MECHANISMS OF BACTERIAL PATHOGENICITY

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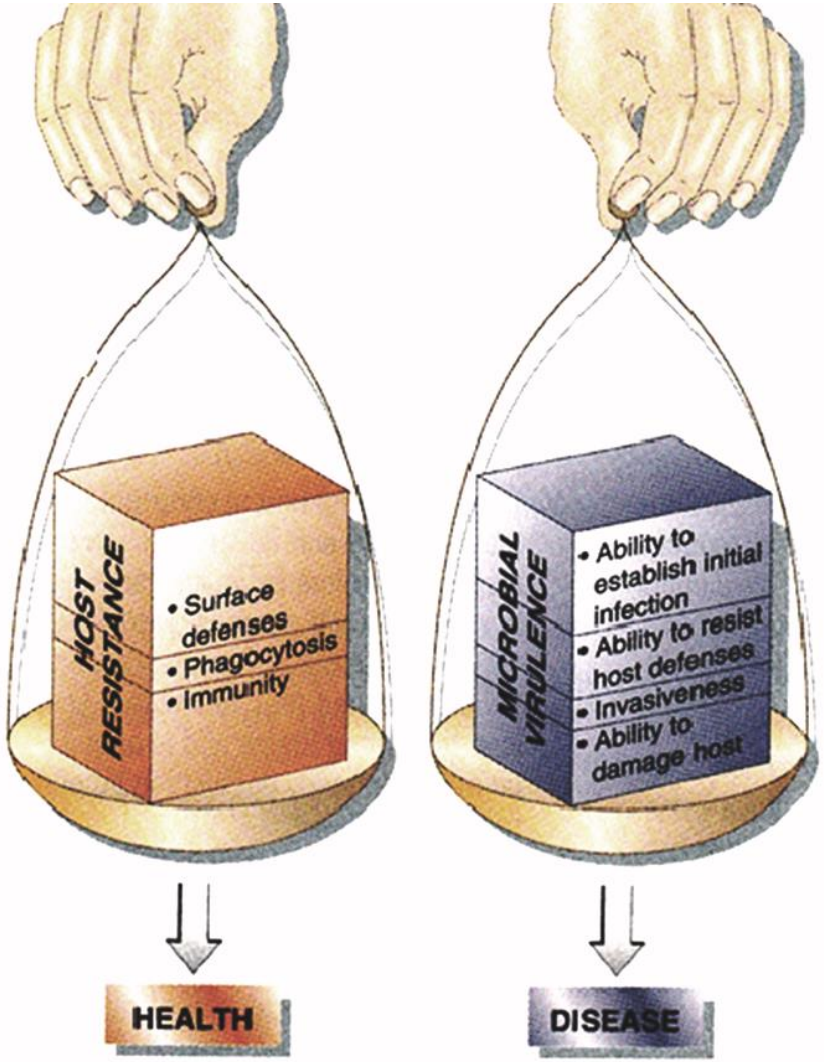


- **Disease**
  - Any deviation from a condition of good health and well-being
- **Infectious Disease**
  - A disease condition caused by the presence or growth of infectious microorganisms

Many properties that determine a microbe's pathogenicity or virulence are unclear or unknown

But, when a microbe overpowers the hosts defenses, infectious disease results!

INTRODUCTION

- The relationship between a host and a pathogen is dynamic, since each modifies the activities and functions of the other
- The outcome of such a relationship depends on:
  - the virulence of the pathogen and
  - the relative degree of resistance or susceptibility of the host, mainly due to the effectiveness of the host defense mechanisms

Pathogenicity and Virulence

Pathogenicity

- The ability of a microbe to cause disease
  - This term is often used to describe or compare species

Virulence

- The degree of pathogenicity in a microorganism
  - This term is often used to describe or compare strains within a species

Pathogenesis is a multi-factorial process which depends on the;

- immune status of the host,
- the nature of the species or strain (virulence factors) and
- the number of organisms in the initial exposure

Pathogenicity

Determinants of pathogenesis

- Transmission
- Adhesion
- Invasion and inflammation
- Toxin production
- Immunopathogenesis

Bacterial pathogenesis

- Infection/entry
- Virulence factors
- Pathogenesis
- Escape of immune surveillance

The pathogenesis of bacterial infection includes initiation of the infectious process and the mechanisms that lead to the development of signs and symptoms of disease

Bacterial Virulence Mechanisms

- Adherence
- Invasion
- Byproducts of growth (gas, acid)
- Toxins
- Degradative enzymes
- Cytotoxic proteins
- Endotoxin
- Superantigen
- Induction of excess inflammation
- Evasion of phagocytic and immune clearance
- Capsule
- Resistance to antibiotics
- Intracellular growth

INNATE IMMUNE SYSTEM

Lysozyme in tears kills Gram-positive bacteria

Removal of particles by turbinates and humidification

Mucus and cilia capture organisms and remove them

Skin: physical barrier

Stomach acid kills ingested pathogens

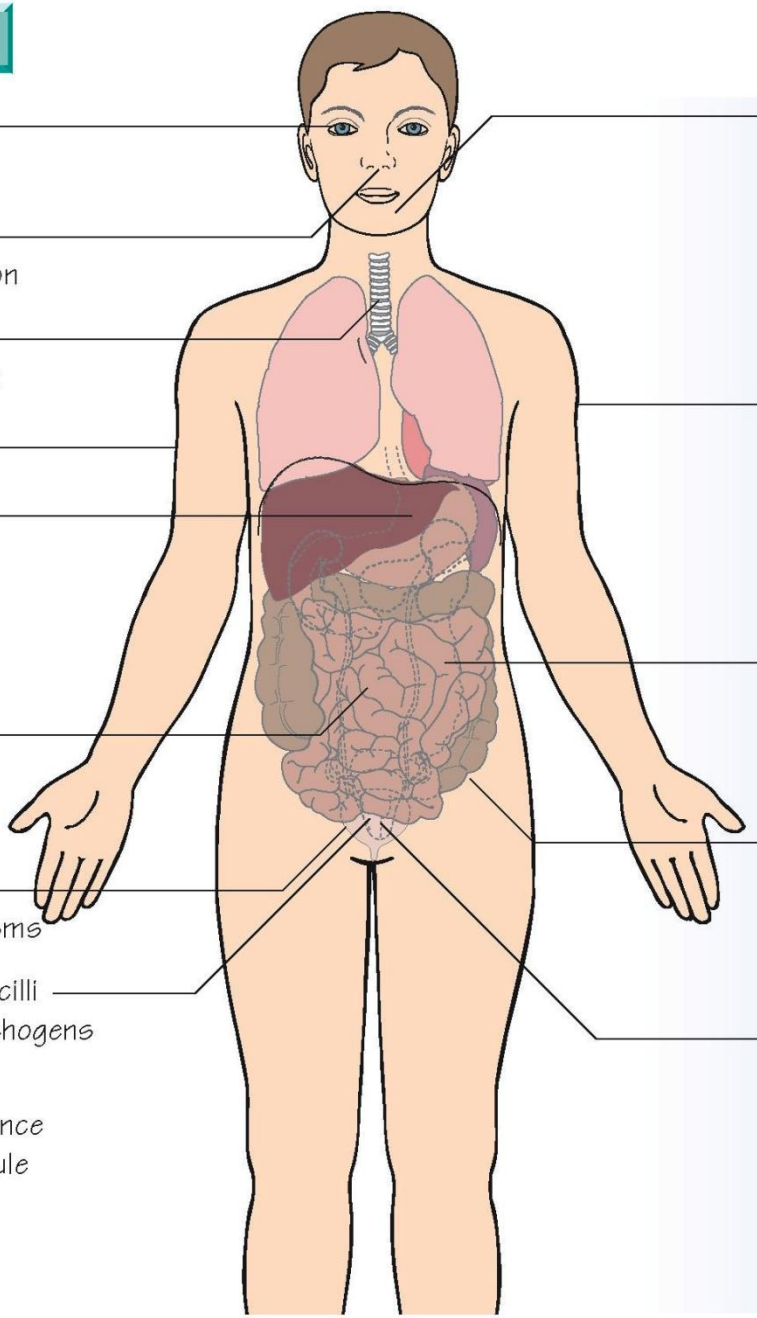
Fatty acids inhibit growth of many bacteria

Competition and toxic products from intestinal flora

Flushing action of urinary flow removes organisms

Low vaginal pH from lactobacilli prevents colonization by pathogens

Whole body:
- Molecular and cellular defence
- Pattern recognition molecule e.g. TLRs
- Neutrophils
- Macrophages



NORMAL FLORA

NASOPHARYNX
- Streptococci
- *Haemophilus*
- *Neisseria*
- Mixed anaerobes
- *Candida*
- *Actinomyces*

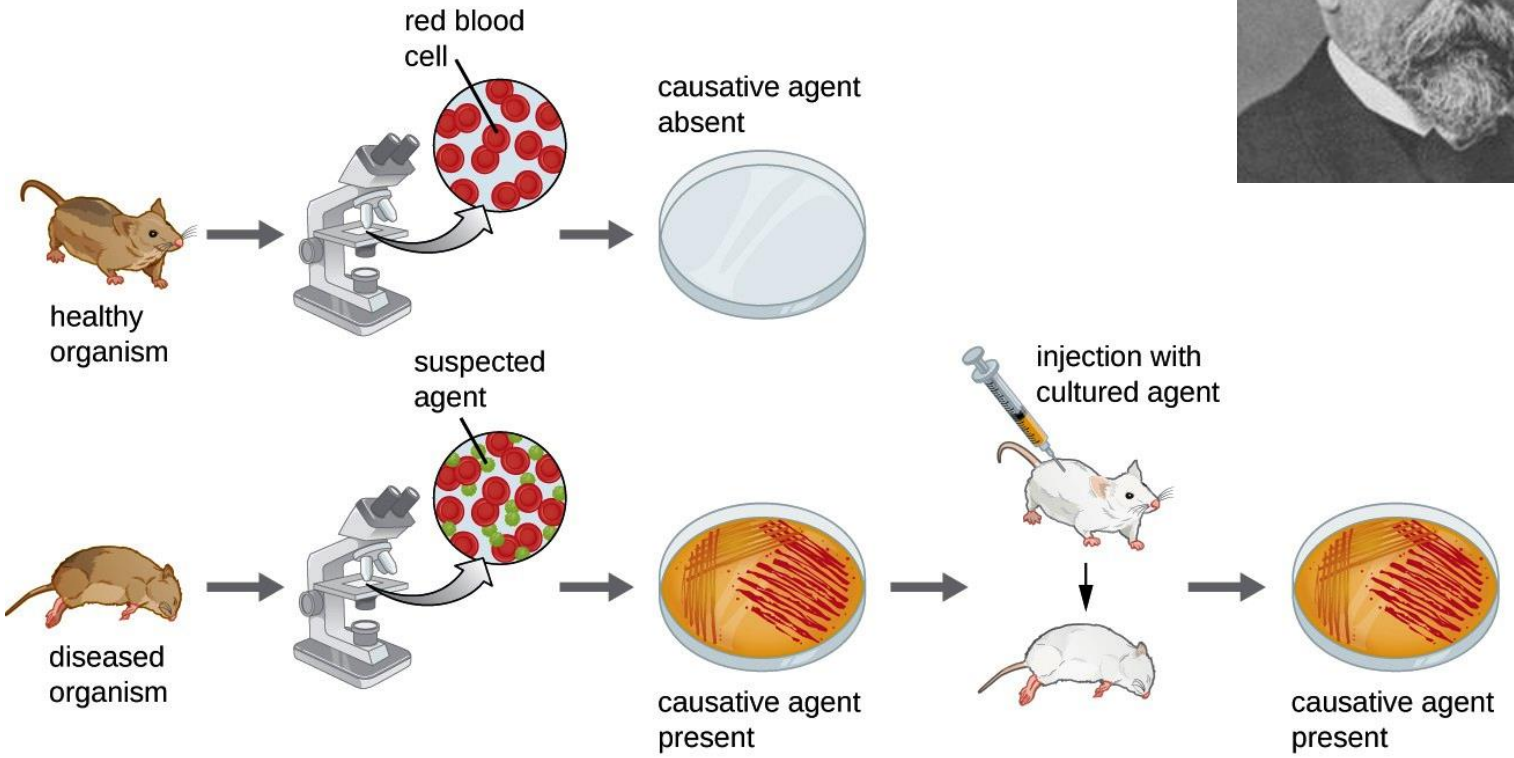
SKIN
- Staphylococci
- Streptococci
- Corynebacteria
- Proprionibacteria
- Yeasts

UPPER BOWEL
- Enterobacteriaceae
- Enterococci
- *Candida*

LOWER BOWEL
- *Bacteroides*
- Bifidobacteria
- *Clostridium*
- Peptostreptococci

VAGINA
- Lactobacilli
- Streptococci
- Corynebacteria
- *Candida*
- *Actinomyces*
- *Mycoplasma hominis*

Koch's postulates
(Robert Koch,1843-1910, Germany)



1. The suspected causative agent must be absent from all healthy organisms but present in all diseased organisms.
2. The causative agent must be isolated from the diseased organism and grown in pure culture.
3. The cultured agent must cause the same disease when inoculated into a healthy, susceptible organism.
4. The same causative agent must then be reisolated from the inoculated, diseased organism.

Exceptions to Koch's Postulates

Modification of Koch's postulates were necessary

1. to establish disease etiology for viruses and bacteria, which cannot be grown on artificial media
2. Some diseases, *e.g.:* pneumonia and nephritis, may be caused by a variety of microbes.
3. Some pathogens, such as *S. pyogenes*, cause several different diseases.
4. Certain pathogens, such as HIV, cause disease in humans only.

