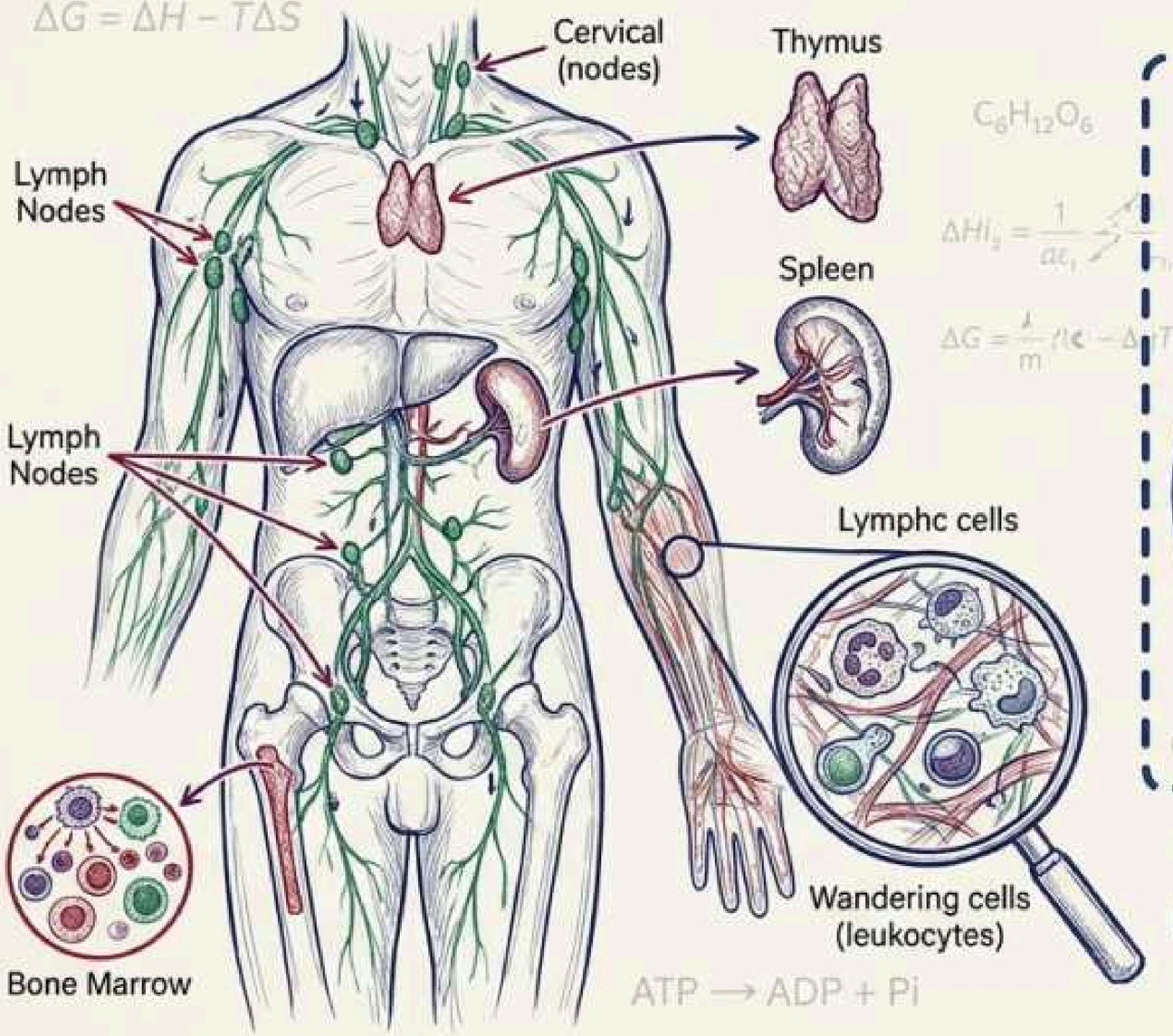
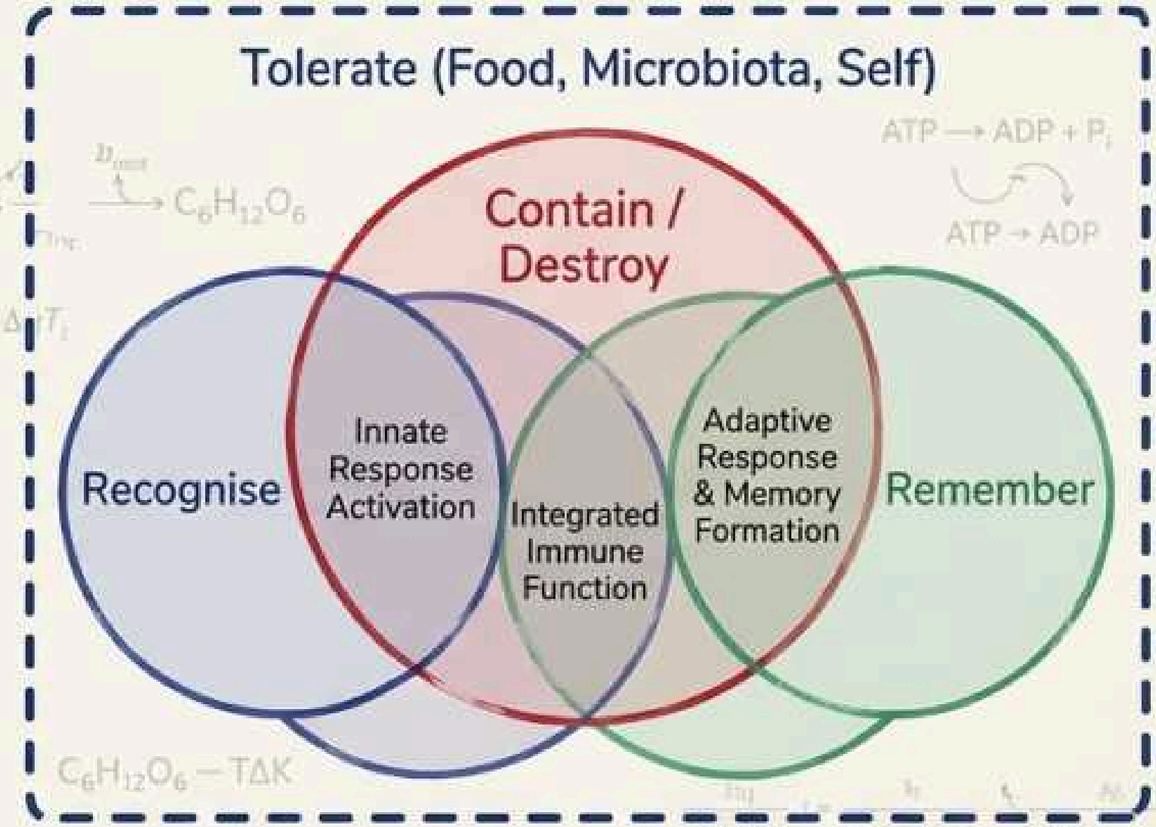
The Organised Defence Network

