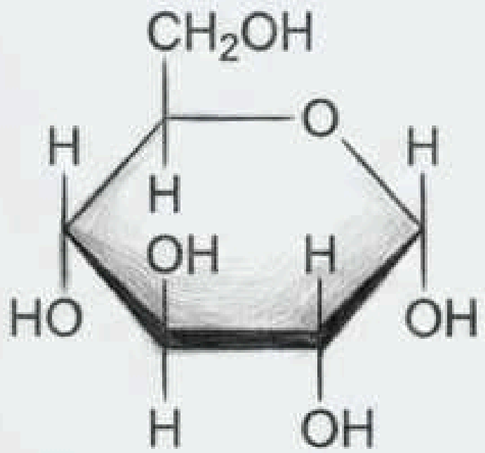
The Four Great Families of Molecules

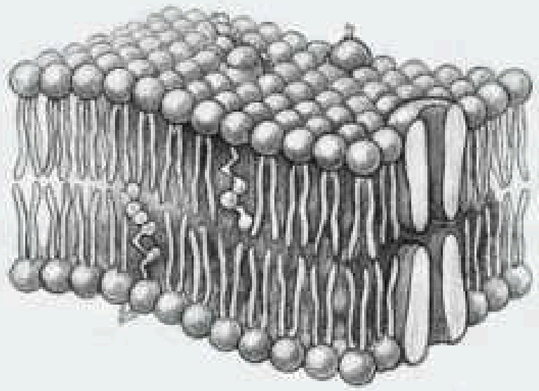
¶ 1.1

Carbohydrates



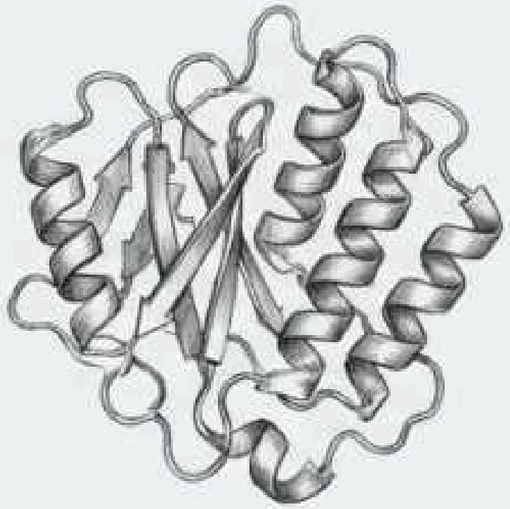
- Fuels and short-term energy stores (e.g., glucose, glycogen).

Lipids



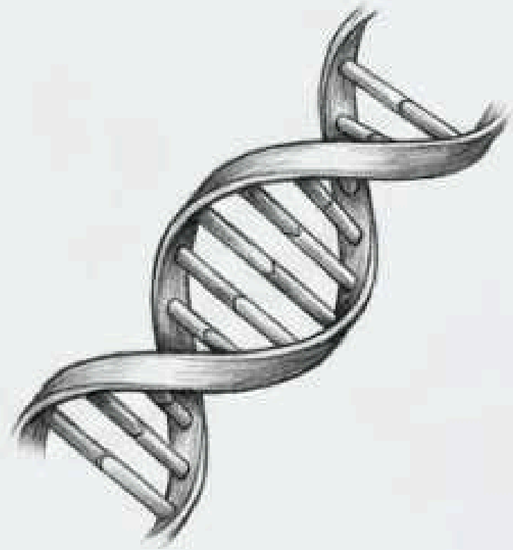
- Long-term energy storage and membranes around every cell (fats, oils).

Proteins



- The primary workers of the cell—functioning as enzymes, carriers, signals, and structural parts.

Nucleic Acids

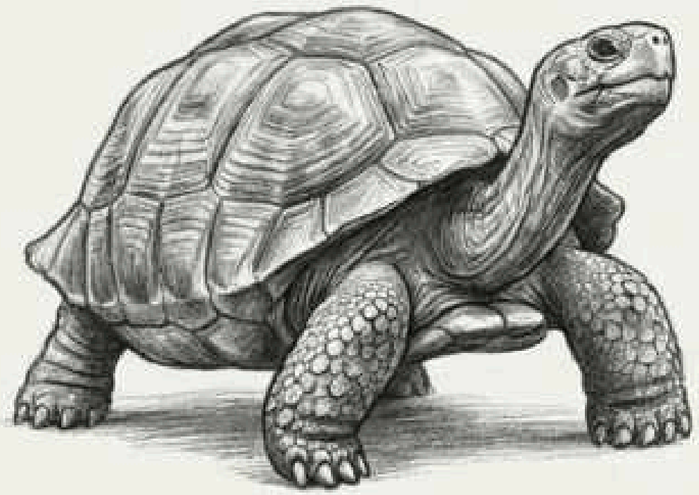


- Store and transmit instructions for making proteins (DNA and RNA).