Molecular Koch's Postulates

Nucleic acid sequence of pathogen should be found in association with disease or diseased organ

Nucleic acid sequence should be absent from healthy individual

Resolution of disease should result in decrease in pathogen associated nucleic acid sequences

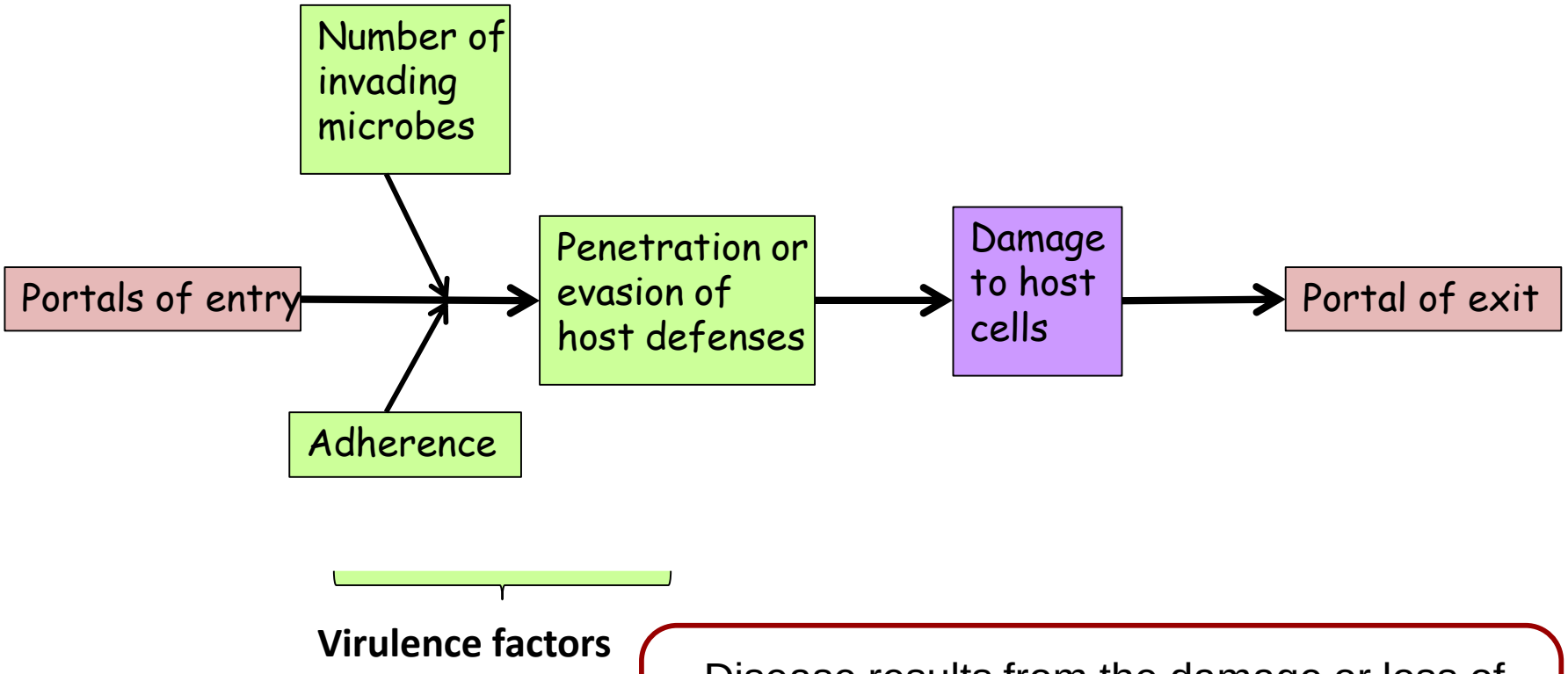
Presence of pathogenic nucleic acid in a healthy individual should predict development of disease

Nature of the microorganism inferred from nucleotide sequence should be consistent with biological characteristics of organism

Sequence based findings should be reproducible

Microbial mechanism of pathogenicity

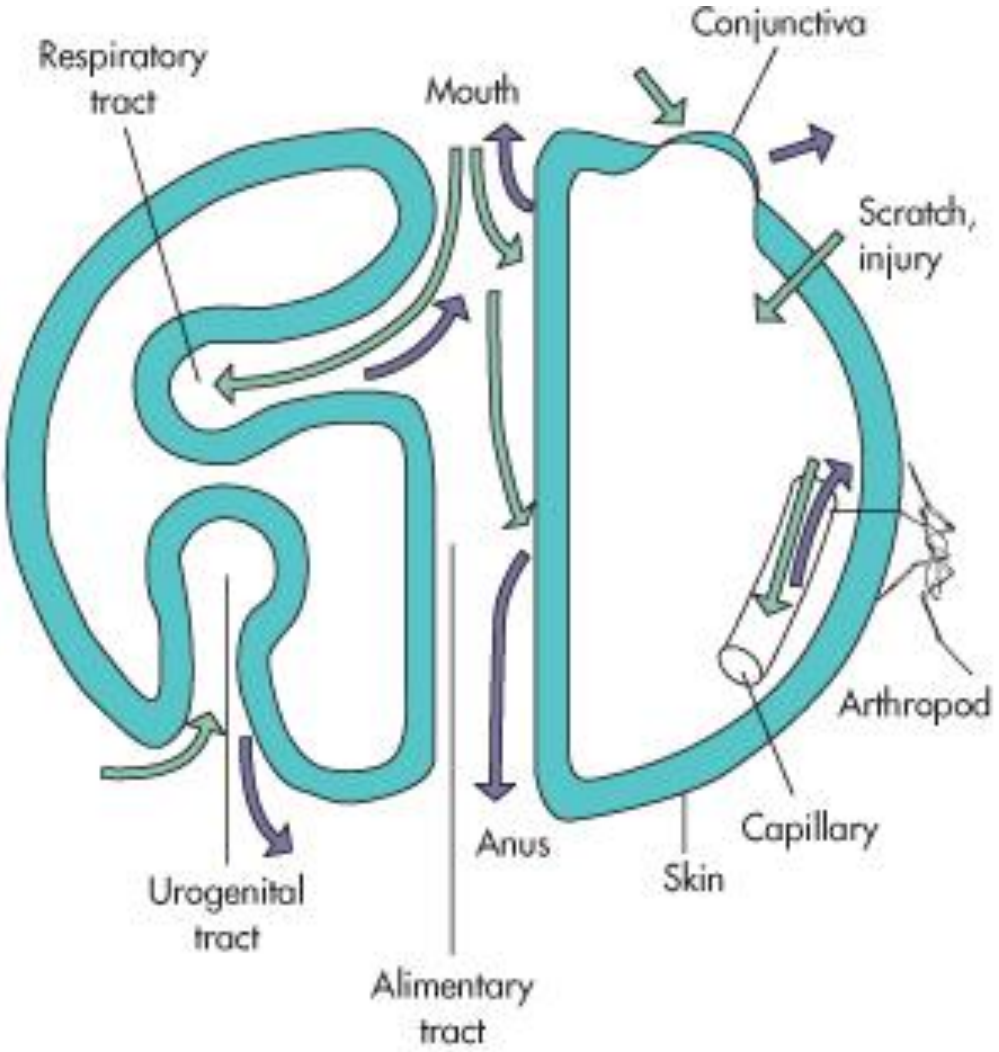
How microorganisms cause disease?



Disease results from the damage or loss of tissue or organ function due to the infection or the host inflammatory responses

Infection/entry

- Ingestion (fecal-oral)
  - *Salmonella, Shigella, Vibrio, Clostridium* etc..
- Inhalation (respiratory)
  - *Mycobacterium, Mycoplasma, Chlamydia* etc..
- Trauma (e.g. burn)
  - *Clostridium tetani*
- Arthropod bite (zoonoses: mosquito, flea, tick)
  - *Rickettsia, Yersinia pestis*, etc.
- Sexual transmission
  - *Neisseria gonorrboeae*, HIV, *Chlamydia*, etc
- Iatrogenic (needle stick, blood transfusion)
  - *Staphylococcus*, HIV, HBV
- Maternal-neonatal
  - HIV, HBV, *Neisseria*, etc.



Body surfaces as sites of microbial infection and shedding. Green arrows indicate infection; purple arrows indicate shedding. (Redrawn from Mims C et al: *Medical microbiology, London, 1993, Mosby-Wolfe.)*

Number of Invading Microbes

- LD_{50} - Lethal Dose of a microbes toxin that will kill 50% of experimentally inoculated test animal
- ID_{50} - Infectious Dose required to cause disease in 50% of inoculated test animals
  - Example: ID_{50} for *Vibrio cholerea* 10^8 cells (100,000,000 cells)
  - ID_{50} for Inhalation Anthrax - 5,000 to 10,000 spores

For shigellosis less than 200 Shigella is enough;
For diarrhea 10^8 Vibrio cholerae are required…

Colonization / Infection

- Colonization occurs whenever any one or more species populate an area.

- Infection, the growth of a parasitic organism within the body. (e.g. Bacteria) (with or without any symptoms)

How do bacterial pathogens penetrate host defenses?

Adherence

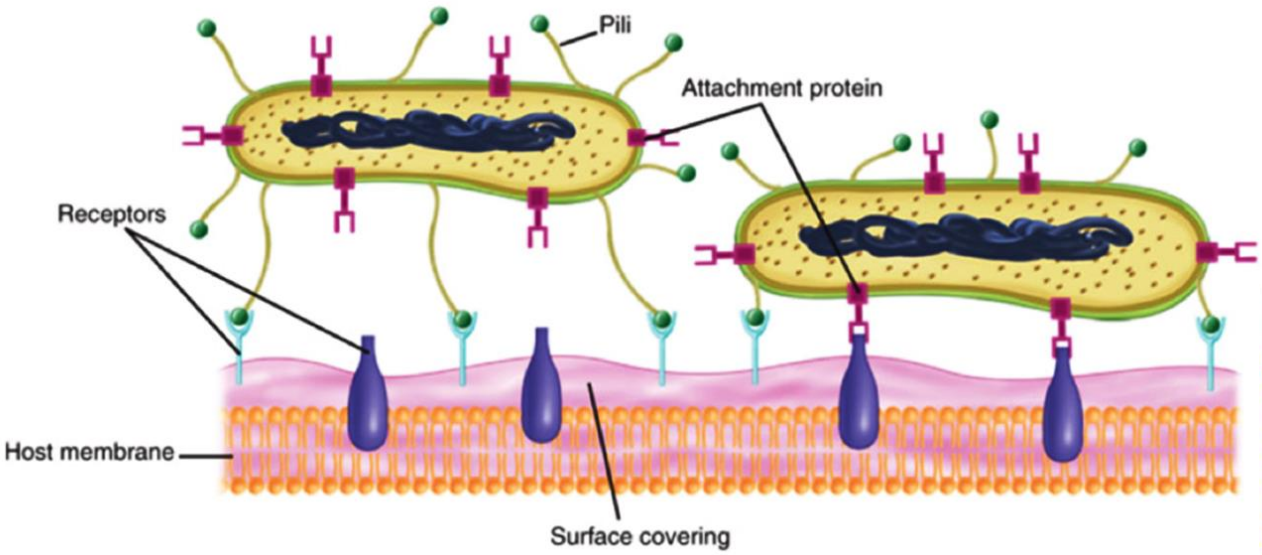
(The process or condition of attachment)

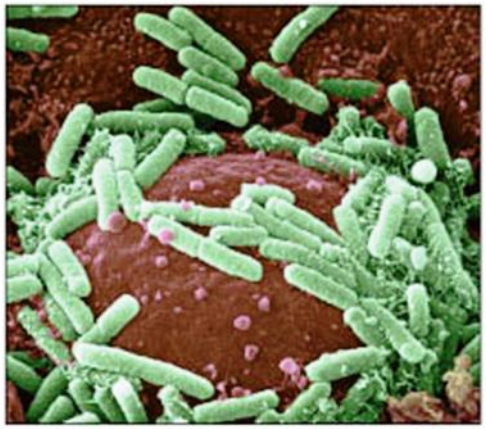
Mechanisms of Adherence to Cell or Tissue Surfaces

The mechanisms for adherence may involve two steps:

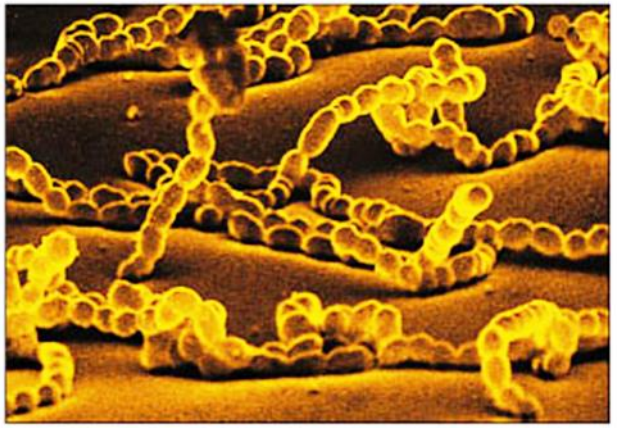
1. Nonspecific adherence: reversible attachment of the bacterium to the eukaryotic surface
   - Sometimes called "**docking**"
2. Specific adherence: irreversible permanent attachment of the microorganism to the surface
   - Sometimes called "**anchoring**"