¶ 3.1

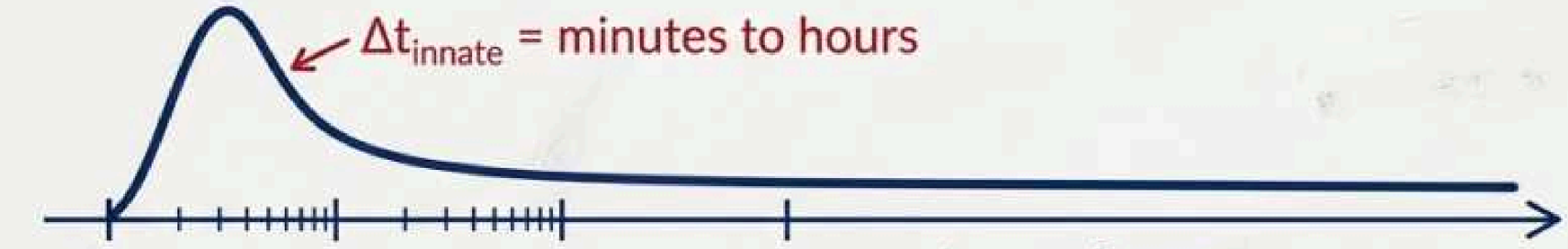


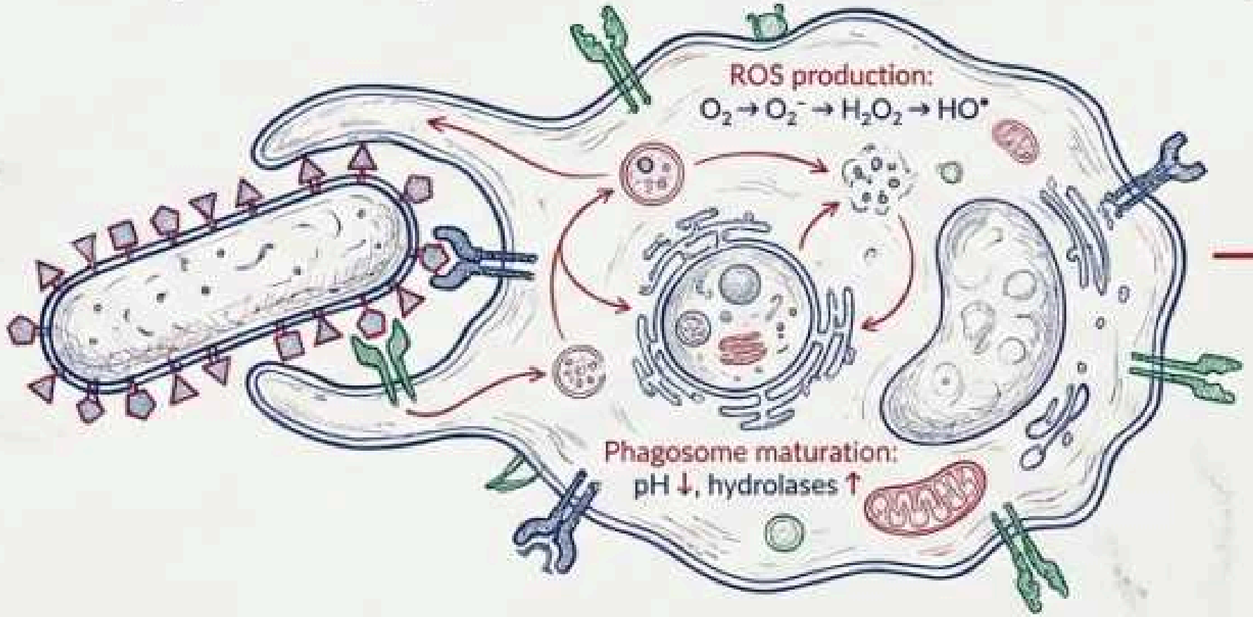


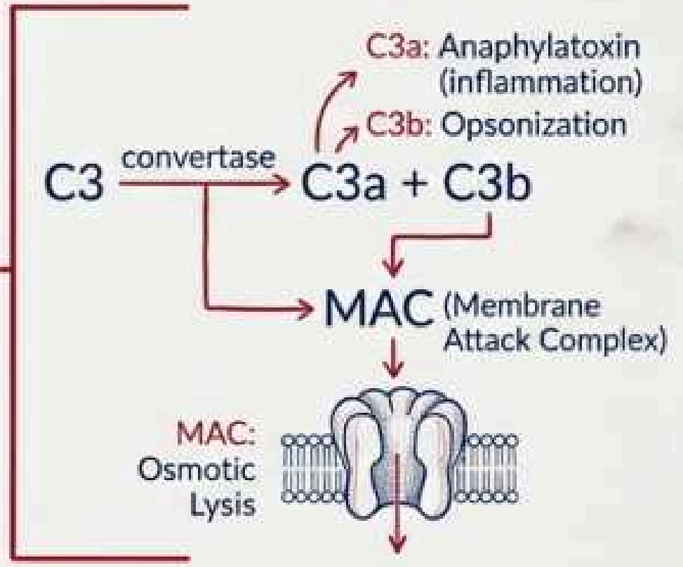
The **primary contest** between host and microbe begins here, functioning as a distributed network once **physical barriers** are breached.

Innate Immunity Acts Rapidly on Broad Signatures

¶ 3.2







- **Sensors:** Refined evolutionary products detecting broad danger, not unique species.
