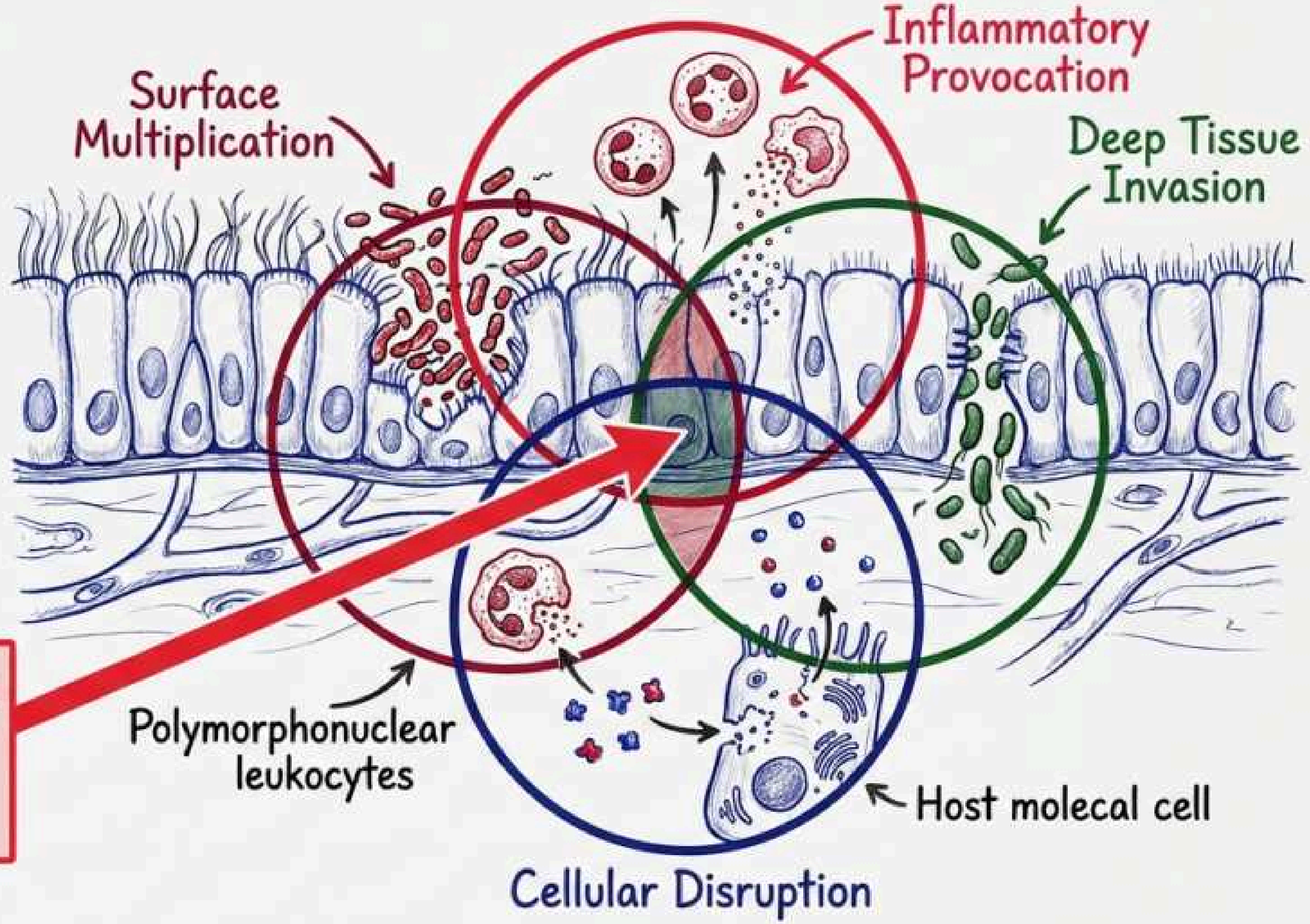
MED 512 B - Lecture 2: Bacterial Weapons

¶ 2.1

Pathogenic Harm
≠
f(Bacterial Count)

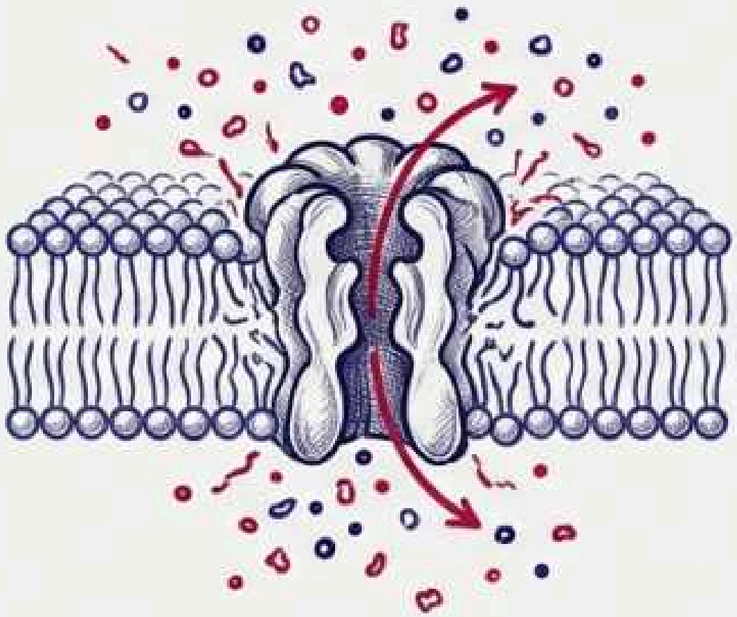


These paths of harm frequently overlap in a single illness.

