Host-pathogen relation

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Our relationship with microbes is very dynamic:

THERE IS A BALANCE BETWEEN:

The disease causing properties of the microbes <<--->> and the antimicrobial defenses of the host.

Microbes:
Aggressive mechanisms including virulence factors

Host defense mechanisms :
None –specific: skin, acid in stomach, and lysozyme in tears and mother milk.
Specific: antibodies and T cells.
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Infection(multiplication of an infectious agent within the body).

When parasitic microorganisms increase in number either within or on the body of the host.

Source of infection:

Exogenous infection: patient, carrier, diseased animal or animal carrier.

Endogenous infection: most are normal flora, cause infection under abnormal condition.

INFECTIOUS DISEASE is disease caused by pathogenic microorganisms.

Pathogen = disease causing agent

Disease = **abnormal state**, deviation from a state of wellness or health

Contamination means that microorganisms are present.

Pathogenicity - The ability of an organism to cause a disease.

50% Lethal dose(LD 50%) : is the number of microorganisms needed to kill half the hosts .

50% Infectious dose(ID 50%) : is the number of organisms required to cause infection in half the host .

Types of the microorganisms

Commensal : a microorganism can lives in a host without causing any disease .

Saprophyte : a microorganism which lives on decaying organic materials.

Pathogen : a microorganism capable of causing disease .

REVIEW SYMBIOSIS: MUTUALISM, PARASITISM, COMMENSALISM

- **Mutualism** is a symbiosis in which both members benefit from the relationship.

- **Parasitism** are those relationships in which one member benefits, and the other one is harmed in some way.

- **Commensalism** is a relationship in which one member benefits, and the other one neither benefits nor is harmed.

OPPORTUNISTS :

These are organisms that normally don't cause disease but will if given an opportunity:

- As in secondary infections

- If resistance is low: - ***Pneumocystis carinii*** - pneumonia in immunocompromised individuals

If they get into the wrong place. -

Ex: ***E. coli*** - Normally in the intestine, but if in the bladder or peritoneal - cavity --- will cause problems.

HOW TO DETERMINE IF AN ORGANISM IS THE ETIOLOGIC AGENT OF DISEASE

REVIEW KOCH'S POSTULATES:

1. The agent must be observed in every case of the disease.

2. The agent must be isolated from a diseased host and grown in pure culture.

3. When purified agent is inoculated into a healthy but susceptible host, it must cause the same disease.

4. The agent must be re isolated from the newly infected, diseased host, and be identical to the previously identified causative agent.

Virulence & Virulence factors

Virulence : The degree of pathogenicity

Virulence factors : Factors that are produced by a microorganism and help bacteria to:

(1) invade the host,

(2) cause disease,

(3) evade host defenses.

