SIDE 1/2 MOLECULES → CELL · Chemistry of life · The four biomolecules · The cell & organelles · Membrane & transport · structure→function throughout

VISUAL · STRUCTURE→FUNCTION

Compiled by **AskSia** · mapped to the BIOL10008 curriculum · asksia.ai/cheatsheet/unimelb-biol10008

0 · The MST Blueprint
READ FIRST

★ The mid-semester test is invigilated, MCQ + short-answer, and it does two things: it makes you **read diagrams** (label a structure, name a stage, read a graph) and **explain mechanisms** (why this structure gives that function). So this sheet pairs **our own schematics** with dense ***structure→function*** notes.

Scope = mostly Topics 1–2 with some Topic-3 genetics. Side 1 = molecules→cell; Side 2 = information→energy→control.

SIA → *Every answer is one move: name the structure, then state the function it forces. "Describe + so-what" beats recall every time.*

1 · Chemistry of Life
L3 · BONDS & WATER

Elements of life: C, H, O, N, P, S. *Carbon* forms **4 covalent bonds** → stable backbone of organic molecules.

Electronegativity = a nucleus's pull on a shared bonding pair (more charge / fewer shells → higher; O>N). The **difference** predicts the bond: non-polar covalent → polar covalent → ionic.

Intermolecular forces (IMFs), weak→strong: *LDF* (temporary induced dipoles; stronger with bigger, less-branched molecules) → **dipole–dipole** → *hydrogen bond* (strongest; needs H bonded to F/O/N near a lone pair) → ion–dipole. IMFs are the hidden lever behind membrane fluidity, protein folding & DNA stability.

Water (from H-bonding): high heat capacity (thermal buffer), cohesion + adhesion (xylem rise), solvent for polar solutes (glucose in blood); *amphiprotic* (donates/accepts H^+). pH = differences in H^+ concentration.

1b · Is It Alive?
L1–2

Living things: common elements · made of **cells** · carry genetic info · grow · respond · mutate · exist in populations & **evolve**. *Viruses* are the borderline case — they mutate & evolve but aren't cells, can't grow or extract their own energy (must hijack a host).

Prokaryote vs eukaryote (heavily tested): both share the genetic code, cytoplasm & a plasma membrane. **Eukaryote** = membrane-bound nucleus + organelles, DNA **linear**. **Prokaryote** = nucleoid (no membrane), no organelles, DNA **circular**; ribosomes also differ in size. Think open-plan studio (prokaryote) vs a house with separate rooms (eukaryote).

2 · The Master Reaction
BUILD VS BREAK

Condensation = two monomers join, a water leaves, a bond forms (glycosidic / peptide / ester). *Hydrolysis* = water is added, the bond breaks. They are exact reverses — **the single most reused idea in the course** (it returns as anabolism vs catabolism in metabolism). A *polymer* is a chain of repeating *monomer* subunits (e.g. starch = many α-glucose). Snapping LEGO together pops out a drop of water (condensation); prying apart needs you to add one (hydrolysis).

3 · The Four Biomolecules
T1 · STRUCTURE→FUNCTION

1 · CARBOHYDRATES

Monomer = **monosaccharide** ($C_6H_{12}O_6$). Glucose/galactose/fructose are *structural isomers*. α- vs β-glucose differ only in the C1 -OH (down = α, up = β) — and that one flip decides everything:

POLYSACCHARIDE	BONDS	FUNCTION
Cellulose (β)	β-1,4	structural; alternating-flipped → straight chains → H-bonded into fibrils (plant wall)
Starch (α)	α-1,4 + 1,6	energy store in plants
Glycogen (α)	α-1,4 + 1,6	energy store in animals (liver)

Functions: structure · signalling (glycoproteins) · energy (glucose) · storage (starch/glycogen). **Glycosidic / ether bond** links C1 of one sugar to C4 of the next (1,4); branches via 1,6. Cellulose **microfibrils** are held by H-bonds between -OH groups of adjacent chains + aggregate LDF — the same IMF idea, repeated.

2 · LIPIDS

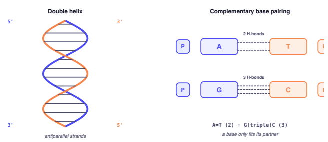
Not true polymers — held by additive **LDF**, large non-polar fraction → *hydrophobic*. **Triglyceride** = glycerol + 3 fatty acids; **phospholipid** = glycerol + 2 tails + charged phosphate head (*amphipathic* → bilayer); **steroid** = fused rings (cholesterol). Saturation = the lever: saturated tails (C–C) pack tight → high LDF → solid (animal fat); unsaturated (C=C **kink**) pack loose → fluid oils. This directly sets **membrane fluidity** later.