- **Arsenal:** Surfaces, Blood Proteins (Complement), Phagocytes.
- **Nuance:** Non-specific does not mean clumsy; it is deeply tailored to universal pathogen signatures.

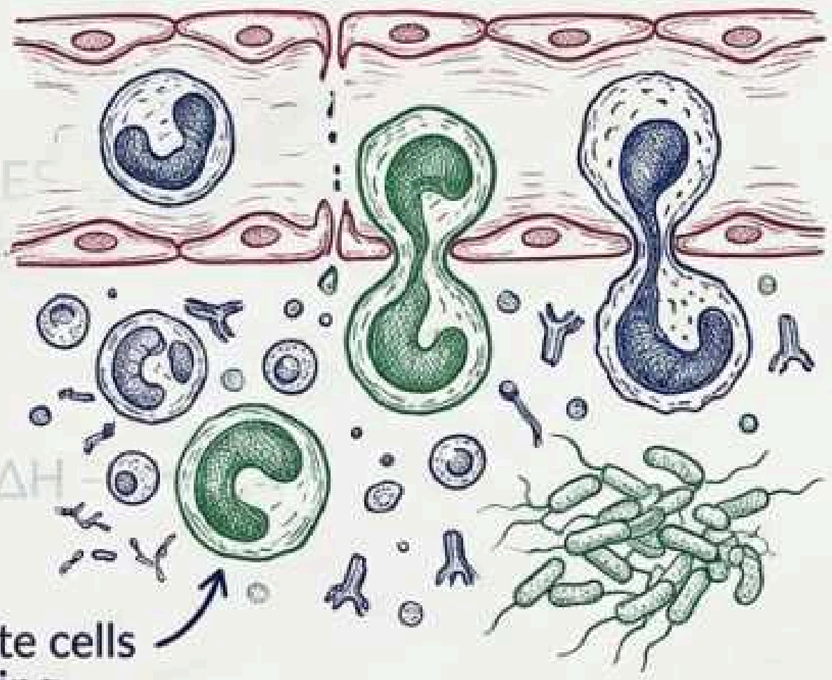
The Dual Tools and Costs of Innate Defence

¶ 3.3

Danger Signal

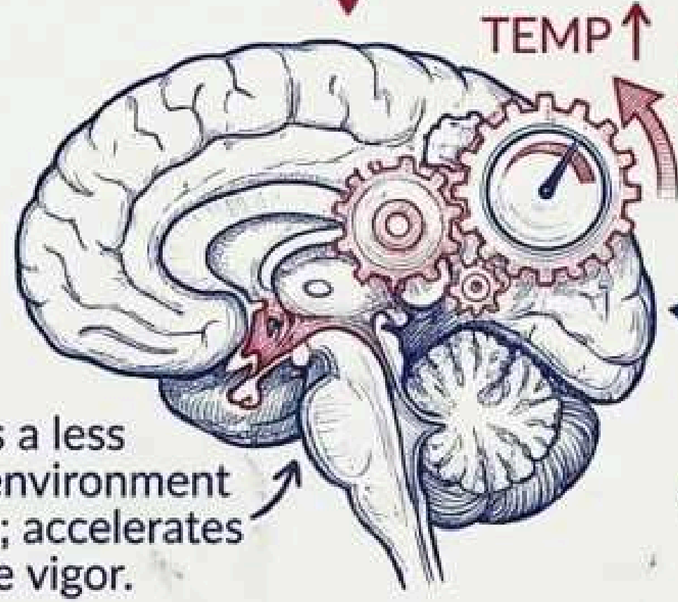
Local (Inflammation)

Recruits cells and proteins to a local site.



Leukocyte cells separatting

Systemic (Fever)

