Cell Biology

Unit 2 · Lesson 3 · Carriers, channels and vesicles

The notes for Lesson 3 of Unit 2: chapters 2.5 to 2.7 of the unit book, with their figures, worked examples, exam traps and quizzes, in one file. The full unit book, with every chapter, the exam question bank and the glossary, is linked at the end.

Lesson 3 of 3 | 3 chapters | Chapters 2.5 to 2.7

About these notes

These are the notes for **Lesson 3 · Carriers, channels and vesicles** of the University free n easy video lessons on Cell Biology Unit 2, Membranes and Membrane Transport. They are chapters 2.5 to 2.7 of the unit book, unchanged, so a page here is the same page in the full book.

2.5 Uniport, symport and antiport systems

Carriers are named by how many solutes they move and in which direction. A **uniporter** moves one solute. A **symporter** moves two solutes in the same direction, and an **antiporter** moves two in opposite directions; together these two are called **cotransporters**, because the movement of one solute is tied to the other. At other universities it appears inside passive and active transport, so this chapter is kept short.

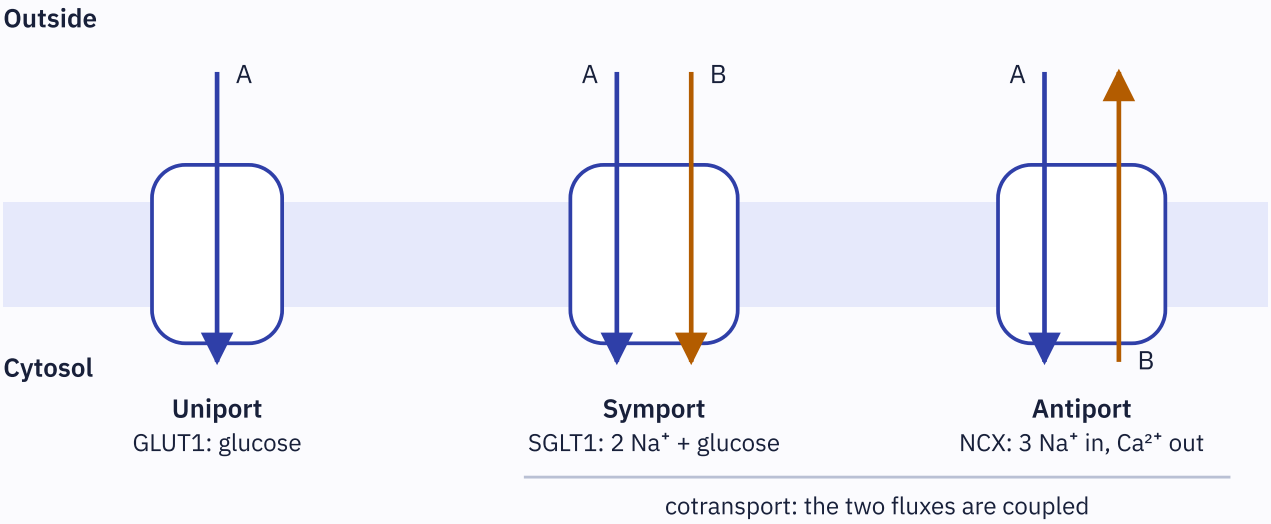


Figure 2.5.1 The three carrier systems. In symport and antiport, A is usually the driving ion (Na^+ in animals, H^+ in bacteria and plants) moving down its gradient, and B is the solute pushed up its gradient. Notice that the arrows show direction only; whether a move is uphill depends on the gradients.

The three systems

- **Uniport**: one solute, facilitated diffusion, always downhill. GLUT1 (glucose into red cells) and GLUT4 (glucose into muscle and fat when insulin moves it to the surface).
- **Symport**: both solutes the same way. SGLT1 in the intestine (2 Na^+ with 1 glucose); Na^+–amino acid symporters; the *E. coli* lactose permease (H^+ with lactose).
- **Antiport**: solutes in opposite directions. NCX in heart muscle (3 Na^+ in, 1 Ca^{2+} out); the Na^+/H^+ exchanger that pushes acid out and controls cytosolic pH; band 3 of the red cell (Cl^- in, HCO_3^- out), which helps blood carry CO_2; the mitochondrial ADP/ATP carrier.

A carrier cannot "half work": a symporter will not move B unless A binds too. That strict coupling is what lets the downhill flow of A pay for the uphill flow of B. Uniporters and cotransporters share the alternating access mechanism of chapter 2.3; only the number of binding sites differs.

