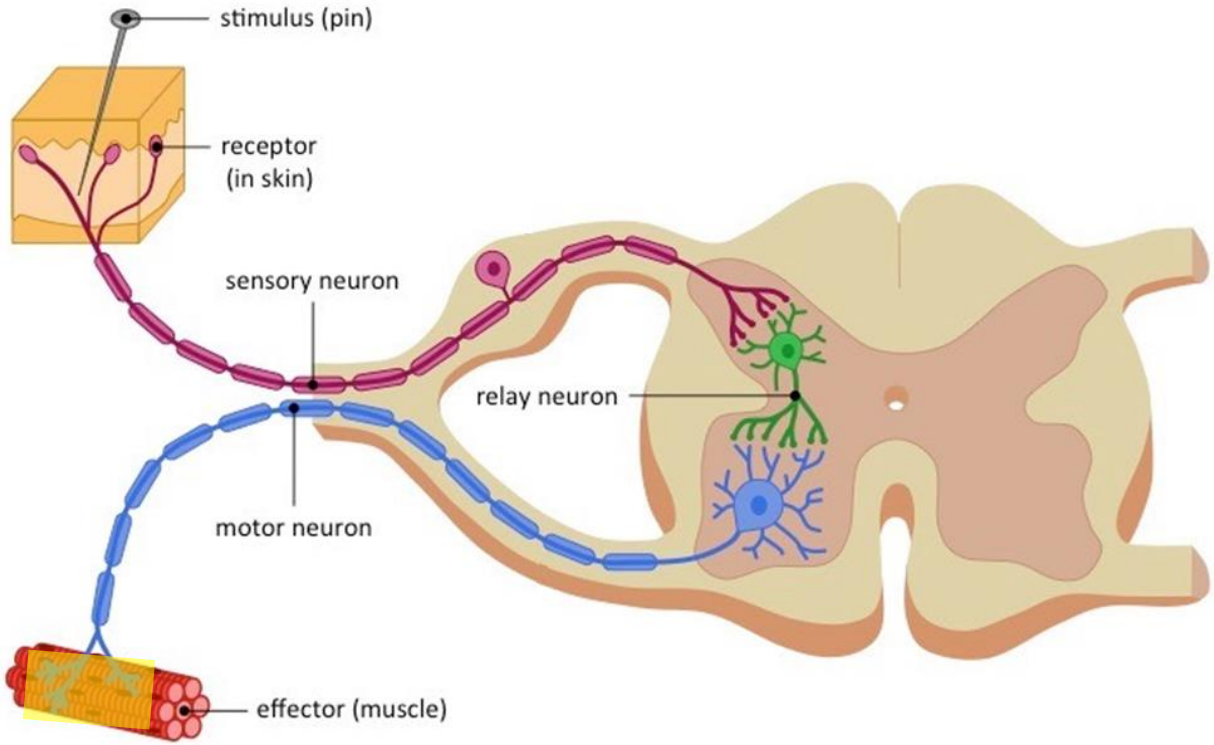


Neuromuscular Junction

Very strong stimulus detected by receptor (E.g. light, sound heat)