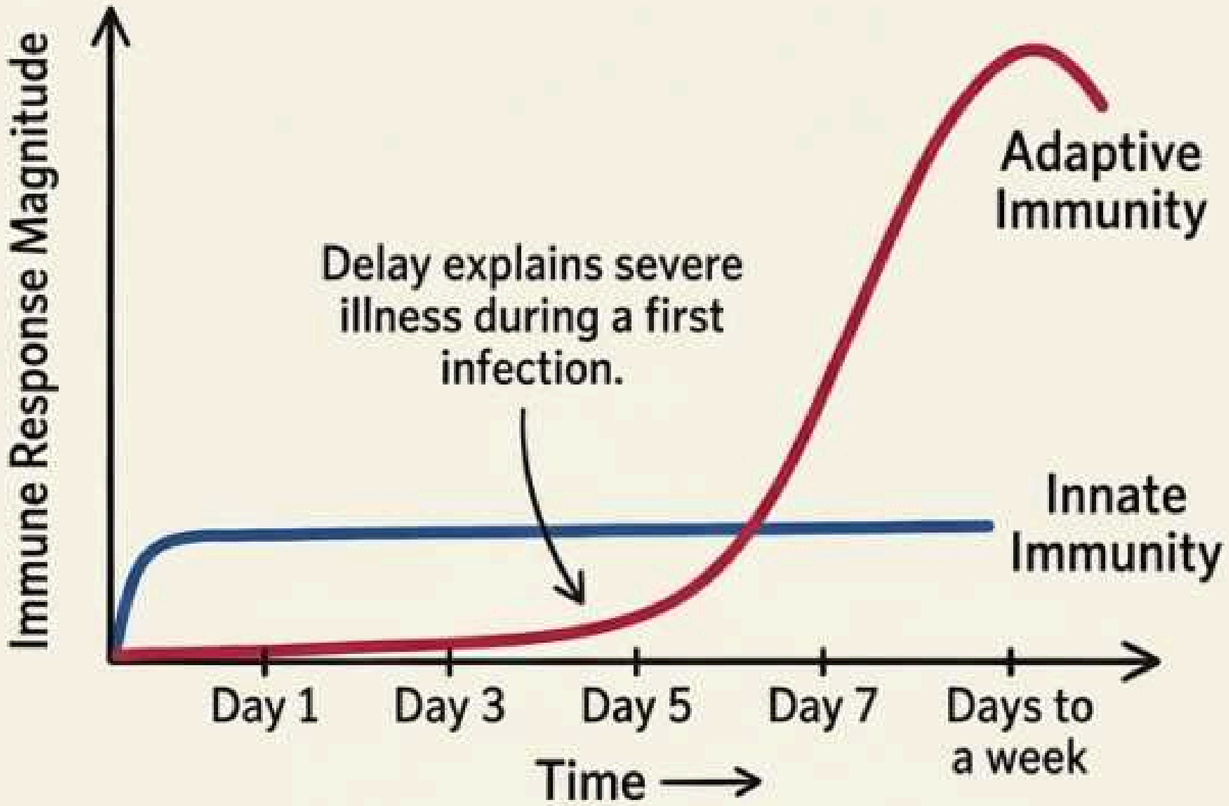


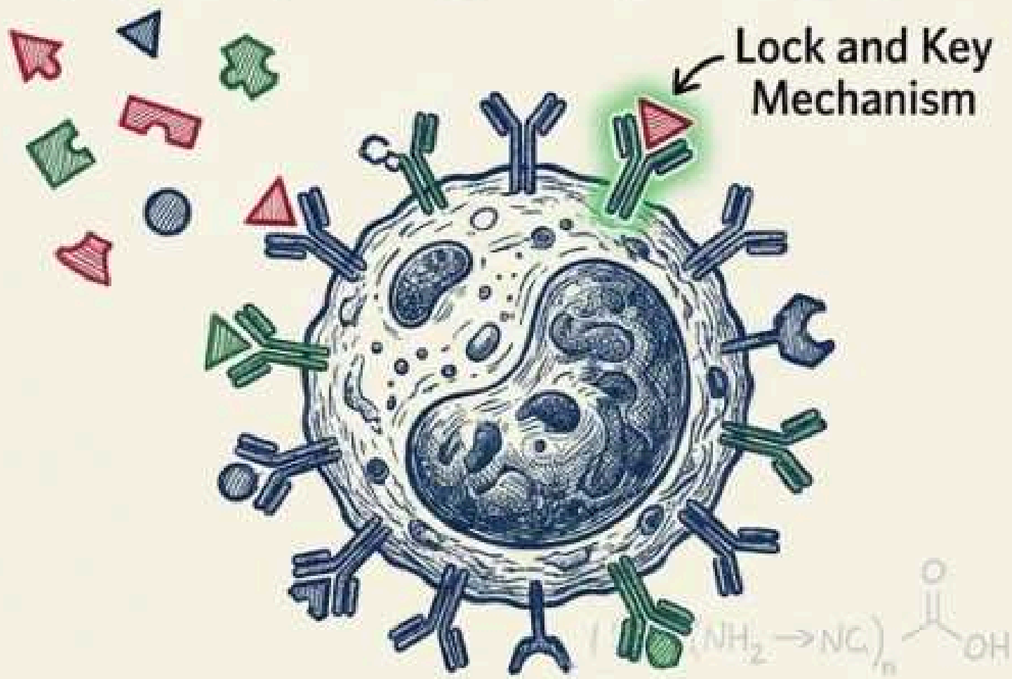
Creates a less hospitable environment for microbes; accelerates immune vigor.

Creates a less hospitable environment for microbes; immune vigor.

Illness is not just microbial load. Extreme or widespread inflammation/fever (especially in infants or compromised lungs) turns protective mechanisms dangerous.