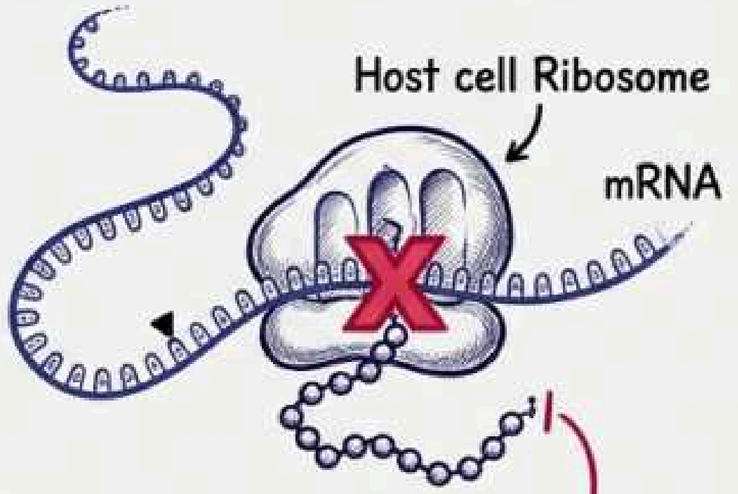
Exotoxins: Acting at a Distance

Exotoxin(protein) + Host Receptor → Cellular Internalization

Membrane Damage



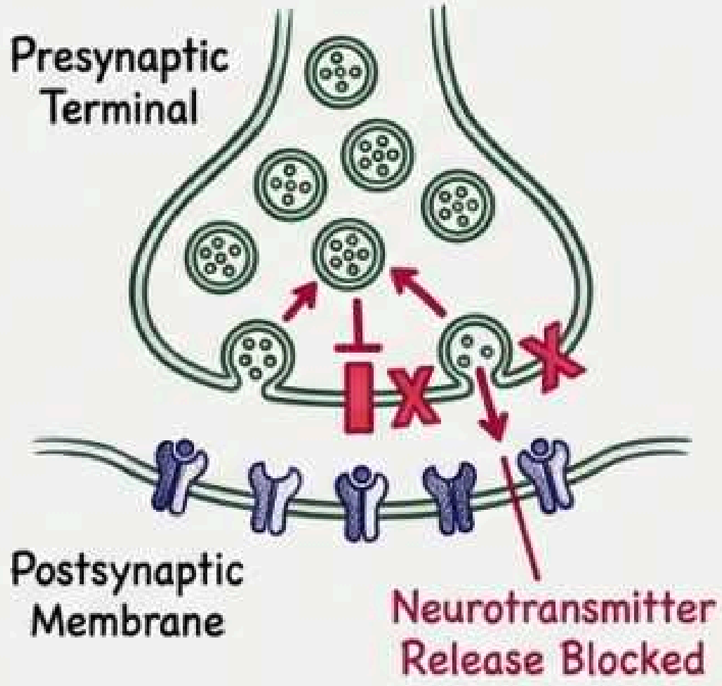
Internal Disruption



mRNA + Ribosome + Amino Acids → ‖

