The Ultimate AP® Biology Unit 3 Cheat Sheet

3.1–3.2 Enzyme Structure & Catalysis

Structure

Enzyme = biological catalyst, usually a protein (some RNA = ribozymes). Speeds rxn; NOT used up or changed.
Active site: pocket w/ specific shape + charge (set by R groups) → binds only matching **substrate**.
Induced fit: substrate binding shifts the active site's shape → tighter fit → strains bonds → rxn goes faster.
Specificity: shape & chemistry must match (e.g., polar ↔ polar, + ↔ −). 3D shape from 1°→4° structure.
Names: often end in -ase (amylase, lipase, ATP synthase). The **induced fit** model replaced lock & key.

How Catalysis Works

Activation energy (E_a): energy needed to start rxn. Enzymes ↓ E_a → more molecules react → rate ↑.

ΔG unchanged: enzyme doesn't change energy of reactants/products or which direction is favored.
Enzyme-substrate complex: E + S ⇌ ES → E + P. The enzyme is freed → reused many, many times over.
Fast: 1 enzyme can turn over thousands of substrates/sec.

Graph feature	Means
Hump height	E_a
Lower hump	Enzyme present
Products below	Exergonic (−ΔG)
Products above	Endergonic (+ΔG)

Cofactors

Cofactor: inorganic helper (Mg^{2+}, Zn^{2+}, Fe^{2+}). **Coenzyme**: organic helper, often vitamin-based (NAD^+, FAD).
Ribozymes: catalytic RNA (e.g., in the ribosome, forming peptide bonds) → a hint of an early RNA world.

⚠ Enzymes lower E_a but never change ΔG, or make an unfavorable (+ΔG) rxn favorable on its own.

✓ Energy diagram Q: the enzyme curve has a lower peak; its start & end points stay at the same heights.

3.3 Environmental Impacts on Enzymes

Temp & pH

Temp ↑ → faster molecules → more collisions → rate ↑ up to **optimum**. Past it → H-bonds break → **denatured**.
Cold: rate ↓ b/c fewer collisions; shape stays intact → activity returns when the enzyme is warmed.
pH: off the optimum → H^+ alters R-group charges → active site shape lost. Pepsin ~pH 2, trypsin ~pH 8.
Optimum varies: human ~37 °C; thermophile enzymes 70 °C+ (extra H-bonds, disulfide bridges).

Concentration

[Substrate] ↑ → rate ↑ until all active sites are busy → **saturation** (the V_{max} plateau on a graph).

[Enzyme] ↑ → V_{max} ↑ (if substrate plentiful).

Graph shapes: temp & pH → hump; [S] → rise then plateau.

Inhibitors

Type	Binds	Fix by ↑[S]?
Competitive	Active site	Yes
Noncompetitive	Allosteric site	No
Irreversible	Covalent	No

Competitive: mimics substrate shape; blocks active site. High [S] outcompetes it → same V_{max}.

Noncompetitive / allosteric: binds elsewhere → shape shift → V_{max} ↓ no matter how high [S] goes up.

Feedback inhibition: end product inhibits an early enzyme in the pathway → stops overproduction.
Allosteric activator: stabilizes active form → rate ↑.
Salinity: ions disrupt ionic bonds b/w R groups → the shape shifts → enzyme activity ↓ (just like pH).

⚠ Denaturation = lost 3D shape, not broken peptide bonds. Damage from high heat is often permanent.

✓ Graph Q: inhibitor curve still reaches the same plateau at high [S] → it's a competitive inhibitor.

3.4 Cellular Energy

Energy Rules

1st law: energy is not created or destroyed, only converted (light → chemical → kinetic, then heat).
2nd law: every transfer loses some energy as heat → entropy ↑. Cells need a constant energy input to live.
Order: life builds order (low entropy) by using energy + releasing heat → total entropy of the universe still ↑.
Free energy (G): energy able to do work. Spontaneous (−ΔG) ≠ fast; enzymes set the actual rxn speed.

Exergonic vs Endergonic

Rxn	ΔG	Energy	Example
Exergonic	−	Released	Cell resp.
Endergonic	+	Absorbed	Photosyn.
Catabolic	−	Breaks	Hydrolysis
Anabolic	+	Builds	Protein syn.

ATP & Coupling

ATP = adenine + ribose + 3 phosphates. Hydrolysis ATP → ADP + P_i releases energy (−ΔG ≈ −30 kJ/mol).

Energy coupling: exergonic ATP hydrolysis drives endergonic rxns; net ΔG must be negative.
Phosphorylation: ATP passes P_i to a molecule → it becomes more reactive (or changes shape: pumps, motor proteins).
ATP cycle: respiration regenerates ATP from ADP + P_i. A cell recycles its whole ATP pool in about a minute.
Pathways: rxns linked in sequence; product of one = substrate of next; each step has its own enzyme.
Energy loss: not enough input → cell can't maintain order → dies. More input than needed → stored (fat, glycogen).