Phospholipids as surfactants: charged head ion-dipole/H-bonds water (hydrophilic), non-polar tail LDFs oils (hydrophobic) — being *amphipathic* lets them sit at a water–oil interface → the basis of the bilayer. Functions: energy store (energy-dense, lightweight), hormones (steroids e.g. estradiol), insulation (blubber), membranes, transport (lipoproteins).

3 · Biomolecules cont.
• NUCLEIC ACIDS & PROTEIN

3 · NUCLEIC ACIDS

Nucleotide = base (A,T/U,C,G) + pentose (deoxyribose/ribose) + phosphate. Base pairing A–T (2 H-bonds), C–G (3 H-bonds) is the most stable fit — and is why DNA self-copies & self-repairs. Chargaff logic: %A≈%T & %C≈%G ⇒ double-stranded; presence of T ⇒ DNA not RNA. Strands are *antiparallel* (5′→3′ opposite) — a zipper whose teeth only fit their partner, run in opposite directions on each side.



Our schematic · DNA double helix — antiparallel strands; A=T (2 H-bonds), G≡C (3); a base only fits its partner.

3 RNAS • SHAPE = JOB

mRNA = transcribed copy, carries codons. **tRNA** = key-shaped; **CCA-3′** end holds the amino acid; the *anticodon* pairs with the mRNA codon — this is how a triplet becomes an amino acid. **rRNA** = folds into the two ribosomal subunits (site of synthesis).

4 · PROTEINS

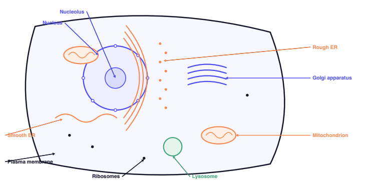
Amino acid = α-C + H + $-NH_2$ + -COOH + variable **R group**. *Peptide bond* = condensation amide. R-groups drive folding (H-bond, ionic, ion-dipole, *disulfide* -S-S-, hydrophobic LDF).

LEVEL	WHAT & FROM
1° Primary	aa sequence, N→C
2° Secondary	α-helix / β-sheet from backbone H-bonds
3° Tertiary	fold from R-group interactions
4° Quaternary	2+ chains (rarely assessed)

Denaturation = loss of 2°/3° shape → loss of function (heat/pH/solvent). **Heat** is most powerful — kinetic vibration overcomes all R-group bonds; change 2° and 3° follows. Shape is function. R-groups are grouped charged / polar uncharged / special / non-polar hydrophobic. **Protein functions:** immune (antibodies), signalling (glycoproteins), transport (lipoproteins), contractile (myosin), enzymes (trypsin, pepsin), structural (keratin, collagen). From a linear sequence (1°) the fold emerges, and the fold is the function.

4 · The Cell & Organelles
L7–8 · THE ROOMS

Each membrane-bound room runs one specialised job — division of labour. The *endomembrane system* is a connected set: **nuclear envelope → ER → Golgi → vesicles/lysosomes → plasma membrane. Rough ER** (ribosome-studded) makes protein; **smooth ER** does not. The ER is continuous with the nuclear envelope.



Our schematic · the eukaryotic cell — each membrane-bound organelle is one specialised 'department'.

ORGANELLE STRUCTURE→FUNCTION

Nucleus (from membrane invagination): DNA stored as *chromatin* = DNA wound on *histones* → nucleosomes → chromosomes. Packs metres of DNA tiny & lets it divide evenly. **Mitochondria & chloroplasts** have **two membranes**; the inner is folded (*cristae* / *thylakoids*) → high surface-area-to-volume → more membrane for ATP-making.

Endosymbiosis (tested): organelles were free-living bacteria engulfed by an early eukaryote. **Evidence chain:** double membrane · own **circular** DNA · own bacteria-like ribosomes · divide by **binary fission**. Secondary endosymbiosis (e.g. *Euglena*) adds a **3rd** membrane (the eaten cell's) + a *nucleomorph*.