The words describe direction, not energy. Some books also call the Na^+/K^+-ATPase an antiport pump, because Na^+ and K^+ move in opposite directions; its energy still comes from ATP, so it is primary active transport (chapter 2.4).

How far can a symporter push?

At the limit, the energy released by the driving ions just equals the energy needed by the solute. For a symporter bringing n Na^+ in with one uncharged solute S:

$$R\,T\,\ln([S]_{in} / [S]_{out}) = n \times |\Delta G(Na^+ \text{ in})|$$

So each extra Na^+ in the stoichiometry multiplies the possible concentration ratio, it does not just add to it.

WORKED EXAMPLE

Question. In an intestinal cell, ΔG for Na^+ entry is −13.18 kJ/mol (from the worked example of chapter 2.4) and RT = 2.577 kJ/mol at 310 K. Find the largest glucose ratio [glucose]in / [glucose]out that a symporter can build with (a) 1 Na^+ per glucose, (b) 2 Na^+ per glucose. `5 marks`

1. (a) ln(ratio) = 1 × 13.18 / 2.577 = 5.11. Ratio = $e^{5.11} \approx$ **166**.
2. (b) ln(ratio) = 2 × 13.18 / 2.577 = 10.22. Ratio = $e^{10.22} \approx$ **2.8×10^4**.
3. Glucose is uncharged, so no voltage term is needed for it.
4. Conclusion: doubling the Na^+ per glucose squares the possible ratio ($166^2 \approx 2.8 \times 10^4$). That is why the 2 : 1 SGLT1 can absorb almost all the glucose from the gut, even when gut glucose is low.

EXAM TRAPS

- "Symport" and "antiport" say nothing about ATP. Name the energy source separately.
- Cotransport covers both symport and antiport; uniport is not cotransport.
- Band 3 is an antiporter (Cl^-/HCO_3^- exchange), not a channel.
- Examiners ask: "Distinguish uniport, symport and antiport with one example each and a diagram."

CHECK YOURSELF

– `REMEMBER` **Define antiport and give one example.** `2 marks`

A carrier that moves two solutes in opposite directions, such as the Na^+/Ca^{2+} exchanger (3 Na^+ in, 1 Ca^{2+} out).

– `REMEMBER` **Which ion drives most symporters in bacteria?** `1 mark`

H^+ (protons), as in the lactose permease.

– `UNDERSTAND` **Why must a cotransporter be strictly coupled?** `2 marks`

If the driving ion could cross alone, its gradient would run down without moving the second solute; coupling makes every downhill ion pay for an uphill solute.

– APPLY **With $|\Delta G(Na^+ \text{ in})|$ = 13.18 kJ/mol and RT = 2.577 kJ/mol, what amino acid ratio can a 1 : 1 Na^+ symporter reach?** 2 marks

ln(ratio) = 5.11, ratio ≈ **166** (for an uncharged amino acid).

– ANALYZE **Digoxin partly blocks the Na^+/K^+ pump in heart cells, and the heart beats more strongly. Explain using NCX.** 4 marks

Less pumping lets cytosolic Na^+ rise, so the Na^+ gradient that drives NCX becomes smaller. NCX removes Ca^{2+} more slowly, Ca^{2+} builds up in the cell and its stores, and more Ca^{2+} is available for each contraction.

BEYOND THE SYLLABUS

This is not for the exam. Transporters of this kind belong to large families named in the SLC (solute carrier) classification, which counts several hundred human genes; many are drug targets or decide how drugs enter cells. Crystal and cryo-EM structures of LacY and LeuT showed the "rocking bundle" form of alternating access. Search next for "SLC transporter family" and "LeuT alternating access".

2.6 Ion channels: ligand-gated and voltage-gated

An ion channel is a membrane protein with a water-filled pore that lets a particular ion flow down its electrochemical gradient. Channels are not open all the time: they are **gated**, opening when a chemical binds (ligand-gated) or when the membrane voltage changes (voltage-gated). Because millions of ions can rush through a single open channel each second, channels are the basis of every fast electrical event in the body, from a nerve impulse to a heartbeat.

You need: the electrochemical gradient and the ion concentrations from chapter 2.4.

Channels versus carriers

A carrier binds its solute and changes shape for every molecule moved; a channel, once open, is a continuous pore. So a channel is much faster: Lodish gives about 10^7–10^8 ions per second for a channel against about 10^2–10^4 molecules per second for carriers (from memory; check). Channel transport is always passive; there is no channel that pumps.

