

MEMBRANE TRANSPORT

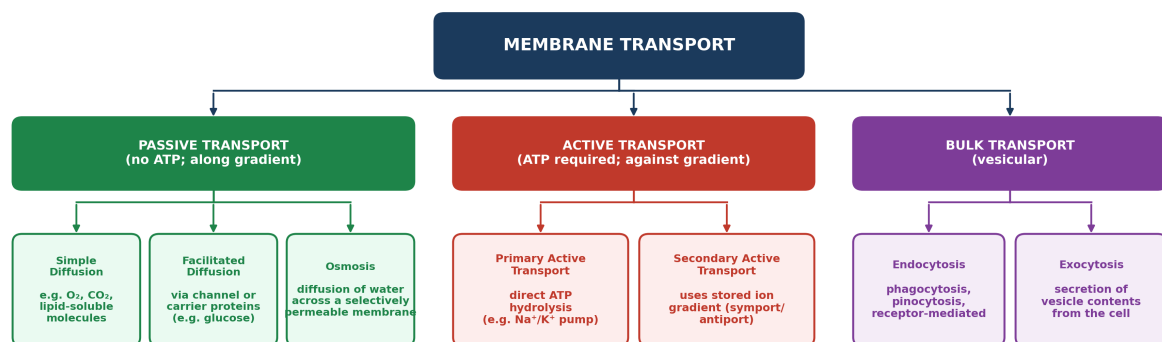
How Cells Move Matter Across Their Boundary – Passive, Active & Bulk Transport

1. Introduction

Every living cell is wrapped in a plasma membrane that is anything but a passive wall – it is a bustling, selectively permeable frontier, constantly deciding what gets in, what stays out, and what leaves. Small, uncharged, lipid-friendly molecules slip across almost unnoticed; larger, polar, or charged solutes need dedicated machinery; and some cargo is so bulky that the membrane must physically fold around it. This traffic across the membrane – collectively called **membrane transport** – keeps the cell's internal environment stable, fuels its metabolism, and allows it to communicate with the world outside.

Membrane transport mechanisms are grouped by whether they require metabolic energy and by the size of the cargo they carry.

Classification of Membrane Transport Mechanisms



Key distinction: passive transport moves substances **DOWN** their concentration gradient and needs no metabolic energy; active transport moves substances **AGAINST** their gradient and requires ATP; bulk transport moves large cargo or bulk fluid via membrane-bound vesicles.

Fig. 1: Membrane transport mechanisms are classified as passive, active, or bulk (vesicular) transport.

Quick facts Passive transport: no ATP, moves **WITH** the gradient | Active transport: uses ATP, moves **AGAINST** the gradient | Bulk transport: moves large cargo via vesicles

2. PASSIVE TRANSPORT – Free Rides Down the Gradient

Passive transport is the cell's "no-cost" option: solutes drift from a region of high concentration to one of low concentration, powered entirely by the random thermal motion of molecules. No ATP is spent – the concentration gradient itself is the energy source.

- **Simple diffusion** – small, nonpolar, lipid-soluble molecules (O_2 , CO_2 , steroid hormones) dissolve directly through the phospholipid bilayer, no protein required.
- **Facilitated diffusion** – polar or charged solutes (glucose, ions) cross via specific membrane proteins: **channel proteins** (open pores, extremely fast) or **carrier proteins** (bind the solute and flip conformation, slower but selective).
- **Osmosis** – a special case of diffusion in which **water** moves across a selectively permeable membrane, from the side with more free water (hypotonic) to the side with more dissolved solute (hypertonic), often

assisted by water-channel proteins called **aquaporins**.