Adherence





E. coli bacteria (green) on human bladder cells.

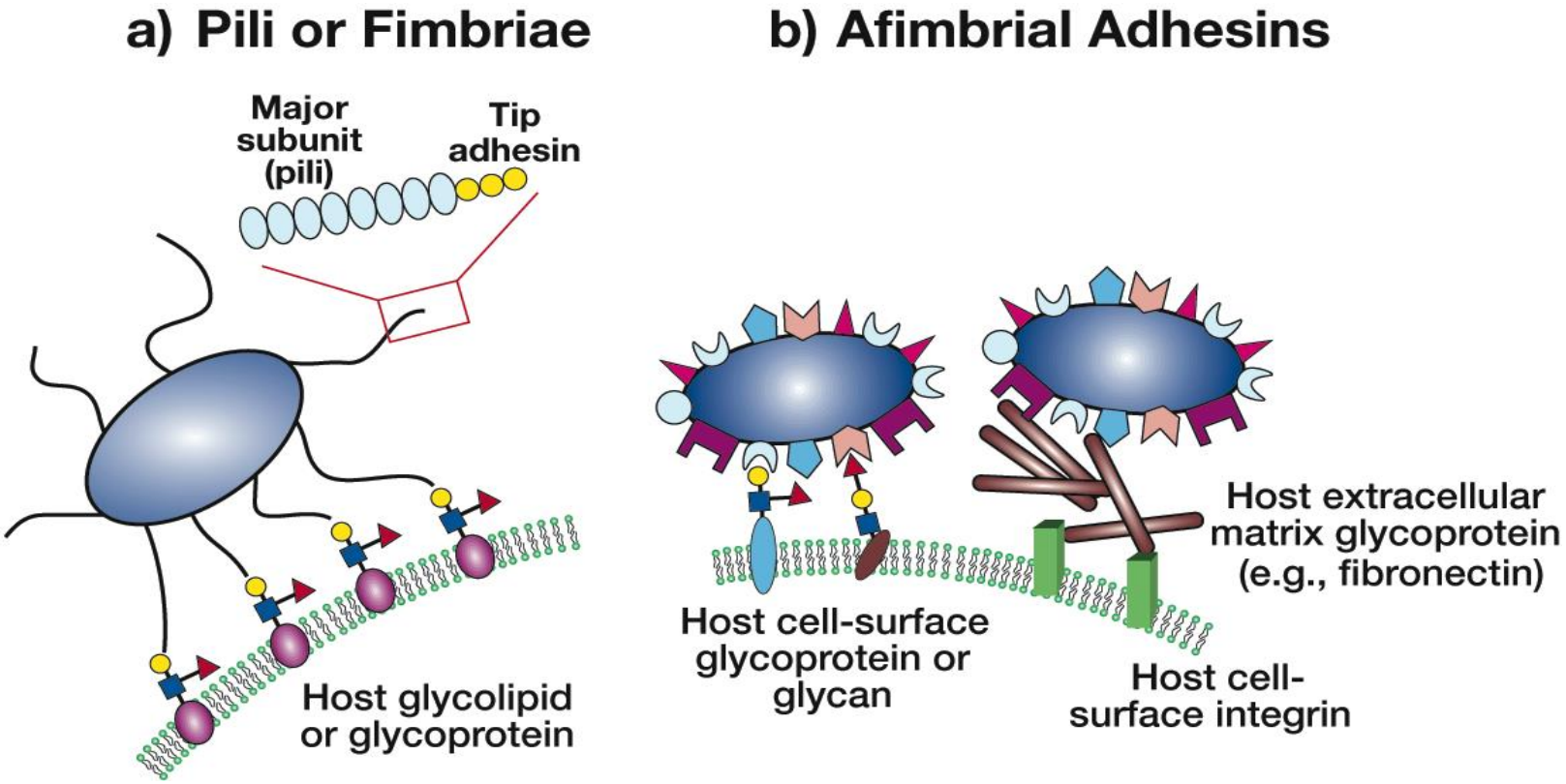


Bacteria adhering to human skin.

Binding sites: **Adhesins & Fimbriae**

Important: Surface hydrophobicity, net surface charge, binding molecules on bacteria and host cell receptor interactions

Examples of mechanisms of bacterial adherence to host-cell surfaces

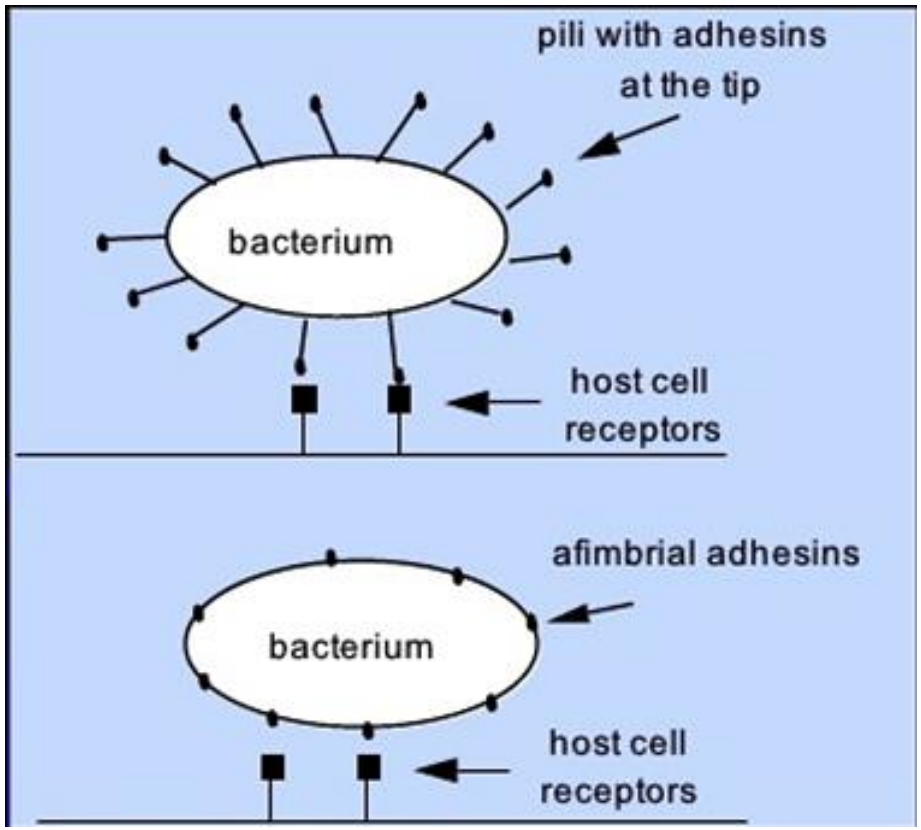


Adhesins and ligands are usually on fimbriae

Neisseria gonorrhoeae
Escherichia coli
Bordetella pertussis

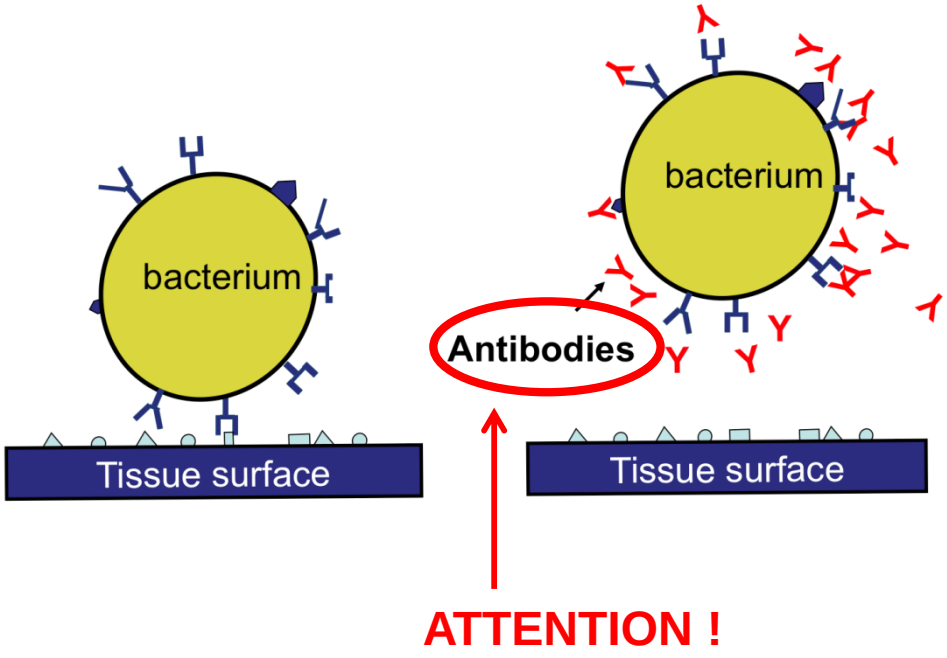
Bacteria have afimrial adhesins

Yersinia spp.
Bordetella pertussis
Mycoplasma pneumoniae

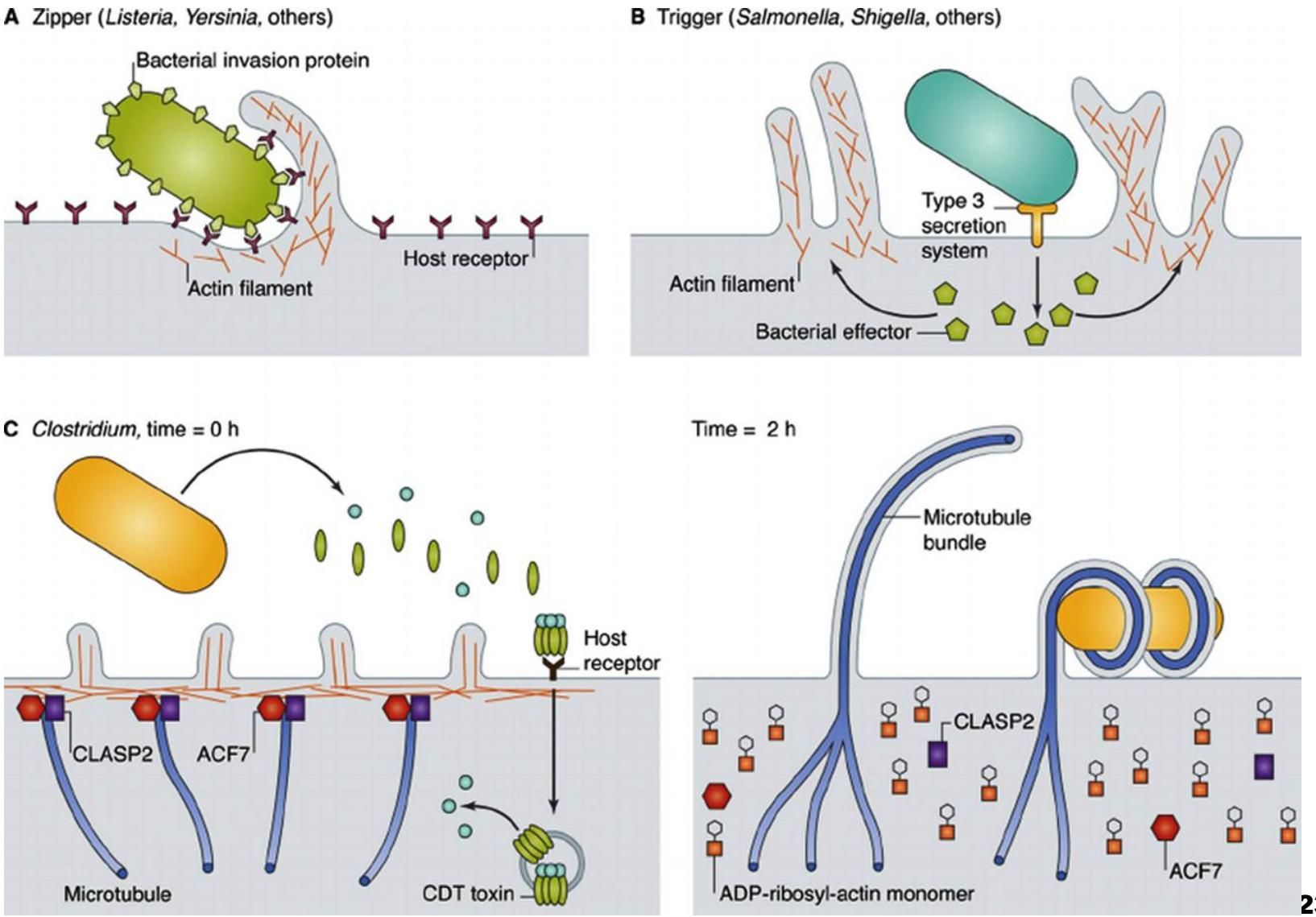


Why is adherence important ?

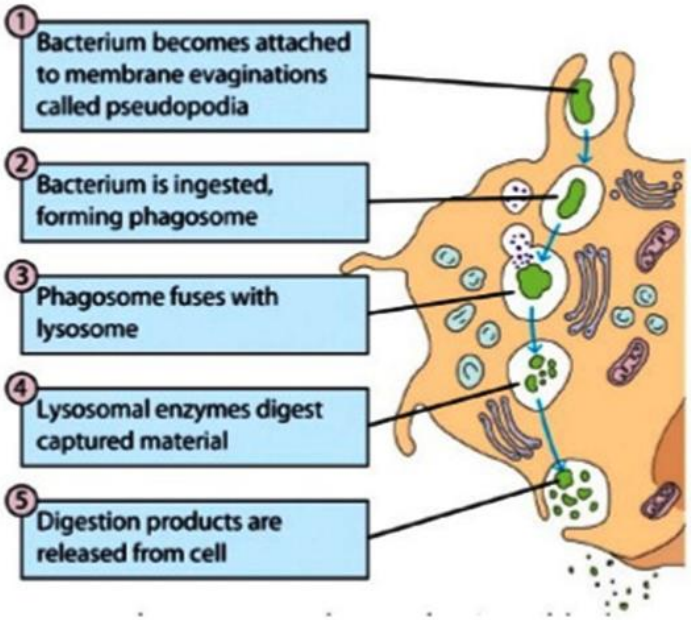
- Ability to colonize and cause disease
- Ability to adhere determines the host specificity
- Potential drug target



INVASION


A Zipper (*Listeria, Yersinia*, others)
Bacterial invasion protein
Host receptor
Actin filament
B Trigger (*Salmonella, Shigella*, others)
Type 3 secretion system
Actin filament
Bacterial effector
C *Clostridium*, time = 0 h
Host receptor
CLASP2
ACF7
Microtubule
CDT toxin
Time = 2 h
Microtubule bundle
CLASP2
ACF7
ADP-ribosyl-actin monomer

How is the microbe killed ?