⚠ Energy is never "used up." It changes form, and some always escapes as heat at every single step.

✓ Coupling Q: add the ΔGs of both rxns. A negative total → the paired reactions can proceed together.

3.5 Photosynthesis: Light Reactions

Big Picture

Equation: $6CO_2 + 6H_2O + light \rightarrow C_6H_{12}O_6 + 6O_2$. Endergonic; converts light energy → chemical energy in sugar.
Where: light rxns in the **thylakoid membranes**; the Calvin cycle in the **stroma** fluid around them.
Origin: first evolved in prokaryotes (cyanobacteria) → O_2 built up in the atmosphere → aerobic life possible.

Step by Step

1) Photosystem II: chlorophyll absorbs light → excited e^- go to the primary acceptor → enter the ETC.
Photosystem: antenna pigments funnel light energy → the reaction-center chlorophyll a pair, then out.
2) Photolysis: H_2O is split → replaces PSII's lost e^-; makes H^+ + **O_2 (a byproduct)** released from the leaf.
3) ETC: e^- energy pumps H^+ from the stroma → into the **thylakoid space** → builds an H^+ gradient there.
4) Photosystem I: light re-energizes the e^- → $NADP^+$ reductase → **NADPH** (e^- carrier for Calvin).
5) ATP synthase: H^+ flows back to the stroma → ATP is made (**chemiosmosis**), just as in mitochondria.

Input	Output
Light, H_2O	O_2 (released)
ADP + P_i	ATP → Calvin
$NADP^+$	NADPH → Calvin

Pigments

Chlorophyll a & b absorb red + blue; reflect green.
Carotenoids widen range. Action spectrum peaks red & blue.
Cyclic e^- flow: PSI only → extra ATP, no NADPH or O_2 → meets the Calvin cycle's higher ATP need (3:2).
Thylakoid stacks (grana) → ↑ membrane surface area → more photosystems + ETC proteins per chloroplast.

⚠ The O_2 released comes from WATER, not CO_2.

✓ Track the H^+: pumped INTO the thylakoid space, then flows OUT through ATP synthase into the stroma.

3.5 Photosynthesis: Calvin Cycle

Three Phases (Stroma)

1) Carbon fixation: **rubisco** attaches CO_2 to RuBP (5-C) → it splits into two 3-C molecules (3-PGA).
Rubisco: the most abundant protein on Earth; it's slow → plants make huge amounts of it in leaves.
2) Reduction: ATP + NADPH convert the 3-C molecules → **G3P** (a 3-C sugar) that leaves the cycle.
3) Regeneration: most G3P + more ATP rebuild RuBP, so the cycle can keep fixing new CO_2 molecules.
Count: 3 CO_2 → 1 net G3P; uses 9 ATP + 6 NADPH. 2 G3P → 1 glucose (so 6 turns of the cycle per glucose).
Light-independent, not "dark": needs ATP/NADPH from the light rxns → it stops soon after darkness falls.

Light Rxns vs Calvin

	Light rxns	Calvin cycle
Location	Thylakoid	Stroma
Uses	Light, H_2O	CO_2, ATP, NADPH
Makes	ATP, NADPH, O_2	G3P (sugar)
Recycles	ADP, $NADP^+$	→ back to light

Predict the Effect

No light → ATP/NADPH run out → Calvin stops → less RuBP is regenerated, so 3-C molecules pile up.
No CO_2 → fixation stops → RuBP piles up; NADPH/ATP build up unused → the light rxns slow down too.
Rate measured by: O_2 made, CO_2 used (water's pH ↑ as CO_2 drops), or how fast punched leaf disks float.
DCPIP lab: blue dye takes e^- from the light rxns → turns clear; a faster fade = a faster light-rxn rate.

⚠ Plants still respire, day & night. Net O_2 output = O_2 from photosynthesis − O_2 used in respiration.

✓ FRQ: link light rxn products (ATP, NADPH) to Calvin inputs, and explain each link of the chain.

3.6 Cellular Respiration: Glycolysis → Krebs

Big Picture

Equation: $C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O$ + ATP. Exergonic. Glucose is oxidized; O_2 is reduced to water.
e^- carriers: NAD^+ → **NADH**, FAD → **$FADH_2$** carry e^- (and their energy) to the ETC in the cristae membrane.

Stage	Where	Net yield
Glycolysis	Cytosol	2 ATP, 2 NADH
Pyruvate ox.	Mito matrix	2 NADH, 2 CO_2
Krebs	Mito matrix	2 ATP, 6 NADH, 2 $FADH_2$
ETC + ATP syn.	Inner memb.	~26–28 ATP

Glycolysis

Glucose (6-C) → 2 pyruvate (3-C). Uses 2 ATP, makes 4 → net 2 ATP + 2 NADH. No O_2 needed for it.
Universal: in nearly all organisms → evidence of shared ancestry; it evolved before there was O_2 in the air.