A channel has two key parts:

- The **selectivity filter**, the narrowest part of the pore, which lets one kind of ion through. In K^+ channels, a ring of backbone carbonyl oxygens sits exactly where the water molecules around a K^+ ion would be. A K^+ ion can shed its water and sit comfortably in the filter; the smaller Na^+ ion does not fit those oxygens well and stays out. Roderick MacKinnon solved this structure and shared the 2003 Nobel Prize in Chemistry.
- The **gate**, which opens and closes the pore in response to a stimulus.

Erwin Neher and Bert Sakmann's **patch clamp** (Nobel Prize 1991) records the current through a single channel. It shows that each channel snaps between fully closed and fully open; a stimulus changes how much of the time it spends open.

Membrane potential and the Nernst equation

When only one ion can cross, it flows until the voltage it builds balances its concentration gradient. That voltage is its **equilibrium potential**, given by the Nernst equation:

$$E = \frac{R\,T}{z\,F}\ln\frac{C_{out}}{C_{in}} \approx \frac{61.5\ \text{mV}}{z}\log_{10}\frac{C_{out}}{C_{in}} \quad \text{at } 37\ ^\circ\text{C}$$

Here E is the equilibrium potential (inside relative to outside), z the ion's charge, and C_out, C_in the concentrations outside and inside. Resting cells have many open K^+ **leak channels**, so the resting potential (about −70 mV) lies close to E_K. Opening Na^+ channels pushes the voltage towards E_Na, which is positive.

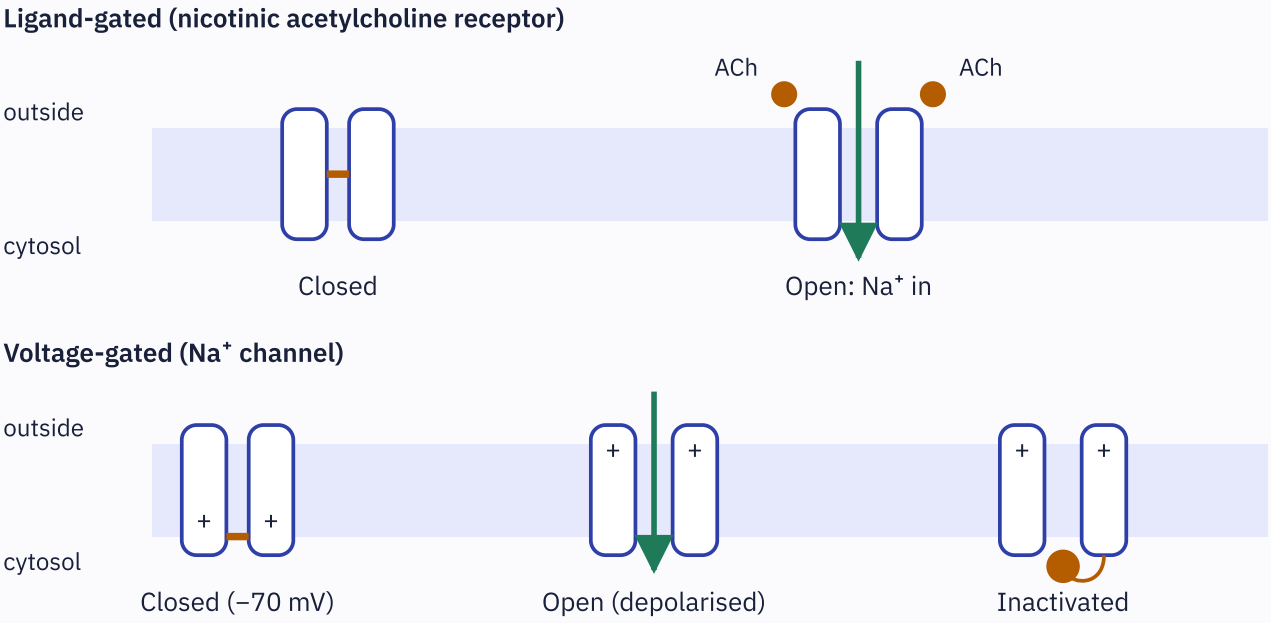


Figure 2.6.1 Top: the nicotinic receptor stays shut until two acetylcholine (ACh) molecules bind, then lets Na^+ flow in. Bottom: in a voltage-gated Na^+ channel the "+" marks the charged S4 voltage sensors; depolarisation moves them outward and opens the gate. Within about a millisecond a cytosolic loop (orange ball) plugs the pore: the channel is inactivated and cannot reopen until the membrane returns to rest.