Adaptive Immunity Provides Delayed, Tailored Precision





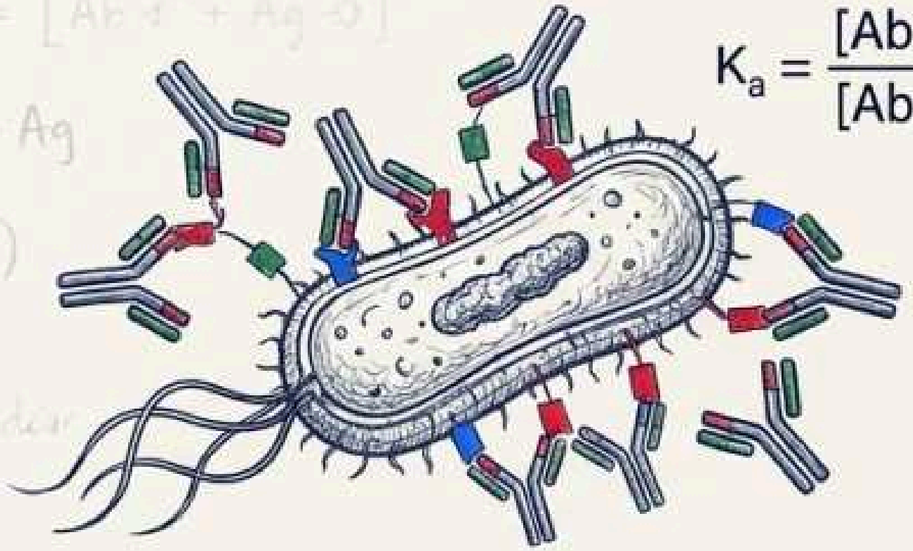
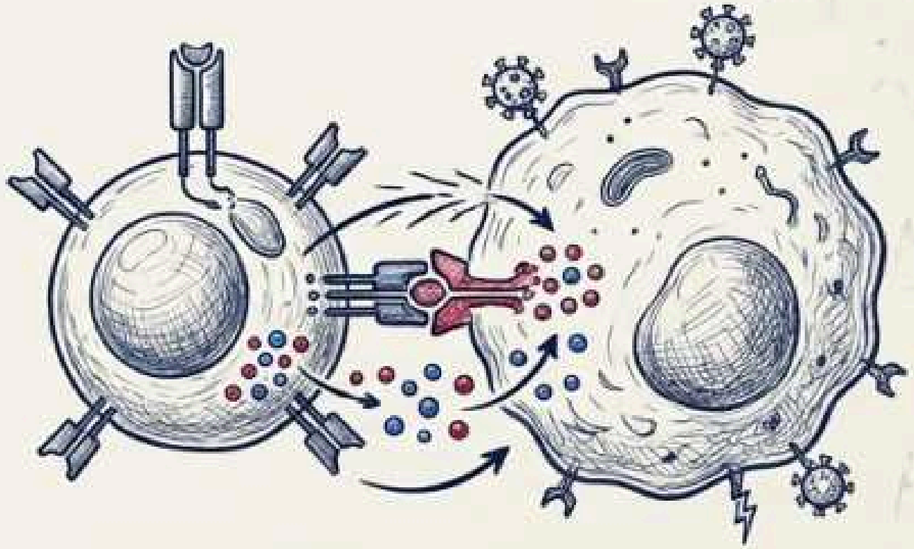
Lymphocyte (T/B cell)

Critical Constraint: The system must be taught self-tolerance.

The system must be taught self-tolerance. Failure in this education leads to autoimmune pathology.

