

Lipids & Membranes

BCH 100 – Introductory Biochemistry · Dr. Radi

By the end of this unit, you can...

- Describe the roles of fats and the structure and naming of fatty acids (systematic, common, ω), and distinguish saturated, mono-, and polyunsaturated
- Identify the major lipid classes (triglycerides, phospholipids, glycolipids, steroids) and the trans-fat problem
- Explain the fluid mosaic model, what controls membrane fluidity, and the types of membrane protein
- Distinguish passive and active transport, and the roles of channels, pumps, and transporters

Today's route



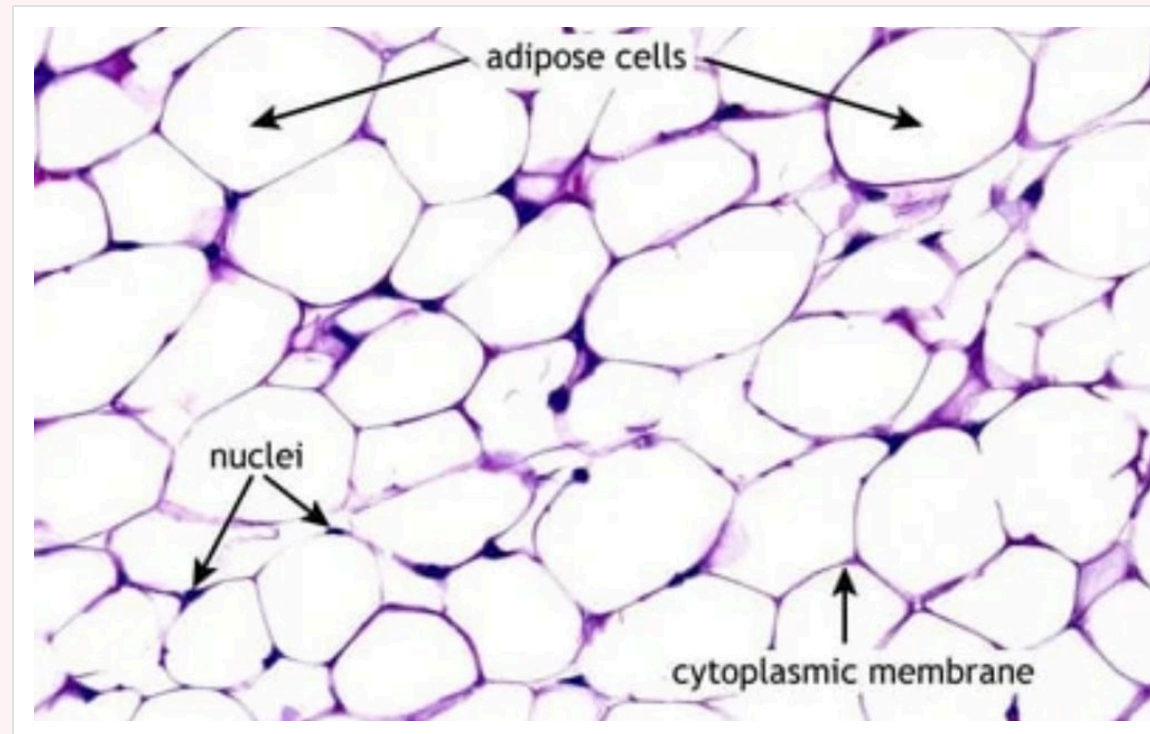
1. **Fatty Acids**
2. **The Lipid Families**
3. **Membranes: Fluid & Mosaic**
4. **Crossing the Membrane**

1 • Fatty Acids

"Meet the fatty acid – a greasy tail with an acid head. One little kink is the difference between butter and olive oil."

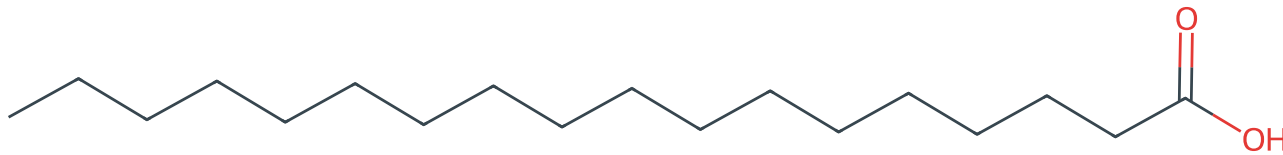
Why fat gets a bad rap it half-deserves

Fat is the body's **best energy store** – it packs **more than twice** the energy of carbs or protein, gram for gram. It also **insulates** you, **cushions** your organs, is the raw material for **steroid hormones**, and builds every **cell membrane** you own. Here's fat doing its main job: **adipocytes**, each a cell nearly filled by a single oil droplet.



A fatty acid: greasy tail, acid head

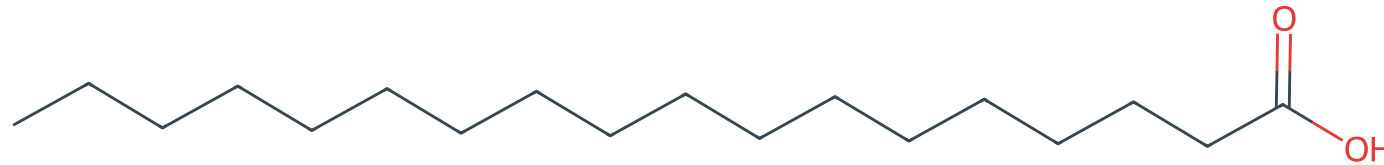
Strip a fat down and you get **fatty acids**: a long **hydrocarbon chain** (greasy, water-hating) capped by a **carboxyl group** (-COOH, the "acid"). That's it – a tail and a head. The chain is where the **energy** is stored; the head is the reactive handle the cell grabs.



One kink changes everything

A **saturated** fatty acid (stearic, 18:0) has no double bonds – it's a **straight** rod. Add **one** double bond and you get a **monounsaturated** one (oleic, 18:1); add **two or more** and it's **polyunsaturated** (linoleic 18:2, α -linolenic 18:3). Each double bond puts a permanent **kink** in the chain. Saturated = straight; unsaturated (mono or poly) = kinked. That bend is about to matter a lot.

Stearic acid (18:0) — saturated · straight



Oleic acid (18:1 cis) — one double bond · KINKED



Butter vs. olive oil

Straight **saturated** chains stack neatly and pack tight – so they're **solid** at room temperature (butter, lard). **Kinked unsaturated** chains can't pack – they stay loose and **liquid** (olive oil, right there in the bottle). Same idea in your cells: more unsaturated fat = a more **fluid** membrane. The kink is why.

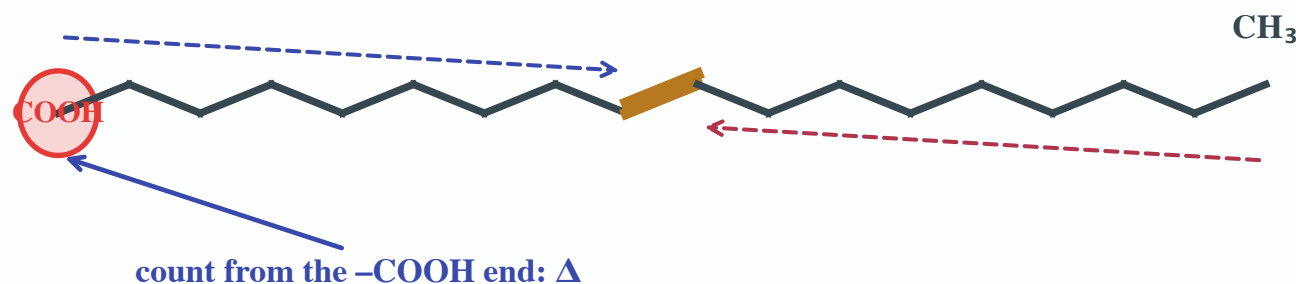


Naming a fatty acid

Fatty acids get named a few ways. **Systematic** and **common** names you memorize (oleic = 18:1). But the useful one is the **omega (ω)** system: count from the **CH₃** end to the first double bond. **ω -3, ω -6, ω -9** – that number names the **family**, and (as you'll see) it's what your body cares about.