Bacterial Virulence factors

1-Adhesion factors

- Capsules, Fimbria , cell wall components

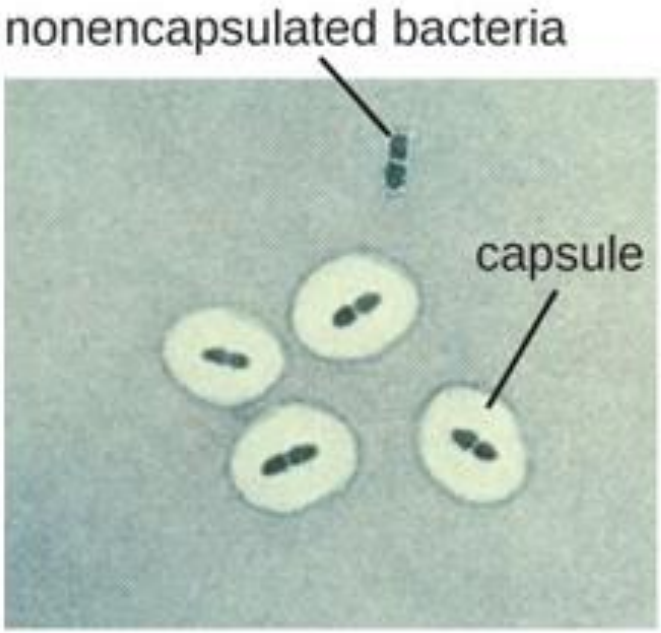
2- Enzymes

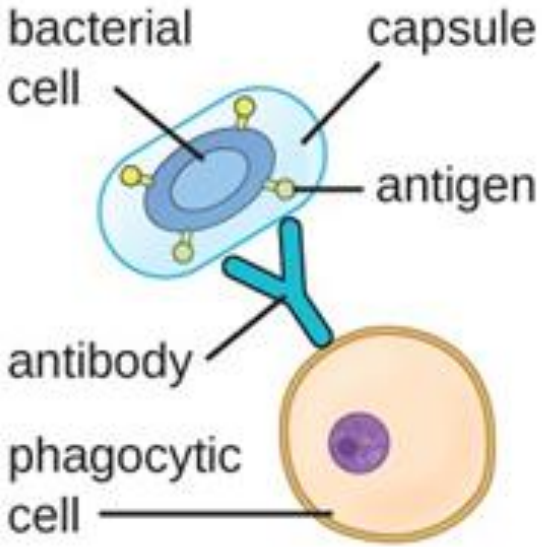
3- Toxins

1-Adhesion factors

1- CAPSULE -- HELPS TO ATTACH BUT ALSO IMPEDES PHAGOCYTOSIS.

ex.: *Streptococcus pneumoniae*, *Klebsiella pneumoniae*,

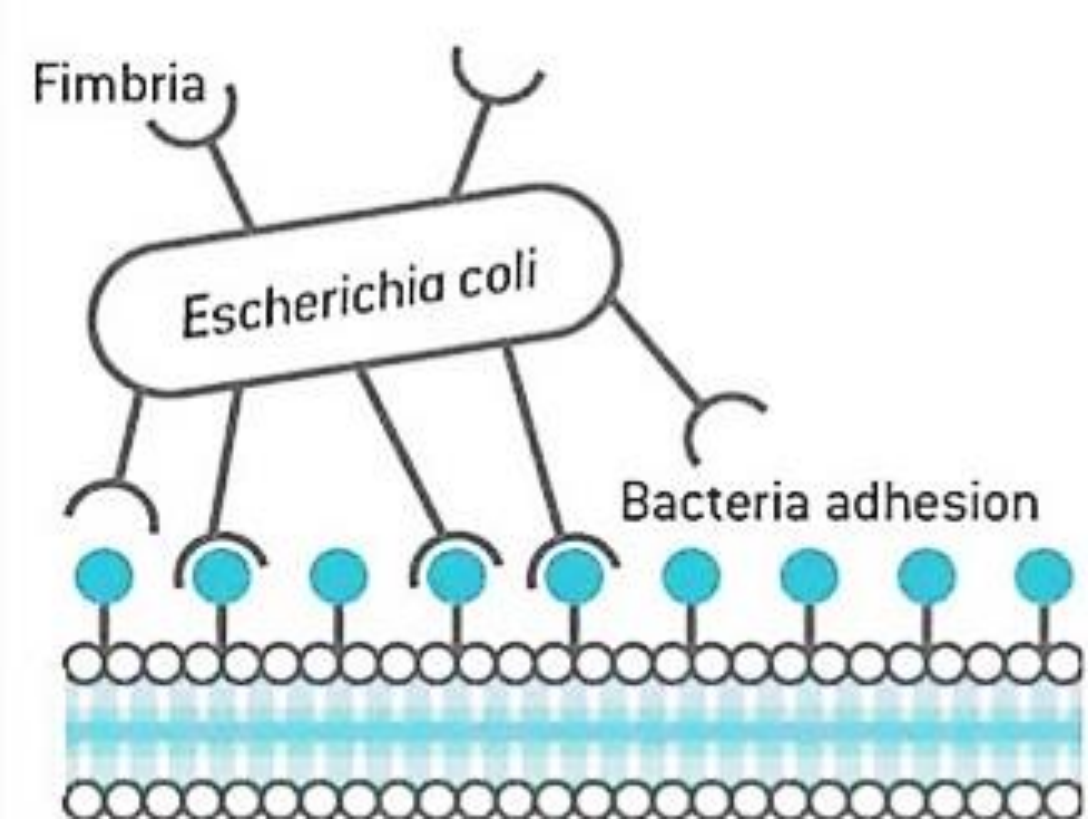




(a) A micrograph of capsules around bacterial cells. (b) Antibodies normally function by binding to antigens, molecules on the surface of pathogenic bacteria. Phagocytes then bind to the antibody, initiating phagocytosis..

2- **FIMBRIAE (PILI)** -- ARE OFTEN TISSUE SPECIFIC.

ex.: *E. coli* strain differences in tissue specificity due to different types of fimbriae
- urinary tract strains differ from enteropathogenic strains in the type of pili they make.