Ligand-gated channels

These channels open when a specific molecule, the **ligand**, binds. Most are receptors for neurotransmitters at synapses, so they turn a chemical signal into an electrical one in under a millisecond.

- **Nicotinic acetylcholine receptor** (nAChR) at the nerve–muscle junction: five subunits around a central pore (two α plus β, δ, and γ or ε). Acetylcholine binds at two sites on the α subunits; both must be filled for the channel to open. The open pore passes cations, mainly Na^+ inward, which depolarises the muscle cell.
- **GABA-A and glycine receptors**: Cl^- channels that inhibit nerve cells.
- **Glutamate receptors** (AMPA, NMDA): excitatory cation channels in the brain; NMDA receptors also let in Ca^{2+}.

An **agonist** binds the receptor and opens it like the natural ligand: nicotine opens nAChR. An **antagonist** binds without opening it and blocks the natural ligand: d-tubocurarine (the arrow poison curare) and α-bungarotoxin, from the venom of the many-banded krait (*Bungarus multicinctus*, a relative of the Indian common krait), block nAChR and paralyse muscle. Benzodiazepines are different again: they do not open GABA-A receptors themselves but make

GABA open them more easily. Receptor pharmacology in general is in Unit 5, Agonists and antagonists.

Voltage-gated channels

Voltage-gated Na^+, K^+ and Ca^{2+} channels share one plan. A voltage-gated K^+ channel is four separate subunits; the Na^+ and Ca^{2+} channels are a single long protein with four similar domains (I to IV). Each subunit or domain has six transmembrane helices, S1 to S6:

1. **Sensing.** Helix S4 carries a positive amino acid (arginine or lysine) at about every third position. At rest the negative inside holds S4 in.
2. **Opening.** Depolarisation (the inside becoming less negative) pushes the S4 helices outward, and the movement pulls open the gate formed by S5 and S6. The loop between S5 and S6 lines the selectivity filter.
3. **Inactivation.** In Na^+ channels a cytosolic loop between domains III and IV swings in and blocks the pore within about a millisecond, even though the membrane is still depolarised. Only after repolarisation does the channel return to closed and become ready again.

In a nerve impulse, Na^+ channels open first and Na^+ rushes in; then they inactivate, and slower K^+ channels open and let K^+ out, bringing the voltage back. Inactivation is why an impulse travels one way and why there is a refractory period (details in Unit 6, Nerve cells). At nerve terminals, voltage-gated Ca^{2+} channels let in the Ca^{2+} that triggers exocytosis of neurotransmitter (chapter 2.7). Tetrodotoxin from pufferfish blocks Na^+ channels; local anaesthetics such as lidocaine block them from the inside, which is how a dentist numbs a tooth.

WORKED EXAMPLE

Question. A nerve cell has K^+ 140 mM inside and 5 mM outside, Na^+ 12 mM inside and 145 mM outside. Calculate E_K and E_Na at 37 °C, and explain why the resting potential is about −70 mV. 6 marks

1. At 37 °C, RT/F × ln 10 = 61.5 mV. For K^+ and Na^+, z = +1.
2. E_K = 61.5 × $\log_{10}(5/140)$ = 61.5 × (−1.447) = **−89 mV**.
3. E_Na = 61.5 × $\log_{10}(145/12)$ = 61.5 × (1.082) = **+67 mV**.
4. At rest, many K^+ leak channels are open and very few Na^+ channels, so the membrane is much more permeable to K^+. The resting potential lies near E_K (−89 mV), pulled a little towards E_Na by the small Na^+ leak, giving about −70 mV.
5. When voltage-gated Na^+ channels open, Na^+ permeability dominates and the potential swings towards +67 mV: the rising phase of the action potential.

EXAM TRAPS

- Channels are passive. "Ion channel pumps ions" is wrong; pumps are carriers that use ATP (chapter 2.4).
- Inactivated is not the same as closed. An inactivated Na^+ channel cannot be opened by depolarisation; a closed one can.
- Nernst uses C_out/C_in for the inside potential. Flipping the ratio gives the wrong sign.
- For Cl^-, z = −1, which flips the sign of the equation.
- The favourite question: "Explain the structure and working of ligand-gated and voltage-gated channels with examples." Give nAChR for one and the Na^+ channel with its S4 sensor for the other.