Oleic acid = 18:1 — systematic, common, and ω all name the same molecule

Δ 9 — double bond starts at carbon 9 (from the acid)

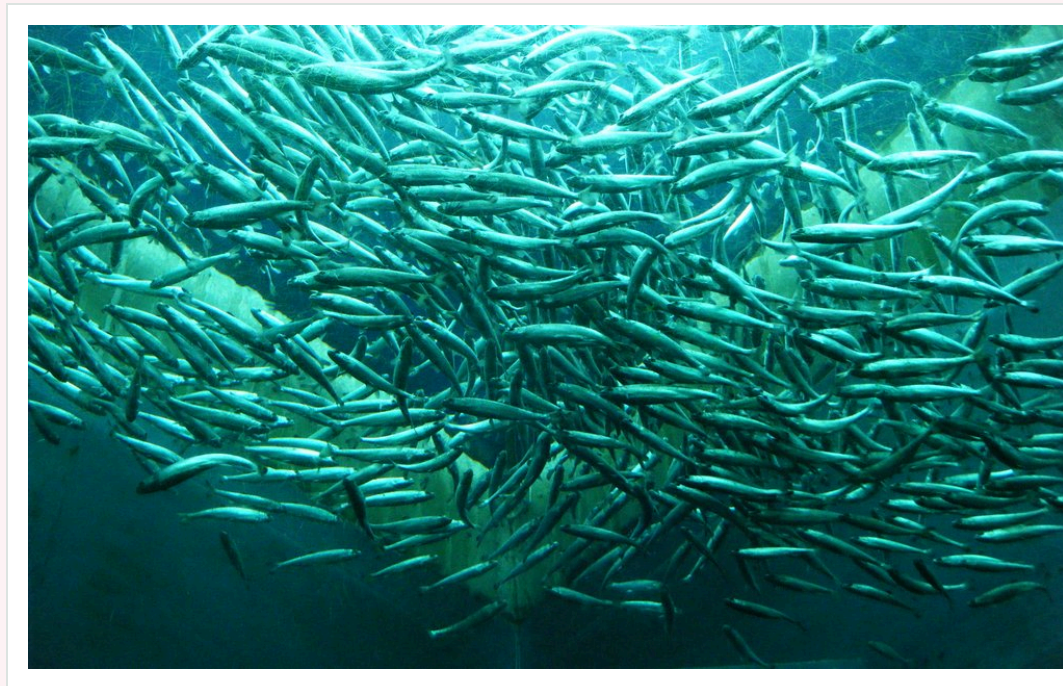


ω -9 — double bond is 9 carbons from the CH₃ (omega) end

18:1 = 18 carbons, 1 double bond · the ω number tells you the family (ω -3, ω -6, ω -9)

The fats you MUST eat

Your body can make most fatty acids – but **not** the **ω -6 (linoleic)** and **ω -3 (α -linolenic)** ones. Those are **essential**: you have to eat them. They build membranes and become signaling molecules. **Oily fish** are loaded with ω -3s – which is why "eat more fish" is on every heart-health list.



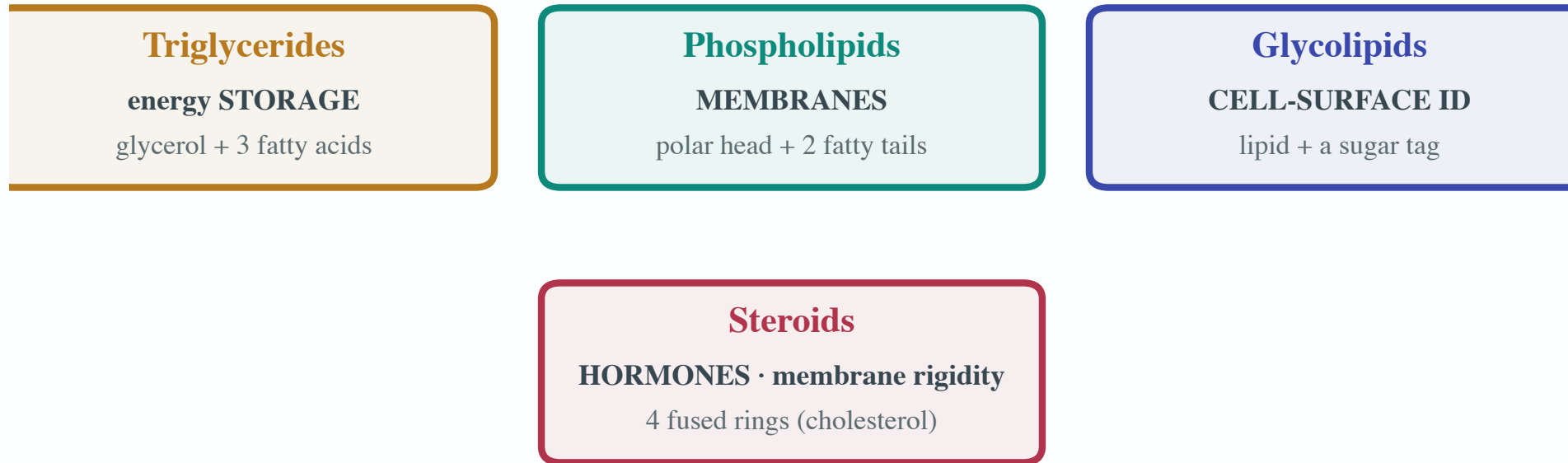
2 • The Lipid Families

"Four families of grease, four totally different jobs – plus the one man-made fat your arteries can't forgive."

One word, four very different molecules

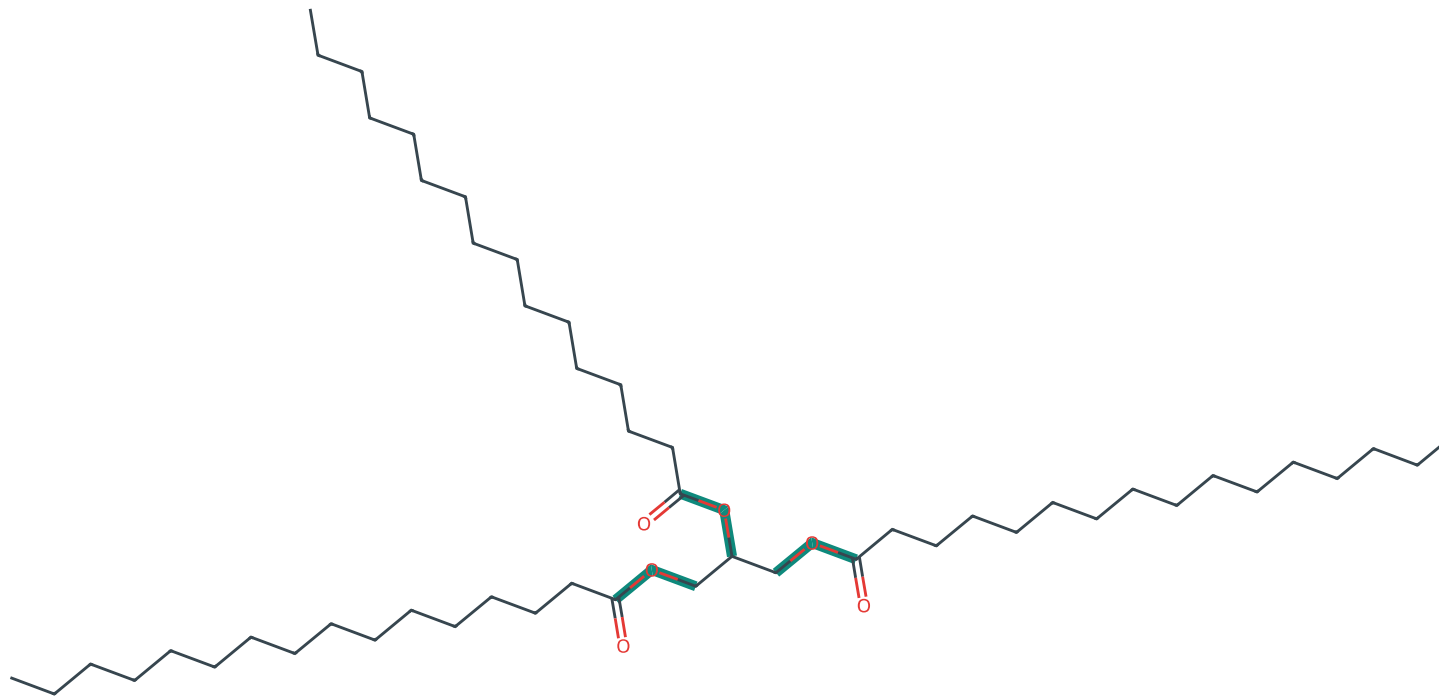
"**Lipid**" isn't a shape – it's an **attitude**: **water-hating**. That's the only thing these four families share. **Triglycerides** store energy, **phospholipids** build membranes, **glycolipids** flag the cell surface, and **steroids** are rigid signaling rings. Same club, wildly different jobs – so let's meet them one at a time.