Pyruvate Oxidation & Krebs

Pyruvate oxidation: pyruvate → acetyl-CoA (2-C) + CO_2 + NADH. Happens after pyruvate enters the matrix.
Krebs cycle: acetyl-CoA + oxaloacetate → citrate → … → oxaloacetate again. 2 turns per glucose molecule.
Carbons: all 6 glucose C leave as CO_2 by the end of Krebs (2 in pyruvate ox., 4 in the Krebs cycle itself).
ATP so far: only 4 per glucose (substrate-level phosphorylation). Most energy now held in NADH + $FADH_2$.
Regulation: high ATP inhibits phosphofructokinase (glycolysis enzyme) → feedback inhibition.
Other fuels: fats → acetyl-CoA; amino acids enter glycolysis/Krebs at several different points.

⚠ The CO_2 you exhale comes from pyruvate ox. & Krebs. The O_2 you breathe ends up in water.

✓ Isotope Q: label glucose's C → watch it appear in CO_2; label the O_2 → it shows up in the H_2O made.

3.6 ETC, Chemiosmosis & Fermentation

Electron Transport Chain

NADH, $FADH_2$ drop off their e^- at proteins in the **inner mito membrane** (the folded cristae) → back to NAD^+, FAD.
e^- pass down chain → energy released → proteins pump H^+ from matrix → **intermembrane space**.
O_2 = final e^- acceptor: takes e^- + H^+ → H_2O. No O_2 → the chain backs up → NAD^+ isn't regenerated in time.
Why O_2: very electronegative → big energy drop → most ATP. **Cyanide** blocks last ETC protein.

Chemiosmosis

H^+ gradient (proton-motive force): H^+ flows back to matrix through **ATP synthase** → spins → ADP + P_i → ATP.

Oxidative phosphorylation = ETC + chemiosmosis → makes most of the cell's ATP (~90% of the total).
Uncouplers (e.g., DNP, brown fat's UCP1): let H^+ leak back → no ATP is made; the gradient's energy → heat.
Prokaryotes: their ETC sits in the plasma membrane; H^+ is pumped out of the cell, not into a space.

No O_2? Fermentation

Type	Product	Who
Lactic acid	Lactate	Muscle, bacteria
Alcoholic	Ethanol + CO_2	Yeast

Purpose: regenerate NAD^+ from NADH → glycolysis keeps going → only 2 ATP/glucose are made this way.
Anaerobic resp. (some prokaryotes): an ETC w/ another final e^- acceptor (SO_4^{2-}, NO_3^-) instead of O_2.
Yield: aerobic ~30–32 ATP/glucose vs 2 → ~15× more.
Facultative anaerobes (yeast) switch.

⚠ Fermentation makes NO extra ATP. Its whole job is recycling NAD^+ so glycolysis can keep running.

✓ Poison Q: block the ETC or remove O_2 → the H^+ gradient drops → ATP synthase stops making ATP.

3.7 Fitness & Unit 3 Exam Strategy

3.7 Variation = Fitness

Molecular variation lets a population handle more environments → ↑ fitness & survival chances.
Pigments: chlorophylls + carotenoids absorb diff. wavelengths → a plant uses more of the light available (deep water, shade, a forest canopy) → more sugar.
Enzyme variants: diff. versions work best at diff. temps/pH → the organism survives a wider range of habitats.
Endotherms: brown fat uncoupling → heat; **ectotherms** use outside heat → lower energy needs overall.
Fermentation & anaerobic paths: let organisms survive in low-O_2 environments (mud, hard-working muscles).

Photo vs Resp. at a Glance

	Photo.	Resp.
Organelle	Chloroplast	Mitochondria
H^+ pumped to	Thylakoid sp.	Intermemb. sp.
e^- source	H_2O	Glucose (NADH)
Final e^- accept.	$NADP^+$	O_2

FRQ & MCQ Moves

Predict: name the step blocked → what piles up → what runs out → the effect on ATP output. 4 links, in order.
Graphs: read the axes; rate = slope. Identify IV, DV & controls (no-enzyme tube, dark tube, boiled enzyme).
Math: rate = Δ amount/Δ time (mL O_2/min); % change = (final − initial)/initial × 100. Always show the units.
Data trap: a plateau may mean saturation or a used-up reactant, not a dead (denatured) enzyme.
Links: Unit 1 protein shape → Unit 2 membrane folds → Unit 8 energy flow through food webs & ecosystems.

⚠ Both organelles use an ETC + ATP synthase. Know which space gets the H^+ in each one (see the table).

✓ Always justify w/ mechanism: "less ATP b/c there's no H^+ gradient" beats a vague "less energy."