Killing occurs by o_2 dependent & independent mechanisms & by both intracellular and extracellular mechanisms

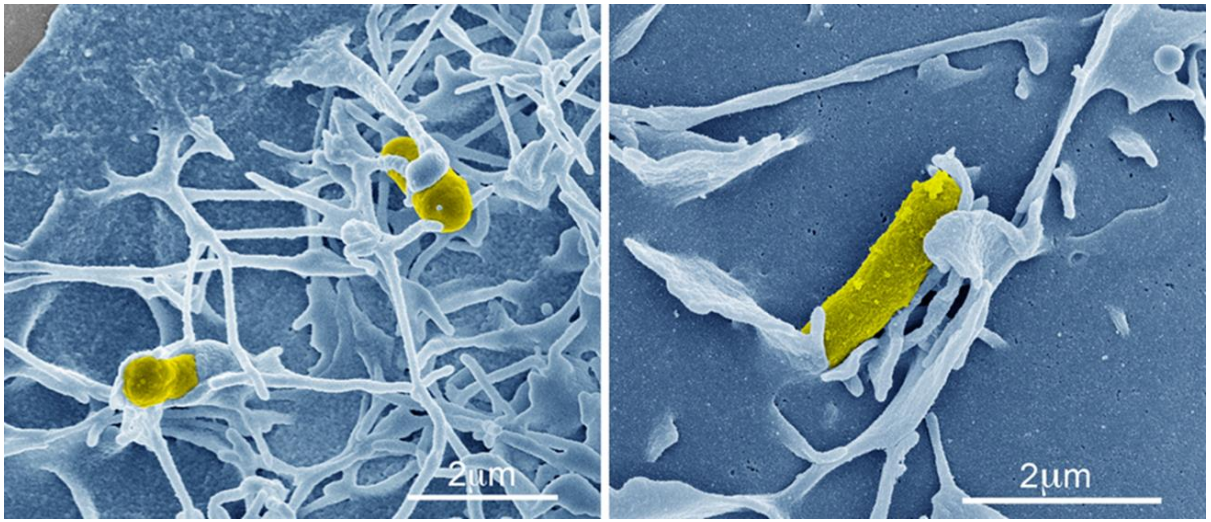
Intracellular bacteria

Intracellular microbes	Examples
A Phagocyte Phagocytosed microbes that survive within phagolysosomes Microbes that escape from phagolysosomes into cytoplasm	Intracellular bacteria: Mycobacteria *Listeria monocytogenes* *Legionella pneumophila* Fungi: *Cryptococcus neoformans* Protozoa: *Leishmania* *Trypanosoma cruzi*
B Nonphagocytic cell (e.g. epithelial cell) Virus Cellular receptor for virus Microbes that infect nonphagocytic cells	Viruses: All Rickettsiae: All Protozoa: *Plasmodium falciparum* *Cryptosporidium parvum*

INVASION

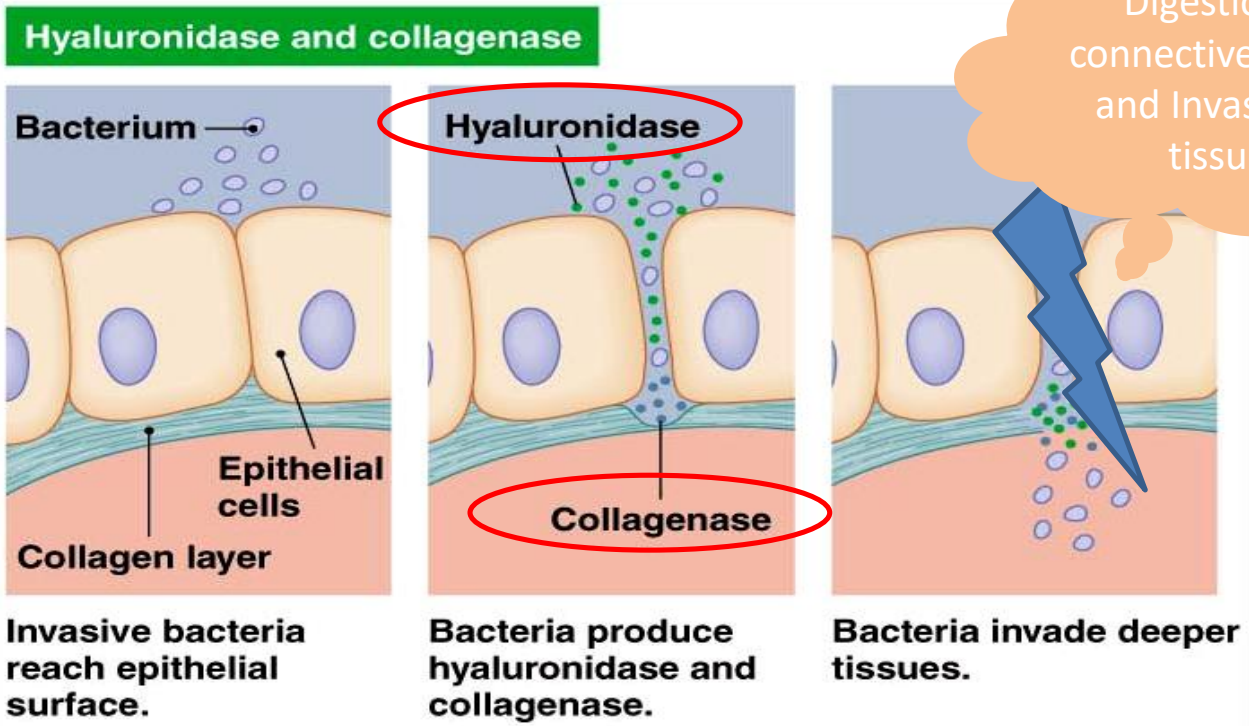
- The invasion of a host by a pathogen may be aided by the production of bacterial extracellular substances which act against the host by breaking down primary or secondary defenses of the body

- Medical microbiologists refer to these substances as **invasins**



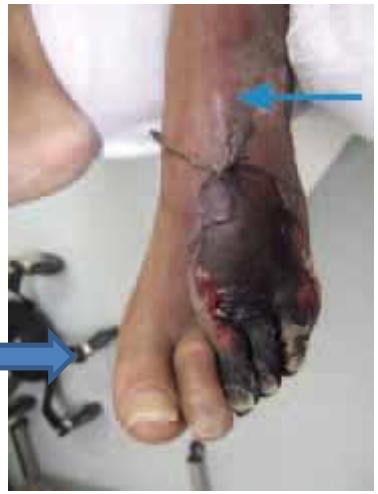
Enzymes

Hyaluronidase and Collagenase



Hyaluronidase: is present in *Staphylococcus aureus* (Skin infections) *and Streptococcus pyogenes (Sore throat)*

Collagenase: is present in Clostridium perfringens (gas gangrene)



Enzymes

- **Neuraminidase**
  - It degrades neuraminic acid (also called sialic acid), an intercellular cement of the epithelial cells of the intestinal mucosa.

- **Streptokinase and staphylokinase**
  - They are produced by streptococci and staphylococci, respectively
  - Kinase enzymes convert inactive plasminogen to plasmin which digests fibrin and prevents clotting of the blood
  - The relative absence of fibrin in spreading bacterial lesions allows more rapid diffusion of the infectious bacteria

Enzymes

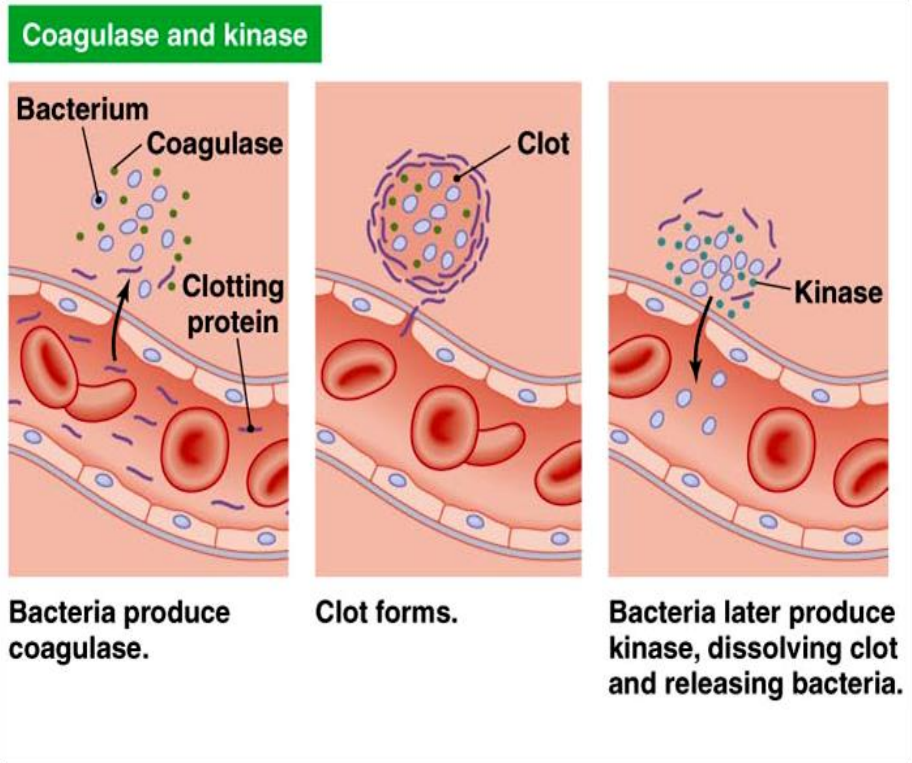
- **Phospholipases**
  - It hydrolyzes phospholipids in cell membranes by removal of polar head groups

- **Lecithinases**
  - It destroys lecithin (phosphatidylcholine) in cell membranes

Enzymes

- **Hemolysins**
  - They destroy red blood cells and other cells (i.e., phagocytes) by lysis

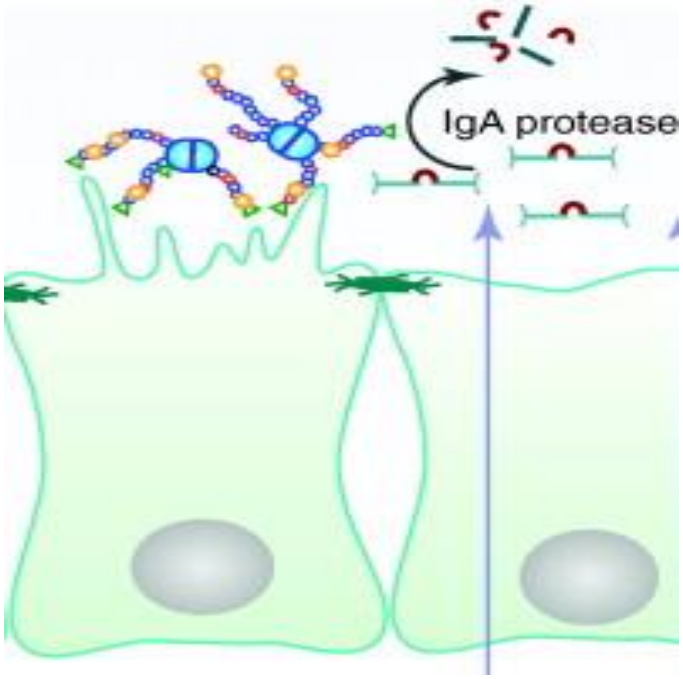
- **Coagulase**
  - Coagulase, formed by *Staphylococcus aureus*, is a cell associated and diffusible enzyme that converts fibrinogen to fibrin which causes clotting

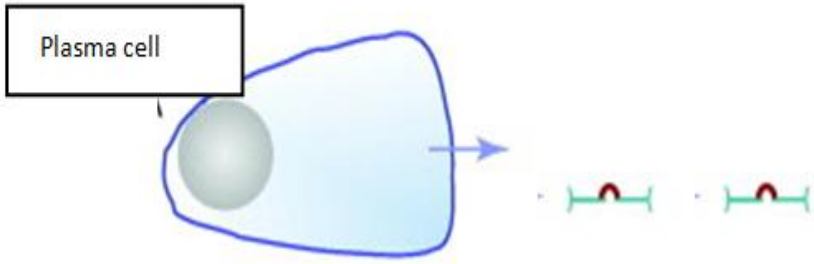


Strains that do not produce coagulase are not pathogenic

IgA protease

- An enzyme that can degrade IgA antibodies
  - eg. *Haemophilus influenza* (causes respiratory tract infection / meningitis)
- Helps overcome mucosal defenses





Some Extra Cellular Bacterial Proteins That Act As Invasins:

Invasin	Bacteria	Involved Activity
Hyaluronidase	Streptococci, staphylococci and clostridia	Degrades hyaluronic of connective tissue
Collagenase	*Clostridium* species	Dissolves collagen framework of muscles
Neuraminidase	*Vibrio cholerae* and *Shigella dysenteriae*	Degrades neuraminic acid of intestinal mucosa
Coagulase	*Staphylococcus aureus*	Converts fibrinogen to fibrin which causes clotting

Invasin	Bacteria	Involved Activity
Kinases	Staphylococci and streptococci	Converts plasminogen to plasmin which digests fibrin
Leukocidin	*Staphylococcus aureus*	Disrupts neutrophil membranes and causes discharge of lysosomal granules
Streptolysin	*Streptococcus pyogenes*	Repels phagocytes and disrupts phagocyte membrane and causes discharge of lysosomal granules
Hemolysins	Streptococci, staphylococci and clostridia	Phospholipases or lecithinases that destroy red blood cells (and other cells) by lysis
Lecithinases	*Clostridium perfringens*	Destroy lecithin in cell membranes

Invasin	Bacteria	Involved Activity
Phospholipases	*Clostridium perfringens*	Destroy phospholipids in cell membrane
Anthrax EF	*Bacillus anthracis*	One component (EF) is an adenylate cyclase which causes increased levels of intracellular cyclic AMP
Pertussis AC	*Bordetella pertussis*	One toxin component is an adenylate cyclase that acts locally producing an increase in intracellular cyclic AMP

EVASION STRATEGIES

Defense	Microbial strategy	Mechanism	Example
Wash-out	Bind to cell	Adhesins	*Neisseria*
	Inhibit ciliary activity	Ciliotoxic/ Ciliostatic molecule	*Bordetella* *Streptococcus*
Ingestion and killing by phagocyte	Disrupt chemotaxis	Leucocidins	*Staphylococcus*
	Inhibit phagocytosis	Capsule	*Streptococcus*
	Inhibit lysosomal fusion	Inhibitory molecule	*Mycobacterium*
	Multiply	Unknown	*Listeria*

EVASION STRATEGIES

Defense	Microbial strategy	Mechanism	Example
Restrict Fe- Lactoferrin Transferrin	Compete	Siderophore	*Mycobacterium Escherichia*
Activate complement	Interfere with alternative pathway	Fully sialylated surface	*Neisseria*
	Inactivate	Elastase	*Pseudomonas*
	Antigen projects beyond surface	Activation occurs at the wrong site	Gram-negatives
	Interfere with complement-mediated phagocytosis	C3b receptor competition, microbe and phagocyte	*Streptococcus*

Mechanisms for escaping host defences

Bacteria;

Bacteria that can evade or incapacitate the host defenses have a greater potential for causing diseases

- Evade recognition and killing by phagocytic cells
  - Antiphagocytic factors
- Inactivate or evade the complement system and antibody
  - Antigenic heterogeneity
- Even grow inside cells to hide from host responses!
  - Intracellular pathogenicity