// PROTEIN SYNTHESIS HALTED

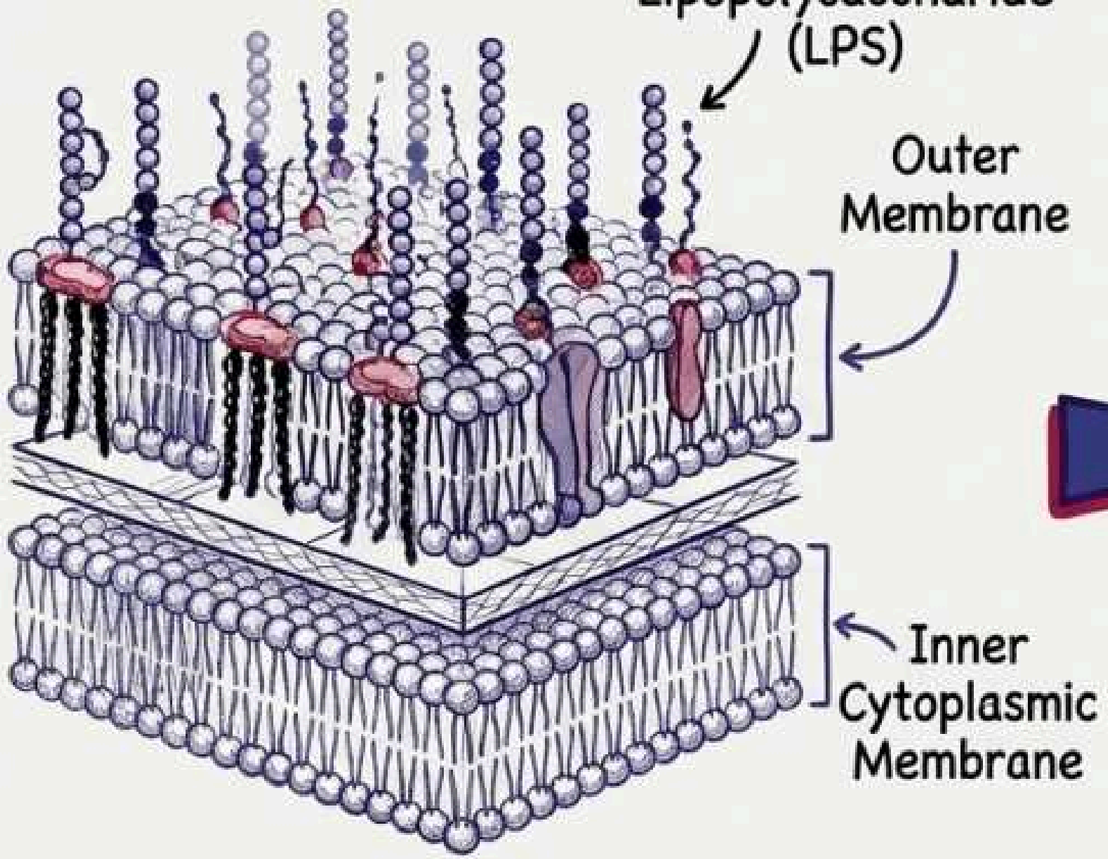
Nerve Interference

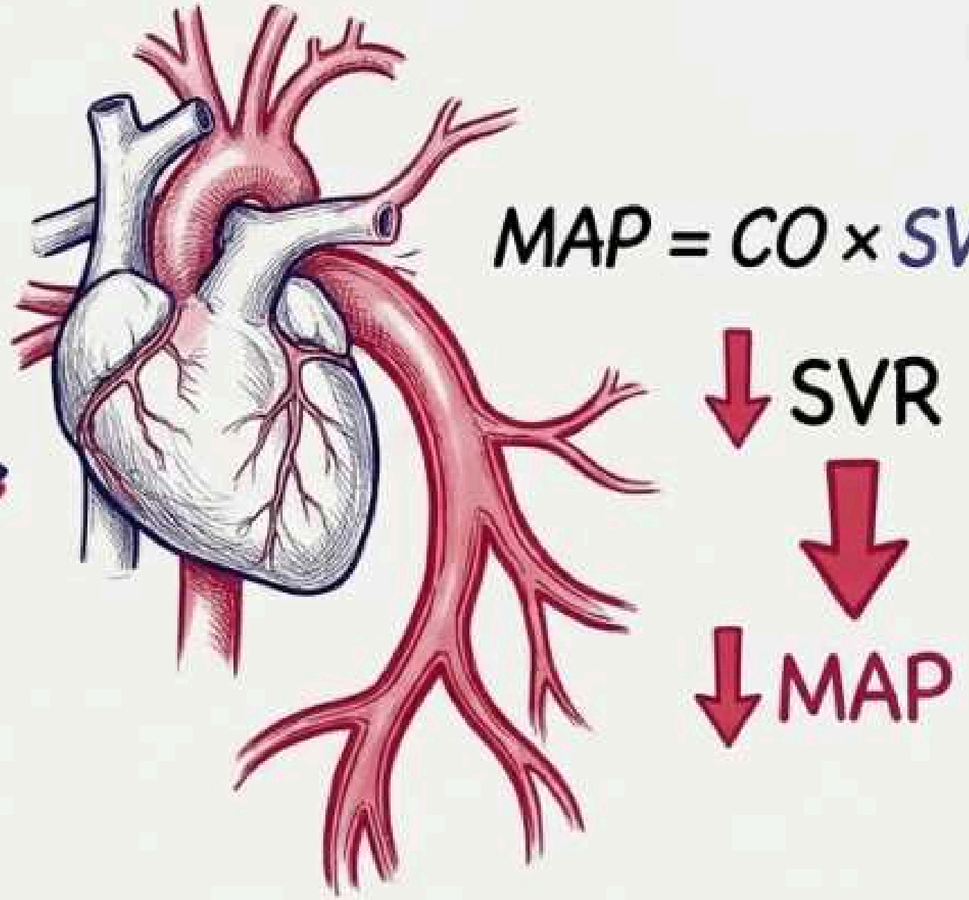


A microscopic amount of a bacterial product accounts for the most striking clinical features, acting far from the bacterial cell itself.