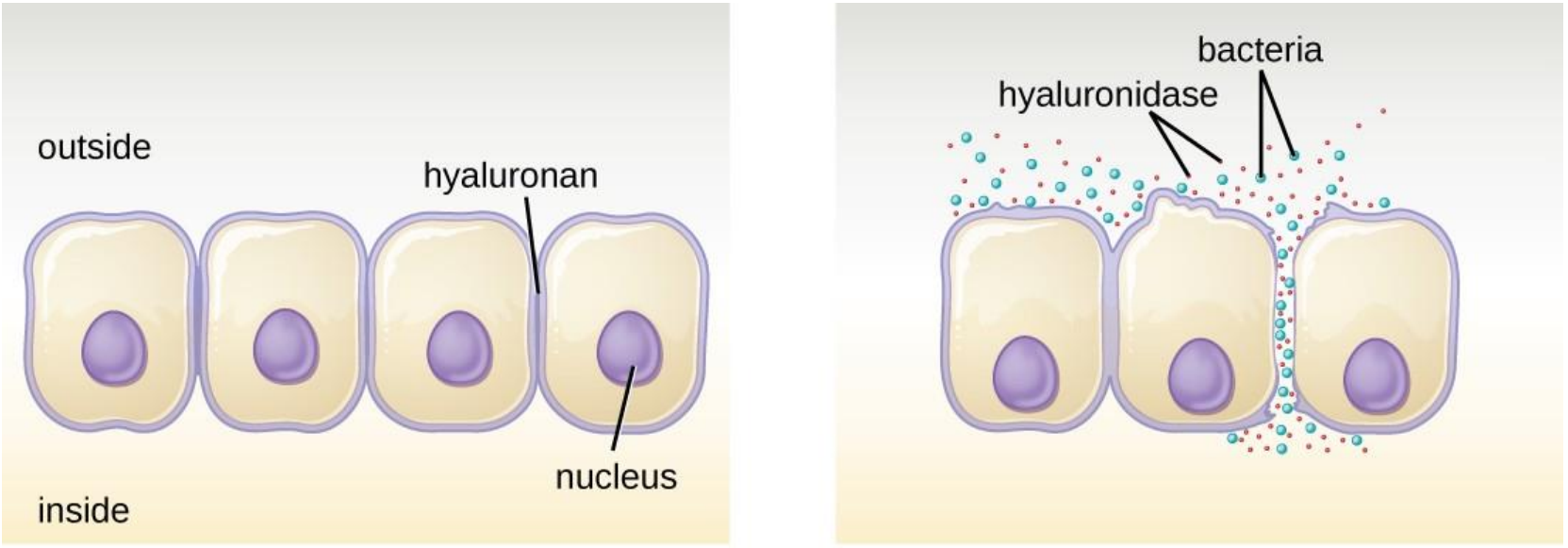
3- THE **CELL WALLS** OF SOME BACTERIA HAVE VIRULENCE PROMOTING PROPERTIES

ex.: *Streptococcus pyogenes* - **protein G** binds to the back end of antibodies preventing their normal function