Four lipid classes — very different jobs, all water-hating



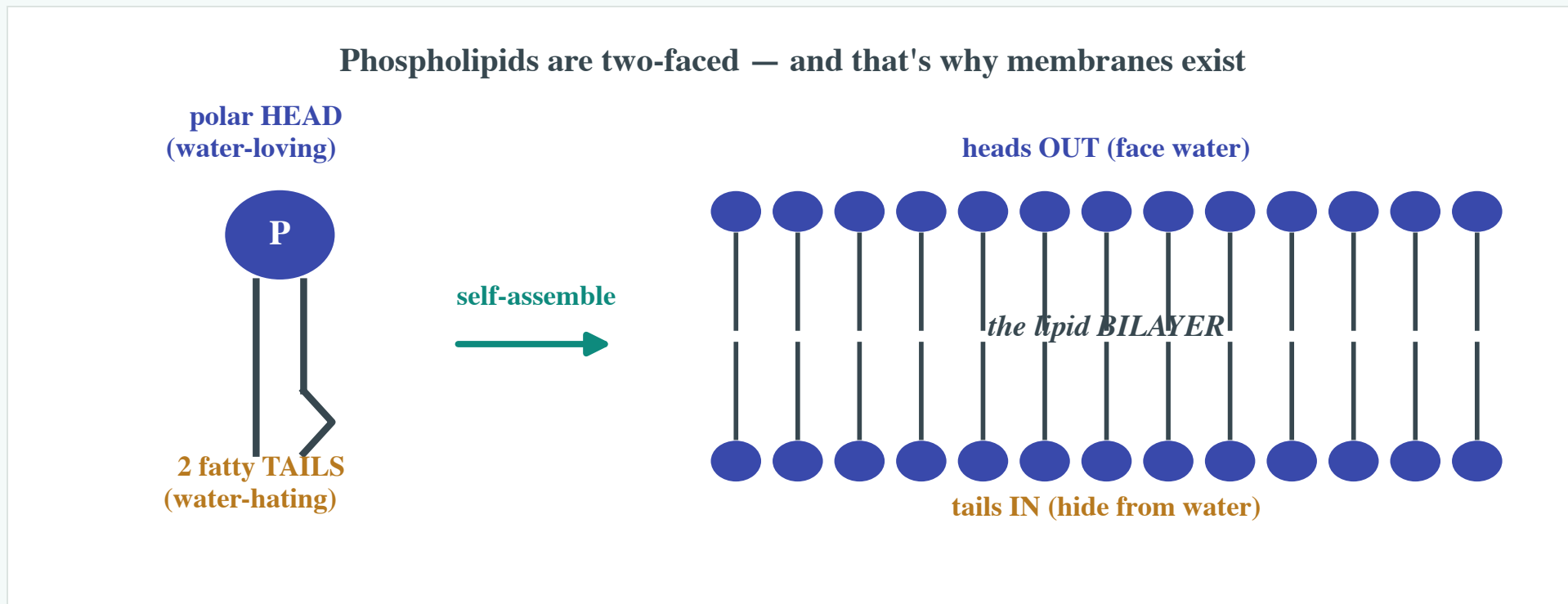
Triglyceride: the fat you store

Take **glycerol** (a 3-carbon backbone) and hang **three fatty acids** off it with **ester bonds** – that's a **triglyceride** (a **triacylglycerol**, TAG). This is the fat in your adipose tissue, in butter, in oil. Three greasy tails, zero polar groups: it's **pure energy storage**, packed away water-free. Snip those ester bonds (a lipase does it) and the fatty acids are freed to burn.



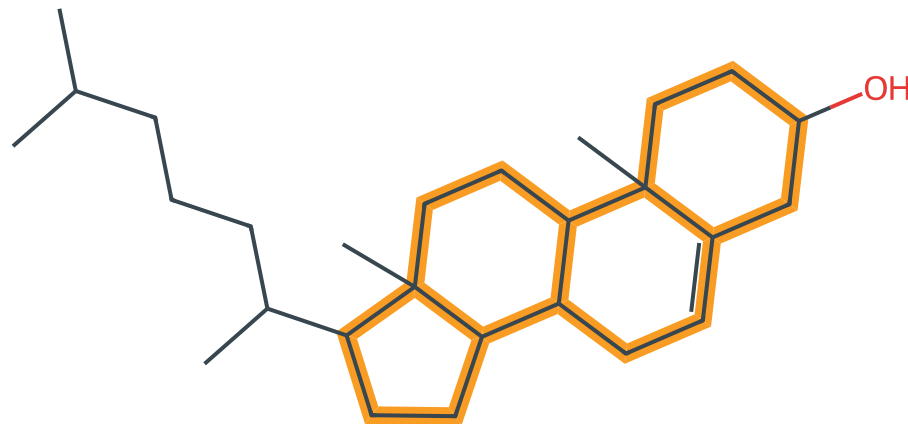
Phospholipid: the two-faced membrane builder

Swap **one** fatty acid for a **phosphate head** and everything changes. Now the molecule is **amphipathic** – a **water-loving head** and **two water-hating tails** on the same body. Drop a crowd of them in water and they **self-assemble**: heads face out, tails hide in. That sheet – the **lipid bilayer** – is the wall around every cell you own. Membranes exist **because** phospholipids can't decide which they are.



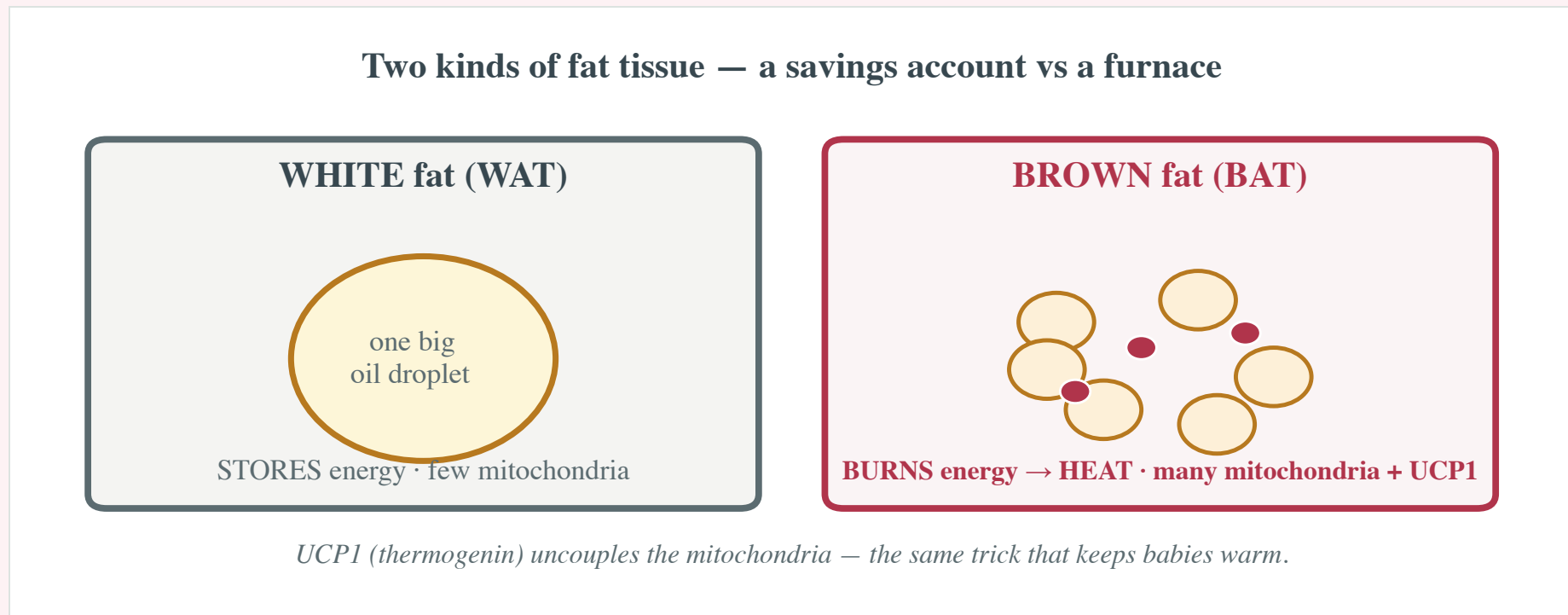
Steroids: four rings, big consequences

No tails here at all. **Steroids** are **four fused carbon rings** – and the parent of the family is **cholesterol**. It wedges into membranes to tune their **stiffness**, and it's the **raw material** your body carves into **steroid hormones** (testosterone, estrogen, cortisol) and **bile salts**. Same four-ring core, redecorated. (Glycolipids, the fourth class, are just a lipid wearing a **sugar** name-tag on the cell surface.)