Endtotoxin: The Structural Danger Signal

¶ 2.3





$MAP = CO \times SVR$

↓ SVR

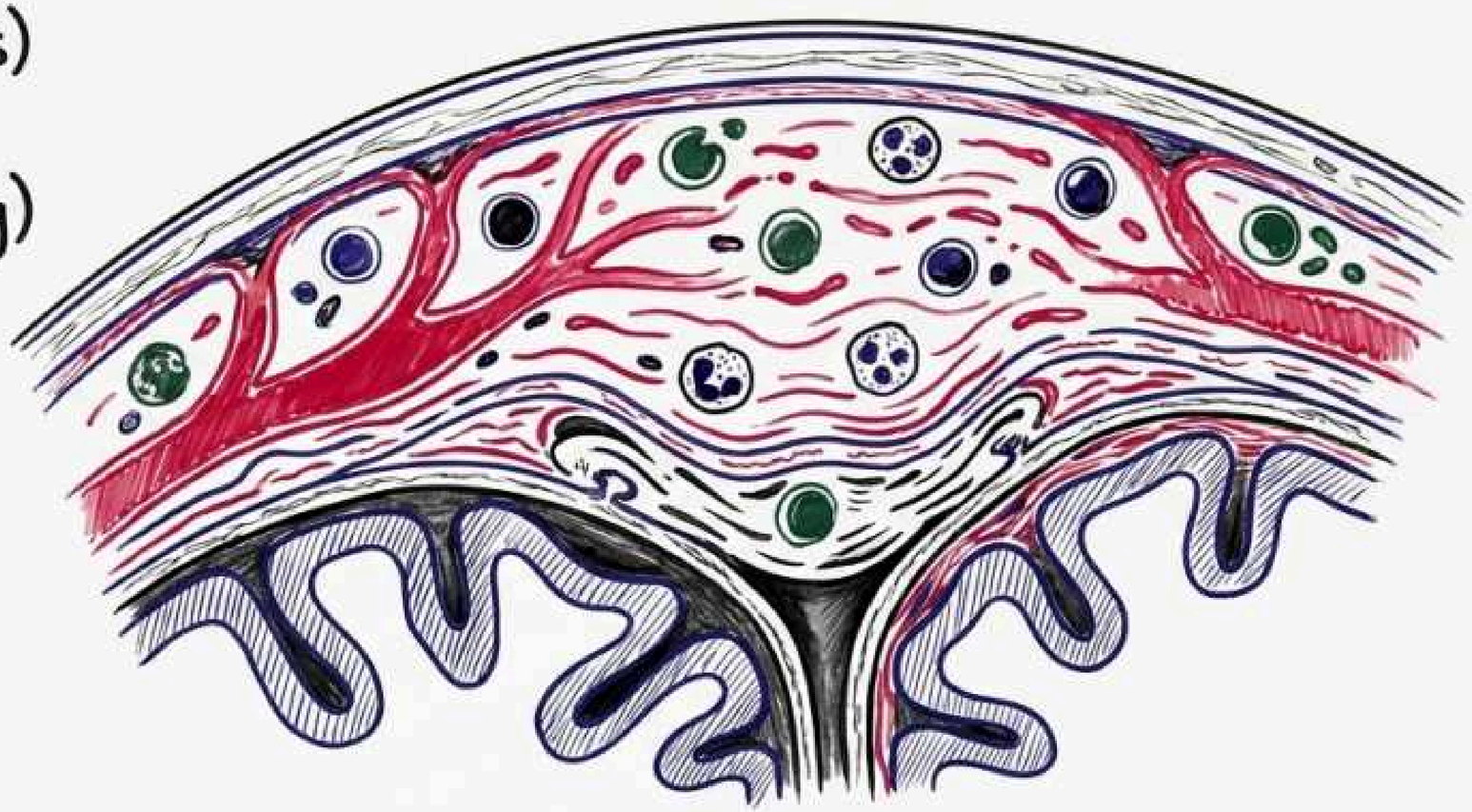
↓ MAP

Not a secreted protein. It is a structural component (LPS) that the immune system reads as a massive danger signal.

The clinical emergency is a spiraling host reaction, requiring urgent medical care.

Host Inflammation: The Double-Edged Sword

Rubor (Redness)
Calor (Heat)
Tumor (Swelling)
Dolor (Pain)

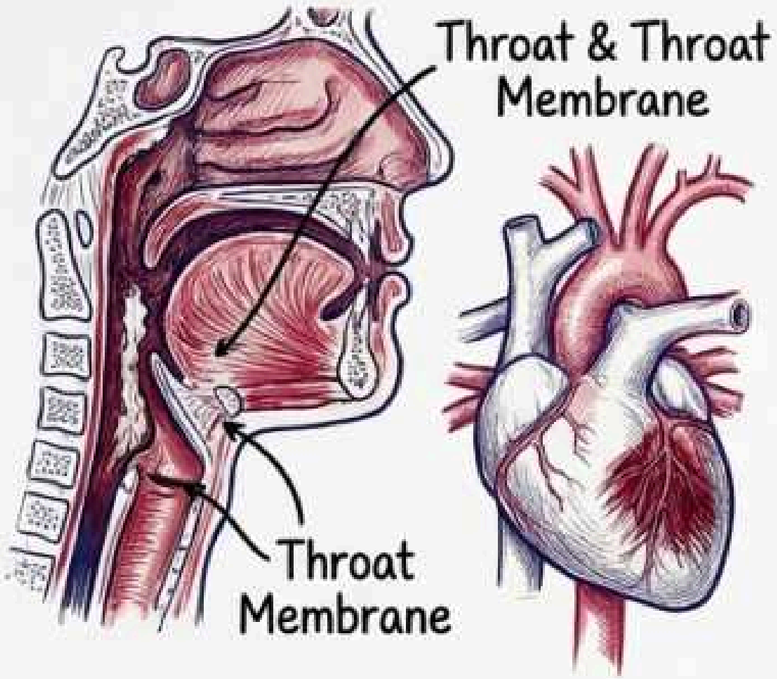


Is the clinical harm stemming from the bacterial product, local growth, or the fire of the host's own response?