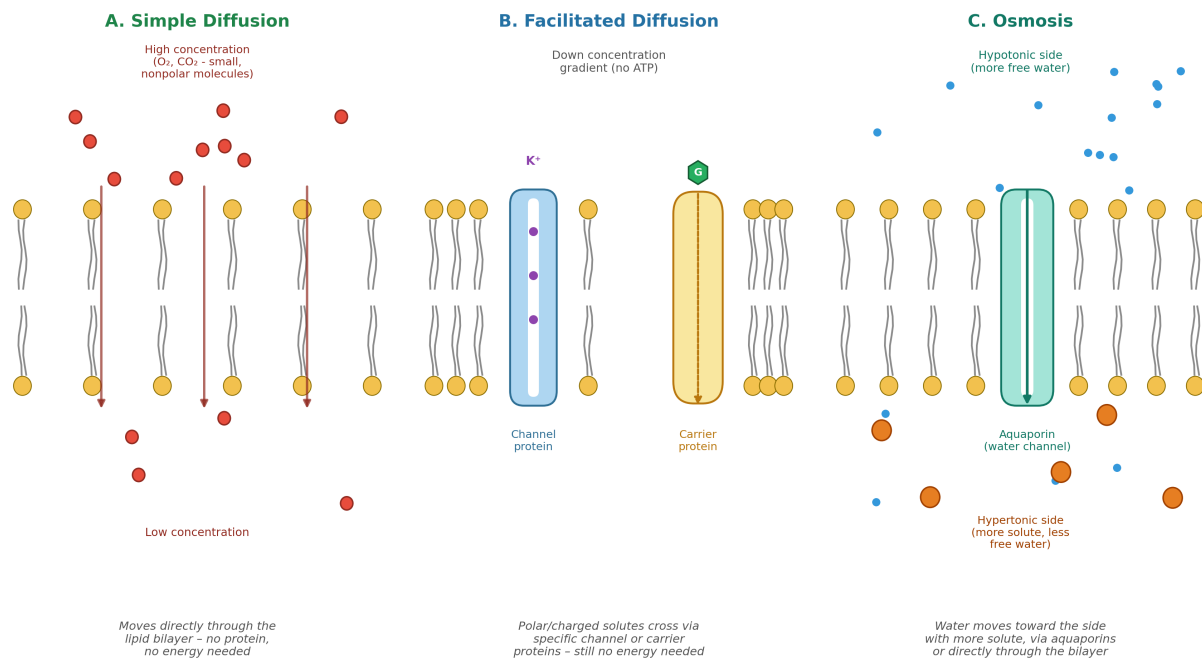


Fig. 2: The three passive transport routes – simple diffusion, facilitated diffusion through channel/carrier proteins, and osmosis through aquaporins.

Tonicity and the animal cell

Solution	Solute concentration vs. cell	Net water movement	Effect on animal cell
Hypotonic	Lower outside	Into the cell	Swells; may burst (lysis)
Isotonic	Equal	None (net)	No change in volume
Hypertonic	Higher outside	Out of the cell	Shrinks (crenation)

3. ACTIVE TRANSPORT – Swimming Against the Current

Sometimes a cell must move a solute *uphill* – from low to high concentration – and that always costs energy. Active transport harnesses **ATP**, either directly or indirectly, to drive solutes against their electrochemical gradient.

(a) Primary active transport

The transporter itself hydrolyses ATP to power solute movement. The classic example is the **Na^+/K^+ -ATPase pump**, found in virtually every animal cell membrane: for every ATP hydrolysed, it exports 3 Na^+ ions and imports 2 K^+ ions, both against their concentration gradients. This single pump maintains the steep ionic gradients that underlie nerve impulses, muscle contraction, and cell volume regulation. Other primary pumps include the **Ca^{2+} -ATPase** (clears calcium from the cytosol) and the gastric **H^+/K^+ -ATPase** (acidifies the stomach).

(b) Secondary active transport

Here, the transporter itself uses no ATP directly – instead it exploits the electrochemical gradient that a primary pump has already built up (usually the Na^+ gradient), letting Na^+ “fall downhill” to drag a second solute along.

- **Symport (co-transport)** – both solutes move in the *same* direction, e.g. the **SGLT** transporter that co-transporters Na^+ and glucose into intestinal and kidney tubule cells.
- **Antiport (counter-transport)** – the two solutes move in *opposite* directions, e.g. the **$\text{Na}^+/\text{Ca}^{2+}$ exchanger (NCX)** that uses the inward Na^+ gradient to pump Ca^{2+} out of the cell.