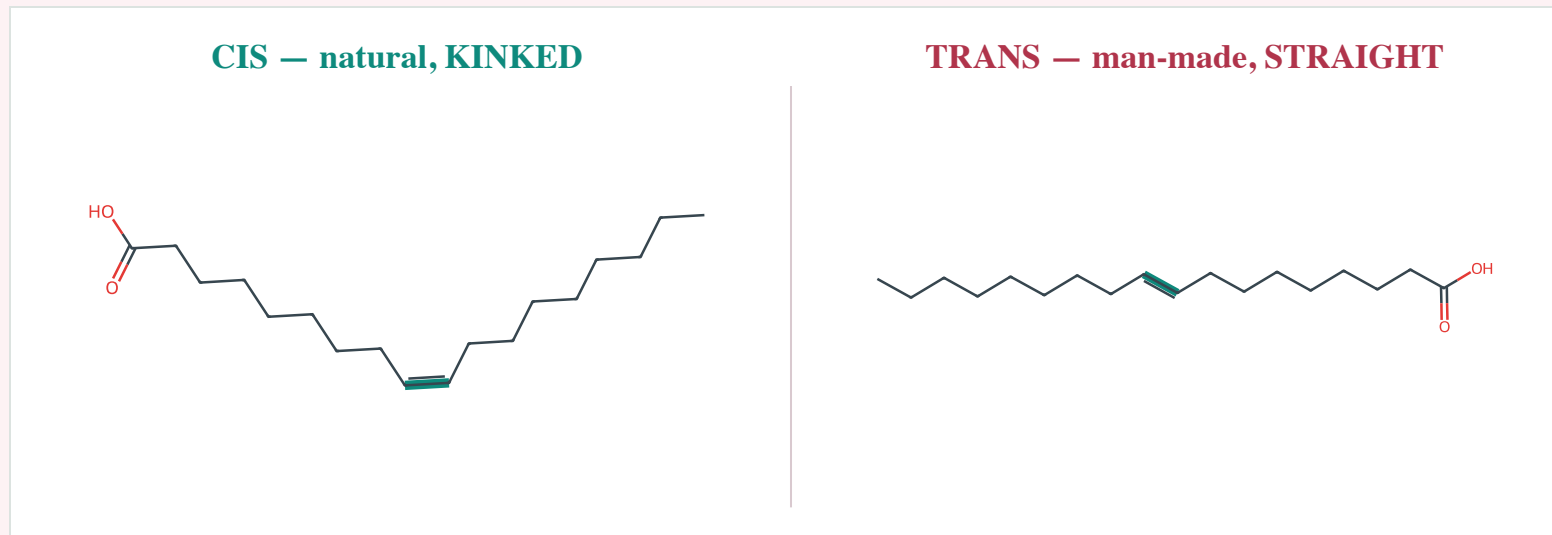
Two kinds of fat tissue: savings vs furnace

Not all adipose is equal. **White fat (WAT)** is the **savings account** – each cell is one big oil droplet, holding energy, insulating, and cushioning. **Brown fat (BAT)** is a **furnace** – many small droplets crammed with **mitochondria** whose protein **UCP1 (thermogenin)** short-circuits the proton gradient to make **heat instead of ATP**. It's how newborns (and hibernators) stay warm, and it's why BAT is a hot target in metabolic research.



THE PROBLEM: trans fats

Nature makes unsaturated fats **cis** – kinked and fluid. Industry **partially hydrogenates** vegetable oil to harden it, and accidentally flips some double bonds to **trans**, which **straightens the chain back out** so it **acts like a saturated fat your body never evolved with**. Trans fats **raise LDL, lower HDL**, and drive **atherosclerosis** – bad enough they're now **banned** from the food supply in much of the world. One flipped bond, one public-health disaster.

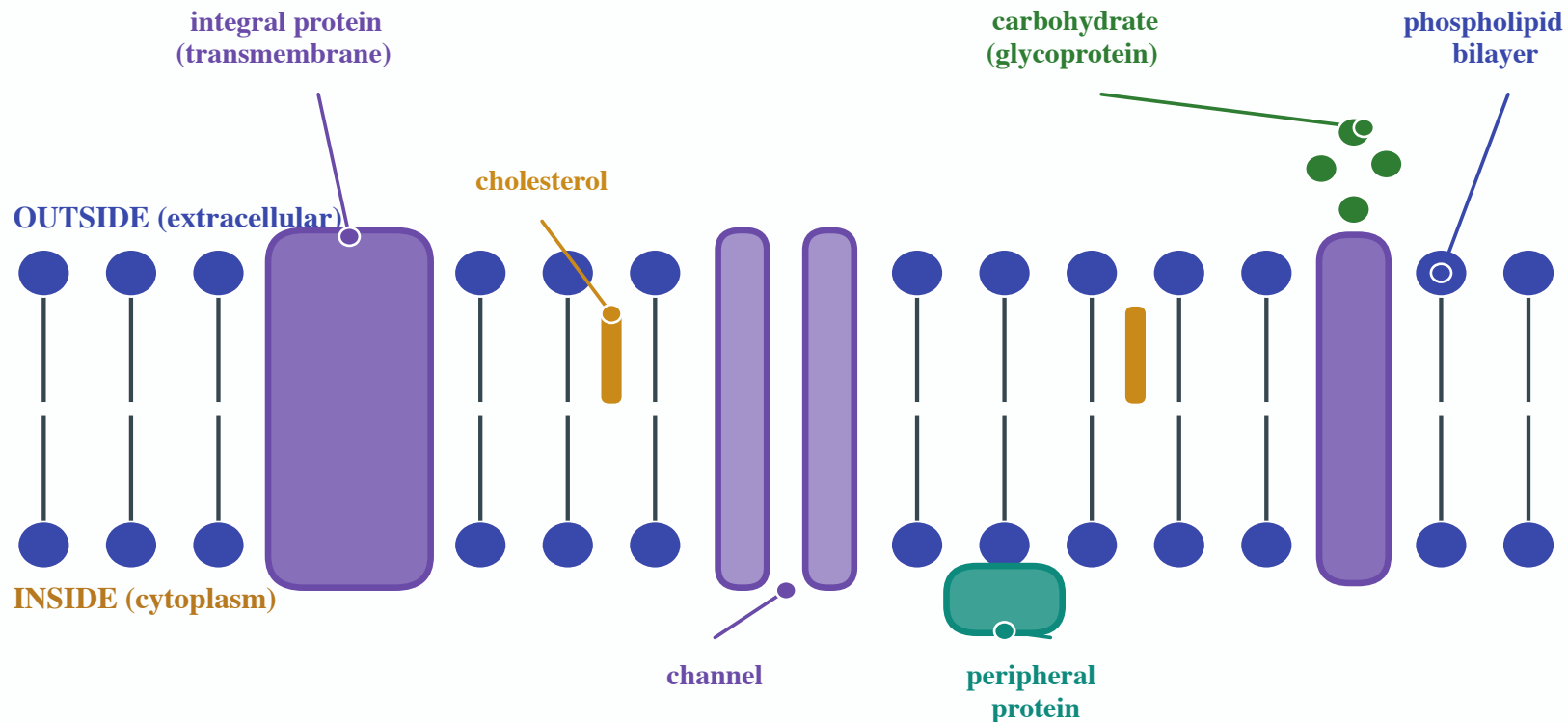


3 · Membranes: Fluid & Mosaic

"The cell membrane isn't a wall – it's a living, drifting oil slick with proteins bobbing in it. Here's what keeps it just fluid enough."