The Catalyst Imperative

¶ 1.2

Uncatalyzed Chemistry



Millions of Years



Catalyzed by Enzymes

Milliseconds

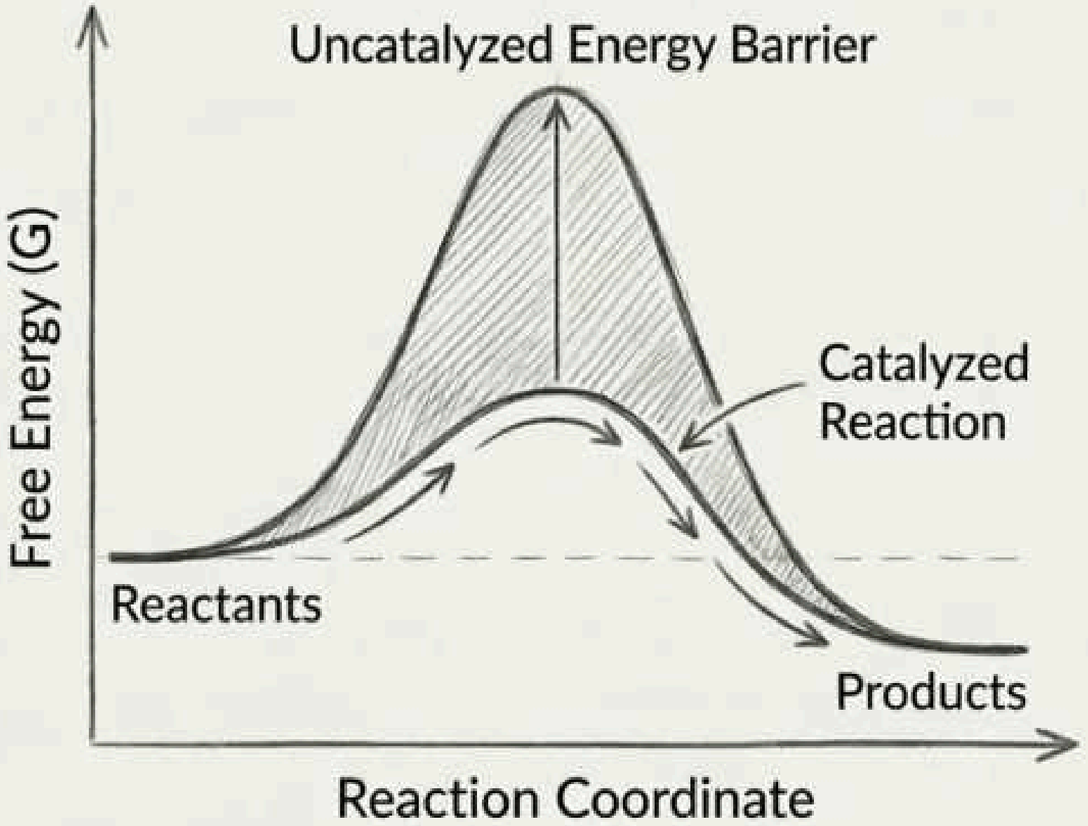
Core Concept: Without help, body chemistry is too slow to sustain life.

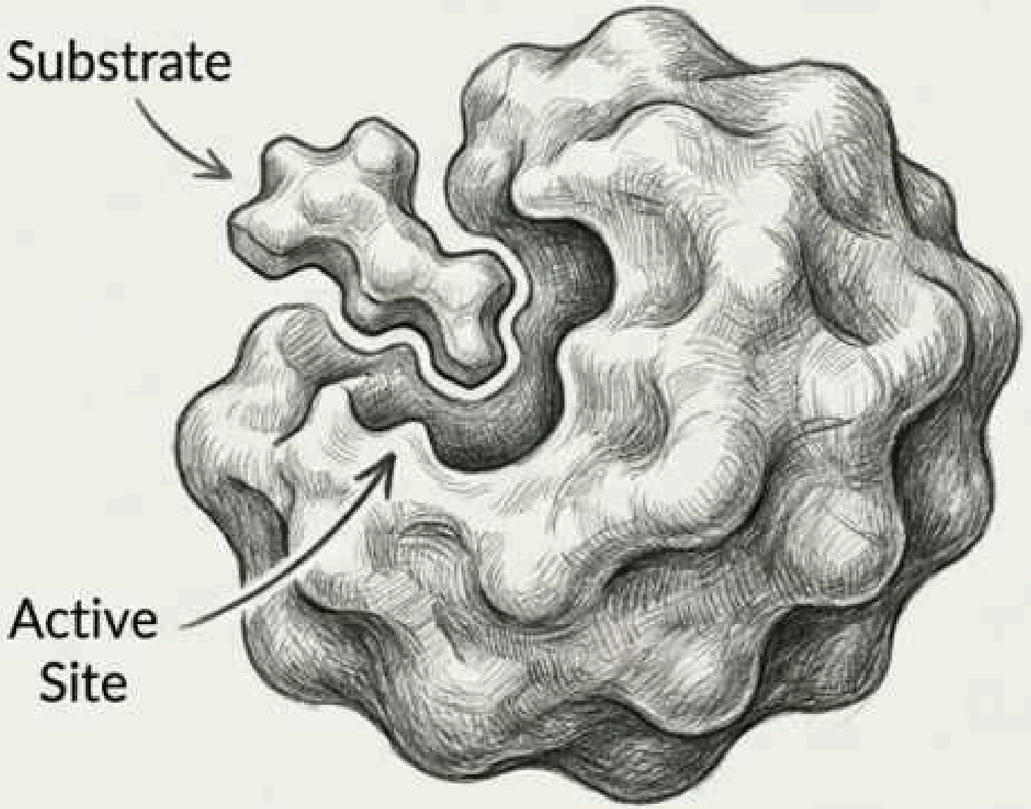
The Enzyme's Role: A reusable protein catalyst that speeds up a specific chemical reaction by many millions of times without being consumed.

Metabolic Scale: There are thousands of different enzymes in the body, precisely because nearly every step of metabolism requires its own dedicated enzyme.