CHECK YOURSELF

– REMEMBER **Which helix is the voltage sensor in voltage-gated channels?** 1 mark

S4, which carries positively charged arginine or lysine residues.

– REMEMBER **Name one agonist and one antagonist of the nicotinic acetylcholine receptor.** 2 marks

Agonist: nicotine (or acetylcholine itself). Antagonist: d-tubocurarine (curare) or α-bungarotoxin.

– UNDERSTAND **How does a K^+ channel let K^+ through but keep out the smaller Na^+?** 3 marks

The filter's carbonyl oxygens are spaced to replace exactly the water shell of a K^+ ion, so K^+ loses its water at little cost. Na^+ is too small to touch all those oxygens at once, so shedding its water is costly and it stays out.

– UNDERSTAND **Why does the resting potential lie close to E_K?** 2 marks

At rest the membrane is far more permeable to K^+ (open leak channels) than to other ions, so the K^+ equilibrium dominates.

– APPLY **Cl^- is 10 mM inside and 110 mM outside. Find E_Cl at 37 °C.** 2 marks

$z = -1$: $E = (61.5 / -1) \times \log_{10}(110/10) = -61.5 \times 1.041 =$ **−64 mV**.

– APPLY **Outside K^+ rises from 5 mM to 10 mM (inside 140 mM). What happens to E_K?** 2 marks

$E_K = 61.5 \times \log_{10}(10/140) =$ **−70 mV**, about 19 mV less negative: the cell depolarises. This is why high blood K^+ is dangerous for the heart.

– ANALYZE **α-Bungarotoxin from krait venom causes paralysis. Which step of nerve–muscle signalling does it block, and why are breathing muscles at risk?** 4 marks

α-Bungarotoxin binds the nicotinic ACh receptors of the muscle end-plate and stops ACh opening them. No Na^+ enters, the muscle does not depolarise and cannot contract. The diaphragm is a skeletal muscle with the same receptors, so breathing fails.

BEYOND THE SYLLABUS

This is not for the exam. Mechanically gated channels, the Piezo proteins, sense touch and pressure; their discovery was part of the 2021 Nobel Prize in Physiology or Medicine. Light-gated channels such as channelrhodopsin let neuroscientists switch chosen neurons on with a flash (optogenetics). Many diseases are channelopathies, from cystic fibrosis (the CFTR Cl^- channel) to some epilepsies and heart rhythm disorders, and automated patch clamp is now a standard drug-safety screen. Search next for "optogenetics channelrhodopsin" and "hERG channel drug safety".

2.7 Endocytosis and exocytosis

Proteins, particles and large volumes of fluid are too big for any carrier or channel, so cells move them inside membrane bubbles called vesicles. In **endocytosis** a patch of plasma membrane folds inward and pinches off, carrying material into the cell. In **exocytosis** a vesicle from inside fuses with the plasma membrane and releases its contents outside. A macrophage swallowing bacteria, a liver cell taking up cholesterol, a pancreatic β cell releasing insulin and a nerve releasing a transmitter all use these two processes.

You need: membrane fluidity from chapter 2.1. The organelles that sort vesicles (Golgi, lysosomes) are in Unit 3.

Kinds of endocytosis

- **Phagocytosis** ("cell eating"): specialised cells such as macrophages and neutrophils extend arm-like pseudopods, driven by actin, around a large particle (a bacterium, a dead cell) and enclose it in a **phagosome**, which then fuses with a lysosome for digestion. It is triggered by receptors that recognise the particle. In protozoa such as *Amoeba* it is a way of feeding.
- **Pinocytosis** ("cell drinking"): nearly all cells continually take in small vesicles of extracellular fluid and whatever is dissolved in it, with no selection. Macropinocytosis takes larger gulps.
- **Receptor-mediated endocytosis**: specific molecules bind receptors that gather in **clathrin-coated pits**, so the cell takes up chosen cargo in concentrated form, even when it is scarce outside.
- **Caveolae**: small flask-shaped pits coated with the protein caveolin, rich in cholesterol and sphingolipids, a clathrin-independent route.

Receptor-mediated endocytosis step by step

1. **Binding.** The ligand, for example a low-density lipoprotein (LDL) particle carrying cholesterol, binds its receptor on the cell surface.
2. **Coat assembly.** Adaptor proteins (AP2) bind the receptor's cytosolic tail and recruit **clathrin**. Clathrin is a three-legged unit (a triskelion of three heavy and three light chains) that assembles into a basket of pentagons and hexagons, bending the membrane into a coated pit.
3. **Pinching off.** The GTPase **dynamin** forms a ring around the neck of the pit and, by hydrolysing GTP, pinches the vesicle free.
4. **Uncoating.** Within seconds the clathrin coat is removed (an ATP-using Hsc70 chaperone helps) and recycled.