Action potential generated in **sensory neurones**

Sensory neurone connected to spinal cord



Synapse with **relay neuron** → action potential passed to **relay neurone**

Synapse with **motor neurones** → action potential passed to **motor neurone**

Cholinergic synapse of neuromuscular junction → Effector/muscle

Effector/muscle carries out response

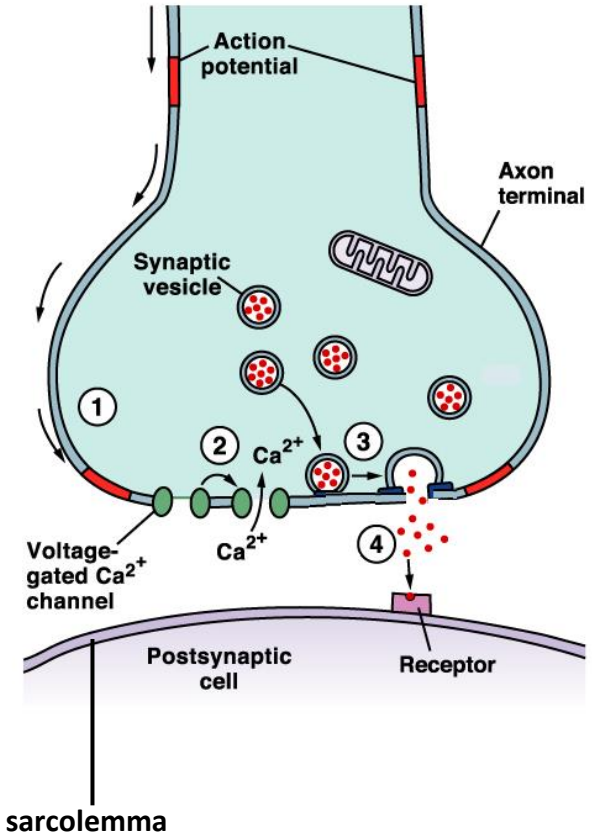
Muscle Contraction

1. Cholinergic synapse of neuromuscular junction

- **Action potential** arrives the presynaptic membrane
- Voltage-gated **Ca^{2+} channels open**
- **Ca^{2+} enter** presynaptic knob

- **Vesicles** containing ACh **fuse** with presynaptic membrane
- ACh released by exocytosis into synaptic cleft
- ACh diffuses across synaptic cleft

- **ACh** bind to **receptors** on sarcolemma (muscle cell membrane)
- Na^+ channel opens
- **Na^+ ions enter** sarcoplasm of muscle cell
- Sacrolemma depolarised