Breaking the Energy Barrier

¶ 1.3





The Active Site: A specialized surface pocket whose shape and chemistry closely fit the starting molecule (the substrate).

The Mechanism: The enzyme grips the substrate in a way that makes the chemical reaction easier by physically lowering the energy barrier it must climb.

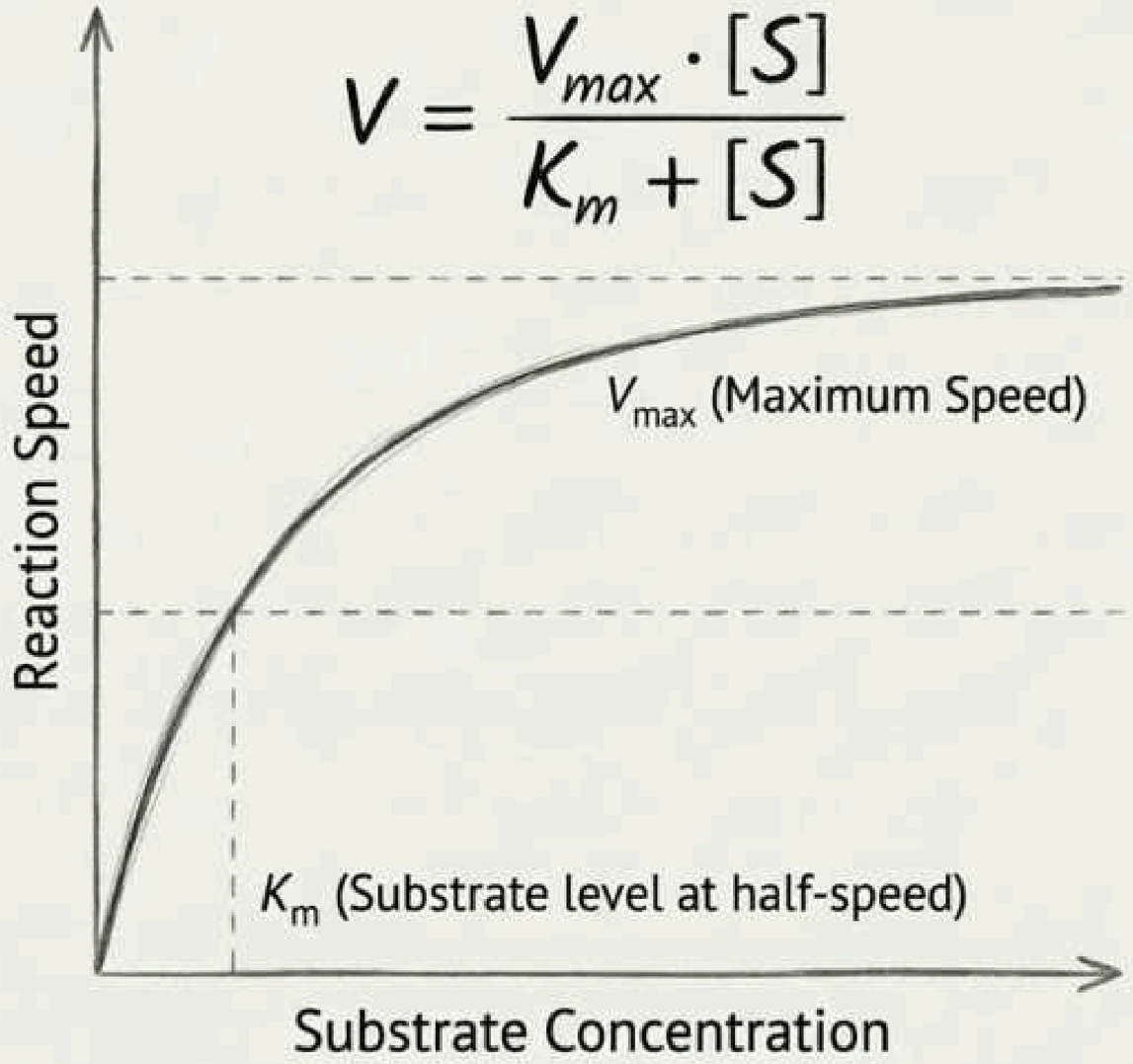
The Reset: Once the reaction completes, products are released and the enzyme is instantly ready for the next molecule.

The Mathematics of Saturation

¶ 1.4

$$V = \frac{V_{max} \cdot [S]}{K_m + [S]}$$

The Limit: Reaction speed increases with substrate, but only until every enzyme molecule is busy (Saturation). Adding more makes no difference.