5. **Sorting in the early endosome.** The vesicle fuses with an early endosome, kept acidic (about pH 6) by V-type H^+ pumps (chapter 2.4). The acid makes LDL fall off its receptor.
6. **Recycling and digestion.** Empty receptors bud off in vesicles and return to the plasma membrane for reuse. The LDL goes on through late endosomes to lysosomes (about pH 5), where it is broken down and cholesterol is released to the cytosol.

Michael Brown and Joseph Goldstein worked out this pathway from patients with **familial hypercholesterolaemia**, whose LDL receptors are missing or cannot gather in coated pits; their blood cholesterol is very high and they develop heart disease early (Nobel Prize in Physiology or Medicine, 1985). Iron enters cells the same way, bound to transferrin. Many viruses, including influenza, exploit the route: they are taken in by endocytosis and use the acid of the endosome to trigger fusion with its membrane.

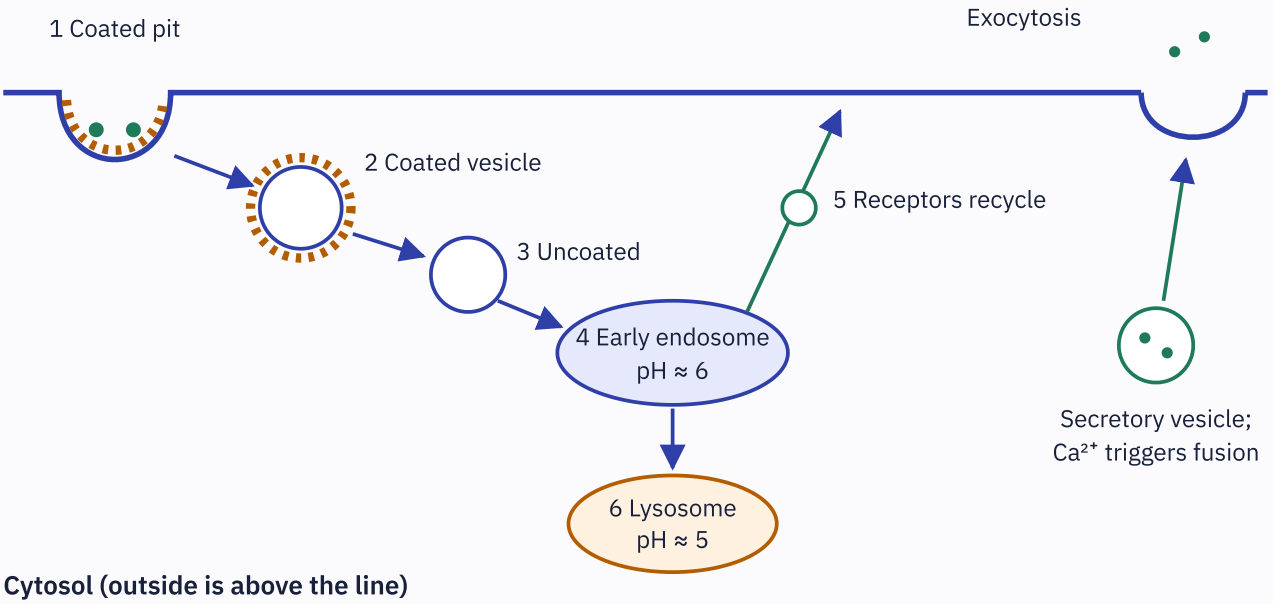


Figure 2.7.1 Left to centre: receptor-mediated endocytosis. Receptors with bound cargo (green dots) gather in a clathrin-coated pit (orange dashes), which pinches off, loses its coat and fuses with the early endosome. The acid releases the cargo; receptors return to the surface (5) while cargo goes on to the lysosome (6). Right: regulated exocytosis, where a secretory vesicle fuses with the plasma membrane and releases its contents outside.

Exocytosis

Proteins made for export are packed into vesicles in the Golgi apparatus (Unit 3). Two routes lead to the surface:

- **Constitutive exocytosis** runs all the time in every cell. It delivers new lipids and membrane proteins to the plasma membrane and secretes material such as extracellular matrix proteins and, in plasma cells, antibodies.