Microbial Evasion of Phagocytosis

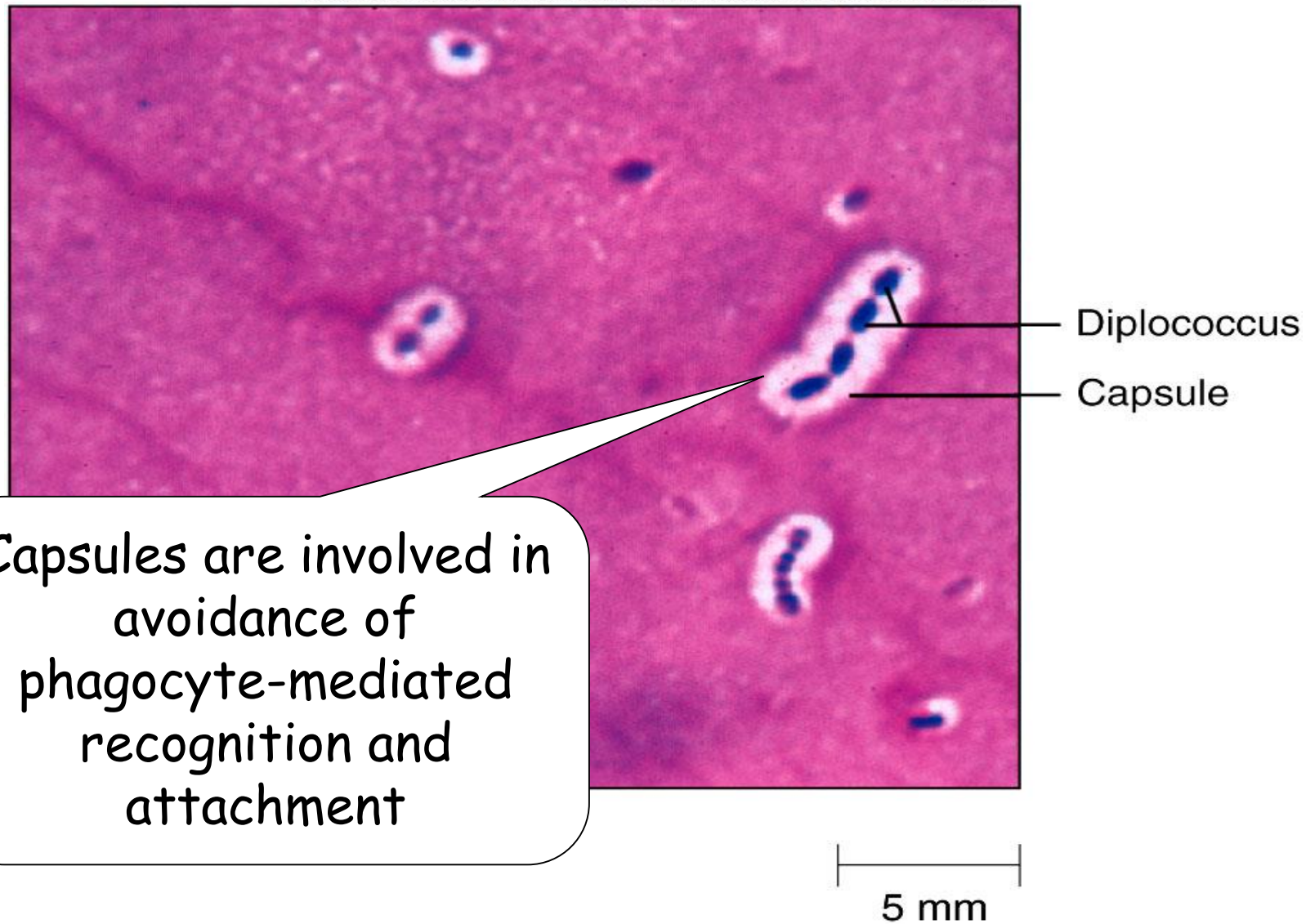
Mechanism	Organisms
Inhibit adherence: M protein, capsules	*Streptococcus pneumoniae*, *Streptococcus pyogenes*
Kill phagocytes: Leukocidins	*Staphylococcus aureus*
Lyse phagocytes: Membrane attack complex	*Listeria monocytogenes*
Escape phagosome	*Shigella* spp.
Prevent phagosom-lysosome fusion	HIV
Survive in phagolysosome	*Coxiella burnetti*, *Mycobacteria* spp.

Antiphagocytic factors

For example;

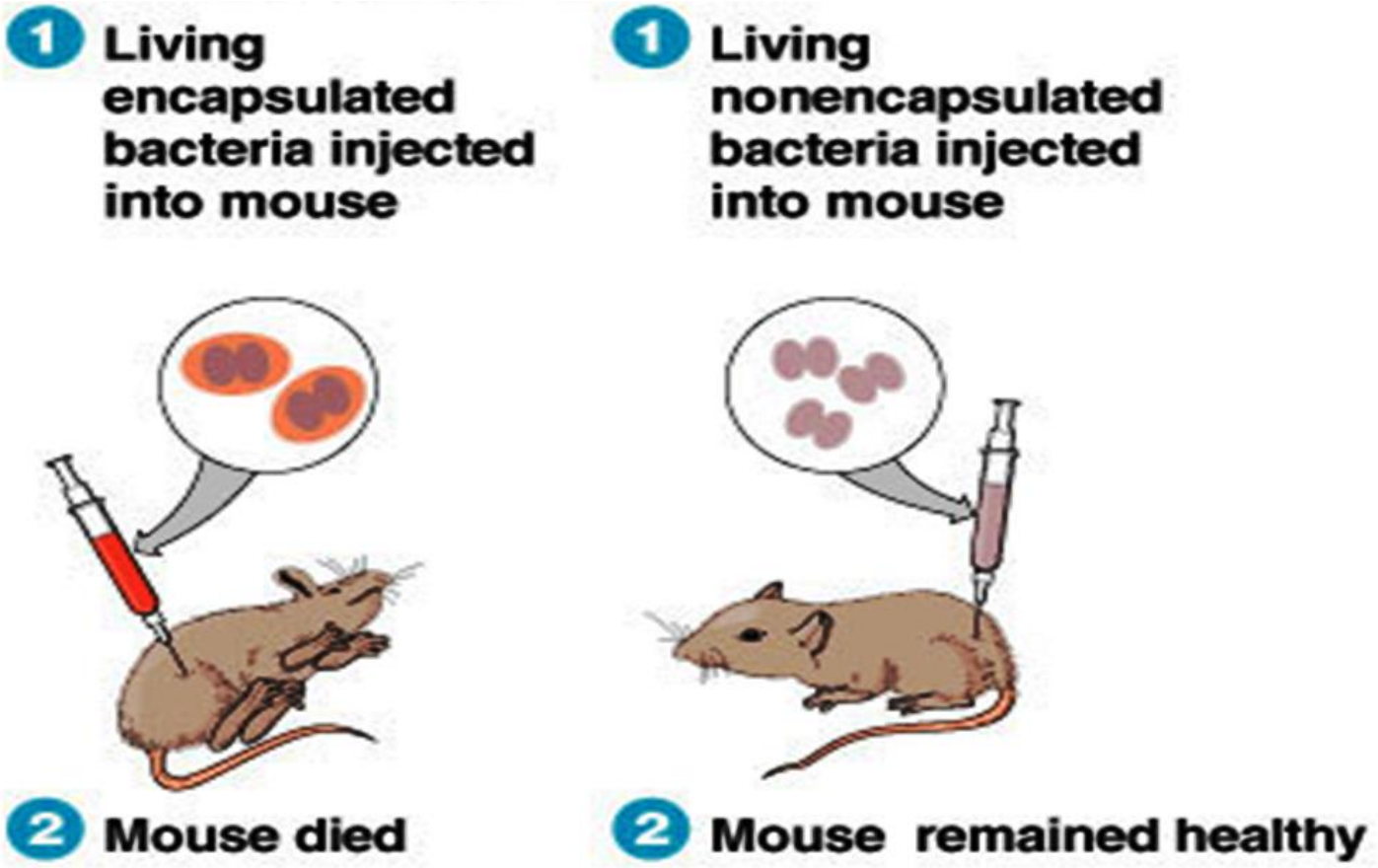
- *Staphylococcus aureus* has surface **protein A**, which binds to the Fc portion of IgG
- Other pathogens such as *Streptococcus pneumoniae* or *Neisseria meningitidis* have **surface factors** that impede phagocytosis
- Many other bacteria have polysaccharide **capsules**

Avoidance of Phagocytosis



Capsules are involved in avoidance of phagocyte-mediated recognition and attachment

Capsules contribute to pathogenesis

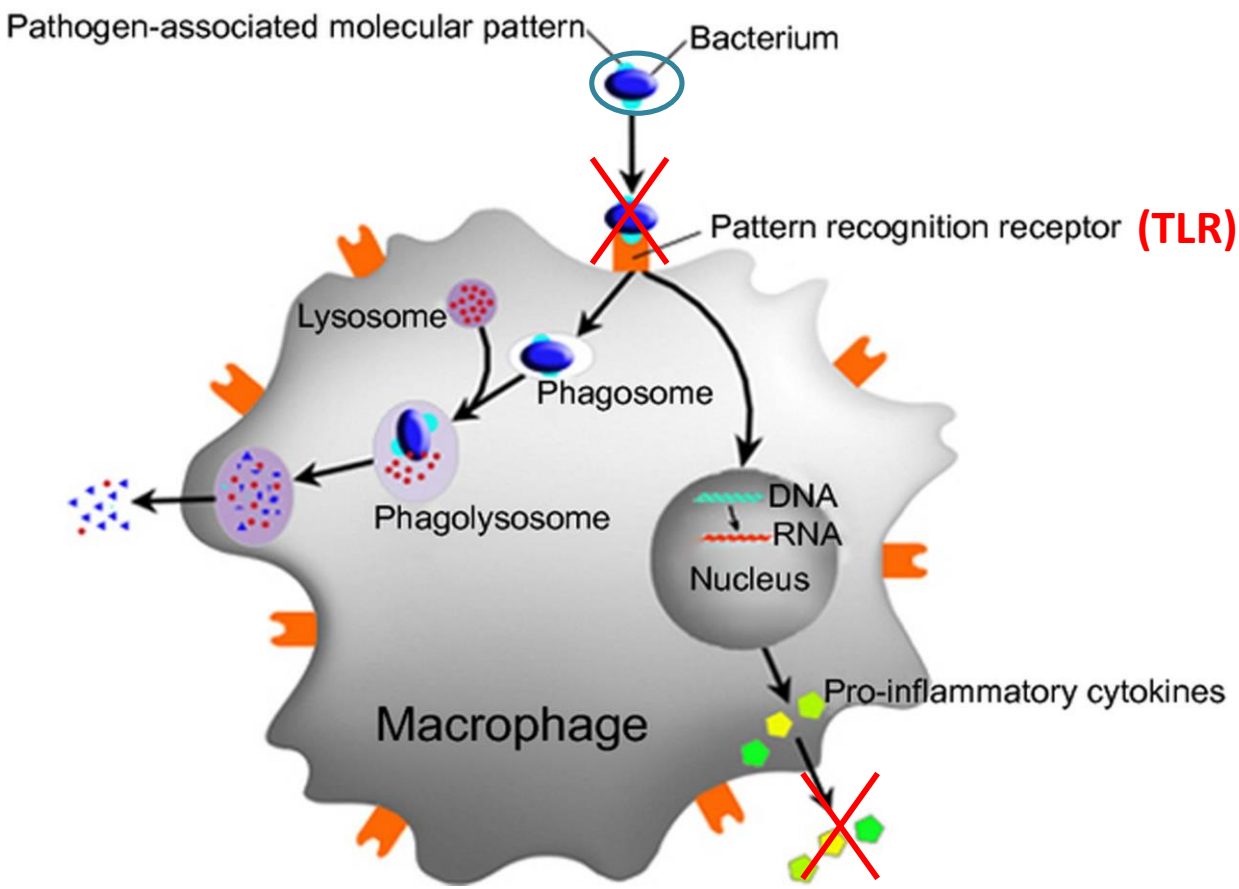


Capsule inhibits adherence of immune cells and resist phagocytosis (Nonpathogens may have capsule)

Antibiotics that work on capsule are formulated (It's easily destroyed if it's encapsulated)

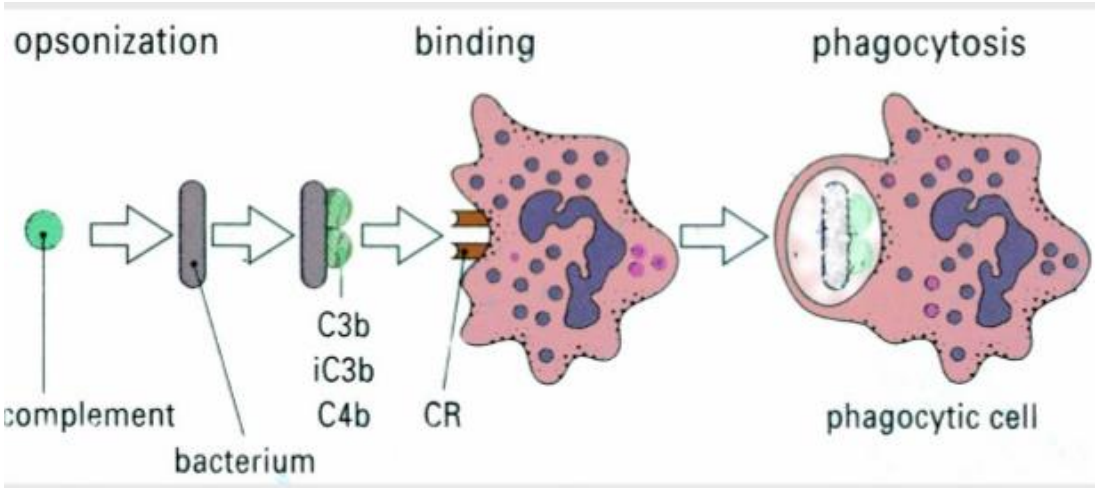
How does the capsule help bacteria evade host defenses ?