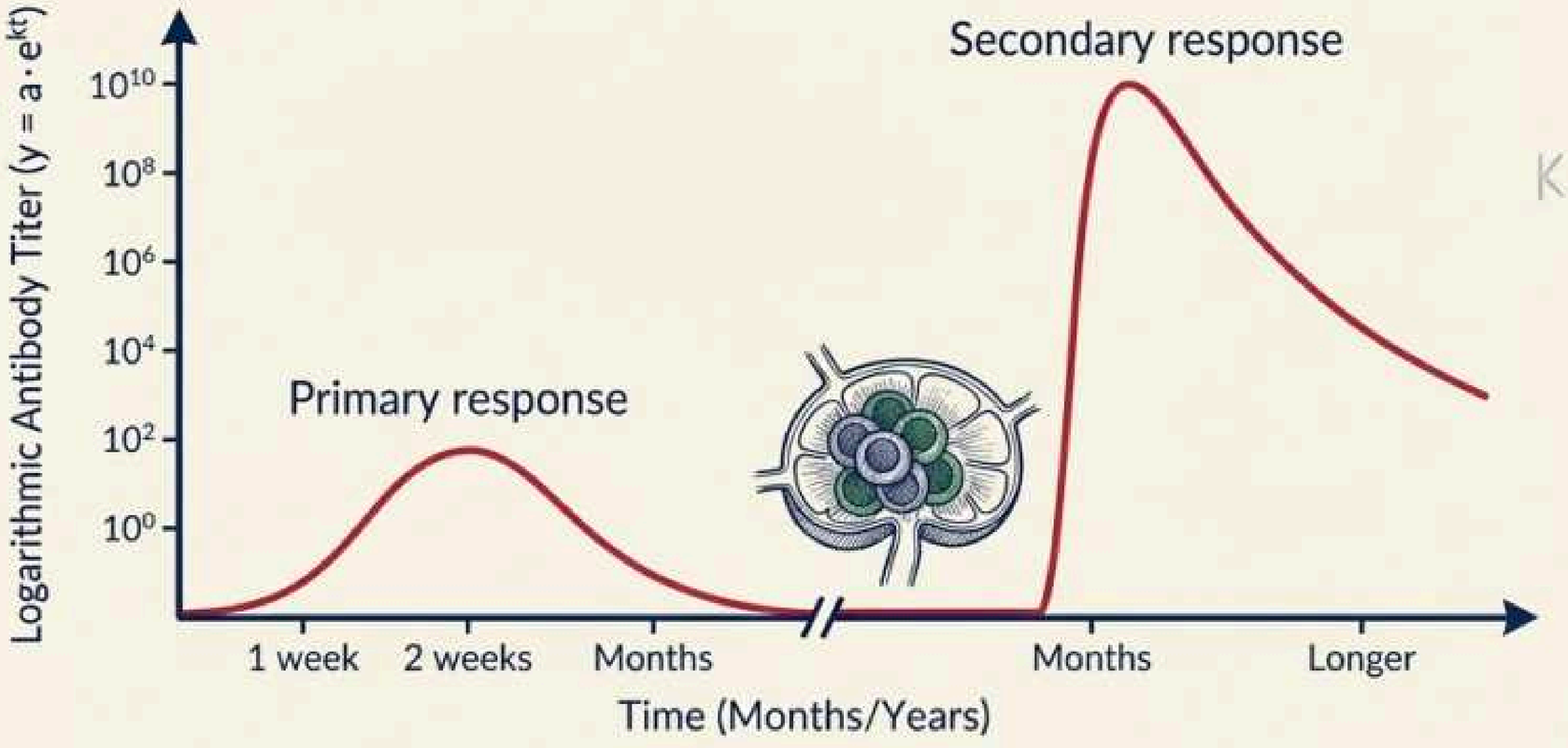
The Two Arms of Adaptive Defence

¶ 3.5

Antibodies (B Cell Lineage)	T Cells (Cell-Mediated)
 $K_a = \frac{[Ab \cdot Ag]}{[Ab][Ag]}$	
Mechanism: Circulating proteins that bind, block, mark for destruction, or enlist complement.	Mechanism: Act via direct cellular contact and signaling. They help other immune cells or actively kill host cells harboring viruses.
Target: Specialized for microbes living outside host cells.	Target: Specialized for microbes living inside host cells.

The two arms cooperate inextricably; a defect in either yields distinct, predictable vulnerabilities.

Immunological Memory and the Principle of Vaccination



Key Limits of Memory:

- It can fade over time.
- It can be evaded by pathogen surface changes.
- Efficacy varies significantly by pathogen.

Historical Context:

Explains why childhood diseases caused severe mortality in unexposed adults during the pre-vaccine era.

Successful Pathogens Deploy Specific Evasion Strategies

¶ 3.7

Hide

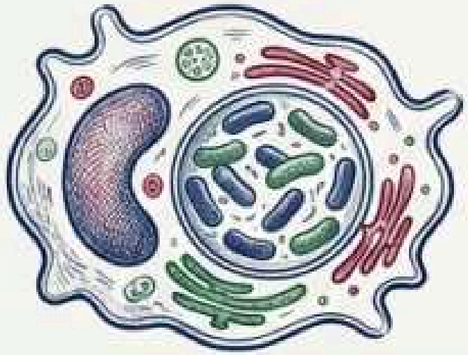


Illustration of bacteria safely multiplying inside a macrophage vacuole.

Cloak

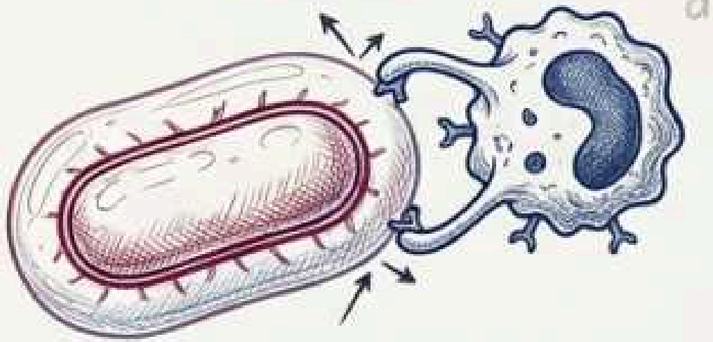
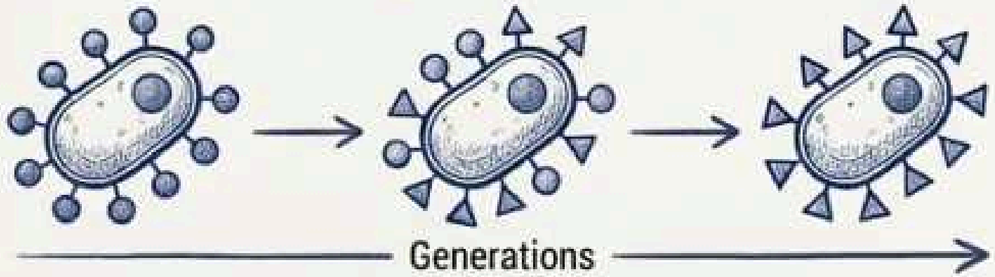


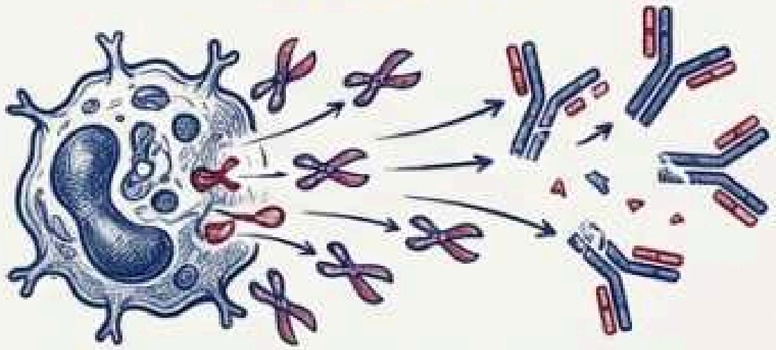
Illustration of a bacterium surrounded by a thick, slippery polysaccharide capsule, with a phagocyte's receptor shown visibly bouncing off.

Change
(Antigenic Variation)



Evolutionary timeline showing microbe surface proteins shifting shape across generations.