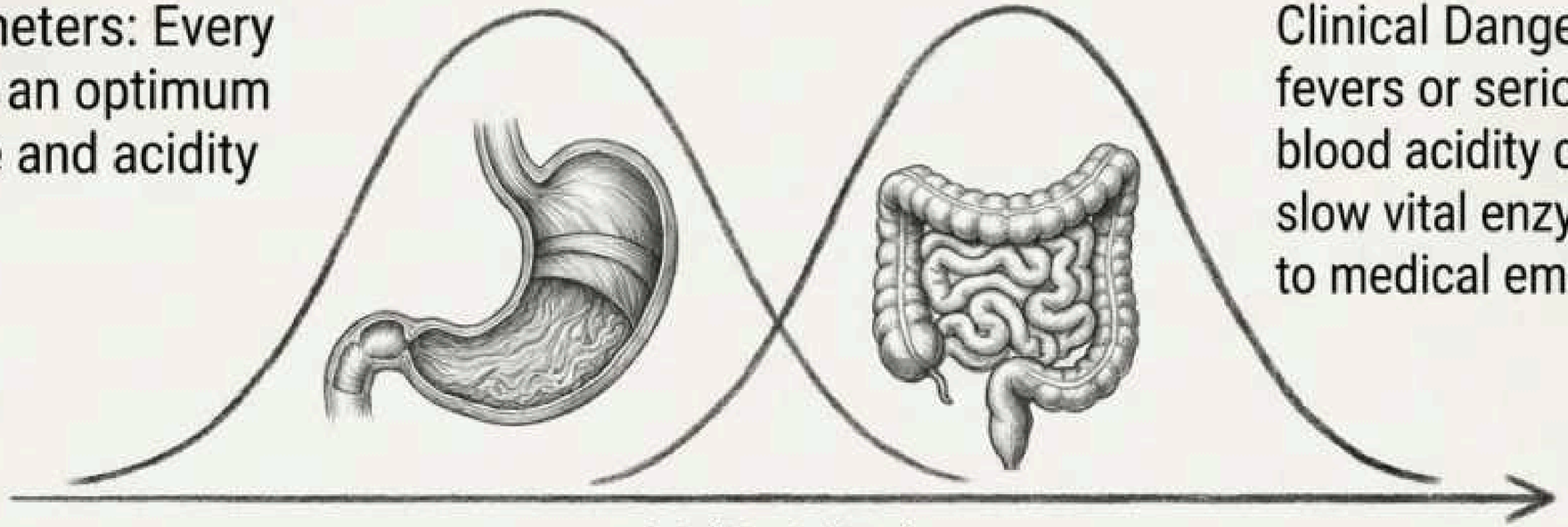
V_{max}: The maximum physical speed of the saturated enzyme.

Half-Speed (K_m): Indicates affinity. A low half-speed value means the enzyme grips its substrate tightly and works efficiently even when substrate is scarce.

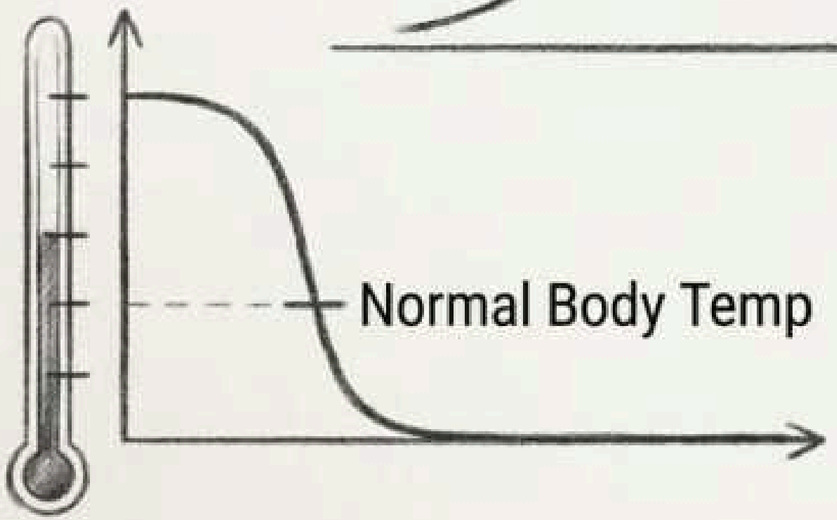
Environmental Vulnerabilities

¶ 1.5

Strict Parameters: Every enzyme has an optimum temperature and acidity (pH).



Clinical Danger: High fevers or serious shifts in blood acidity can physically slow vital enzymes, leading to medical emergencies.



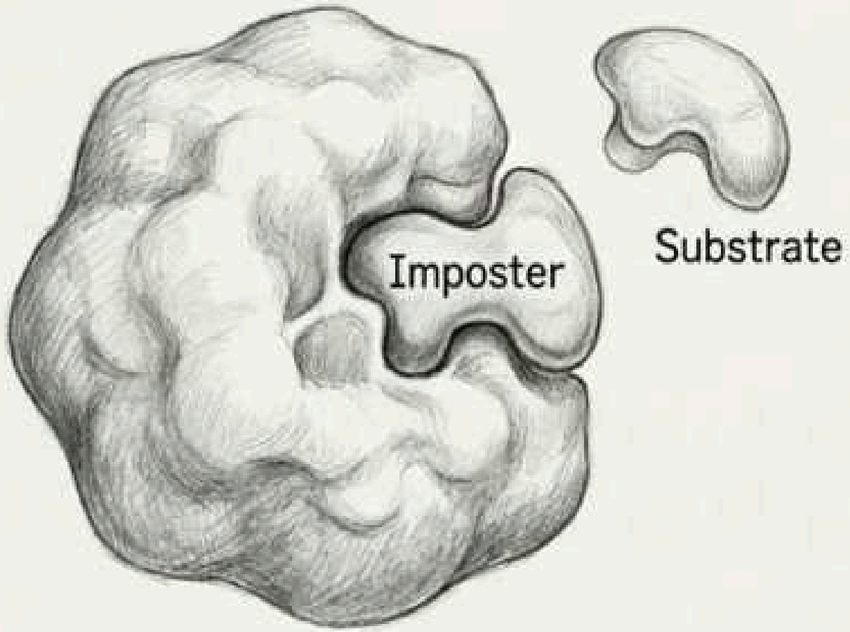
Regional Design: Stomach enzymes are purpose-built for strong acid, whereas intestinal enzymes require a mildly alkaline environment to function.