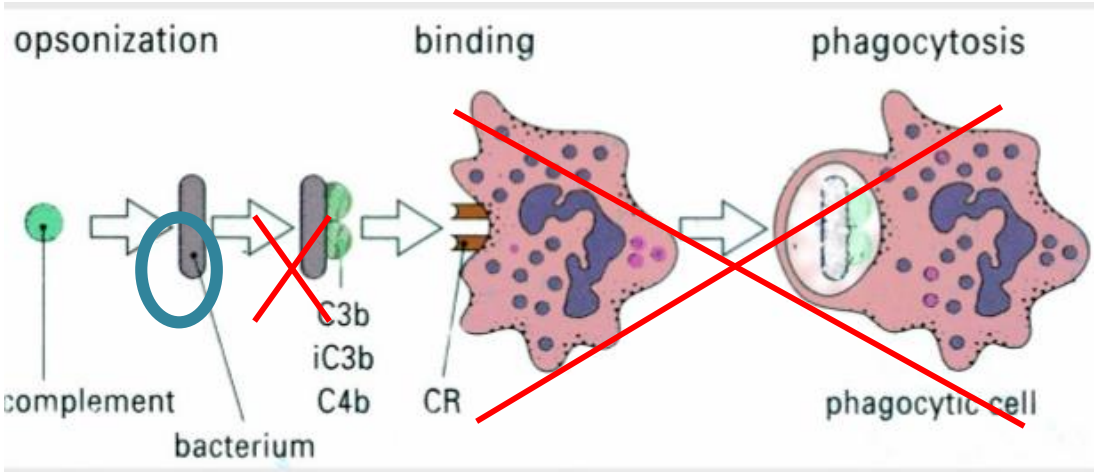
Capsules allow escape of TLR recognition



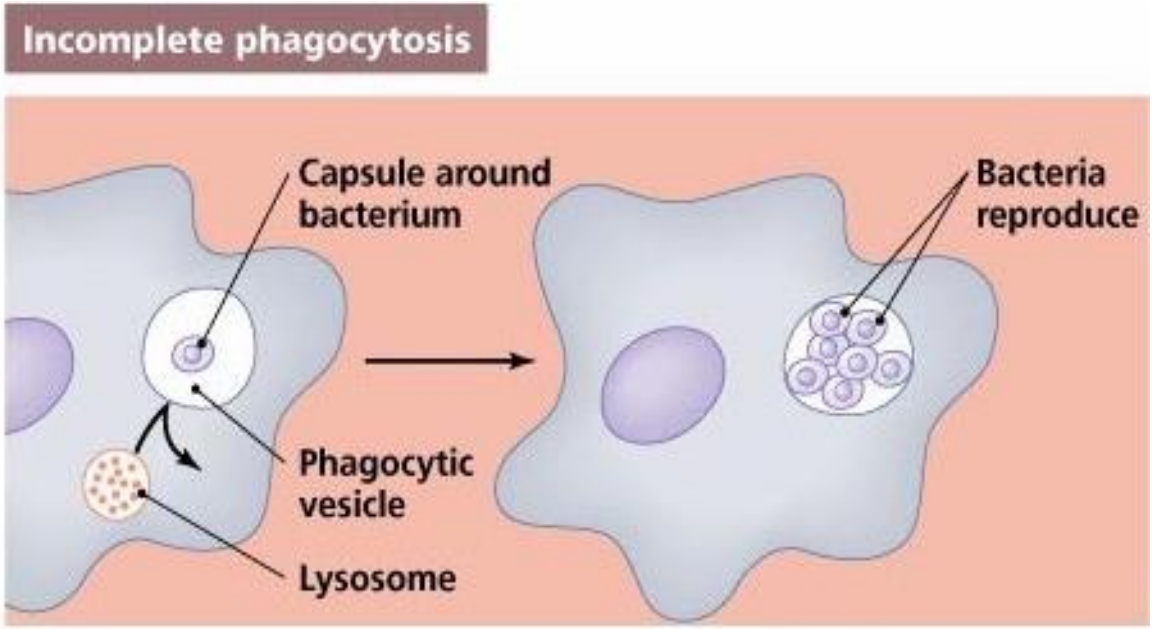
No immune response

Capsule inhibits phagocytosis





Capsule allows survival inside phagocytes

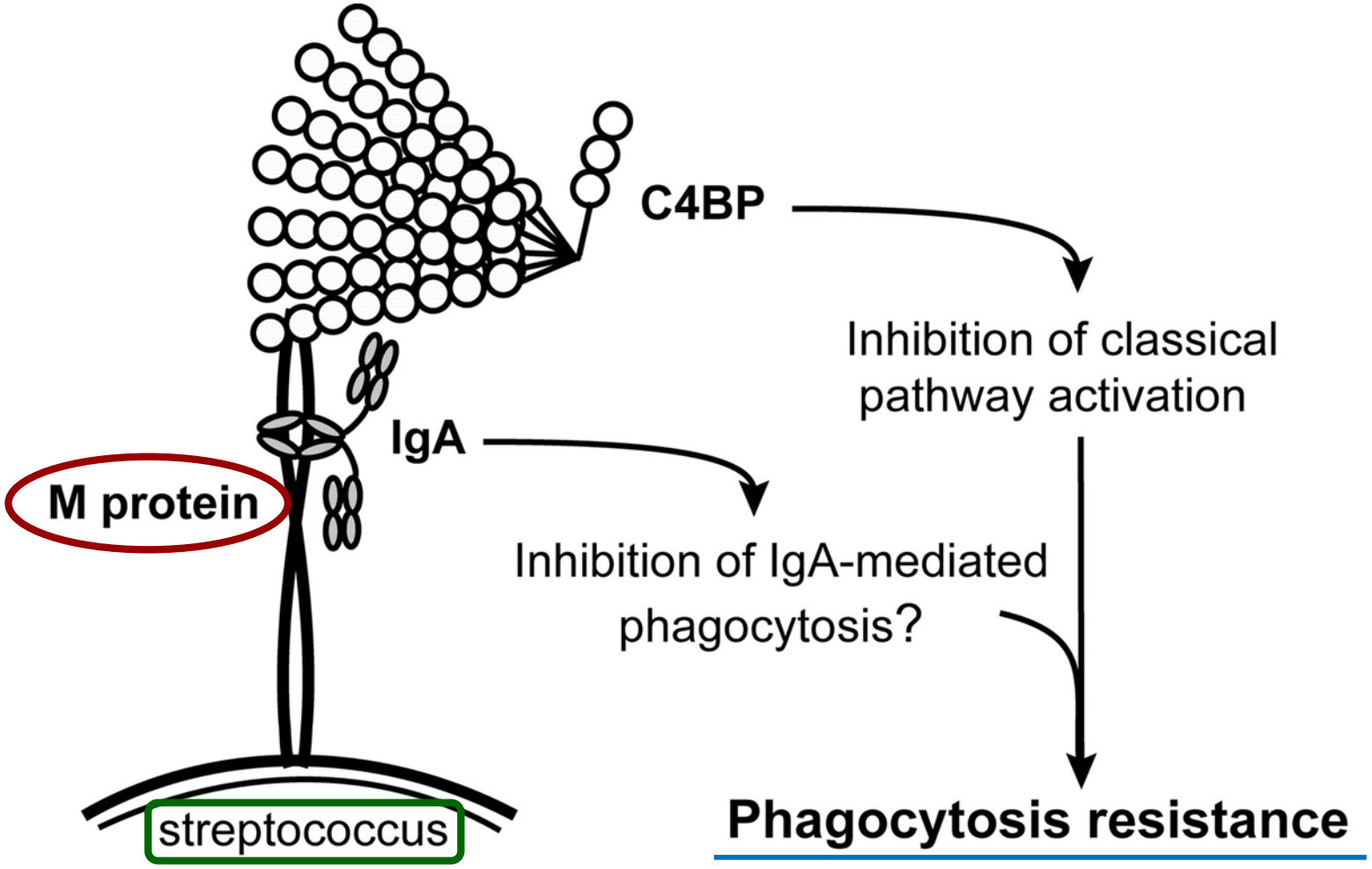


Capsule may help resist digestion by lysosomal enzymes

Encapsulated microorganisms

- *Staphylococcus aureus*
- *Streptococcus pneumoniae*
- *Streptococcus pyogenes*
- *Streptococcus agalactiae*
- *Bacillus anthracis*
- *Bacillus subtilis*
- *Neisseria gonorrhoeae*
- *Neisseria meningitidis*
- *Haemophilus influenzea*
- *Escherichia coli*
- *Klebsiella pneumoniae*
- *Salmonella* spp.
- *Yersinia pestis*
- *Campylobacter fetus*
- *Pseudomonas aeruginosa*
- *Bacteroides fragilis*
- *Cryptococcus neoformans*

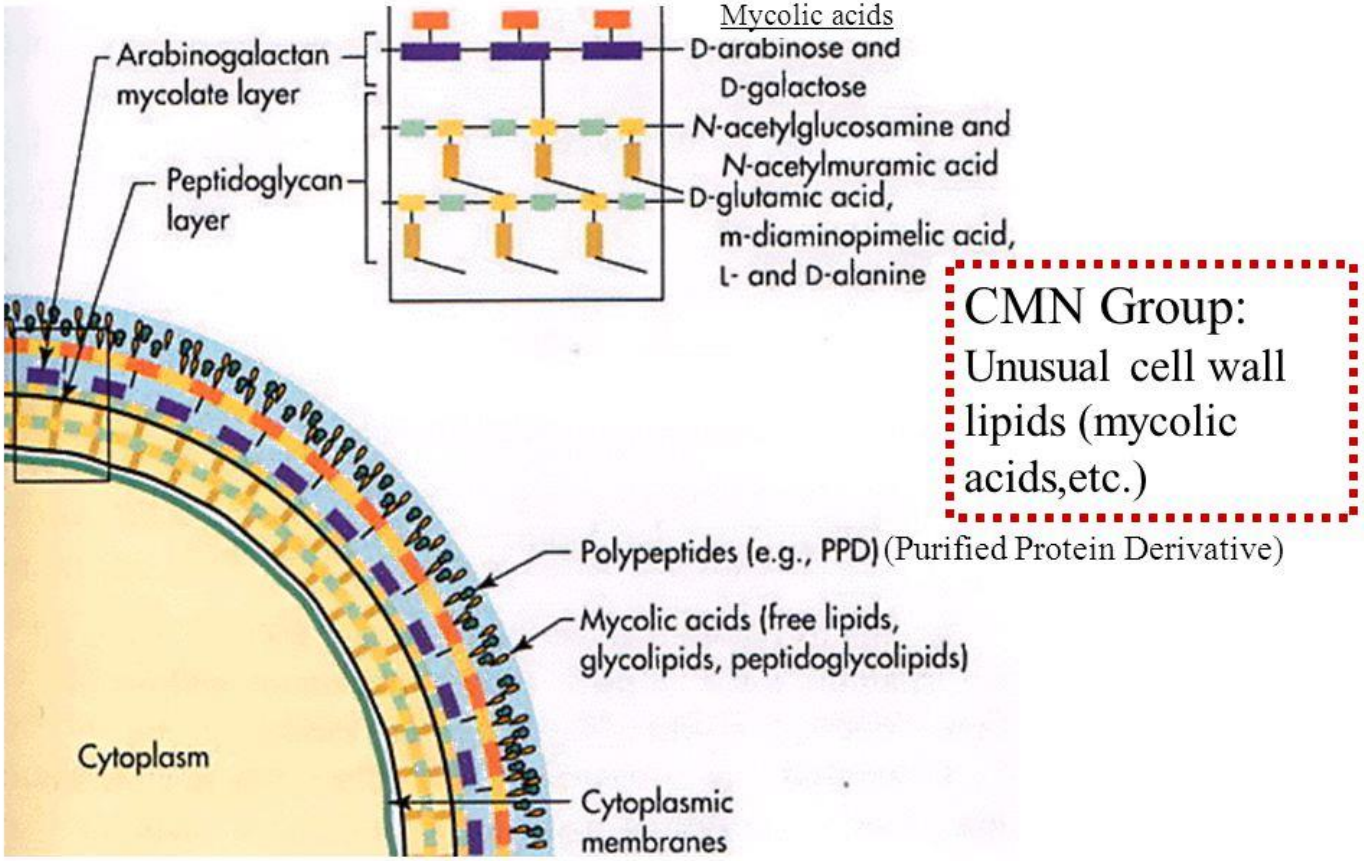
Antiphagocytic factors



waxy lipid

Antiphagocytic factors

Lipid-Rich Cell Wall of Mycobacterium



CMN Group:
Unusual cell wall lipids (mycolic acids,etc.)