Classic Toxin Diseases: Targets & Effects

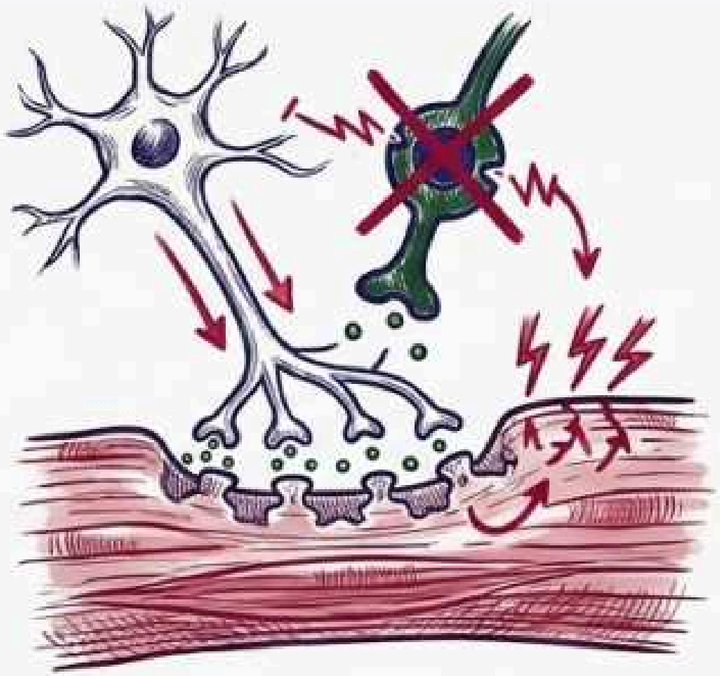
¶ 2.5

Diphtheria



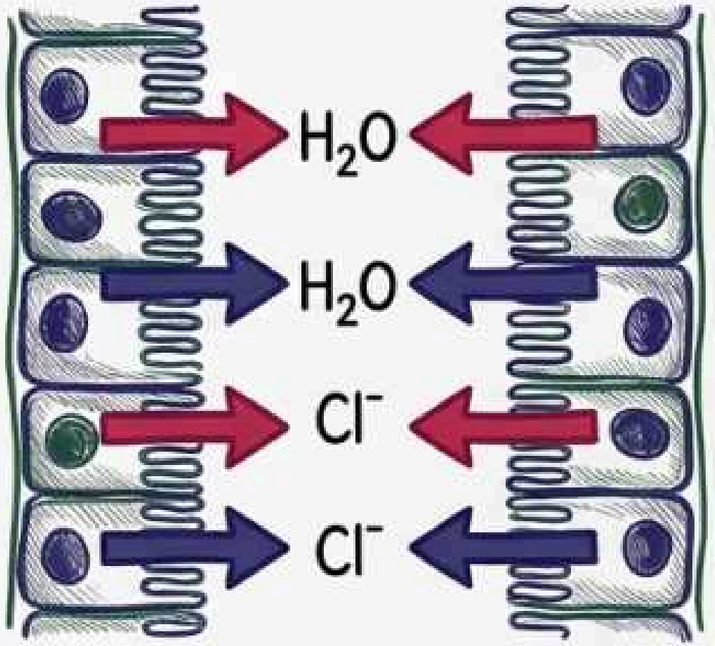
Throat membrane obstruction & cardiac damage

Tetanus



Inhibitory interneuron block → unyielding muscle spasms

Cholera



Massive watery fluid efflux → immediate dehydration danger

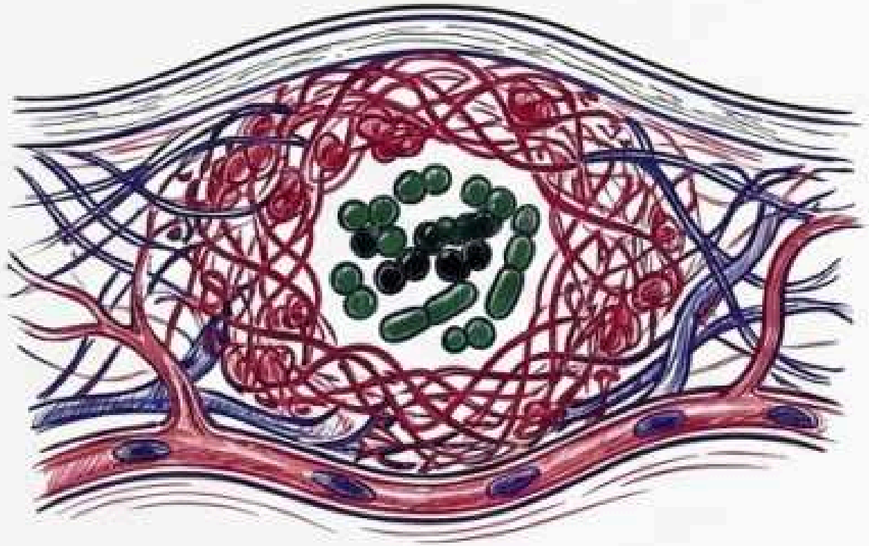
Sanitation, immunisation, and antitoxins have shifted these from ordinary terrors to clinical history.