The fluid mosaic model

The membrane around your cells is **two things at once**. It's **fluid** – the phospholipids and proteins aren't nailed down; they **drift sideways** like boats on a sea. And it's a **mosaic** – that lipid sea is studded with a patchwork of **proteins, cholesterol, and sugar tags**. Put those together and you have the **fluid mosaic model**, biology's picture of every membrane you own.

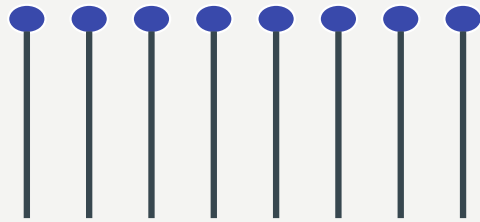


What keeps it just fluid enough

A membrane has to stay **soft enough to work** but not fall apart – and three things tune that. **Saturation:** straight saturated tails pack tight (**less fluid**); kinked unsaturated tails can't pack (**more fluid**). **Chain length:** shorter tails = more fluid. **Temperature:** heat loosens it, cold **gels** it. Same trick as butter vs. oil – now doing a job inside your cells.

What controls how fluid a membrane is

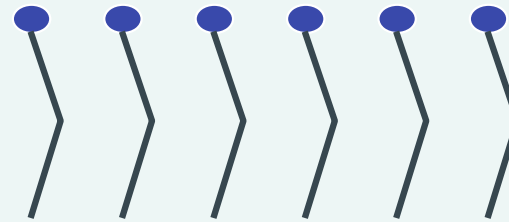
Mostly SATURATED tails



straight → pack TIGHT

LESS fluid (more solid)

More UNSATURATED tails



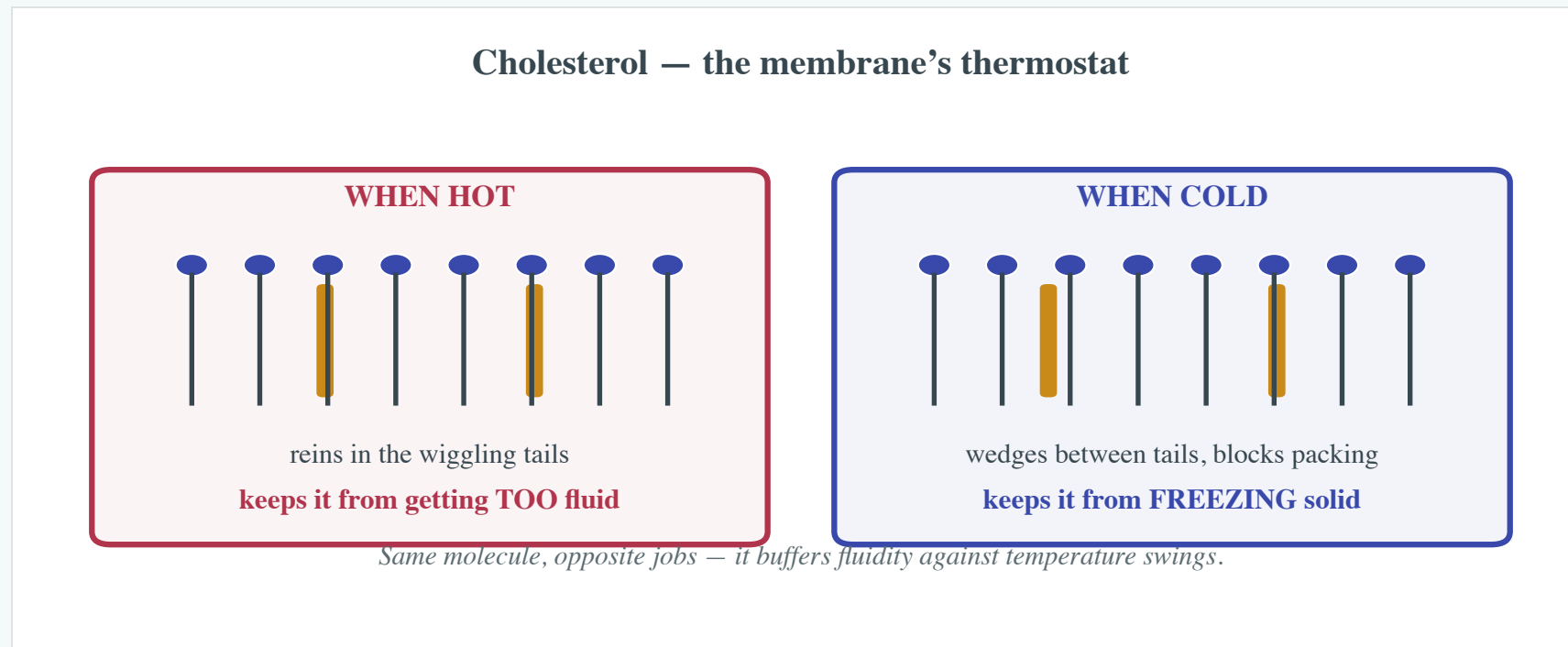
kinks → can't pack (gaps)

MORE fluid

Also: shorter chains → more fluid · ↑ temperature → more fluid, ↓ temperature → gels

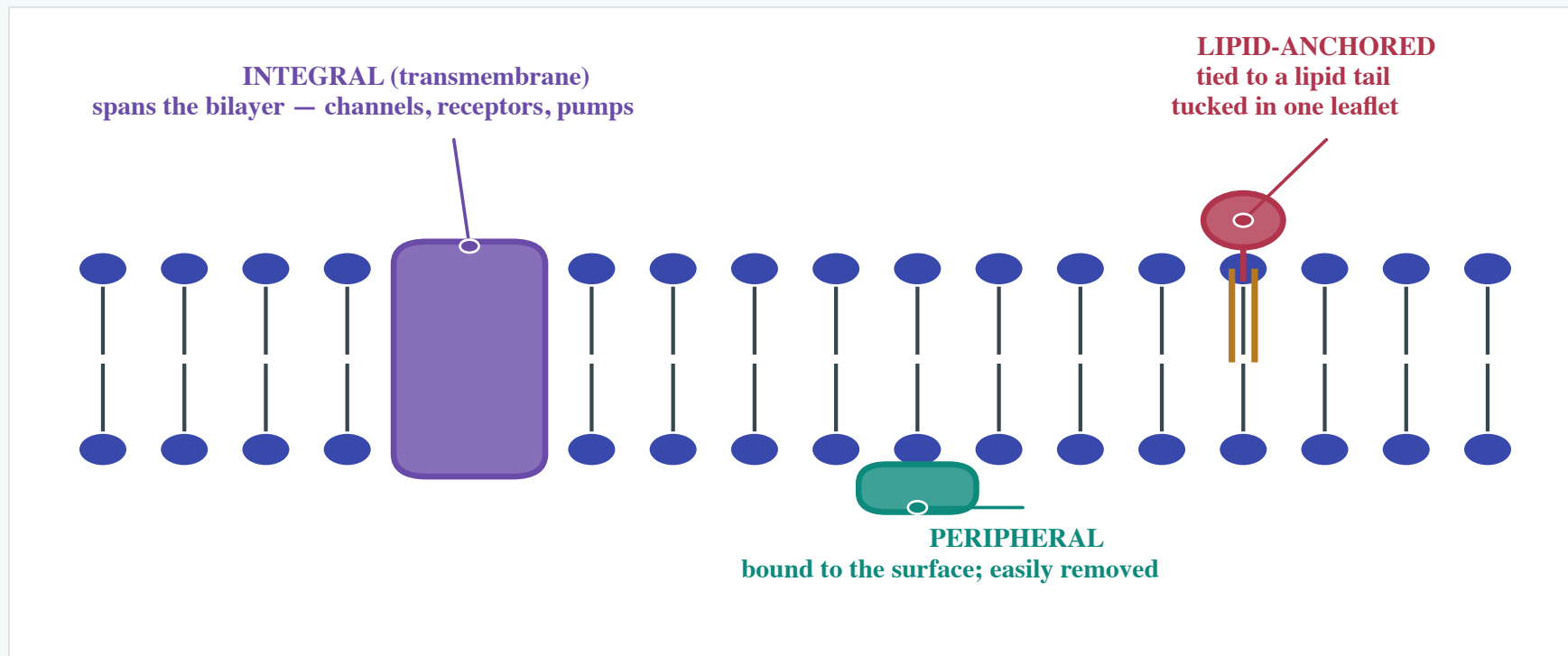
Cholesterol: the membrane's thermostat

Cholesterol wedges in among the phospholipid tails and does something clever – it works **both directions**. When the membrane gets **hot** and too runny, cholesterol **reins in** the flailing tails and firms it up. When it gets **cold** and starts to solidify, cholesterol **wedges between** the tails and blocks them from packing – keeping it from **freezing**. One molecule, a built-in **fluidity buffer**.