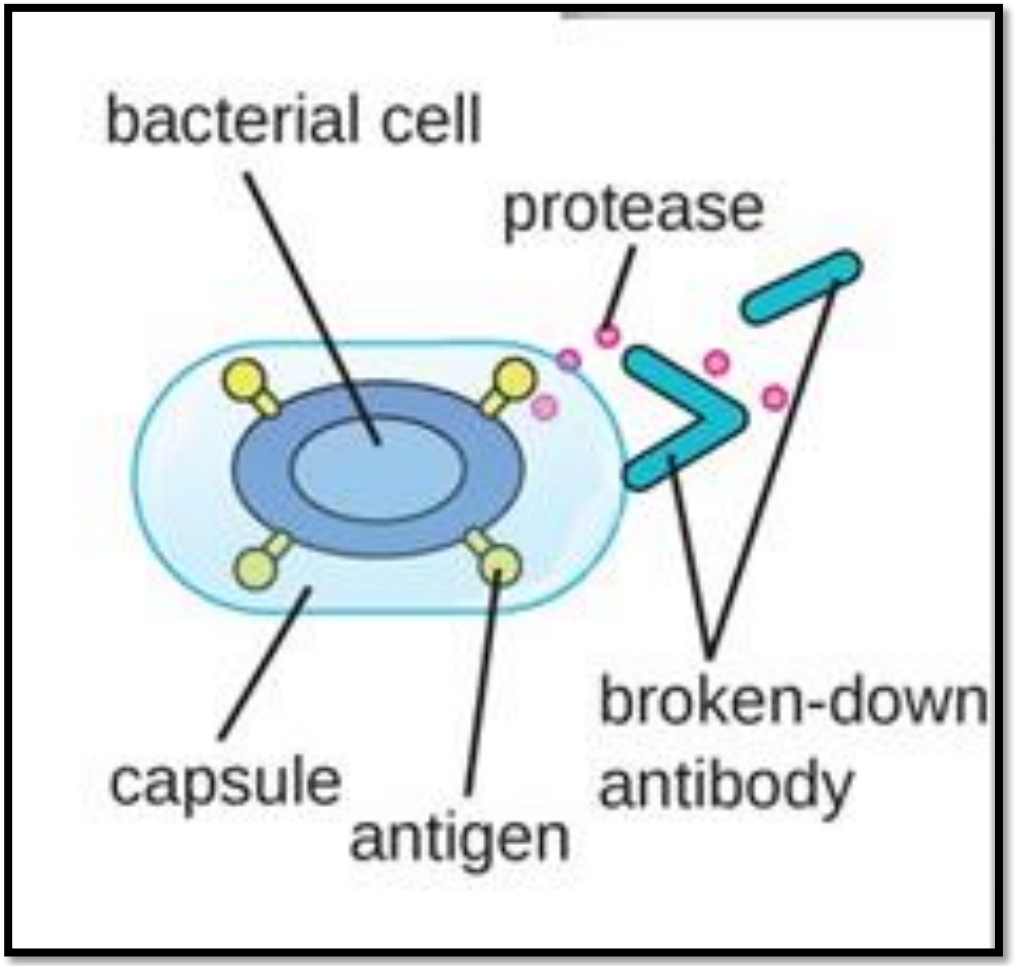
Staphylococcus aureus has **protein A** - which works the same way.

2-BACTERIAL ENZYMES

- Leukocidins -- destroy phagocytic leukocytes, which then release their own digestive enzymes onto tissues. *S. aureus* and *S. pyogenes*.
- Hemolysins -- destroy RBC'S -- *S. aureus*, *S. pyogenes* and *C. perfringes*
- Coagulase -- makes a fibrin clot around the organism. -- *S. aureus*
- Bacteria kinases -- digest fibrin clots –
- Hyaluronidase -- dissolves hyaluronic acid
- Collagenase (gelatinase) -- dissolves collagen -- *C. perfringes*
- Siderophores -- scavenge iron
- Protease ----acts on Immunoglobulins (IgA)



(a) Hyaluronan is a polymer found in the layers of epidermis that connect adjacent cells. (b) Hyaluronidase produced by bacteria degrades this adhesive polymer in the extracellular matrix, allowing passage between cells that would otherwise be blocked.



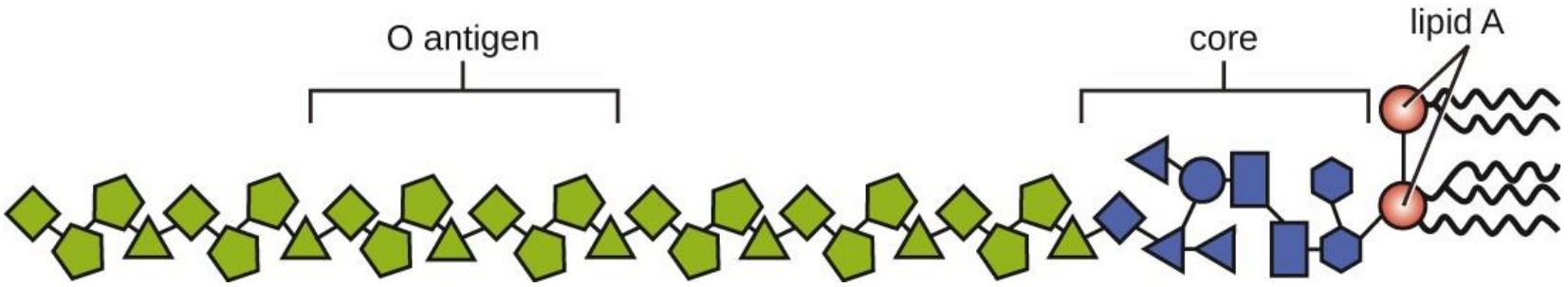
Some bacteria produce proteases, virulence factors that break down host antibodies to evade phagocytosis

BACTERIAL TOXINS

The ability of a pathogen to produce toxins to cause damage to host cells is called **toxigenicity**.