THE SECRETORY PATHWAY

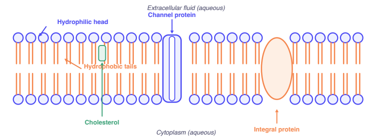
A flagship integrative answer: gene on → transcription → mRNA out a nuclear pore → translated on rough-ER ribosome → folded in ER → vesicle to **cis Golgi** → modified → leaves **trans Golgi** as a lysosome → fuses with the food vacuole → digestion. It links transcription + translation + endomembrane in one chain. Evolutionary origin of rough ER: some bacteria have ribosomes on the inner face of the cell membrane; invagination of that membrane is thought to give rough ER.

4b · Counting Membranes
ENDOSYMBIOSIS MST

Membrane count = how many "swallowings" happened. **2** membranes = primary (chloroplast/mitochondrion). **3** = secondary (*Euglena*; the extra = the eaten cell's plasma membrane). **4** = cryptomonads (with 4 genomes: nuclear & nucleomorph linear/eukaryotic; plastid & mitochondrial circular/bacterial). Russian-doll logic — each shell = one more cell eaten. A *nucleomorph* is the vestigial remnant nucleus of the engulfed eukaryote.

5 · Membrane & Transport
L7 · THE BORDER

Fluid mosaic: a phospholipid **bilayer** — hydrophilic heads out, hydrophobic tails in — with embedded proteins. An amphipathic molecule self-organises into a barrier that is fluid yet sealed to ions.



Our schematic · fluid-mosaic membrane — heads out, tails in; cholesterol & proteins embedded.

TUNING FLUIDITY (STRUCTURE→FUNCTION)

Cholesterol: polar -OH head H-bonds the phospholipid heads; non-polar rings LDF the tails. At higher temp → more LDF → tighter packing → **lower fluidity** (a buffer). At low temp a cell raises its **unsaturated** phospholipids — kinks keep spacing, stop solidifying (taught via *Tetrahymena*). Cholesterol acts as a fluidity buffer / shock-absorber.

Crosses easily: small, non-polar, uncharged (O_2, CO_2). **Cannot:** large or charged. Water is small but polar → slow osmosis, fast via *aquaporins*.

PASSIVE VS ACTIVE

	PASSIVE	ACTIVE
Direction	down gradient	against gradient
Energy	none	ATP (usually)
Examples	diffusion, osmosis, facilitated (aquaporin)	primary pump; secondary co-transport

Primary active: ATP pumps an ion low→high, building a gradient. **Secondary active**: a co-transported ion flowing *down* its gradient drags another substance *up* (e.g. sucrose/H^+ symporter). Carriers: *uniporter* · *symporter* (same way) · *antiporter* (opposite). **Bulk:** endocytosis (incl. *phagocytosis*) / exocytosis.

SIA → *A ligand-gated channel like an aquaporin is still facilitated diffusion — passive. "Channel/carrier" does not mean "active"; only moving against the gradient (spending ATP or an ion gradient) is active.*

5b · Why It Selects
THE PAYOFF

The cell trades free passage for control: passive crossing is free but uncontrolled; spending ATP on pumps buys the power to set concentrations, hold an ion gradient, and store energy it can later spend (secondary transport). Pump uphill into a tank (primary), then let the downhill flow turn a wheel (secondary). A plant **sucrose/H^+ symporter** is the textbook secondary case: the H^+ gradient (built by a primary pump) flows back in and drags sucrose up with it. The cell stores energy in the gradient itself, then spends it to move other cargo.

6 · The Cytoskeleton
L9 · THICKNESS = JOB

FILAMENT	PROTEIN	FUNCTION
Micro-filament ~7nm	actin	shape, **contractile ring**, streaming, pseudopodia
Intermediate 8–12nm	keratin	anchor nucleus/organelles, nuclear lamina
Microtubule ~25nm	tubulin	move organelles, cilia/flagella, **spindle**

MST inference: a drug that wrecks an amoeba's shape & movement has hit microfilament (actin) assembly.

7 · Classification
L1 · SORTING LIFE

Prokarya = two domains, **Bacteria** + **Archaea**. Four eukaryotic kingdoms sort by wall, nutrition & cellularity:

KINGDOM	NUTRITION	WALL
Plantae	autotroph	cellulose
Animalia	heterotroph	none
Fungi	heterotroph	**chitin**
Protista	both	varied

MST trap: a cell with no chloroplast **cannot** be an autotroph. *Autotroph* = makes its own food (e.g. via a chloroplast); *heterotroph* = gets energy from other organisms. A *phylogenetic key* is read as a branching key to find nearest relatives / oldest lineage (tested with Australian elapid snakes). Note the course flags uni/multicellular as a simplification.