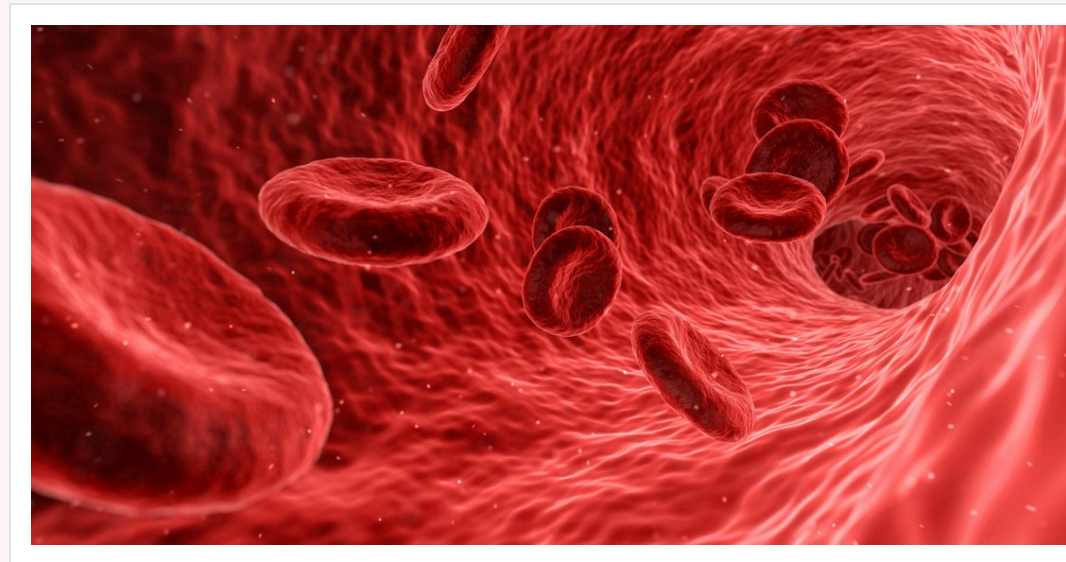
The proteins in the mosaic

The membrane's real machinery is its **proteins**, and they ride in **three ways**. **Integral (transmembrane)** proteins pass all the way through the bilayer – these are your **channels, pumps, and receptors**. **Peripheral** proteins cling loosely to one surface and lift off easily. **Lipid-anchored** proteins float above the membrane, tied down by a **lipid tail** tucked into one leaflet. Different mountings, different jobs.



Why "fluid" isn't optional

Here's fluidity earning its keep. A **red blood cell** is about **8 μm** across – but it has to squeeze single-file through **capillaries half that width**, millions of times, without bursting. Only a **fluid, flexible membrane** can fold, deform, and spring back like that. Make the membrane too stiff (too much cholesterol, too little unsaturation) and cells get **rigid and fragile** – the membrane's softness is a feature, not a flaw.

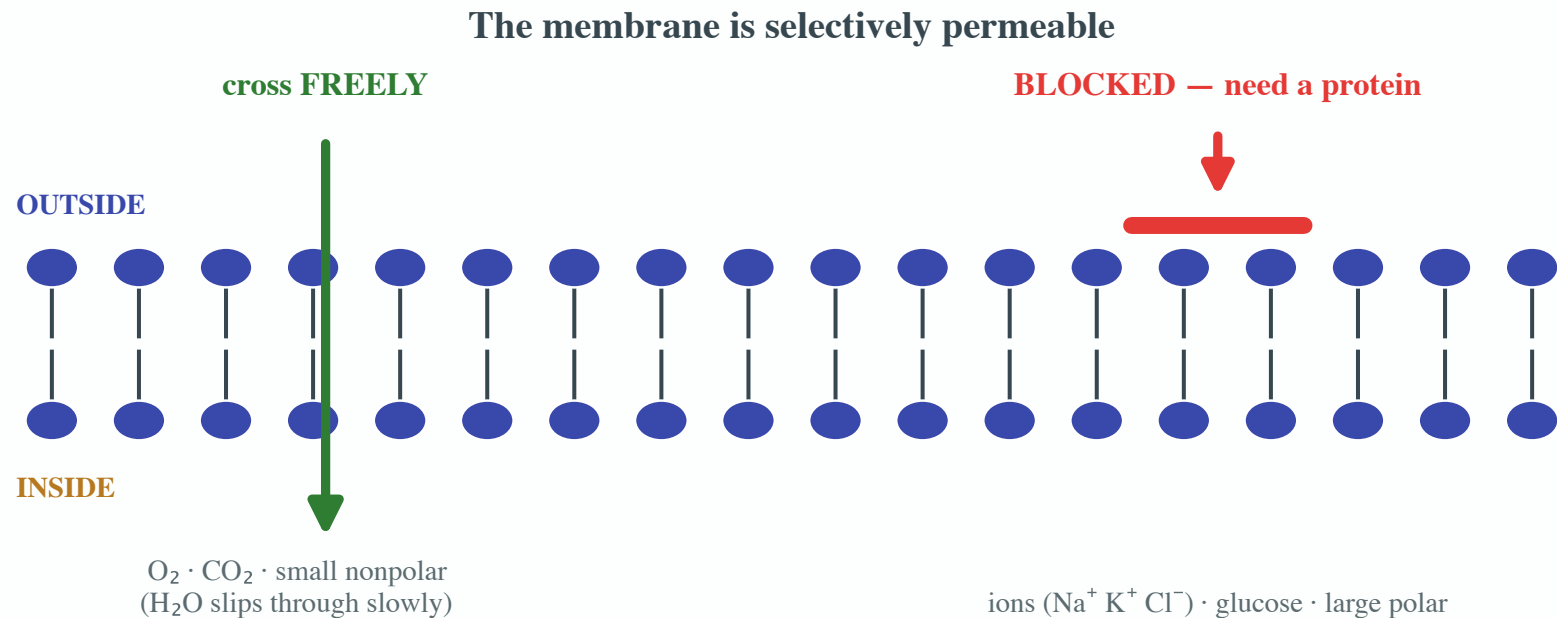


4 · Crossing the Membrane

"The membrane keeps almost everything out – so how does anything get in? Two answers: coast downhill for free, or pay ATP to climb."