Toxins can be categorized as **endotoxins** or **exotoxins.** The lipopolysaccharide (LPS) found on the outer membrane of gram-negative bacteria is called **endotoxin**

The lipid component of endotoxin, lipid A, is responsible for the toxic properties of the LPS molecule.



Lipopolysaccharide is composed of lipid A, a core glycolipid, and an O-specific polysaccharide side chain. Lipid A is the toxic component that promotes inflammation and fever.

Exotoxins

Can be classified as **CYTOTOXINS, NEUROTOXINS, ENTEROTOXINS**

Erythrogenic toxin , Tetanus toxin , *S. aureus* enterotoxin

Unlike the toxic lipid A of endotoxin, **exotoxins** are protein molecules that are produced by a wide variety of living pathogenic bacteria. Although some gram-negative pathogens produce exotoxins, the majority are produced by gram- positive pathogens.

Comparison of Endotoxin and Exotoxins Produced by Bacteria

PROPERTY	EXOTOXINS	ENDOTOXINS
Source	G+ and G- bacteria	G- bacteria
Secreted from bacterial cell	yes	no
Chemistry	polypeptides	Lipopolysaccharides
Location of genes	On plasmid or Bacteriophage	chromosome
Toxicity	High (1)µ g	low(10-100) mg
Antigenicity	induce high of antibodies	poorly antigen
Vaccines	Convert to toxoid (vaccine)	Not convert to toxoid (no vaccine)
Heat stability	destroyed at 60 C°	Stable at 100C° For 1 hr.

MODE OF TRANSMISSION

1. **CONTACT TRANSMISSION:**
   a) Direct contact -- Touch, kiss, sex, animals
   b) Indirect: **-** -- doorknobs, towels, phones, needles and other medical equipment.

2. **VEHICLE TRANSMISSION:**
   a) Waterborne -- **oral-fecal transmission**
   b) Foodborne -- unproperly cooked, fecal contamination
   c) Airborne -- droplet nuclei in dust -- must travel more than 1 meter.

3. **Vector** -- and infected animal (usually an insect) that transmits to humans

SPREAD OF INFECTION IN A POPULATION

RESERVOIR - Can think of the reservoir as the source of the organism

1) **Human source**

2) **Animal source**

3) **Non-living source:**

- **Soil**
- **Water**

Stages of bacterial infection:

1. Transmission from external source,t here are four important portal of entry (respiratory tract , gastrointestinal tract, genital tract & skin).
2. Evasion of primary host defense(The process whereby bacteria, animal parasites, fungi, and viruses enter host cells or tissues and spread in the body).
3. Adherence to cells.
4. Colonization: site of adherence.
5. Symptoms of disease :by toxins ,or invasion & inflammation.
6. Host response: specific & non-specific immunity.
7. Progression or resolution of the disease.

DISEASE PRESENTATION:

a) Symptoms –Commonly felt by the patient.

b) Signs – Commonly observed by a physician.

c) Syndrome – a combination of both symptoms and signs.

The development of disease

1. The incubation period - initial infection and the first appearance of signs or symptoms.

2. The prodromal period -short duration. Period of initial mild signs or symptoms.

3. The period of illness -period of maximum presentation of signs and symptoms.

4. The period of decline -signs and symptoms start to decline.

5. The period of convalescence - regain strength

Sepsis: The presence of pathogens & their toxins in tissue or blood.

Bacteremia: The presence of visible bacteria in circulating blood.

Toxemia: The presence of microbial toxins in blood.

Endotoxemia : is the presence of endotoxins in the blood.

Septicemia : illness that occurs when poisonous substances (toxins) produced by certain bacteria enter the bloodstream.

Pyemia : is caused by pyogenic microorganisms in the blood.

Pyogenic infection refers to bacterial infection that leads to the production of pus.

Diseases in a population:

Sporadic disease: outbreak occurs in population occasionally and at irregular intervals eg, many gastrointestinal diseases from contaminated food(Typhoid fever).

Endemic disease- describes a disease that is present permanently in a region or population(example: Malaria is an infectious disease that is endemic to Africa).

Epidemic disease - describe a situation where a disease spreads rapidly to a large number of people in a given population over a short time period(examples: smallpox, measles).

Pandemic disease - is the term used to describe an epidemic when the spread is global (eg, worldwide Covid-19).

Thanks