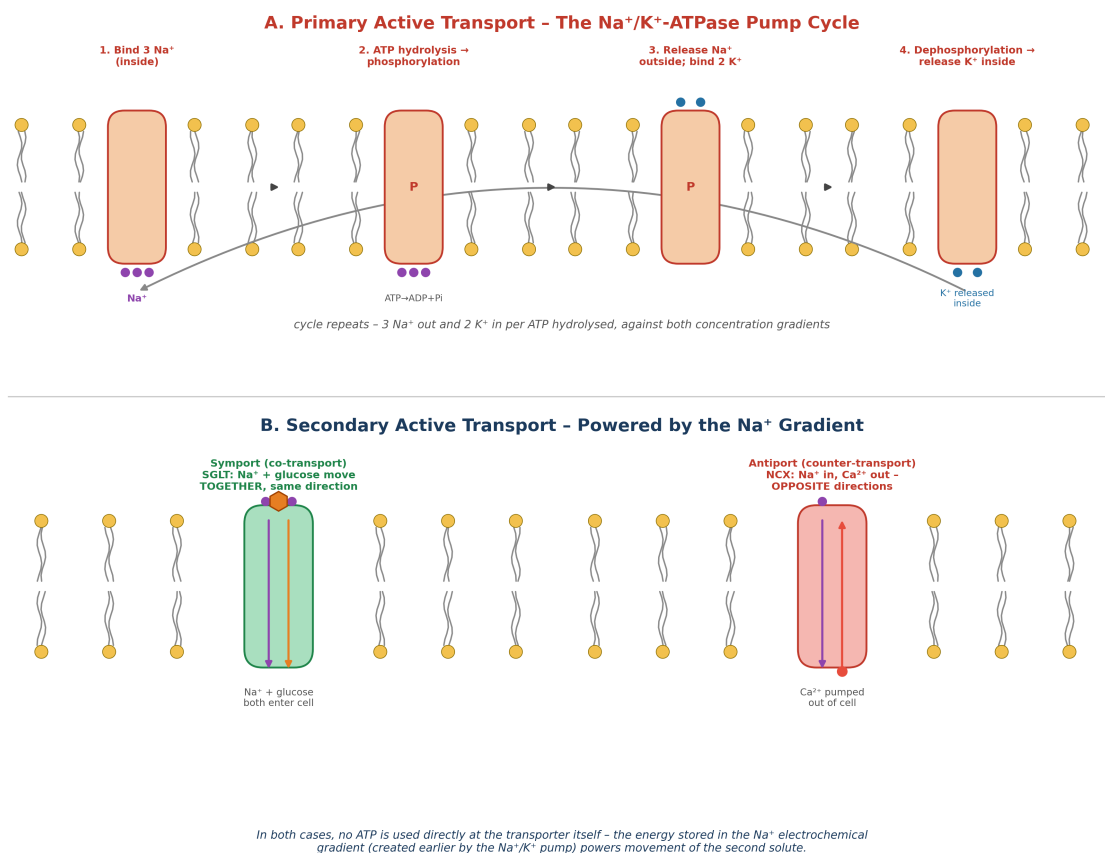


Fig. 3: (A) The Na^+/K^+ -ATPase pump cycle (primary active transport). (B) Symport and antiport driven by the stored Na^+ gradient (secondary active transport).

4. Bulk (Vesicular) Transport

Some cargo – bacteria, large protein complexes, litres of extracellular fluid – is far too big for any channel or carrier. The cell instead wraps the material in a piece of membrane, forming a vesicle that buds off or fuses with the plasma membrane. This, too, consumes ATP (to remodel the cytoskeleton and membrane), so bulk transport is considered a form of active transport in the broad sense.

- **Phagocytosis** (“cell eating”) – large particles such as bacteria are engulfed by pseudopodia and enclosed in a phagosome; central to the innate immune defences of macrophages and neutrophils.
- **Pinocytosis** (“cell drinking”) – non-specific, continuous uptake of small droplets of extracellular fluid and dissolved solutes.
- **Receptor-mediated endocytosis** – a highly specific route in which a ligand (e.g. LDL cholesterol, iron-transferrin) binds a surface receptor, triggering internalisation via a clathrin-coated pit.
- **Exocytosis** – the reverse process: a secretory vesicle travels to, and fuses with, the plasma membrane, releasing its contents outside the cell.