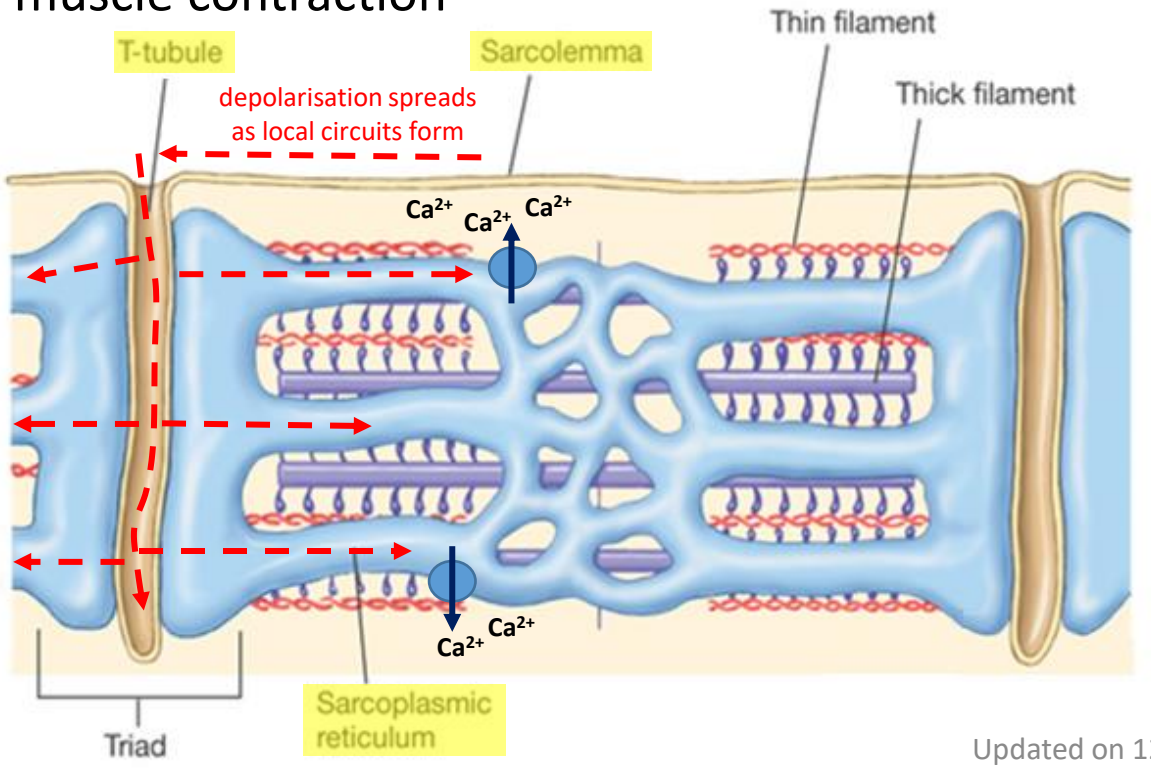


Muscle Contraction

2. Depolarisation and Ca^{2+}

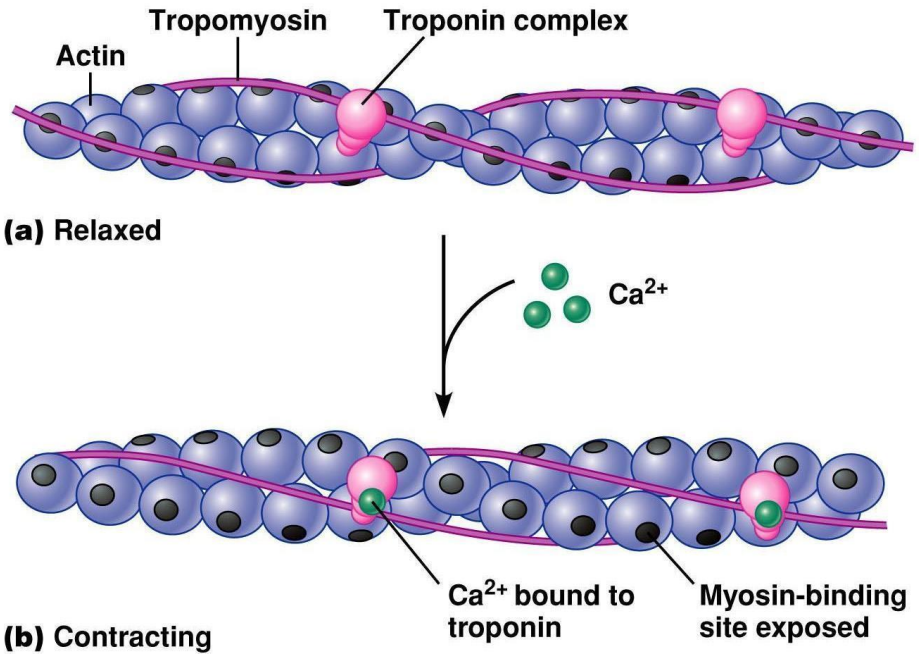
- **Depolarisation spreads** via **T-tubules** → **sarcoplasmic reticulum (ER)**
- Sarcoplasmic reticulum **depolarized**

- Voltage-gated **Ca^{2+} channels open**
- **Ca^{2+} diffuse out** from sarcoplasmic reticulum → **sarcoplasm**
- Ca^{2+} initiates muscle contraction



Muscle Contraction

3. Troponin and tropomyosin



When muscle is relaxed:

- **Troponin** = attached to tropomyosin
- **Tropomyosin** = blocks myosin-binding site on actin

When muscle contracts:

- Ca^{2+} in sarcoplasm bind to **troponin**

→ Troponin changes shape and moves **tropomyosin**

→ **Exposes myosin-binding site on actin**

→ Allows **myosin head** to attach and form **cross-bridge** with actin

Muscle Contraction

4. **Sliding Filament Model**

1) Myosin head with ADP and Pi form **cross-bridges with actin**

→ Pi is released

2) **Myosin head tilts** and **pulls actin**

→ **Power stroke moves actin** towards M line

→ Myofibril / sarcomere shortens

→ ADP released from myosin head

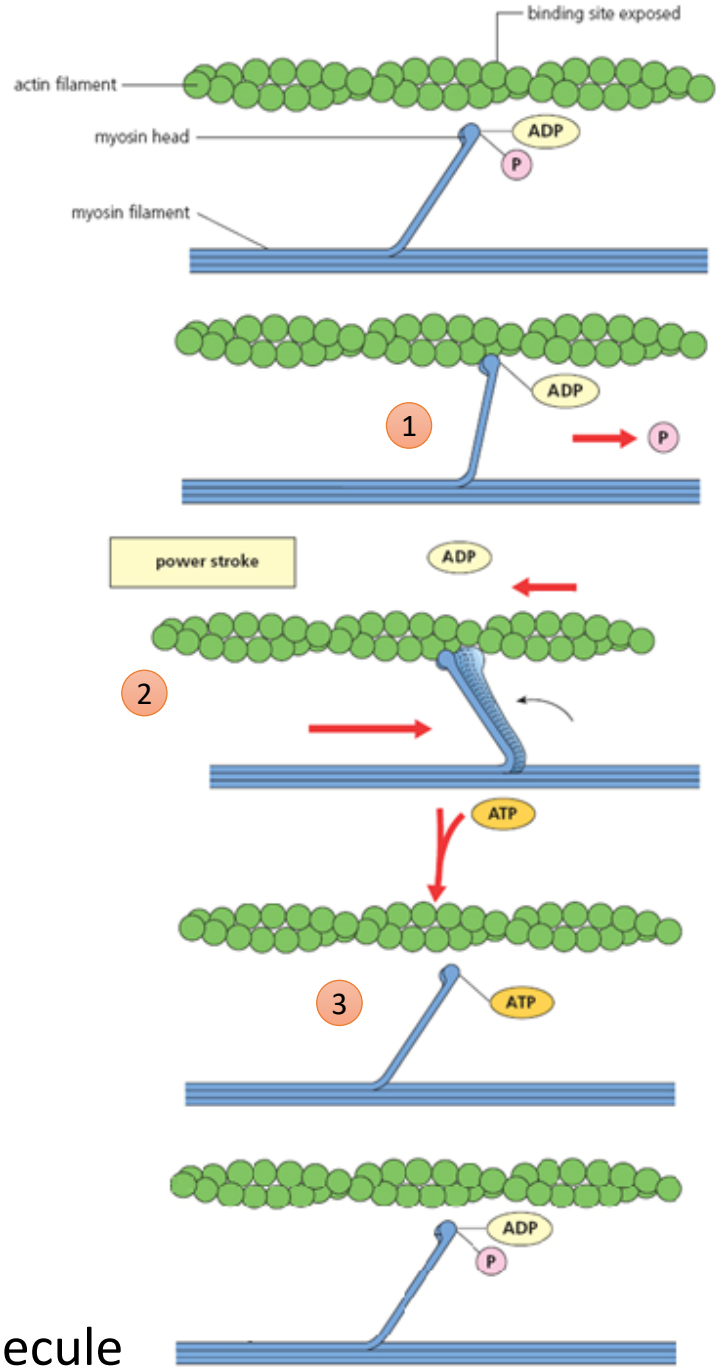
3) **ATP** binds to myosin head

→ ATPase hydrolyses ATP into **ADP and Pi**

→ **Myosin head lets go** of actin

→ Myosin **moves back** to original position

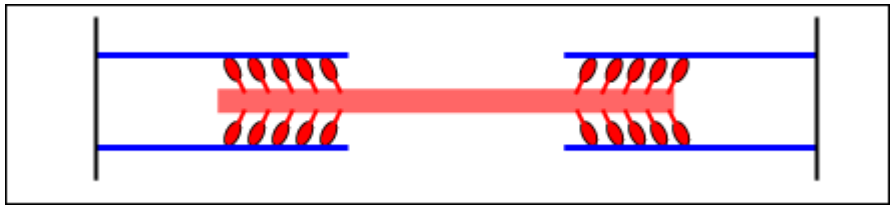
4) **Process repeated** at site further along actin molecule