- **Regulated exocytosis** happens only in secretory cells and only on a signal. Vesicles full of product wait near the membrane until a signal, usually a rise in cytosolic Ca^{2+}, triggers fusion. Examples: insulin from pancreatic β cells when blood glucose rises, digestive enzymes from pancreatic acinar cells, and neurotransmitters at a synapse after voltage-gated Ca^{2+} channels open (chapter 2.6).

Fusion is done by **SNARE** proteins. A v-SNARE on the vesicle (synaptobrevin, also called VAMP) twists together with t-SNAREs on the target membrane (syntaxin and SNAP-25) into a tight four-helix bundle, pulling the two bilayers so close that they merge. Rab GTPases help the vesicle find the right target first. At synapses, the Ca^{2+} sensor **synaptotagmin** releases the final step. After fusion the ATPase NSF pulls the SNARE bundle apart for reuse. Botulinum and tetanus toxins are enzymes that cut SNAREs, which is why they paralyse. James Rothman, Randy Schekman and Thomas Südhof shared the 2013 Nobel Prize in Physiology or Medicine for this vesicle traffic machinery.

Endocytosis removes membrane from the surface, and exocytosis adds it back. The two must balance, or the cell would shrink or grow. In **transcytosis** a vesicle taken in on one face of an epithelial cell is carried across and released on the other face; this is how antibodies from a mother's milk cross the gut wall of a newborn rodent.

WORKED EXAMPLE

Question. A cultured fibroblast has a plasma membrane area of 1500 μm². It forms 2500 coated vesicles per minute, each a sphere 100 nm in diameter. (a) What area of membrane is taken in per minute? (b) What percentage of the surface is this, and how long would it take to internalise an area equal to the whole surface? (c) What does this imply? `6 marks`

1. Surface area of one vesicle = $4\pi r^2 = \pi d^2$, with d = 100 nm = 0.1 μm. Area = $3.142 \times (0.1)^2 = 0.0314$ μm².
2. (a) Per minute: $2500 \times 0.0314 =$ **78.5 μm²**.
3. (b) Fraction = 78.5 / 1500 = 0.052, about **5.2% per minute**. Time for the whole surface = 1500 / 78.5 ≈ **19 minutes**.
4. (c) The cell does not shrink, so membrane must return to the surface at the same rate, by receptor recycling and exocytosis. Membrane flows in a continuous loop.

EXAM TRAPS

- Pinocytosis is non-selective; receptor-mediated endocytosis is selective. Do not call clathrin-coated uptake "pinocytosis" without the qualifier.
- Dynamin uses GTP, not ATP, to pinch off the vesicle.
- The receptor is not digested; it recycles. Only the ligand goes to the lysosome (for LDL).
- Constitutive exocytosis needs no signal; regulated exocytosis needs one, usually Ca^{2+}.
- The favourite question: "Explain receptor-mediated endocytosis with the LDL example and a neat diagram."

CHECK YOURSELF

– REMEMBER **Name the coat protein of coated pits and the protein that pinches the vesicle off.** 2 marks

Clathrin; dynamin (a GTPase).

– REMEMBER **Name the v-SNARE and the two t-SNAREs of a synapse.** 2 marks

v-SNARE: synaptobrevin (VAMP). t-SNAREs: syntaxin and SNAP-25.

– UNDERSTAND **Distinguish phagocytosis from pinocytosis.** 3 marks

Phagocytosis takes up large particles, is done by specialised cells, is triggered by receptors and uses actin-driven pseudopods to form a phagosome. Pinocytosis takes up fluid and solutes in small vesicles, continuously and in almost all cells, without selection.

– UNDERSTAND **Why does the early endosome need to be acidic?** 2 marks

The lower pH weakens ligand–receptor binding, so cargo such as LDL falls off and the empty receptor can recycle to the surface.

– APPLY **A cell forms 1000 vesicles per minute, each 80 nm across. How much membrane area is that per minute?** 2 marks

Each: $\pi \times (0.08\ \mu m)^2 = 0.0201\ \mu m^2$. Total = **20.1 µm² per minute**.

– APPLY **Predict what happens to insulin release if Ca^{2+} is removed from the medium around β cells.** 2 marks

Release falls sharply, because regulated exocytosis needs a rise in cytosolic Ca^{2+} from outside to trigger vesicle fusion.