Enzymes: Tools for Crossing Host Partitions

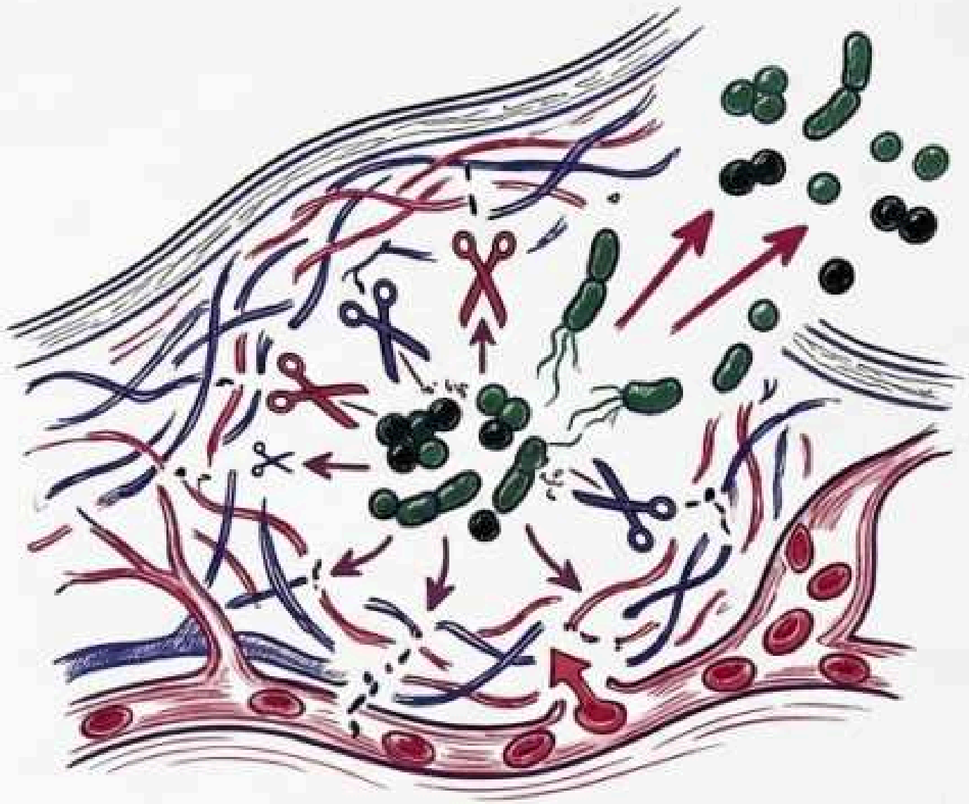
¶ 2.6

Enzymes ≠ Cellular Poisons (Toxins)

Enzymes = Spatial Spread Tools



Host Fibrosis / Abscess



Enzymatic Degradation & Spread

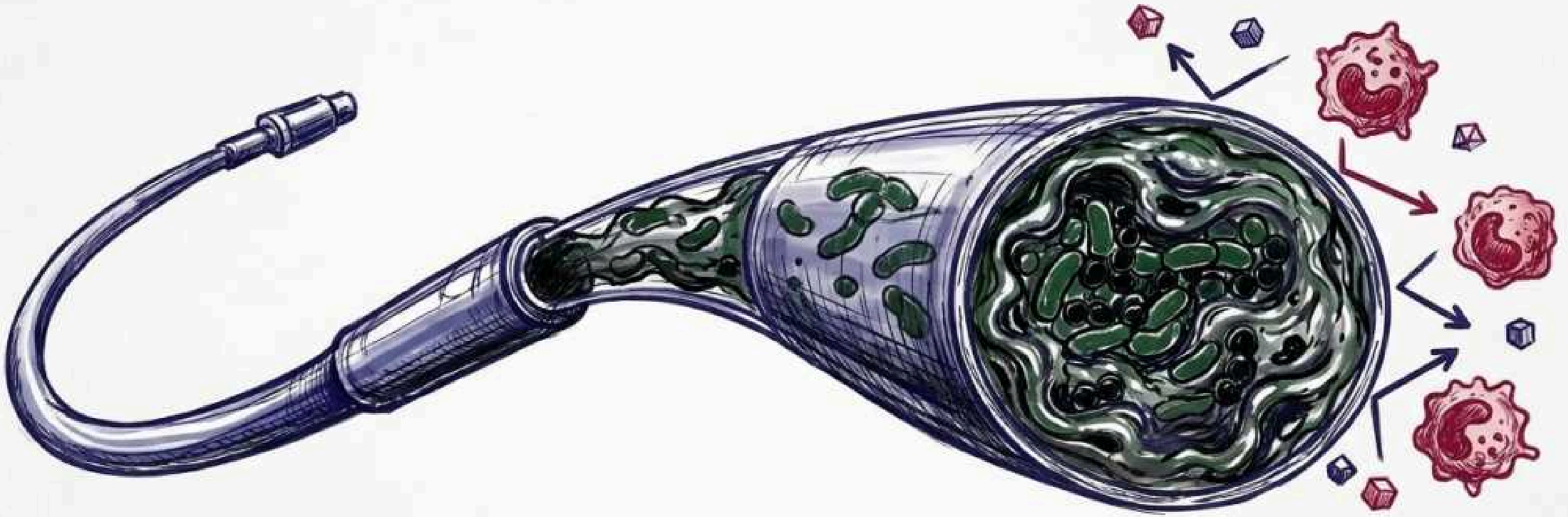
Disease is often the visible trace of a local struggle: the host attempts to wall off the infection, while bacterial enzymes break down those very partitions.