¶ 1.6

The Mechanisms of Inhibition

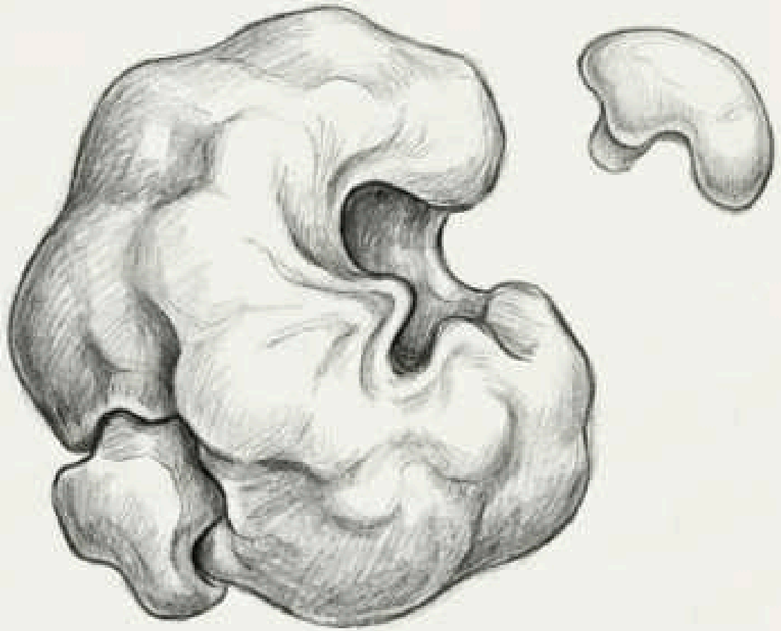
Context: Countless drugs and poisons function simply by blocking enzymes.

Competitive Inhibition



Resemble the substrate and fight for the active site. Key vulnerability: Can be overcome by flooding the system with extra substrate.

Non-Competitive Inhibition

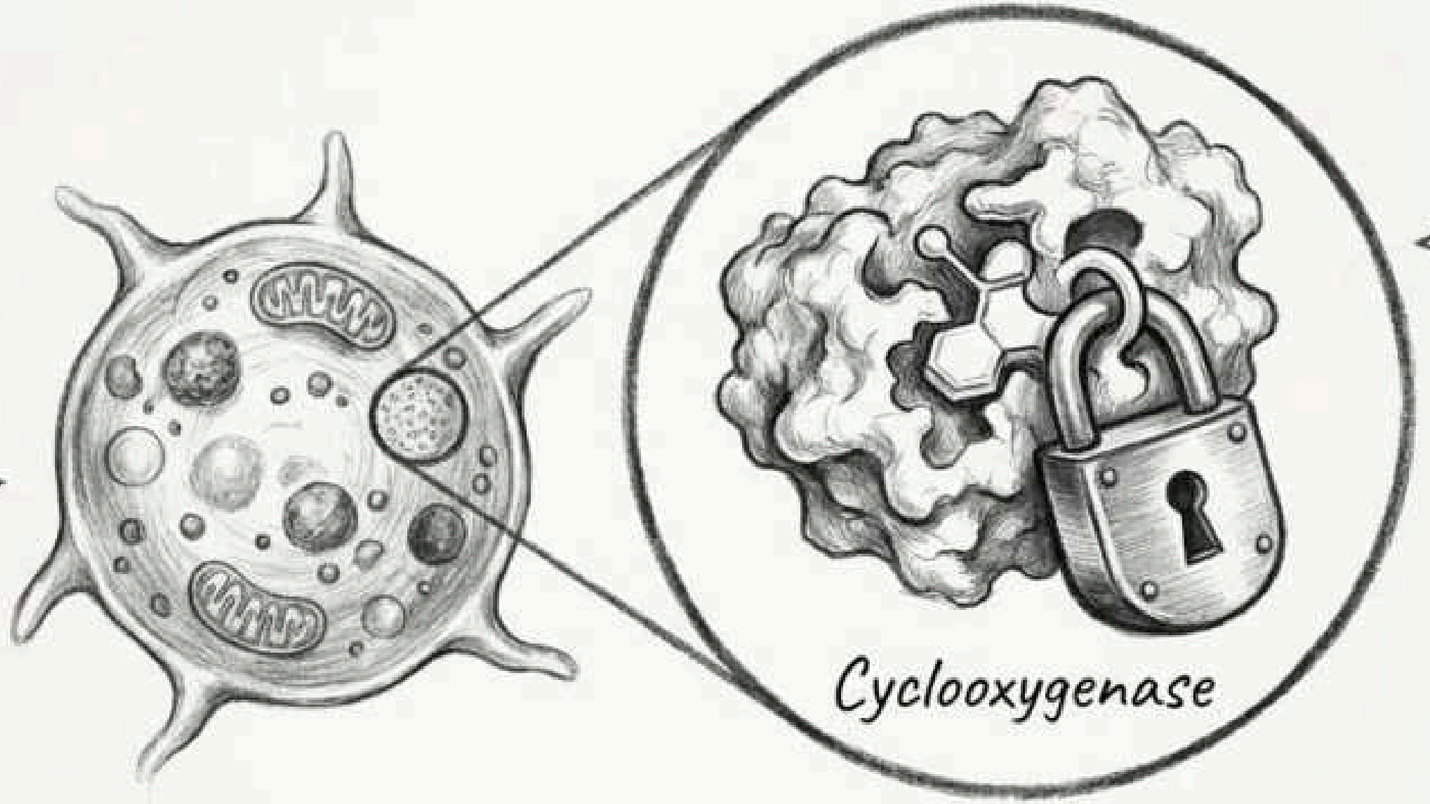


Bind elsewhere on the enzyme, reducing its activity regardless of how much substrate is present. (Note: Some of these bind permanently).