Corynebacterium, Mycobacterium, Nocardia

Intracellular pathogenicity

- Avoid entry into phagolysosomes
  - Live within the cytosol of the phagocyte
- Prevent phagosome-lysosome fusion
  - Live within the phagosome
- Resistant to lysosomal enzymes
  - Survive within the phagolysosome

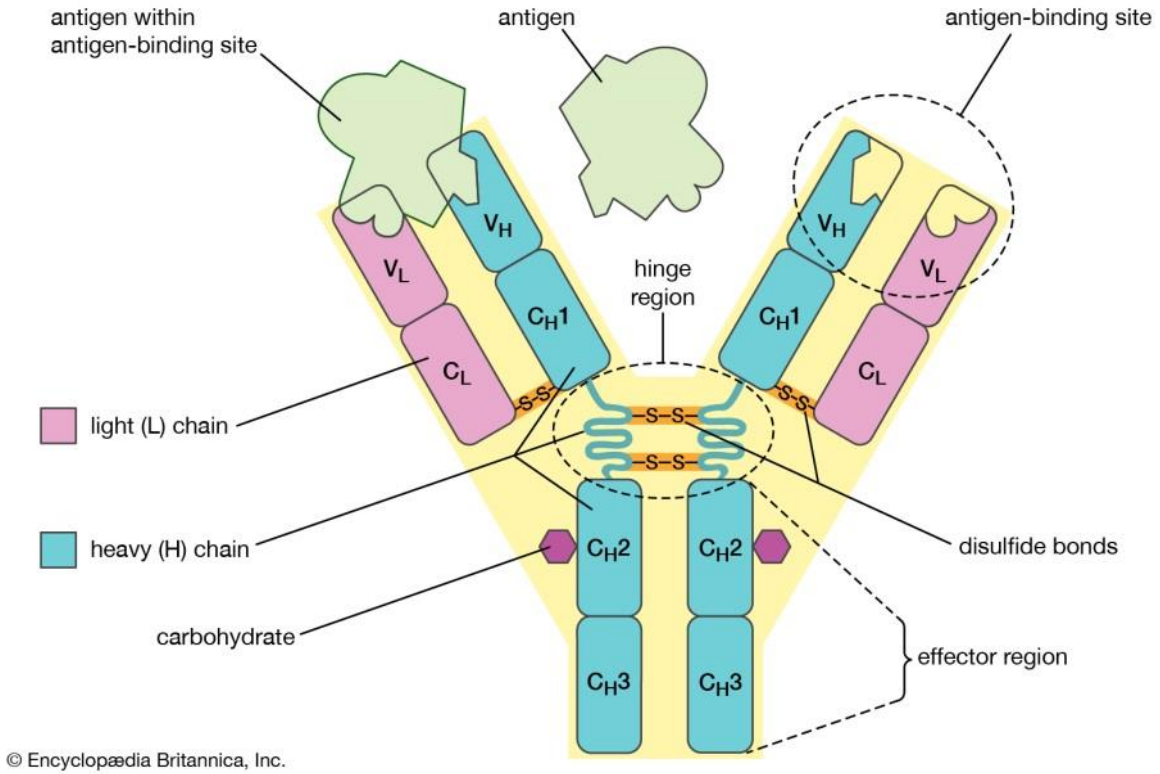
Examples of intracellular microorganisms

- *Mycobacterium* spp.
- *Brucella* spp.
- *Francisella* spp.
- *Rickettsia* spp.
- *Chlamydia* spp.
- *Listeria monocytogenes*
- *Salmonella typhi*
- *Shigella dysenteriae*
- *Yersinia pestis*
- *Legionella pneumophila*

Antigenic Variation

- Some pathogens can alter their surface antigen to avoid immune system detection

When the host creates an immune response, the pathogen has an already altered antigen which is formed by an alternative gene activation



Mechanisms of Antigenic Variation

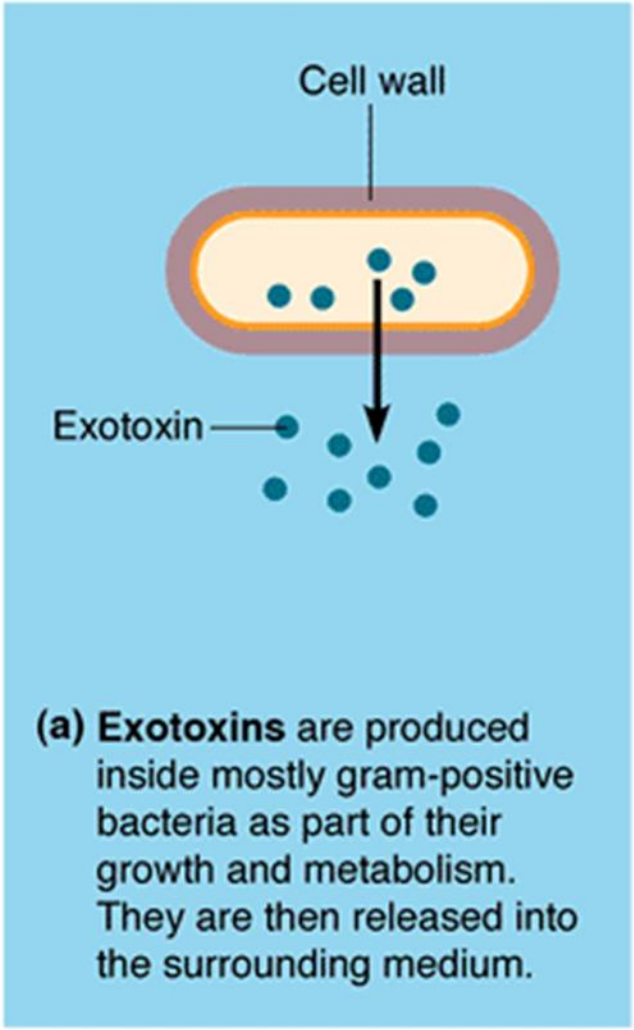
Type	Example	Disease
High mutation rate	HIV Influenza	AIDS Influenza
Genetic reassortment	Influenza Rota virus	Influenza Diarrhea
Genetic rearrangement	*Borrelia* *N.gonorrhoeae* *Plasmodium* spp.	Lyme disease Urethritis Malaria
Large diversity of serotype	Rhinoviruses *S.pneumoniae*	Colds Pneumonia

Mechanisms of Bacterial Pathogenicity

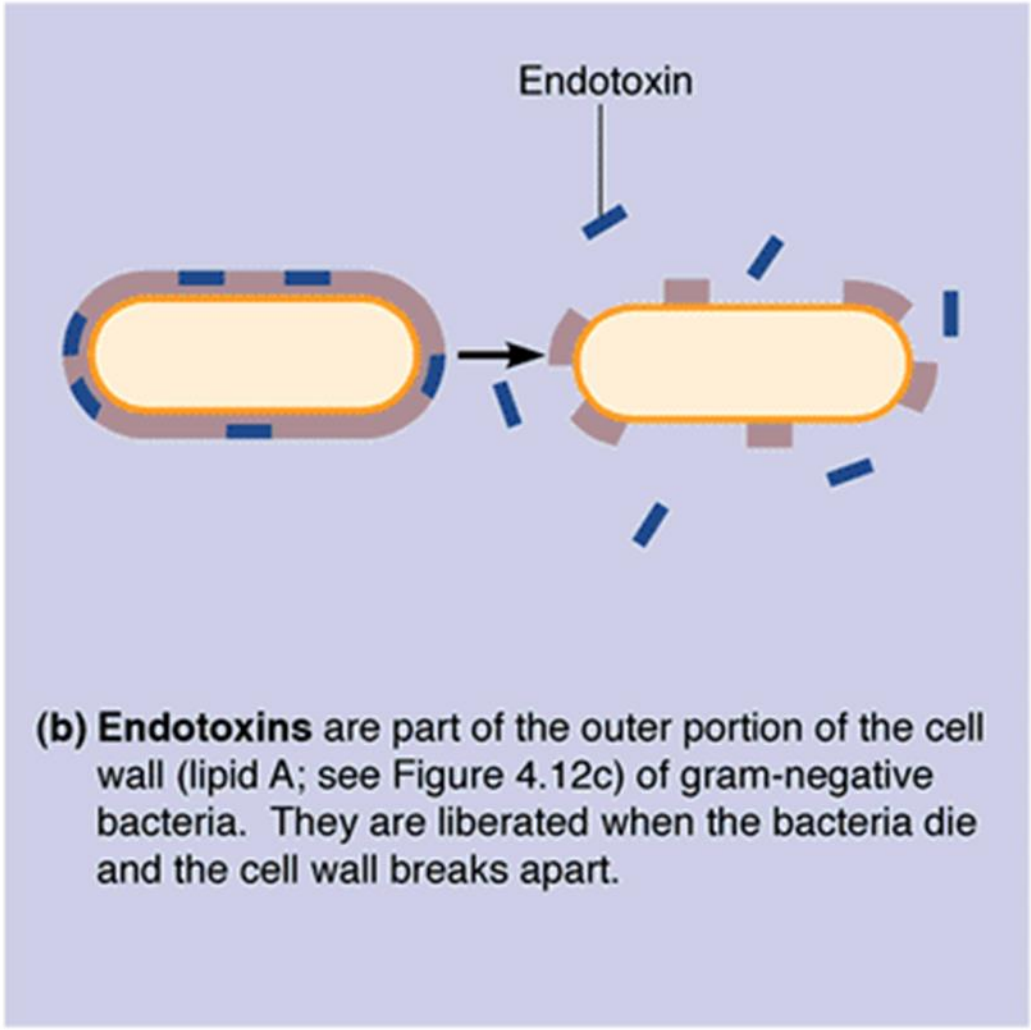
- **Toxigenesis:** Ability to produce toxins
- Bacteria may produce two types of toxins:

1. Exotoxins and
2. Endotoxins

  - **Exotoxins** are released from bacterial cells and may act at tissue sites removed from the site of bacterial growth
  - **Endotoxins** are cell-associated substance (classic sense, **endotoxin** refers to the lipopolysaccharide component of the outer membrane of Gram-negative bacteria)



(a) **Exotoxins** are produced inside mostly gram-positive bacteria as part of their growth and metabolism. They are then released into the surrounding medium.



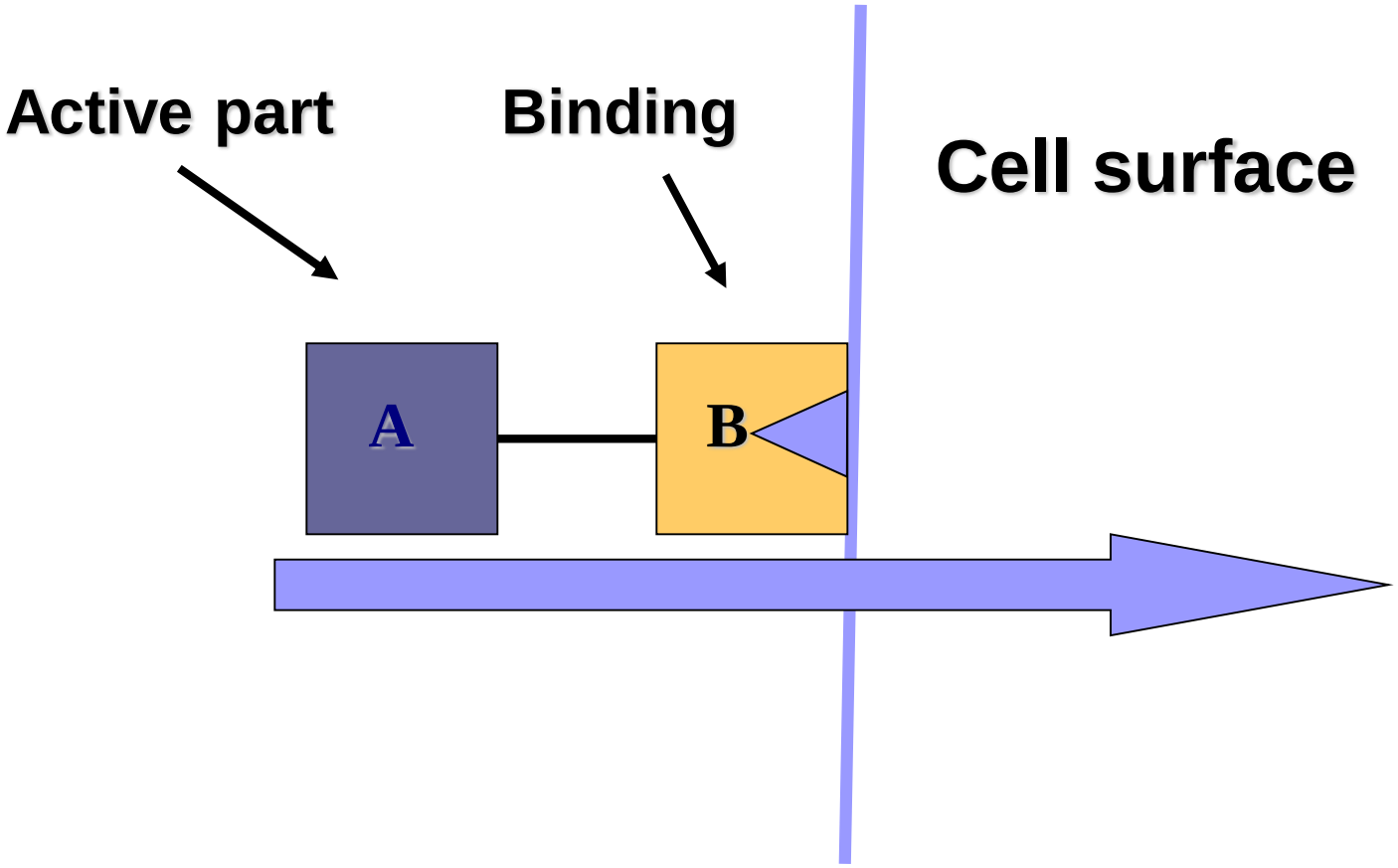
(b) **Endotoxins** are part of the outer portion of the cell wall (lipid A; see Figure 4.12c) of gram-negative bacteria. They are liberated when the bacteria die and the cell wall breaks apart.