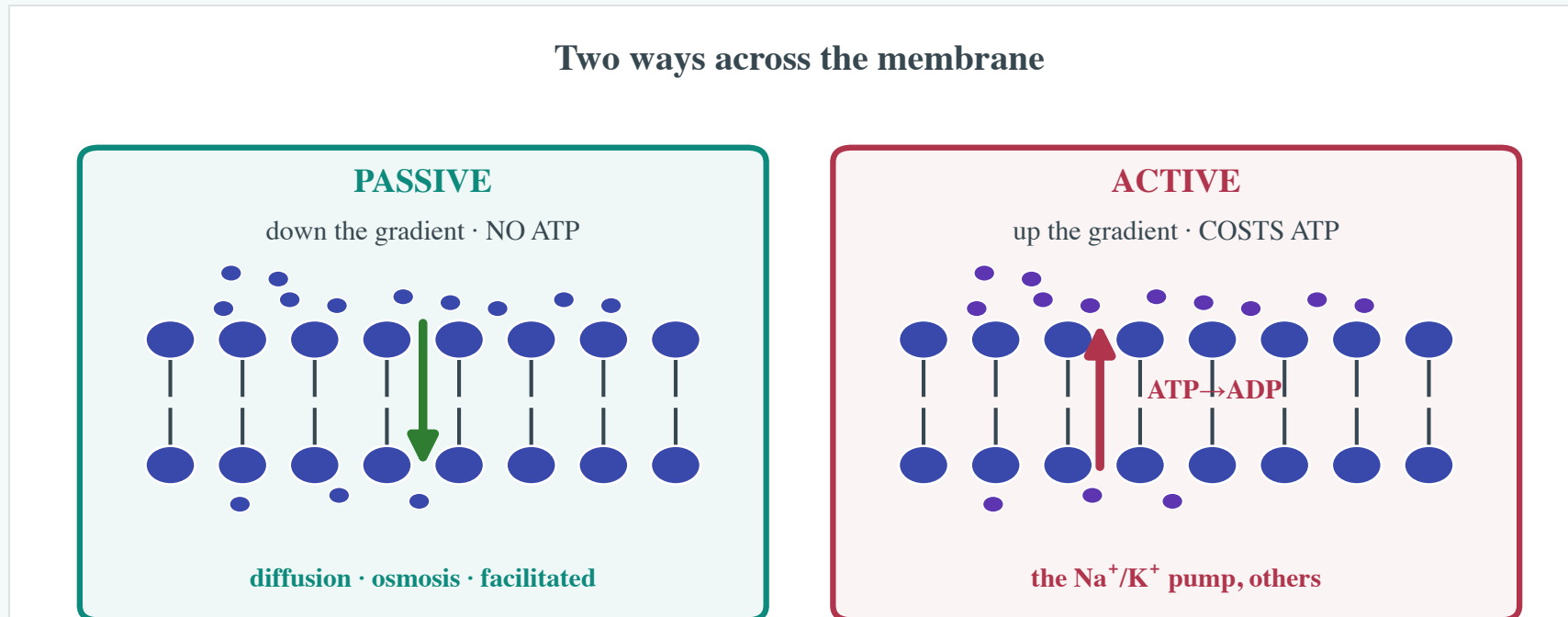
The membrane is a picky gatekeeper

That greasy bilayer is a great wall – which is a problem, because cells need to **let things in and out**. What crosses on its own is short: **gases** (O_2 , CO_2) and **small nonpolar** molecules slip right through; water trickles through slowly. But **ions** (Na^+ , K^+ , Cl^-), **glucose**, and anything **large or charged** are stuck at the door. They need a **protein** to let them through – and that's what the rest of this lecture is about.



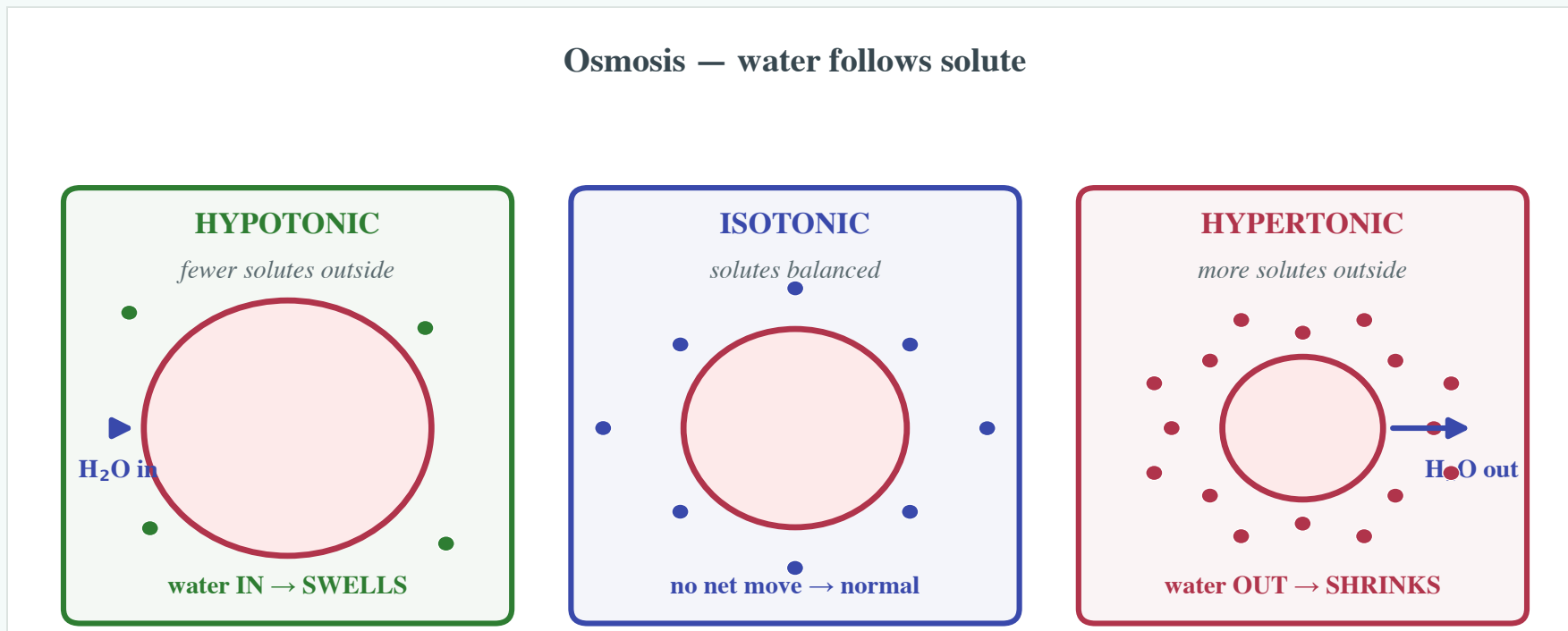
Two ways across: free vs paid

Every crossing is one of two kinds. **Passive transport** goes **down** the concentration gradient – from crowded to empty – so it's **free**, no energy needed (that covers simple **diffusion**, **osmosis**, and **facilitated diffusion** through a protein). **Active transport** goes the other way, **up** the gradient from empty to crowded – which nature never does for free, so it **costs ATP**. Downhill is free; uphill you pay.



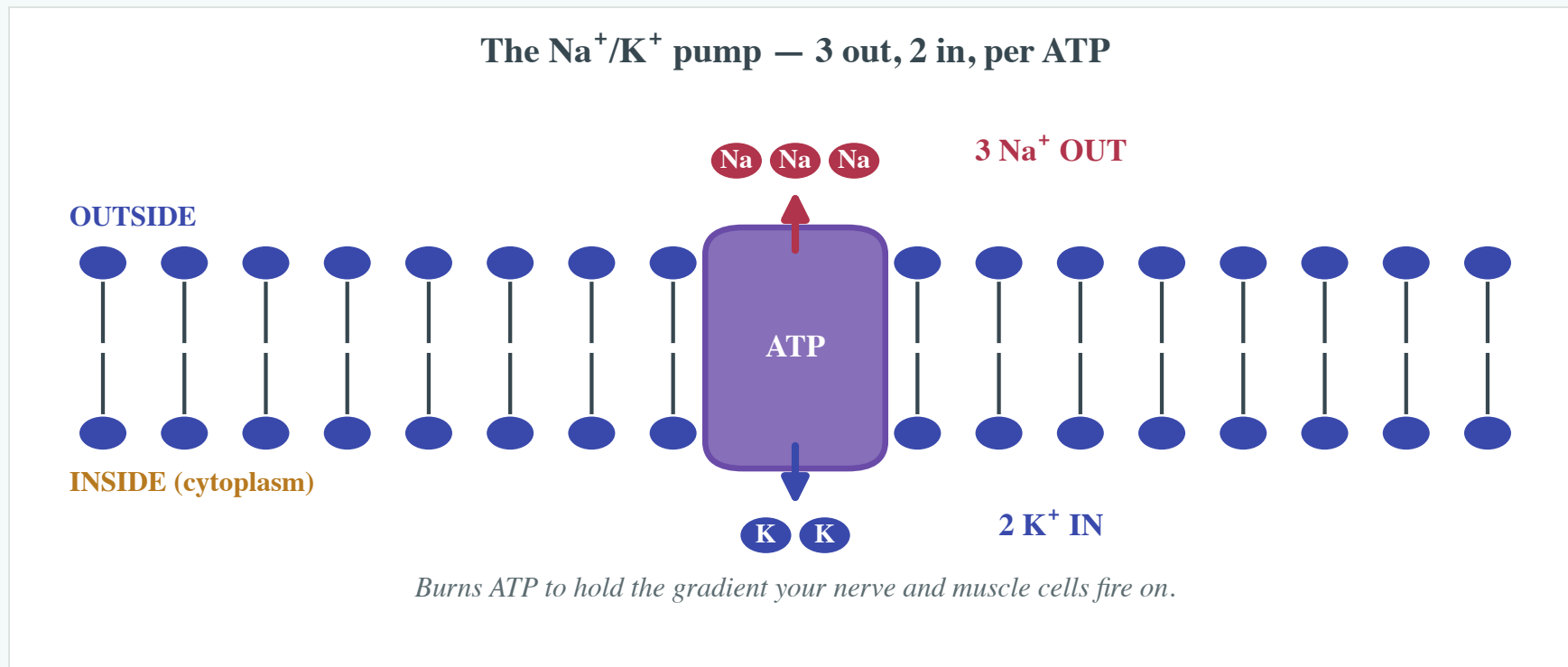
Osmosis: when water is the thing that moves