Biofilms: Protected Micro-Communities

¶ 2.7

Planktonic (Free-floating & Vulnerable)

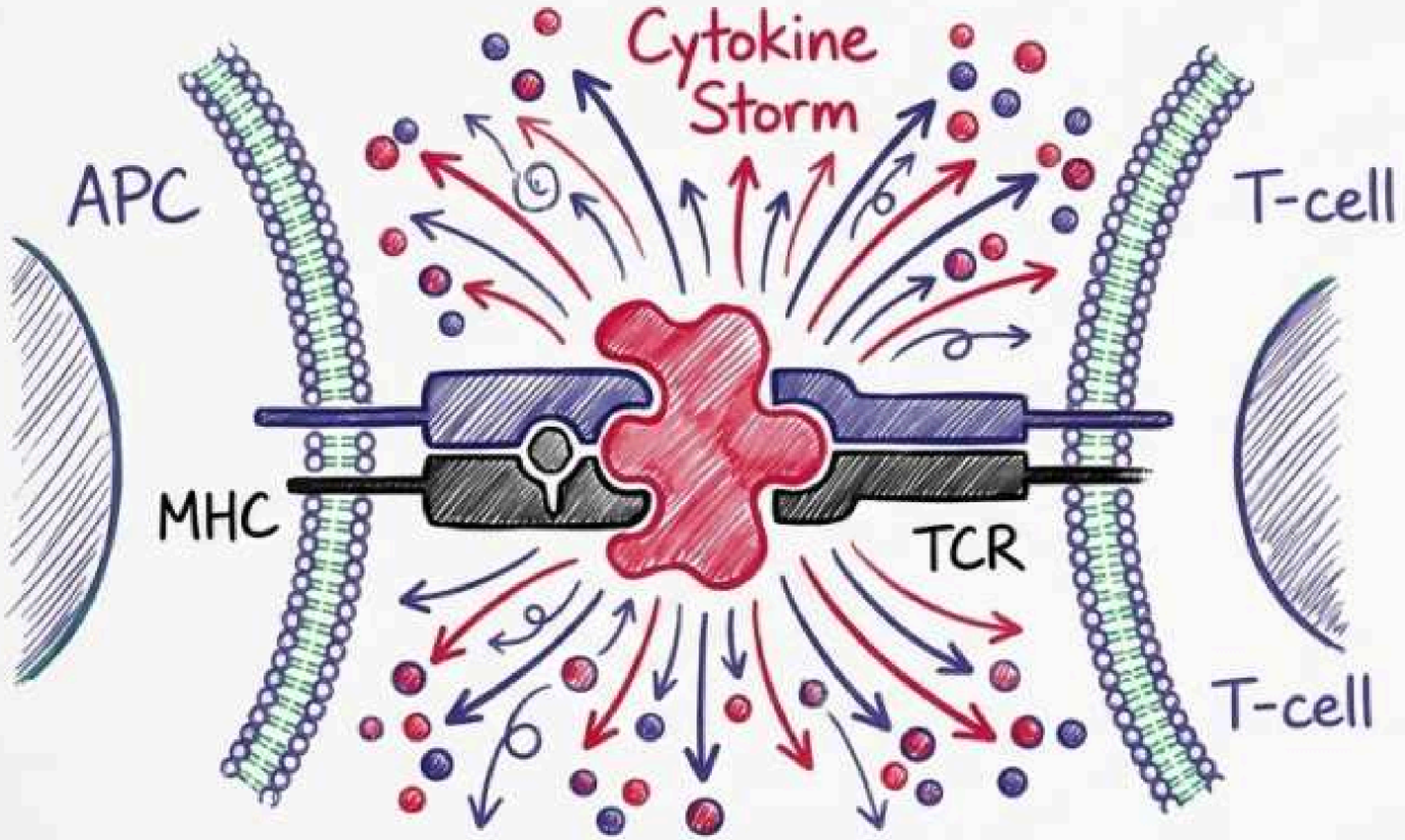
Biofilm (Embedded & Resistant)



Explains persistent infections on devices or in protected niches that complicate the simple picture of a single bacterium paired with a single drug.

Superantigens: Over-Stimulating Immunity

¶ 2.8



T_{cell} Activation:

Normal Antigen (<0.1%)

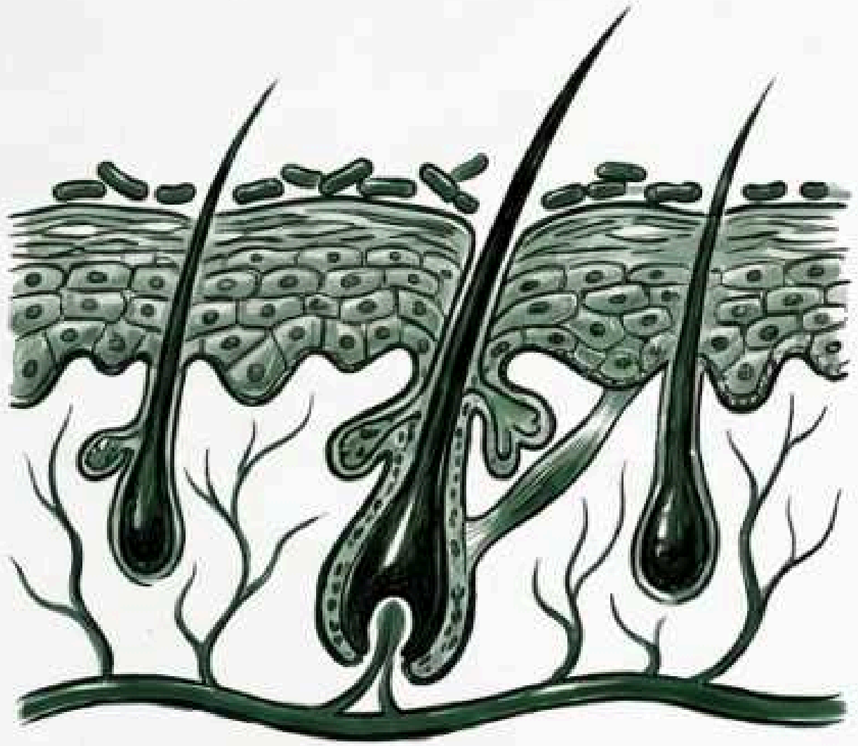
≪ Superantigen (~20%)

The recognition machinery is driven too hard, triggering a massive share of T-cells simultaneously and resulting in toxic shock.