Bulk (Vesicular) Transport

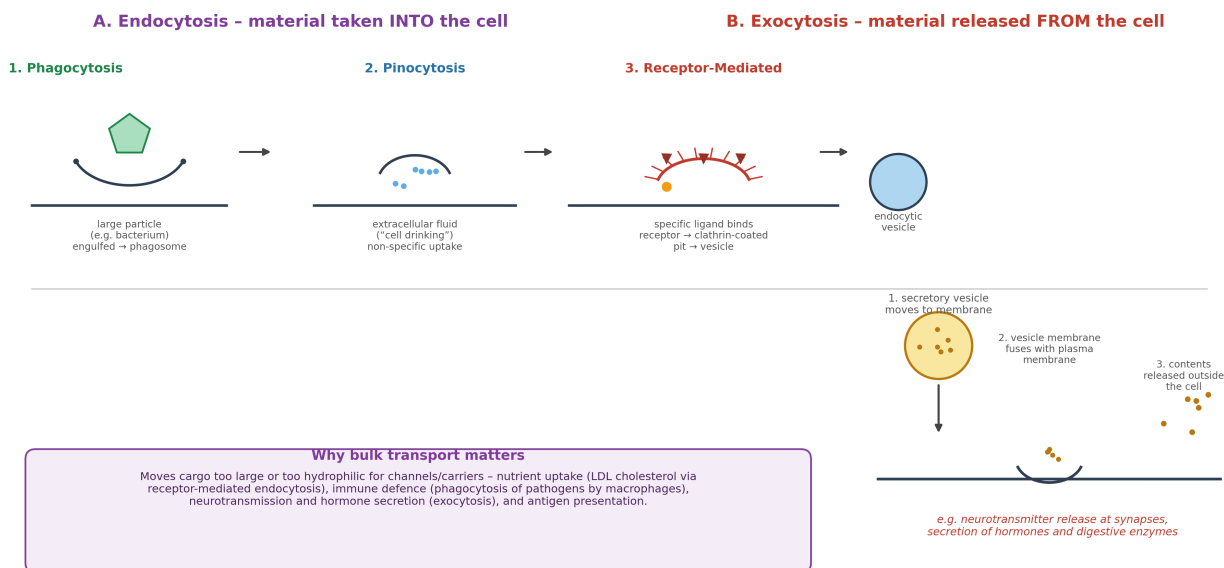


Fig. 4: The three main routes of endocytosis and the three-step sequence of exocytosis.

5. Comparison at a Glance

Feature	Passive transport	Active transport	Bulk transport
Energy source	Concentration gradient only	ATP (direct or indirect)	ATP (vesicle formation/fusion)
Direction	Down the gradient	Against the gradient	Either, via vesicles
Carrier needed?	Sometimes (facilitated diffusion)	Always (pump/transporter)	Vesicle, not a carrier protein
Typical cargo	Gases, water, small ions/solutes	Ions against gradient (Na^+ , K^+ , Ca^{2+} , H^+)	Bacteria, fluid droplets, secretory granules
Example	O_2 entering a red blood cell	Na^+/K^+ -ATPase pump	Macrophage engulfing bacteria

6. Factors Affecting the Rate of Diffusion

- **Concentration gradient** – a steeper gradient drives faster net diffusion.
- **Temperature** – higher temperature increases molecular kinetic energy and diffusion rate.
- **Surface area** – a larger membrane surface area allows more simultaneous crossings (e.g. the alveolar and intestinal epithelium are both extensively folded to maximise this).
- **Membrane thickness / diffusion distance** – a thinner membrane or shorter diffusion path speeds up transport.
- **Molecular size and polarity** – smaller, less polar molecules cross the lipid bilayer more readily than larger or charged ones.
- **Number of channel/carrier proteins** – for facilitated diffusion, the rate depends on how many transport proteins are available and can saturate at high solute concentrations.

7. Why Membrane Transport Matters

Membrane transport is not a peripheral biochemical detail – it is the quiet engine behind nearly every major physiological process in the body.

- **Nerve impulse conduction** – voltage-gated Na^+ and K^+ channels (facilitated diffusion) generate the action potential, while the Na^+/K^+ pump (primary active transport) resets the resting potential afterwards.
- **Muscle contraction** – the sarcoplasmic reticulum's Ca^{2+} -ATPase pumps calcium back out of the cytosol, allowing muscle relaxation between contractions.
- **Kidney and gut absorption** – Na^+ -glucose and Na^+ -amino acid symporters reclaim filtered nutrients in the kidney tubule and absorb them across the intestinal lining.
- **Cell volume regulation** – osmosis, tightly coupled to active ion pumping, keeps cells from swelling and bursting or shrinking and shrivelling.
- **Immune defence** – phagocytosis lets macrophages and neutrophils engulf and destroy invading bacteria.
- **Chemical signalling** – exocytosis releases neurotransmitters at synapses and hormones such as insulin into the bloodstream.
- **Pharmacology** – many drugs act directly on membrane transporters, making this one of medicine's richest targets for intervention.

8. Clinical Significance

- **Cystic fibrosis** – a mutated CFTR chloride channel disrupts facilitated diffusion of Cl^- across epithelial membranes, producing thick, sticky mucus in the lungs and pancreas.
- **Digoxin (cardiac glycoside)** – inhibits the Na^+/K^+ -ATPase pump in heart muscle, indirectly raising intracellular Ca^{2+} and strengthening contraction; used in heart failure.
- **Cholera** – the cholera toxin locks CFTR channels open, driving massive Cl^- and water loss into the gut and causing severe dehydration.
- **Type 2 diabetes** – impaired insulin-triggered translocation of GLUT4 glucose transporters to the cell surface underlies insulin resistance.
- **Familial hypercholesterolaemia** – defective LDL receptors impair receptor-mediated endocytosis of LDL cholesterol, leading to dangerously high blood cholesterol.

9. Summary

- **Passive transport** (simple diffusion, facilitated diffusion, osmosis) needs no ATP and moves solutes down their gradient.
- **Active transport** (primary, e.g. Na^+/K^+ pump; secondary, e.g. symport/antiport) uses ATP directly or indirectly to move solutes against their gradient.
- **Bulk transport** (endocytosis and exocytosis) moves large cargo across the membrane inside vesicles.
- Together these mechanisms underlie nerve signalling, muscle contraction, nutrient absorption, immune defence, and are frequent targets of disease and drug action.

End of notes – Membrane Transport