Osmosis is just diffusion of **water** – and water moves **toward the side with more solute** (trying to dilute it). This is why the fluid around a cell matters. In a **hypotonic** solution (less solute outside), water rushes **in** and the cell **swells** – and can burst. In a **hypertonic** one (more solute outside), water leaves and the cell **shrivels**. Only an **isotonic** solution leaves it happily unchanged.



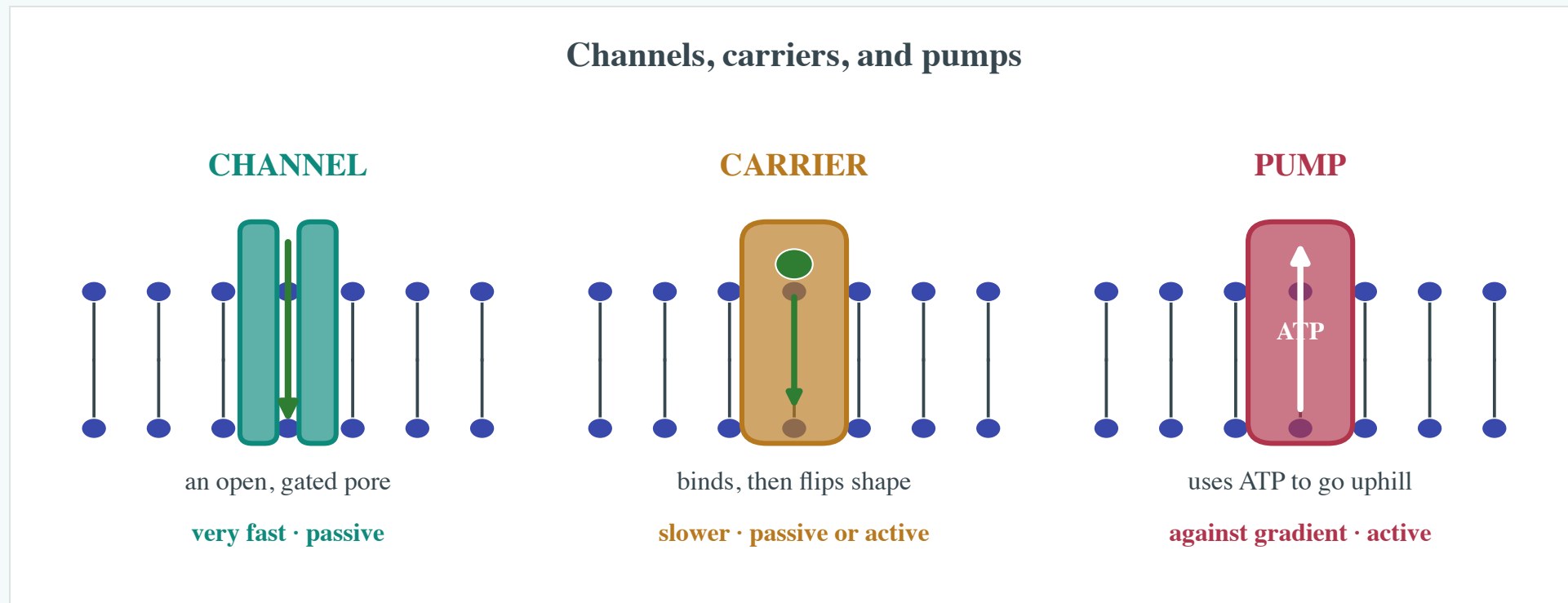
Paying to pump: the Na^+/K^+ pump

The star of active transport sits in every one of your cells. The **Na^+/K^+ pump** grabs **3 Na^+** and pushes them **out**, grabs **2 K^+** and pulls them **in** – both **against** their gradients – and it burns **one ATP** each cycle to do it. That relentless pumping builds the **ion gradient** your nerve and muscle cells discharge to fire. It's so important it eats a big slice of your **resting energy budget**.



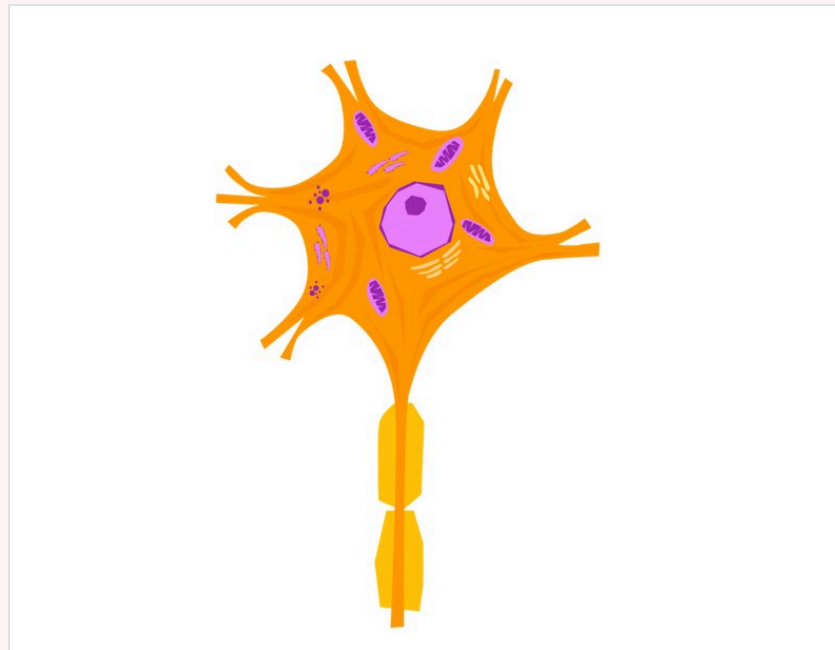
The three doormen: channels, carriers, pumps

The proteins that move things come in three flavors. A **channel** is an open (often gated) **pore** – ions stream through fast, passively. A **carrier** (transporter) **binds** its passenger and **flips its shape** to ferry it across – slower, and either passive or active. A **pump** is a carrier that **spends ATP** to force cargo **uphill**. Channels and passive carriers coast down the gradient; pumps push against it.



The pump that runs your mind

Why should you care that a pump moves 3 Na⁺ for 2 K⁺? Because that tiny imbalance is the **battery** behind every **thought, heartbeat, and muscle twitch**. Neurons spend up to a **fifth to a third of your resting ATP** just running Na⁺/K⁺ pumps to keep that battery charged. Stop the pumps – as the poison **ouabain** or a failing blood supply does – and the gradient collapses, cells swell, and excitable tissue goes silent.



Can you...?

- ☐ describe the roles of fats and the structure and naming of fatty acids (systematic, common, ω), and distinguish saturated, mono-, and polyunsaturated?
- ☐ identify the major lipid classes (triglycerides, phospholipids, glycolipids, steroids) and the trans-fat problem?
- ☐ explain the fluid mosaic model, what controls membrane fluidity, and the types of membrane protein?
- ☐ distinguish passive and active transport, and the roles of channels, pumps, and transporters?

If any box stays empty, the practice site has a drill for it.