8 · Origin of Life
L1–2 · THE STORY

Life began with the first **liquid water** (movement + UV protection); earliest fossils = *cyanobacteria in stromatolites* (~3.5 bya). Great Oxygenation: cyanobacterial photosynthesis ($6CO_2+6H_2O \rightarrow C_6H_{12}O_6+6O_2$) flipped the air to O_2 → ozone ($O_2 \rightarrow O^* + O^*$; $O^* + O_2 \rightarrow O_3$) → land became habitable → aerobic, multicellular eukaryotes. **Why the ocean first?** No ozone meant UV was lethal on land; water attenuates UV and shields early life.

SIA → *Stromatolites on Mars? They trap water + microbes; Mars once had water — so they could hold trapped life. Classic MST short-answer.*

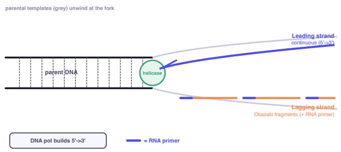
8b · Water, Again
IT TIES TOGETHER

Every "why does water do that?" answer is one weak bond — the **hydrogen bond** — repeated billions of times: high heat capacity (marine-iguana thermoregulation), cohesion + adhesion (xylem rise), solvent action (glucose in blood). Individually weak, collectively a strong web — like holding hands in a crowd.

9 · DNA Replication
L9 · SEMI-CONSERVATIVE

Semi-conservative: each new DNA keeps **one old strand + one new**. The intact old strand lets the cell proofread → fewer mutations.

The antiparallel problem: DNA pol III only builds **5′→3′** (reads template 3′→5′). So at the fork one strand is the continuous **leading** strand; the other is built in pieces (*Okazaki fragments*) as the **lagging** strand, primed by RNA primers.



Our schematic · replication fork — leading strand continuous; lagging strand in Okazaki fragments (5′→3′ only).

Other players: helicase (unwinds), gyrase, primase, DNA pol I, ligase. **Prokaryote** = circular DNA, **single origin**; **eukaryote** = linear, **multiple origins**.

MST link — DNA repair (base-excision, nucleotide-excision, mismatch) works *because* DNA is double-stranded: the complementary strand is the template for the fix.

9b · DNA Packaging
WHY IT COILS

DNA → wound on *histones* → *nucleosomes* → coiled into chromosomes. **Metaphase** chromosomes are tightly coiled (visible); **interphase** loosely coiled (not visible). Extreme coiling packs long DNA into a tiny, divisible package — like string wound on yo-yos, then coiled again and again. The advantage is twofold: it fits metres of DNA into a microscopic nucleus, and it lets the genome divide evenly at mitosis.

9c · The Fork Players
WHO DOES WHAT

- **Helicase** — unwinds the double helix at the fork
- **Primase** — lays the RNA primer to start each piece
- **DNA pol III** — builds the new strand 5′→3′
- **DNA pol I** — swaps RNA primer for DNA
- **Ligase** — seals the Okazaki fragments together

Think of painting both sides of a road: one painter walks forward smoothly (leading), the other backs up in dabs (lagging).

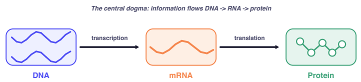
9d · Prok vs Euk Replication
CONTRAST

	PROKARYOTE	EUKARYOTE
DNA	circular	linear
Origins	single	multiple
Division	binary fission	mitosis

Multiple origins let a big linear genome copy fast enough — many forks fire at once rather than one crawling round a loop.

10 · The Central Dogma
L · DNA→RNA→PROTEIN

Information flows DNA → RNA → protein. Read a sequence in the MST: identify the template strand, the transcription direction, then the peptide from a codon table.



Our schematic · the central dogma — transcription copies DNA to mRNA; translation builds protein.

TRANSCRIPTION

RNA polymerase binds a *promoter*, opens a transcription bubble, reads the **template strand 3′→5′**, adds NTPs to build mRNA **5′→3′**, stops at a termination signal, releases mRNA. Three phases: initiation / elongation / termination.

TRANSLATION

Ribosome subunits + mRNA assemble; initiator tRNA (Met) sits at the **P site**; a charged tRNA enters the **A site**; a peptide bond forms; the ribosome advances; spent tRNA exits the **E site**; a **stop codon** (UAA/UAG/UGA) ends it, releasing the polypeptide.

THE GENETIC CODE

Triplet **codons**; *redundancy* (several codons → one amino acid, e.g. UUA & CUG = Leu); **reading frame** matters. Start = **AUG** = Met.