Interfere

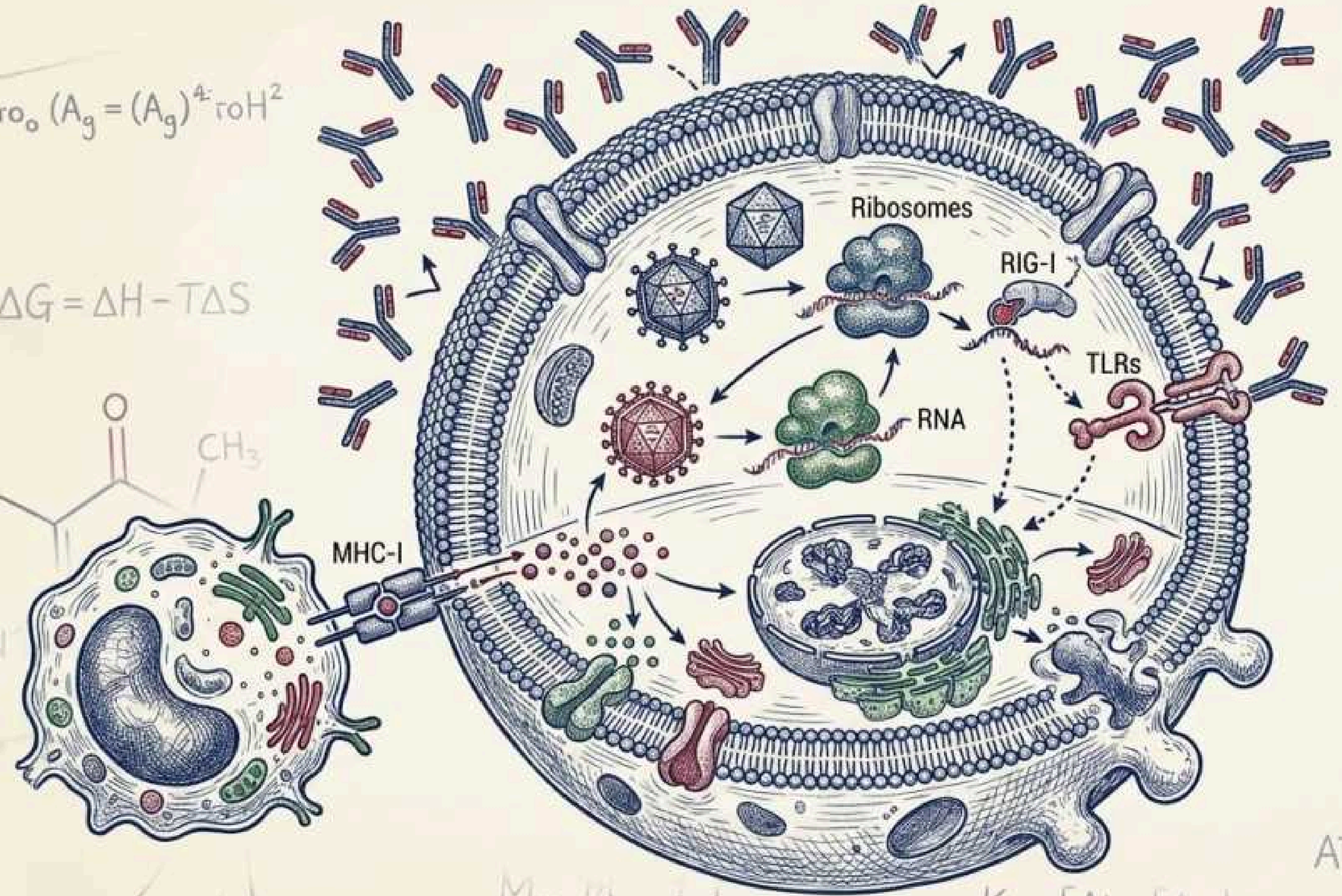


Pathogen releasing scissor-like enzymes actively cleaving and destroying Y-shaped antibodies.

The host does not always win. Textbooks categorize these as strategies, but in reality, pathogens are highly specialized survivors.

The Intracellular Lifestyle Forces the Host to Sacrifice Cells

¶ 3.8

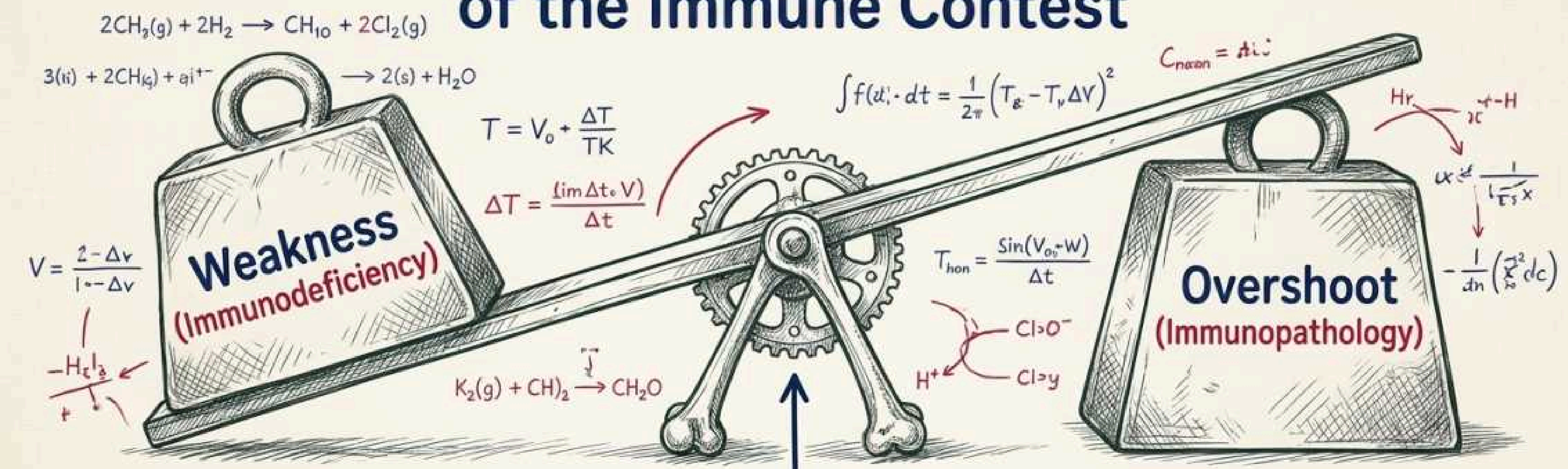


The Trade-Off:

- **Microbial Advantage**: Shielded from blood and surface defenses.
- **Microbial Cost**: Entirely dependent on the host cell's machinery and survival.