Permanent Blockade: The Aspirin Example

¶ 1.7

The Target: Aspirin permanently blocks cyclooxygenase, the enzyme responsible for chemical signals governing pain, fever, inflammation, and blood clumping.



The Clinical Reality: Because platelets lack the machinery to make new enzymes, a single dose of aspirin permanently disables them.



Lifespan: The anti-clumping effect lasts for the entire remaining life of the platelet—about a week to ten days (Preview for MED 511 B).

The Cellular Control Panel

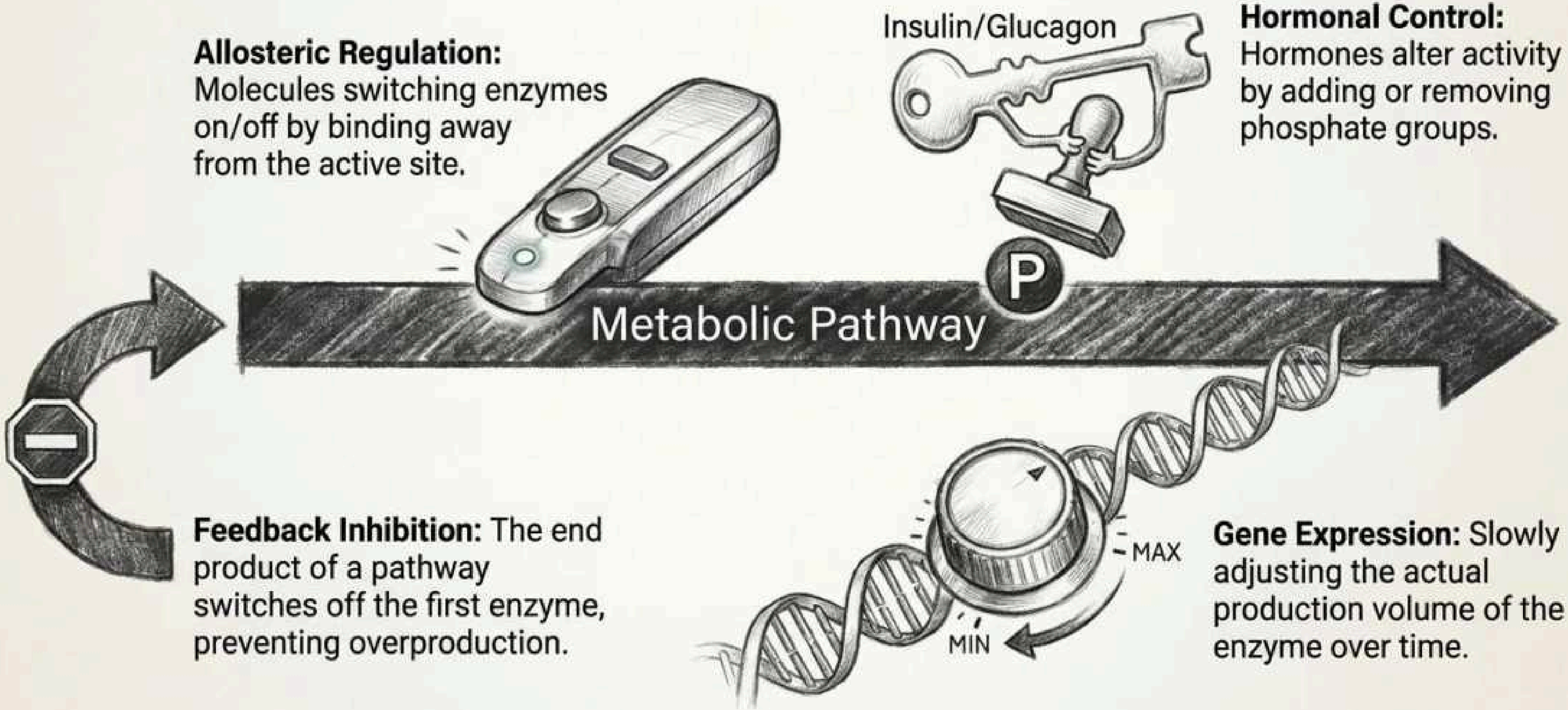
¶ 1.8

Goal: Ensuring pathways run only when needed.

Allosteric Regulation:
Molecules switching enzymes on/off by binding away from the active site.

Insulin/Glucagon

Hormonal Control:
Hormones alter activity by adding or removing phosphate groups.