10b · DNA vs RNA
QUICK CONTRAST

	DNA	RNA
Strands	usually 2	1
Sugar	deoxyribose	ribose
Bases	A T C G	A U C G
Forms	DNA	mRNA, tRNA, rRNA

Picture the players: mRNA = the recipe card, tRNA = the waiter carrying one ingredient, rRNA = the kitchen bench where it all comes together. Shape = job: a flat message, a key-shaped adaptor, a machine that reads.

10c · Reading a Sequence
MST DRILL

1. **Find the template** — RNA pol reads it 3′→5′
2. **Build mRNA 5′→3′** — complementary, with U for T
3. **Split into codons** from the start (AUG)
4. **Read the codon table** → the peptide, N→C

Mind the **reading frame**: shift by one base and every downstream codon changes. Because the code is redundant, a base change can be silent — but a frameshift rarely is.

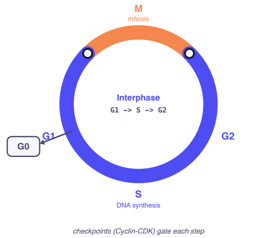
10d · The Ribosome Sites
A · P · E

- **A site** — arrival of the next charged tRNA
- **P site** — holds the growing peptide
- **E site** — spent tRNA exits

The ribosome ratchets one codon at a time until a **stop codon** is reached and the polypeptide is released.

11 · Cell Cycle & Mitosis
L9 · REGULATED DIVISION

Eukaryotic cell cycle: G0 (resting) / G1 (prep) / S (DNA synthesis) / G2 (prep) / M (mitosis). Gated at checkpoints by *Cyclin–CDK* complexes — the cell only divides after passing each.



Our schematic · the cell cycle — checkpoints (Cyclin-CDK) gate each step.

MITOSIS STAGES

STAGE	WHAT HAPPENS
Prophase	chromosomes condense, spindle forms
Metaphase	chromosomes align at the middle (plate)
Anaphase	sisters pulled apart by spindle
Telophase	nuclei reform; cytokinesis

Microtubules build the spindle & move chromosomes; *microfilaments* (actin) form the contractile ring. **Cytokinesis**: animals pinch via an **actin ring**; plants build a **cell plate** from fused Golgi vesicles. Prokaryotes don't do mitosis — they split by *binary fission* (B/C/D phases).

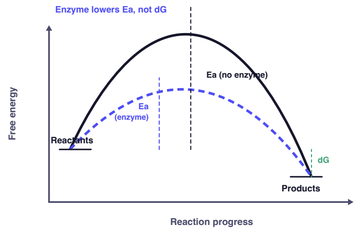
11b · Genetics • gene→trait
T3 · L19–21

A *gene* = a DNA sequence expressed as an RNA/polypeptide; carried on *chromosomes* & inherited. *Genotype* (makeup) vs *phenotype* (observable). *Allele* = a variant; **homozygous/heterozygous**; **dominant/recessive**. *Mutation* = the primary source of variation (alters RNA/polypeptide).

Dominance, molecularly: complete (one product masks) · *incomplete* (blended — purple×white primula → all **lilac**; lilac×lilac → 1:2:1) · *co-dominance* (both fully shown).

12 · Enzymes
L11–12 · CATALYSTS

An *enzyme* = a biological *catalyst* (usually protein) that speeds a reaction by lowering the *activation energy (Ea)*; it is **not consumed**. The substrate binds the *active site* (specific shape/chemistry) → enzyme–substrate complex → products (lock-and-key / induced fit). Specific shape → specific catalysis → reusable.



Our schematic · reaction profile — the enzyme lowers Ea but leaves ΔG unchanged (it speeds the trip, not the destination).

Key MST point: the enzyme lowers Ea, NOT ΔG. Same reactants & products, same energy difference — just a lower hill to climb (tunnel through the hill, don't move it).

Rate factors: temperature, **pH** (each enzyme has an **optimum** — pepsin ~pH 2–4, trypsin ~pH 8; read on an activity-vs-pH bell curve), substrate concentration, inhibitors. Beyond the optimum, denaturation drops activity. *Apoenzyme* (protein only) vs *holoenzyme* (+ cofactor/coenzyme) — both terms tested in MCQs. The enzyme doesn't move the destination, it digs a tunnel through the hill instead of climbing over it.

REGULATION

Allosteric regulation: a molecule binds a site **other than** the active site, changing shape/activity. *Feedback inhibition*: a pathway's end-product switches off an earlier enzyme. **MST:** block ATP at the **allosteric** site of PFK and glycolysis keeps running despite high ATP (lost negative feedback). Feedback inhibition is a thermostat — the product shuts off its own production once there is enough. **Allosteric enzymes** are precisely the ones regulated this way (common MCQ).