Endotoxins vs Exotoxins

Exotoxins	Endotoxins
Excreted by living cells	Integral part of the cell wall of gram negative bacteria
Produced by both gram positive and negative bacteria	Found only in gram negatives
Polypeptides	Lipopolysaccharide complexes
Relatively unstable	Relatively stable
Highly antigenic	Weakly immunogenic
Toxoids used as vaccines	No vaccine
Highly toxic	Moderately toxic
Usually bind to specific receptors on cells	Specific receptors not found on cells
Usually do not produce fever in the host	Induces TNF and IL1; produce fever
Frequently controlled by extrachromosomal genes	Synthesis directed by chromosomal genes

Exotoxins

A - B Toxins: Such toxins consist of two components. One binds to cell surfaces and the other passes into the cell membrane or cytoplasm where it acts (i.e. cholera and diphtheria toxins)



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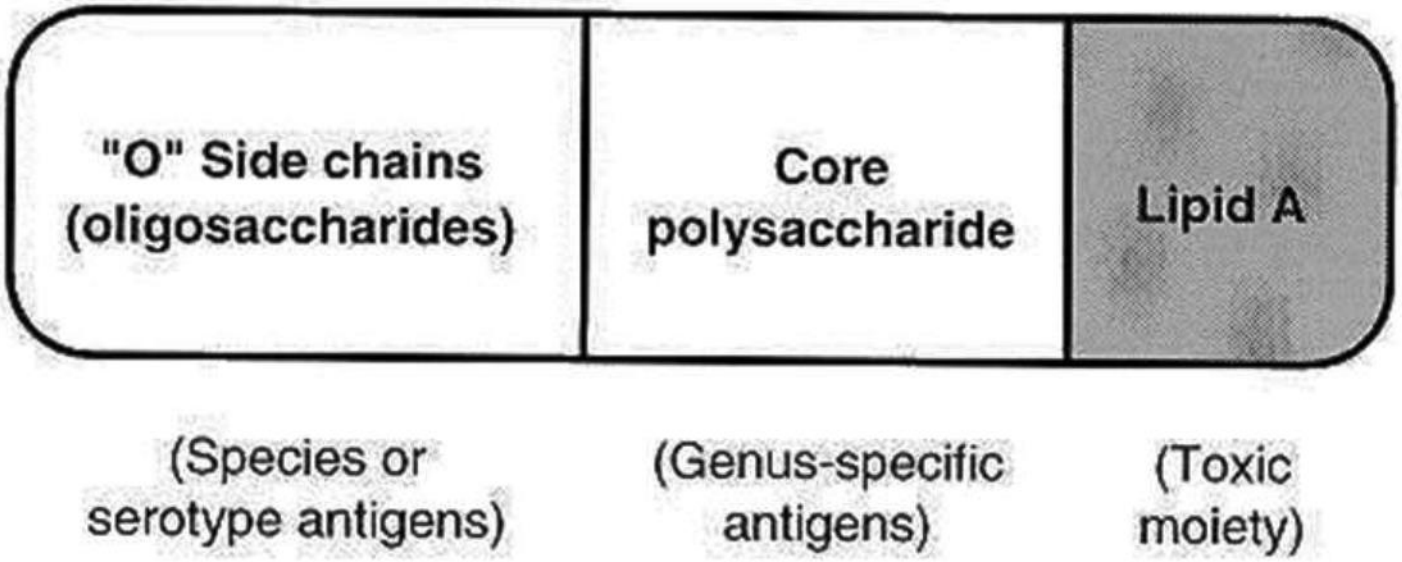
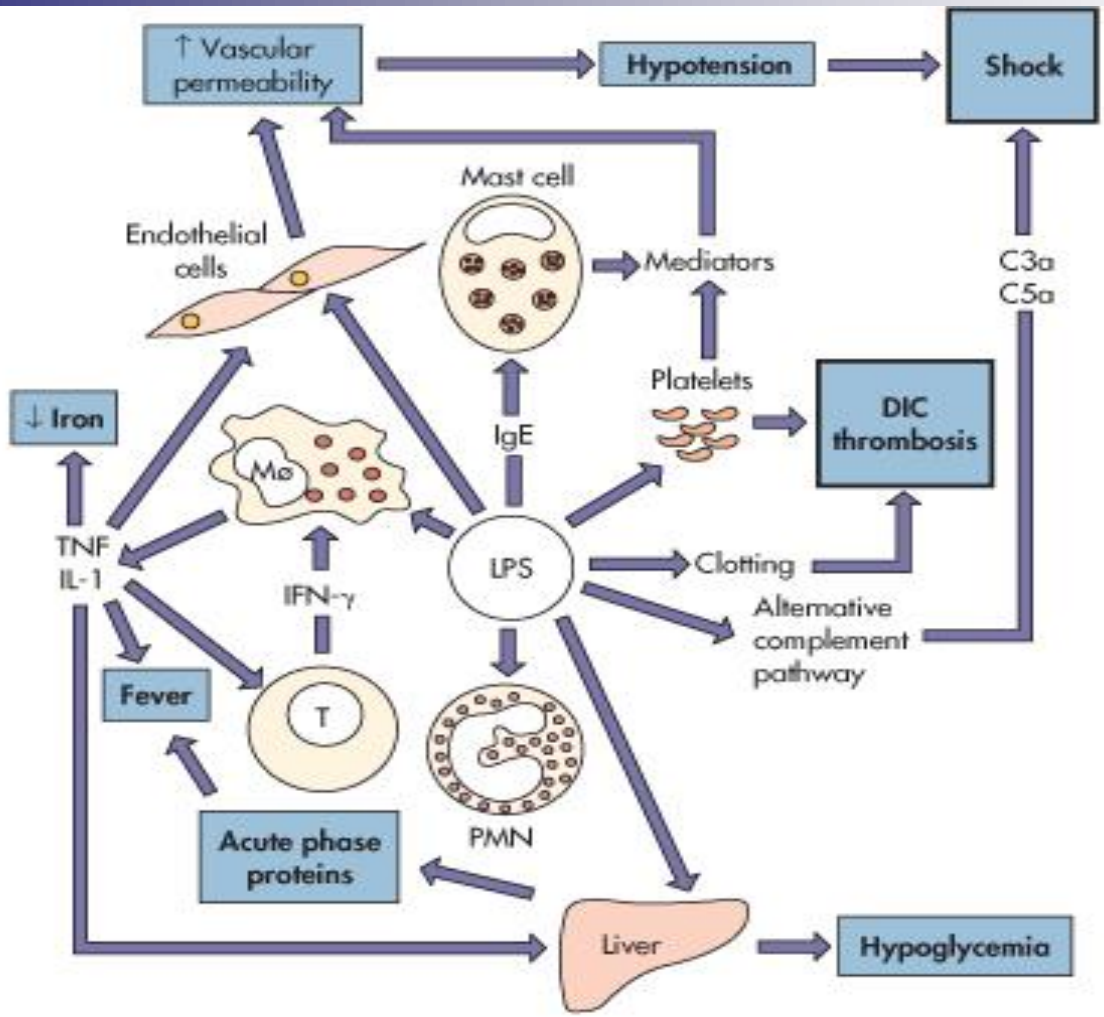
Examples of exotoxins produced by pathogenic bacteria

Microorganism	Disease	Toxin
Bacillus anthracis	Athrax	Anthrax toxin (EF) Lethal toxin
Bordetella pertussis	Pertussis	Adenylate cyclase toxin (pertussis AC)
Clostridium botulinum	Botulism	Botulinum toxin
Clostridium difficile	Pseudomembranous colitis	Enterotoxin
Clostridium perfringens	Gas gangrene	Alfa toxin
	Food poisoning	Enterotoxin
Clostridium tetani	Tetanus	Tetanospasmin
Corynebacterium diphteriae	Diphteriae	Diphteria toxin

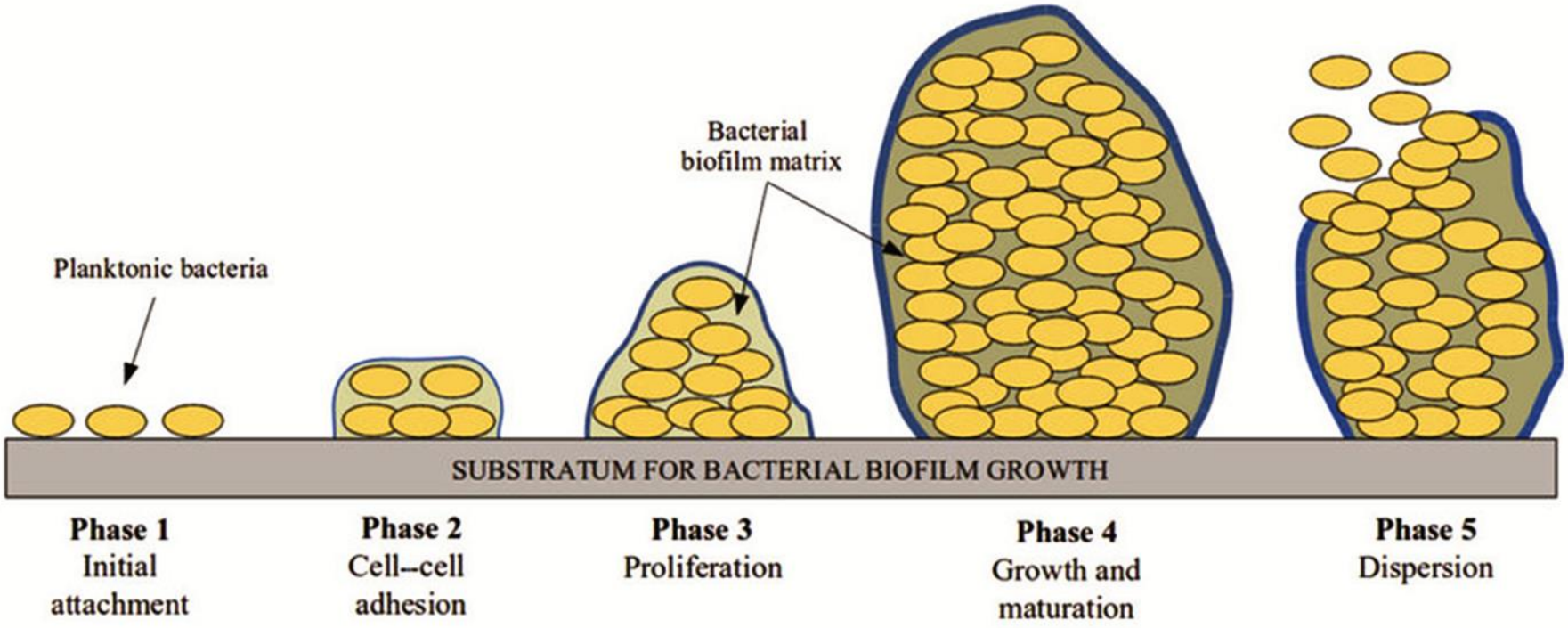
Basic structure of endotoxin (lipopolysaccharide) from Gram-negative bacteria





The many activities of lipopolysaccharide (LPS). **This bacterial endotoxin activates almost every immune mechanism, as well as the clotting pathway, which together make LPS one of the most powerful immune stimuli known.** DIC, Disseminated intravascular coagulation; IFN-γ interferon-γ; IgE, immunoglobulin E; IL-1, interleukin-1; PMN, polymorphonuclear (neutrophil) leukocytes; TNF, tumor necrosis factor. (Redrawn from Mims C et al: *Medical microbiology, London, 1993, Mosby-Wolfe.)*

Formation of bacterial biofilms



Quorum sensing: Ability to sense population density and alter gene expression

What triggers biofilm formation ?

- Bacterial attachment to surfaces
- Nutritional depletion
- Sub-inhibitory concentrations of antibiotics
- A critical population density of bacteria

How do biofilms help bacteria ?

- Protection from immune repsonse
- Protection from antibiotics
- Help tide over periods of low nutrition
- Microbial chatter (communication)

Biofilms: Why do they matter ?

- Key mechanism in bacterial pathogenesis
- Important part of food chain
- Major cause of corrosion of metal pipes

Modes of infectious disease transmission

- Contact transmission
  - Direct contact (person-to-person): syphilis, gonorrhoeae, herpes
  - Indirect contact (fomites): enterovirus infection, measles
  - Droplet (less than 1 meter): whooping cough, strep throat
- Vehicle transmission
  - Airborne: influenza, tuberculoses, chickenpox
  - Water-borne (fecal-oral infection): cholera, diarrhea
  - Food-borne: hepatitis, food poisoning, typhoid fever
- Vector transmission
  - Biological vectors: malaria, plaque, yellow fever
  - Mechanical vectors: E. coli diarrhea, salmonellosis

Transmission

- Specific bacterial species (or strains within a species) initiate infection after being transmitted by different routes to specific sites in the human body

Four major routes of transmission:

1. Skin i.e. through cuts or wounds
2. Gastro intestinal tract i.e. by ingestion of food or water
3. Respiratory tract i.e.in airborne droplets
4. Genital tract i.e. through sexual contact

Transmission

Disease can be directly transmitted in two ways:

- **Horizontal disease transmission** – from one individual to another in the same generation (peers in the same age group)
  - Horizontal transmission can occur by either direct contact (licking, touching, biting) or indirect contact air – cough or sneeze (vectors or fomites that allow the transmission of disease without physical contact)

- **Vertical disease transmission** – passing a disease causing agent vertically from parent to offspring,
  - Such as perinatal transmission

Types of Infections (Diseases)

- Communicable Disease (Infection): An infection that can be transmitted from one individual to another either directly by contact or indirectly by fomites and vectors

- Noncommunicable Disease: A disease that is not transmitted from one individual to another

Types of Infections

- **Asymptomatic Infection:**
  - A disease is considered asymptomatic if a patient is a carrier for a disease or infection but experiences no symptoms.
  - A condition might be asymptomatic if it fails to show the noticeable symptoms with which it is usually associated
  - Also called **subclinical infections**. The term clinically silent is also used
- **Symptomatic Infection:**
  - A disease is considered symptomatic if a patient is a carrier for a disease or infection and express symptoms . e.g. fever.

Types of Infections

- **Pandemic infection:**
  - Pandemic is an epidemic of infectious disease that is spreading through human populations across a large region; for instance multiple continents, or even worldwide. e.g. HIV
- **Epidemic infection:**
  - An epidemic occurs when new cases of a certain disease, in a given human population, and during a given period. e.g. Dengue
- **Endemic infection:**
  - Endemic infection Prevalent in or restricted to a particular region, community, or group of people. e.g. cholera

Infection results in:

- Acute infection vs. chronic infection
  - **Acute Infection**
    - An infection characterized by sudden onset, rapid progression, and often with severe symptoms
  - **Chronic Infection**
    - An infection characterized by delayed onset and slow progression

Infection results in:

- Primary infection vs. secondary infection
  - **Primary Infection**
    - An infection that develops in an otherwise healthy individual
  - **Secondary Infection**
    - An infection that develops in an individual who is already infected with a different pathogen

Infection results in:

- Localized infection vs. systemic infection
  - **Localized Infection**
    - An infection that is restricted to a specific location or region within the body of the host
  - **Systemic Infection**
    - An infection that has spread to several regions or areas in the body of the host

Infection results in:

- Clinical infection vs. subclinical infection
  - **Clinical Infection**
    - An infection with obvious observable or detectable symptoms
  - **Subclinical Infection**
    - An infection with few or no obvious symptoms

Infection results in:

- **Opportunistic infection**
  - An infection caused by microorganisms that are commonly found in the host's environment. This term is often used to refer to infections caused by organisms in the normal flora.

Infection results in:

Carrier state:

- A carrier state occurs when someone has been exposed to a pathogen (such as TB).
- A person may live as a carrier of a pathogen and would never know it
- Unless tested antibodies against the pathogen it lives in the host's body and can be passed on to others, without ever actually causing measurable harm to the host.

Latent state:

- In a latent state, the virus is simply "at rest".
- Person infected with the pathogen will show signs of the disease, at some time in the individual's life.
- Common in many diseases,
- Complimented by the "lytic" or active state of the pathogen/disease.

THE END

THANKS FOR LISTENING ☺

References

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- Sherris Medical Microbiology; 6th Ed; Ryan KJ, Ray CG; McGraw Hill Education; 2014