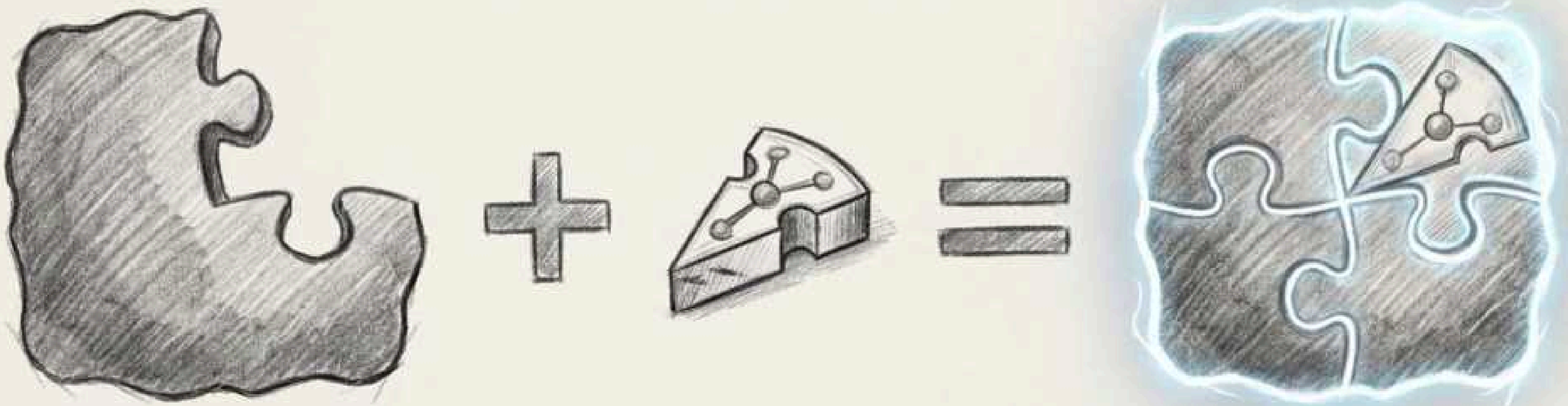


Feedback Inhibition: The end product of a pathway switches off the first enzyme, preventing overproduction.

Gene Expression: Slowly adjusting the actual production volume of the enzyme over time.

Essential Helpers: Cofactors and Coenzymes

¶ 1.9



Apoenzyme
(Inactive)

Cofactor
(Metal Ion or B-Vitamin)

Holoenzyme
(Active)

The Requirement: Many enzymes are inactive without non-protein helpers.

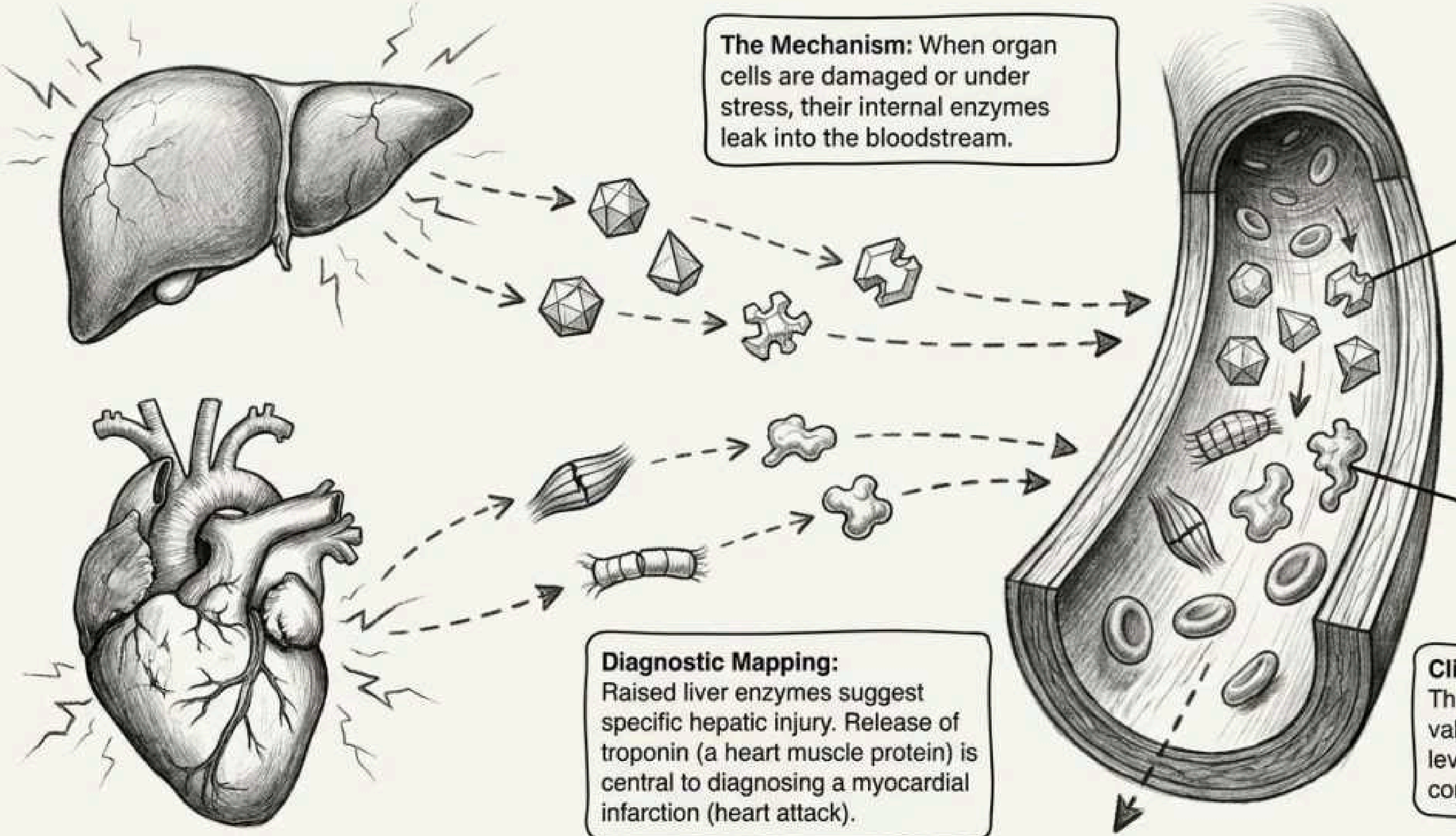
The Helpers: Metal ions (Zinc, Iron) act as structural aides. Coenzymes are small organic molecules, frequently made from vitamins.