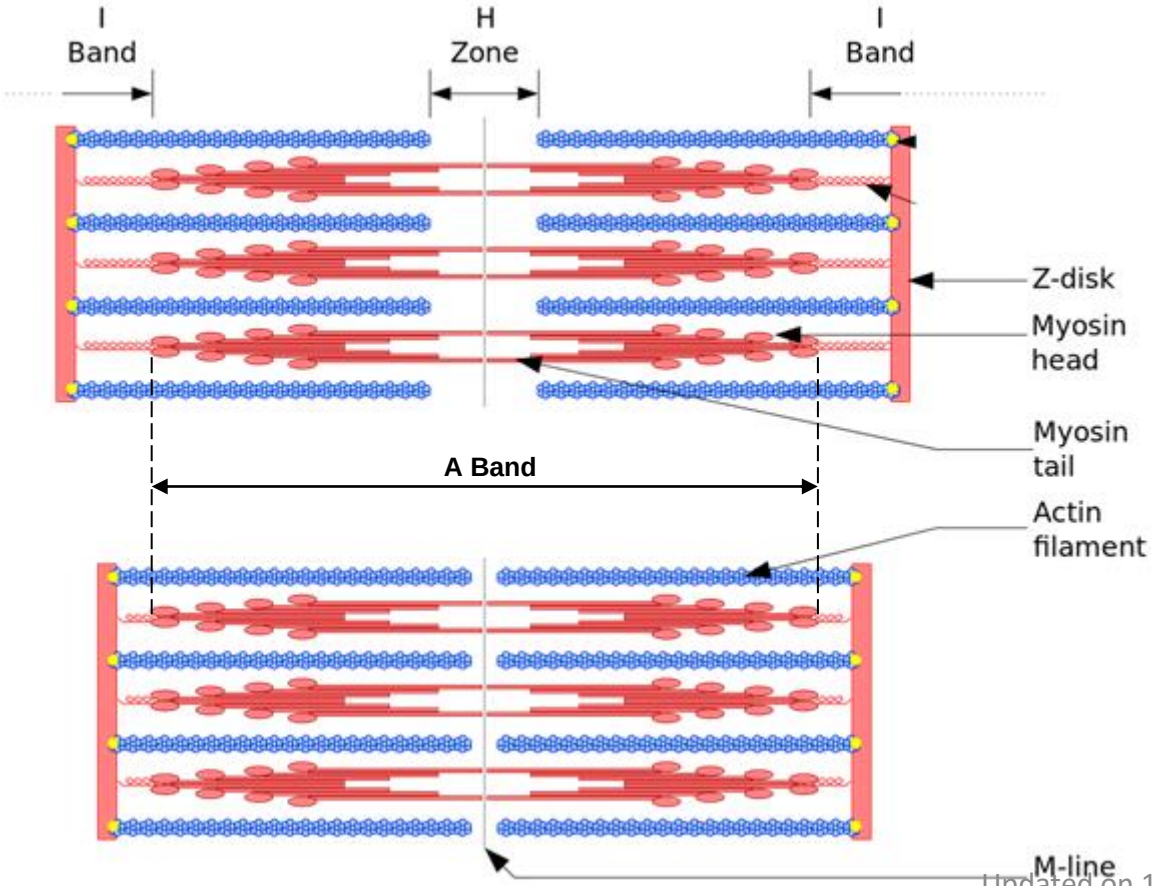


Muscle Contraction

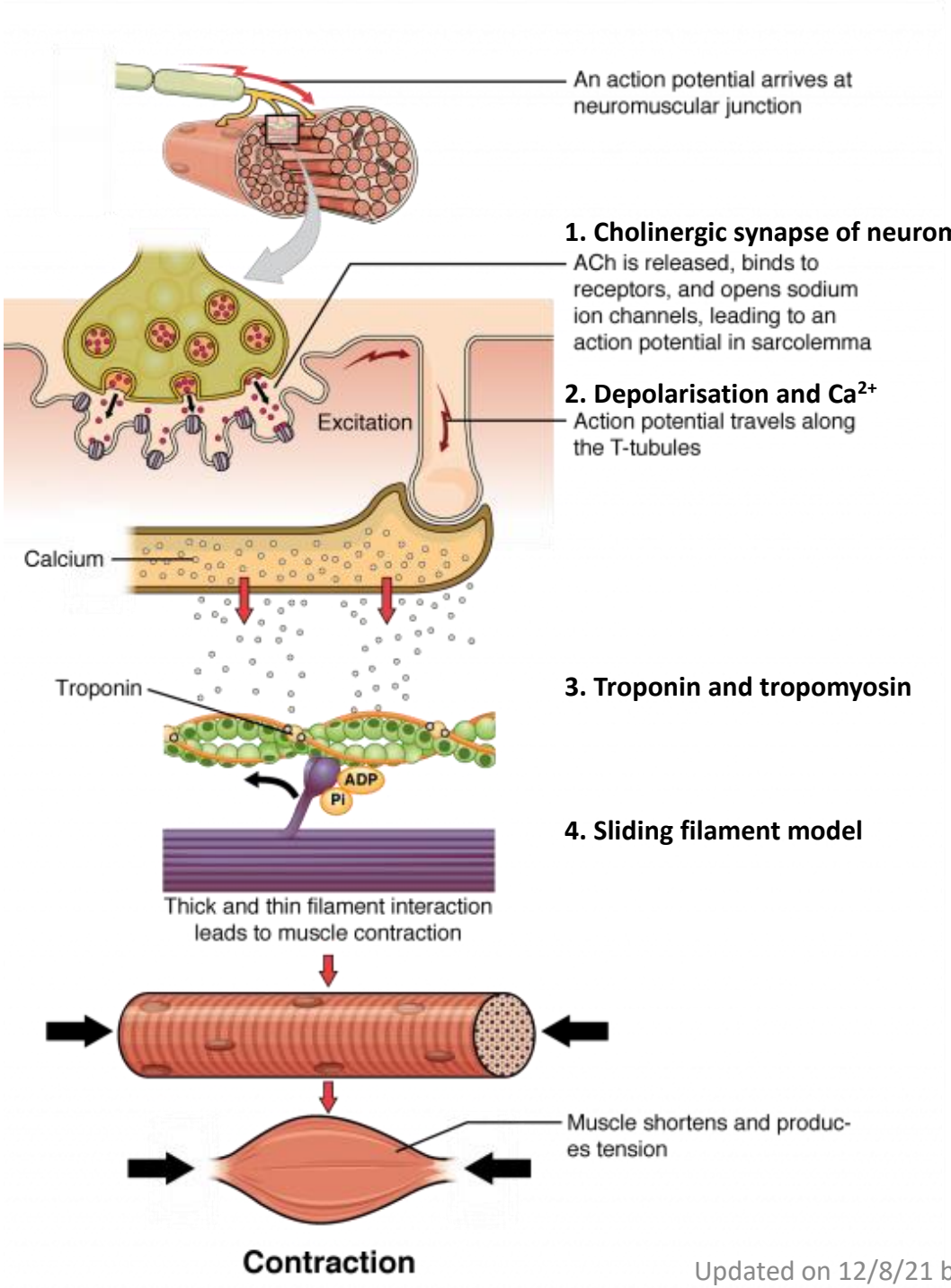
Sarcomere shortens during muscle contraction

- H band shortens
- I band shortens
- A band remains the same





Summary


An action potential arrives at neuromuscular junction
1. Cholinergic synapse of neuromuscular junction
ACh is released, binds to receptors, and opens sodium ion channels, leading to an action potential in sarcolemma
2. Depolarisation and Ca^{2+}
Action potential travels along the T-tubules
Excitation
Calcium
Troponin
3. Troponin and tropomyosin
ADP
Pi
4. Sliding filament model
Thick and thin filament interaction leads to muscle contraction
Muscle shortens and produces tension
Contraction

Muscle Relaxation

When action potential stimulation stops….