– ANALYZE **A patient's LDL receptors bind LDL normally but cannot gather in coated pits. Explain why the blood cholesterol is high.** 4 marks

Binding alone does not bring LDL in; receptors must enter coated pits to be endocytosed. Without that step, LDL stays on the surface and in the blood, cells sense low cholesterol and make more of their own, and plasma LDL cholesterol rises: a form of familial hypercholesterolaemia.

BEYOND THE SYLLABUS

This is not for the exam. Drug delivery depends on endocytosis: antibody–drug conjugates and lipid nanoparticles enter cells this way, and a major challenge is helping their cargo escape the endosome before it is digested. Cells also release tiny vesicles called exosomes, now studied as disease markers in "liquid biopsy". Biotech production of therapeutic proteins in CHO cells relies on the constitutive secretory pathway. Search next for "endosomal escape lipid nanoparticles" and "exosomes liquid biopsy".

Key terms of this lesson

TERM	MEANING OR FORMULA	CHAPTER
Symport limit	RT ln([S]in/[S]out) = n|ΔG(Na^+ in)|	2.5
Nernst equation	E = (61.5 mV/z) $\log_{10}$(C_out/C_in) at 37 °C	2.6
Receptor-mediated endocytosis	Receptor → clathrin-coated pit → dynamin → endosome (pH ≈ 6) → lysosome; receptor recycles	2.7
SNAREs	v-SNARE (synaptobrevin) + t-SNAREs (syntaxin, SNAP-25) drive fusion	2.7

Glossary (terms used in this lesson)

Antiport Carrier that moves two solutes in opposite directions.

Clathrin Three-legged protein that forms the basket-like coat of coated pits and vesicles.

Cotransport	Coupled transport of two solutes: symport or antiport.
Dynamin	GTPase that pinches a coated vesicle off the membrane.
Electrochemical gradient	Combined driving force on an ion from its concentration difference and the membrane voltage.
Endosome	Acidic compartment that receives endocytosed vesicles and sorts their contents.
Equilibrium potential	Voltage at which an ion's electrical and concentration forces balance; given by the Nernst equation.
Inactivation	Channel state in which the pore is blocked despite an open-favouring stimulus.
Km	Solute concentration at which a carrier works at half its maximum rate.
Ligand	Molecule that binds specifically to a receptor or channel.
Permease	Older name for a carrier protein (transporter).
Phagocytosis	Uptake of large particles by specialised cells into a phagosome.
Pinocytosis	Non-selective uptake of extracellular fluid in small vesicles.
Selectivity filter	Narrowest part of a channel pore, which picks the ion that passes.
SNARE	Proteins on vesicle (v-SNARE) and target (t-SNARE) membranes that zip together to drive fusion.
Symport	Carrier that moves two solutes in the same direction.
Uniport	Carrier that moves a single solute down its gradient.

The full unit book

Every chapter of Unit 2, the unit summary, the exam question bank with model answers and the full glossary: Cell Biology · Unit 2 · Membranes and Membrane Transport (one file, works offline).

Sources and further reading

- B. Alberts, A. Johnson, J. Lewis and others, *Molecular Biology of the Cell*, Garland Science; chapters on membrane structure and membrane transport. The 4th edition (2002) is free to read on NCBI Bookshelf; values quoted in this book from Alberts were checked there.
- H. Lodish, A. Berk and others, *Molecular Cell Biology*, W. H. Freeman; chapters on biomembranes and transmembrane transport (from memory; check the transport-rate figures against your edition).

- G. Karp, *Cell and Molecular Biology: Concepts and Experiments*, Wiley; chapter on the plasma membrane (from memory; check).
- G. M. Cooper, *The Cell: A Molecular Approach*, Sinauer; chapter on the plasma membrane (from memory; check).
- S. J. Singer and G. L. Nicolson, "The fluid mosaic model of the structure of cell membranes", *Science*, 1972 (from memory; check).
- Alberts et al., *Molecular Biology of the Cell*, 4th ed., "The Lipid Bilayer", NCBI Bookshelf: https://www.ncbi.nlm.nih.gov/books/NBK26871/ (opened; lipid diffusion, flip-flop, cholesterol and asymmetry figures).
- Alberts et al., *Molecular Biology of the Cell*, 4th ed., "Carrier Proteins and Active Membrane Transport", NCBI Bookshelf: https://www.ncbi.nlm.nih.gov/books/NBK26896/ (opened; Na^+/K^+ pump stoichiometry and energy share, P-type and ABC pumps, Ca^{2+} levels).

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