Clinical Link: B vitamins form the coenzymes for energy metabolism. A lack of vitamins halts these enzymes, causing systemic disease.

Cellular Leakage as Clinical Clues

¶ 1.10

The Mechanism: When organ cells are damaged or under stress, their internal enzymes leak into the bloodstream.

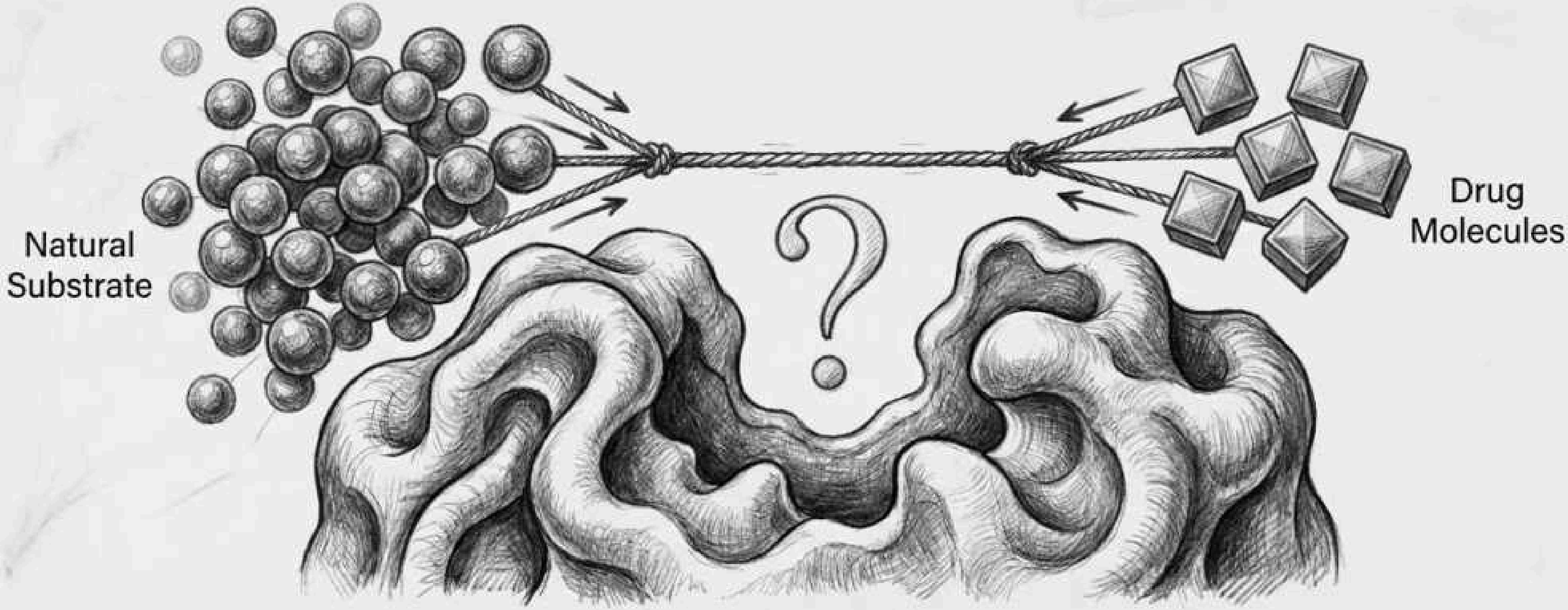


Diagnostic Mapping:
Raised liver enzymes suggest specific hepatic injury. Release of troponin (a heart muscle protein) is central to diagnosing a myocardial infarction (heart attack).

Clinical Caveat:
These levels are valuable clues, but a level alone never makes a complete diagnosis.

The Substrate Competition (Lecture Question)

¶ 1.11

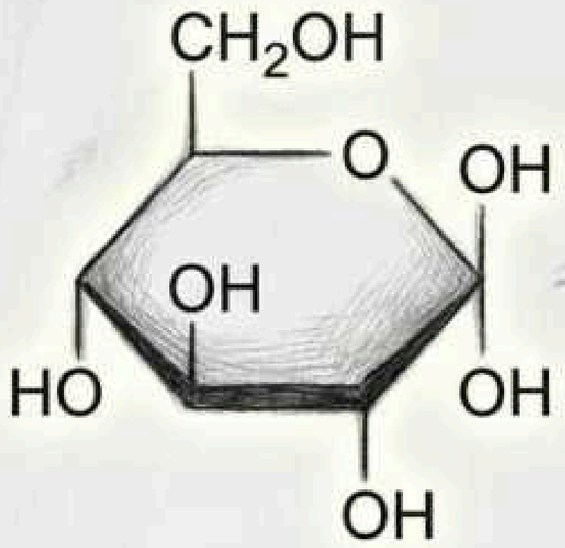


The Prompt: Why might a drug that competes with a natural substrate need to be given in larger amounts when the level of that substrate is high?

Application: Formulate your answer using the principle of active site competition and the physical probabilities of binding.

Prologue to Energy

¶ 1.12



Next in Lecture 2:
We will follow glucose—the body's most familiar fuel—step-by-step through the intricate first stage of energy extraction.