The Spectrum of Presence: Colonizer vs. Pathogen

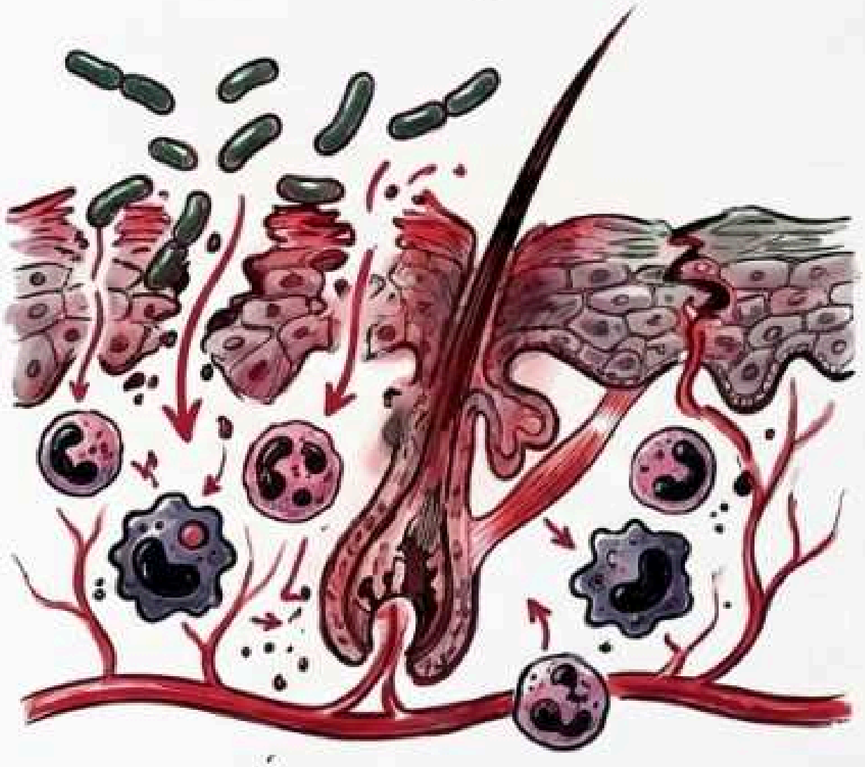
¶ 2.9

Quiet Colonizer



1. Toxin gene acquisition
2. Shift to a new anatomical site
3. Physical barrier breach
4. Changed host immunity

Pathogen

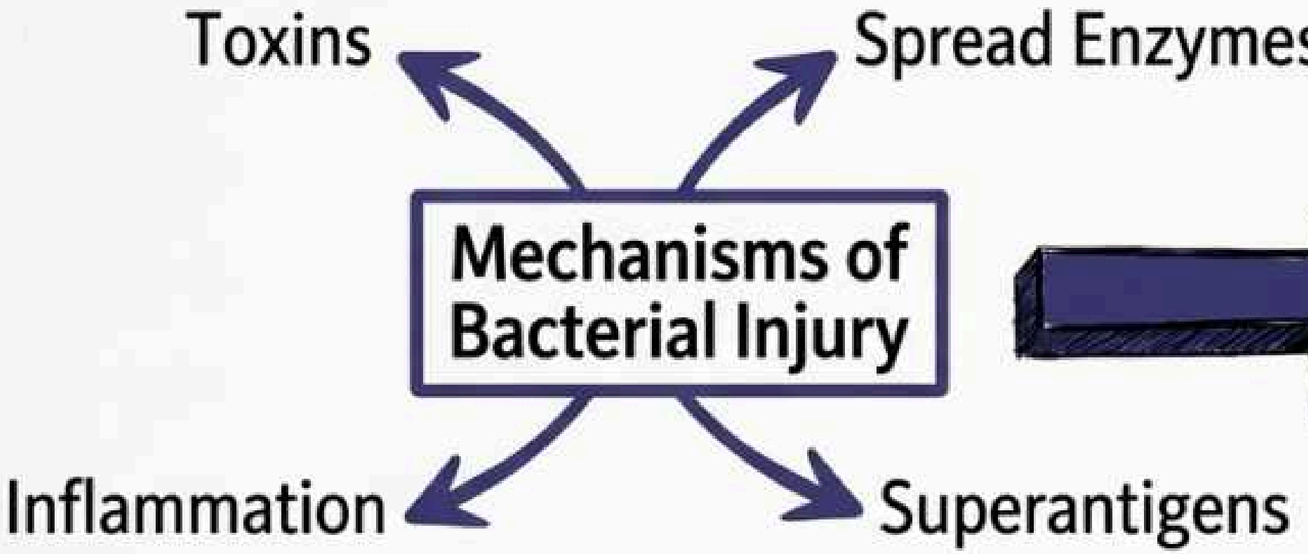


A positive laboratory result ≠ an automatic sentence of severe disease.

Strain differences are real. Host differences are real. A fair reading holds both in view.

Conclusion & The Next Contest

¶2.10



Lecture 3: Immune Defenses & Microbial Evasion



The contest is always two-sided. The same species can be colonizer or pathogen according to strain and circumstance.

Recommended Reading: Grokipedia (Pathogens, Robert Koch).