The Pathological Extremes of the Immune Contest

¶ 3.9



Inherited, malnutrition, drugs, HIV.

Result: Opportunists and ordinary pathogens cause unusually severe disease.

The Everyday Success

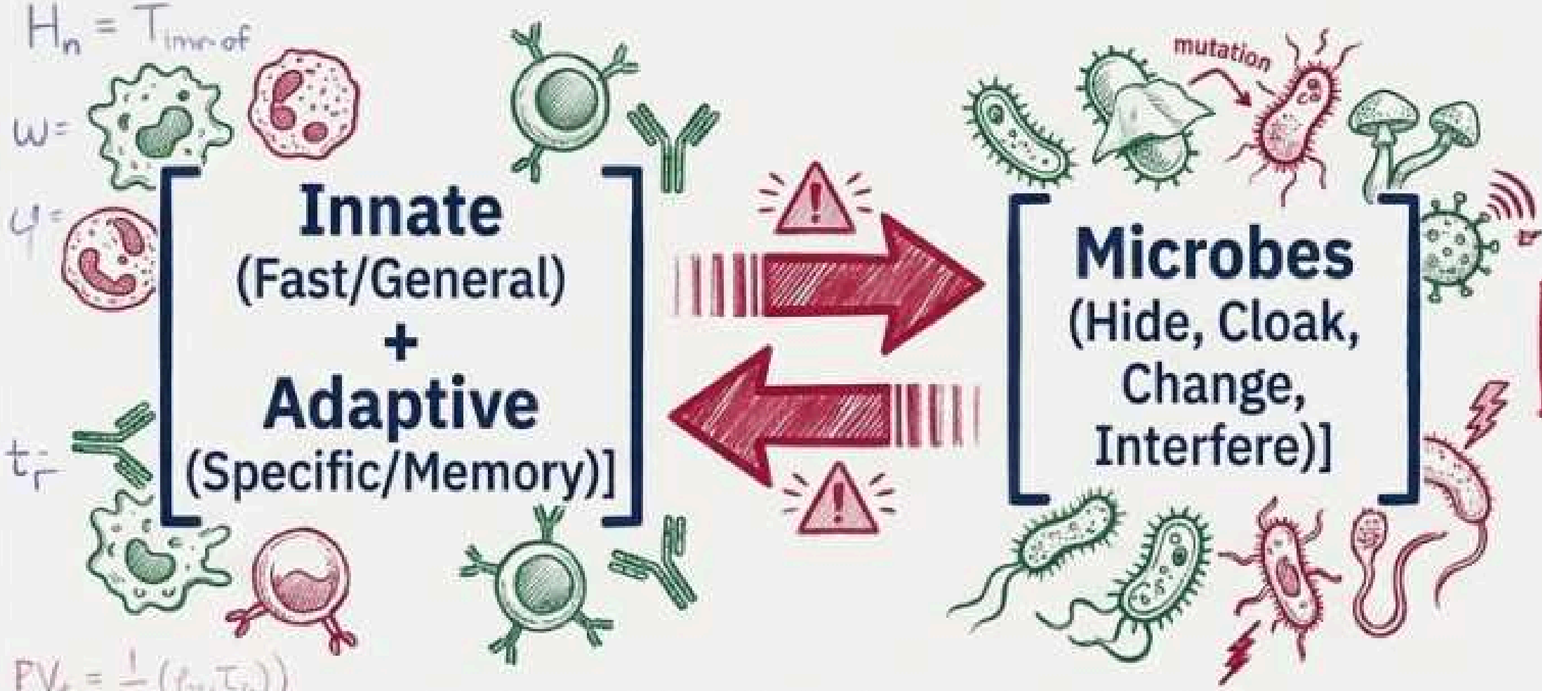
The clearance and containment of infections that happen continuously without the host ever noticing.

Response too strong, widespread, or off-target.

Result: Shock, chronic inflammation, direct tissue injury (often the worst moments of an infection).

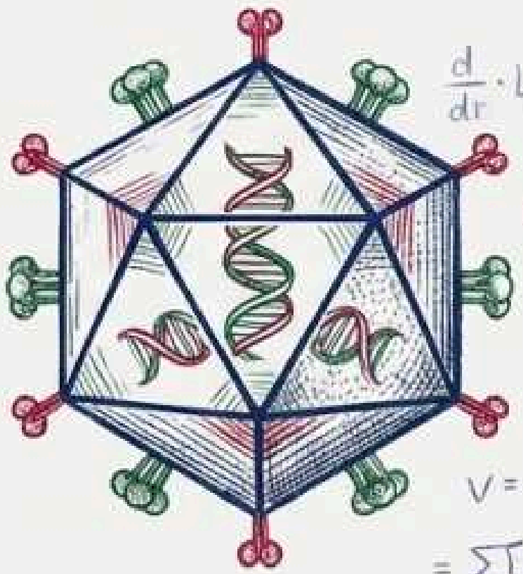
¶ 3.10

Synthesis of the Contest and Next Steps



System subject to Failure or Overshoot.

Up Next:
Lecture 4 - Viruses



Taking the intracellular lifestyle to its absolute limit.

Further Study:

- Grokipedia: Immune System, Innate & Adaptive
- Medicine 504 B (Brief Course in Immunology)