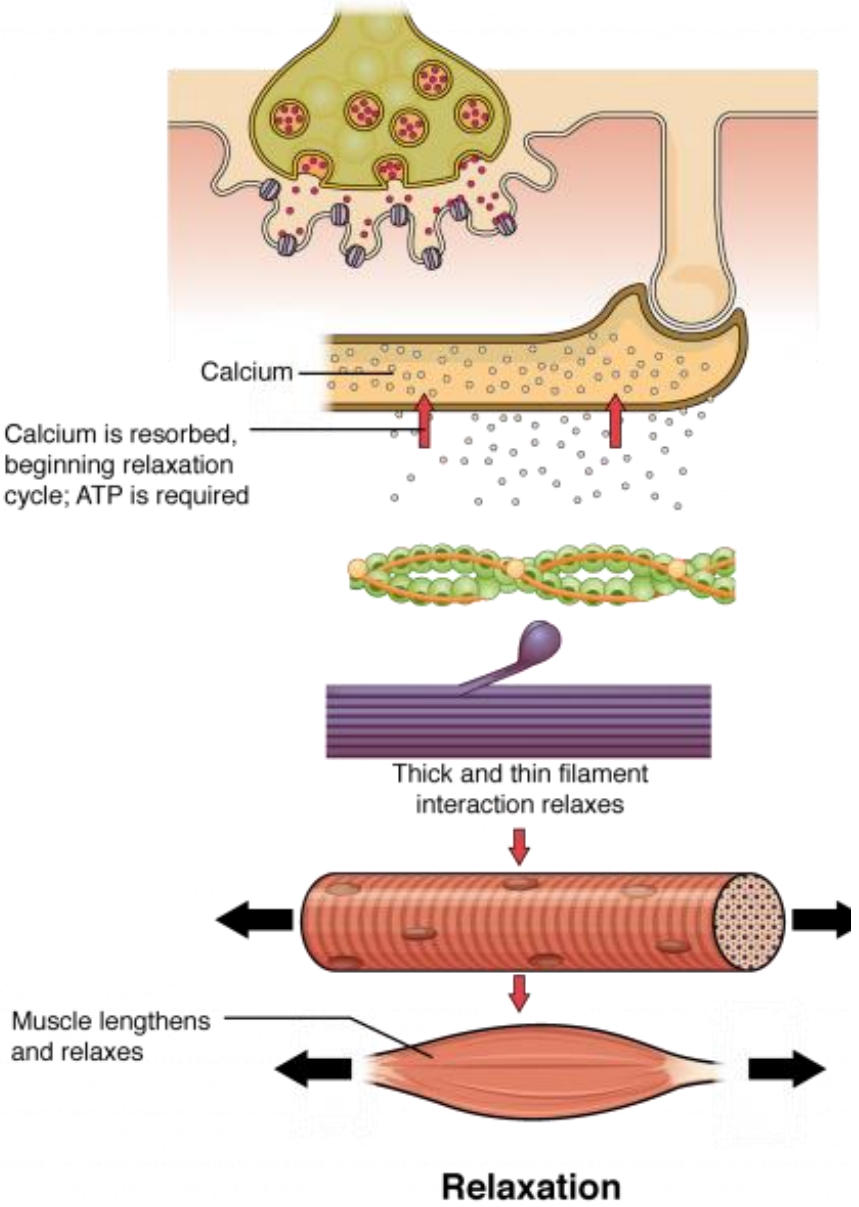
- **Ca^{2+} is actively pumped into sarcoplasmic reticulum**

→ Ca^{2+} do not bind to troponin on actin filament

→ Tropomyosin moves to block myosin-binding sites on actin filament

→ Filaments slide back to original position

→ Muscle relaxes



Source of ATP

- Muscles uses a lot of ATP
- Only small amount of ATP present in muscle

More ATP is synthesized by….

1. **Aerobic respiration** in mitochondria
2. **Lactate pathway** in sarcoplasm

3. **Creatine phosphate** in sarcoplasm
- Immediate source of energy once ATP is used up

creatine phosphate + ADP ⇌ creatine + ATP

creatinine → blood plasma → ultrafiltration @ kidney → urine

- Reversible when the demand of ATP reduced
- If not, creatine converted to creatinine and excreted in urine

Chapter Outline (Part 2 of 3)

Striated Muscle Contraction

- Structure
  - Muscle fibre
  - Myofibrils
  - Sarcomere
  - Actin and Myofilaments

- **Muscle Contraction**
  - Roles of neuromuscular junctions, transverse system tubules (T-tubules) and sarcoplasmic reticulum
  - Roles of troponin, tropomyosin, calcium ions and ATP
  - **Sliding filament model**

Chapter Outline (Part 3 of 3)
To Be Continued…

Control and Communication in Plants

Electrical communication in plants

- **Venus fly trap**

Chemical communication in plants

- Role of **auxin** in elongation growth
- Role of **gibberellin** in germination of wheat/barley
- Role of gibberellin in stem elongation (incl. role of dominant allele, *Le*)