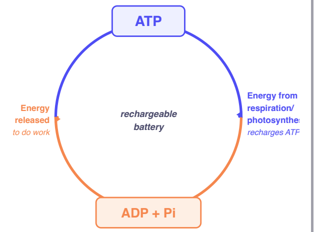
SIA → *"Enzyme used up?" No — it is released unchanged and reused, like a key that turns a lock then comes back out. On a pH graph, the peak marks the optimum; either side, activity falls and eventually denatures.*

12b · Optima & Inhibition
READ THE GRAPH

Each enzyme is tuned to **where it works**: pepsin peaks in stomach acid (~pH 2–4), trypsin in the basic intestine (~pH 8) — athletes who each peak only in their own climate. A bell curve's apex is the optimum; the slopes are reduced activity, then irreversible denaturation. Raising substrate concentration speeds the rate until enzymes saturate, then it plateaus. Inhibitors lower activity — competitive ones block the active site; allosteric/non-competitive ones bind elsewhere and reshape the enzyme.

13 · Metabolism & ATP
L10,13 · THE CURRENCY

ATP = the cell's energy currency; its job is to capture & transfer free energy. Hydrolysing a high-energy phosphate bond (**ATP → ADP + Pi**) releases energy.



Our schematic · the ATP cycle — a rechargeable battery: hydrolysis releases energy, respiration/photosynthesis recharge it.

ATP is a rechargeable battery, not a fuel tank — constantly cycled, never stockpiled. *Energy coupling*: endergonic **anabolic** building is driven by being coupled to the exergonic hydrolysis of ATP.

CATABOLISM VS ANABOLISM

Catabolism = break big→small, **releases** energy (protein→aa, fat→FA+glycerol, ATP hydrolysis). *Anabolism* = build small→big, **requires** energy (nucleotides→nucleic acids). It's the same condensation/hydrolysis logic from biomolecules — build vs break. The cell pays for building by burning down.

WHERE ATP IS MADE (CONTEXT)

Glycolysis (cytosol; control at PFK) → TCA (matrix) → *oxidative phosphorylation* on the inner membrane: an electron transport chain builds a **H^+ gradient** that drives *ATP synthase* (chemiosmosis). In prokaryotes the chain sits on the **inner face of the plasma membrane**. No O_2 → *fermentation* regenerates NAD^+ (yeast→ethanol+CO_2; muscle→lactate). **MST:** a low-O_2, yeast-like Mars cell does ethanol fermentation. The proton gradient is the real driver — like water behind a dam turning a turbine; a thylakoid gradient set up artificially makes ATP even without light (acid-bath experiment).

14 · Cell Signalling
L18 · SENSE & RESPOND

Three stages: reception → transduction → response. A **ligand** binds a *receptor*; transduction often runs through **second messengers** (e.g. *cAMP*); then a cellular response.

Specificity: a cell responds only if it has the matching receptor. Why one hormone does different things in different tissues: the same second messenger (cAMP) hits **different target proteins** in different cells — same doorbell, different households. Signals are classed by source & distribution (autocrine, paracrine, endocrine/hormonal, direct contact). So specificity comes from which receptors & target proteins a cell carries — same messenger, different outcome.

15 · structure→function recap
THE SPINE

1. **IMFs explain everything** — saturation→packing→fluidity; R-groups→fold→function; base pairing→DNA stability.
2. **Condensation/hydrolysis = anabolism/catabolism** — build-vs-break recurs.
3. **Surface-area-to-volume** — folded inner membranes (cristae/thylakoids) maximise reaction surface.
4. **Endosymbiosis evidence** — double membrane + circular DNA + own ribosomes + binary fission.
5. **Specificity via shape** — active sites, receptors, anticodon–codon, base pairing: right shape unlocks function.

16 · MST Diagram Discipline
BEFORE YOU SUBMIT

- **Read the axes/labels first** — a graph's units & a diagram's arrows carry the answer.
- **Name the stage/structure exactly** — "metaphase", "leading strand", "allosteric site".
- **State the function the structure forces** — never stop at naming.
- **Watch direction** — 5′→3′, down vs against gradient, Ea vs ΔG.
- **For "why" questions, give the IMF / shape reason.**

SIA → *If the question shows a picture, the marks are in labelling it precisely and explaining the mechanism it depicts — not in retelling the topic.*