Prof Dr. Aysam Mahmoud Fayed - Aysam.Mahmoud.Ali@uomus.edu.iq
Dr. walaa Sabri Rahem walaa.Sabri.Raheem@uomus.edu.iq

# Specific Adherence of Bacteria to Cell and Tissue Surfaces

Several types of observations have provided indirect evidence for **specificity of adherence** of bacteria to host cells or tissues:

1. **Tissue tropism**. Particular bacteria are known to have an apparent preference for certain tissues over others, e.g. *S. mutans* is abundant in dental plaque but does not occur on epithelial surfaces of the tongue.

**2. Species specificity**. Certain pathogenic bacteria infect only certain species ,e.g. Group A streptococcal infections occur only in humans.

**3. Genetic specificity within a species**: certain strains or races within a species may be genetically immune to a pathogen, e.g. certain pigs are not susceptible to *E. coli* K-88 infections; males are not susceptible to mastitis; females are not susceptible to orchitis.

## Mechanisms of Adherence to Cell or Tissue Surfaces

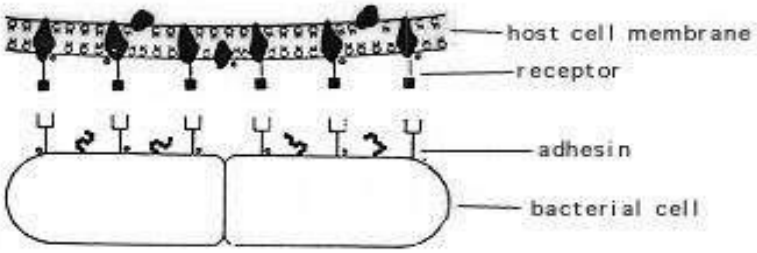
The mechanisms for adherence may involve two steps:

1. **Nonspecific adherence: reversible attachment** of the bacterium to the eukaryotic surface (sometimes called "docking") involves nonspecific attractive forces which allow approach of the bacterium to the eukaryotic cell surface. Possible interactions and forces involved are:

1. Hydrophobic interactions
2. Electrostatic attractions
3. Atomic and molecular vibrations resulting from fluctuating dipoles of similar frequencies
4. Brownian movement

5. Recruitment and trapping by biofilm polymers interacting with the bacterial glycocalyx (capsule)

2. **Specific adherence: irreversible permanent attachment** of the microorganism to the surface (sometimes called "anchoring").The usual situation is that reversible attachment precedes irreversible attachment but in some cases, the opposite situation occurs or specific adherence may never occur .Specific adherence involves permanent formation of many specific lock-and-key bonds between complementary molecules on each cell surface.




**2. Multiply:** Multiplication of bacterium at the site of entry **.**

**3. Invasion**

**Invasiveness** is the **ability of a pathogen to invade tissues.** 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**. Most invasins are proteins (enzymes) that act locally to damage host cells and/or have the immediate effect of facilitating the growth and spread of the pathogen. The damage to the host

as a result of this invasive activity may become part of the pathology of an infectious disease.

Invasiveness encompasses.

(1) mechanisms for colonization (adherence and initial multiplication), (2) production of extracellular substances

("invasins"), that promote the immediate invasion of tissues and

(3) ability to bypass or overcome host defense mechanisms which facilitate the actual invasive process.

**4. Spread within the host**: using

**"Spreading Factors**" is a descriptive term for a family of bacterial. enzymes that affect the physical properties of tissue matrices and intercellular spaces, thereby promoting the spread of the pathogen such as Hyaluronidase, Collagenase , Neuraminidase , Hemolysins.

**5. Ability to persist within the host (evade the host immune response)**

The consisting of polysaccharides, capsules**,** Outer membrane proteins, IgAses, Antigenic variation. All these factors are considered as virulence factors for pathogen that can be overcome the human or animals immune system.

**6. SHEDDING**

For many pathogenic bacteria multiplication within the human body provides a means of generating large numbers of progeny, thus increasing the numbers that can be shed into the environment.

Prof Dr. Aysam Mahmoud Fayed - Aysam.Mahmoud.Ali@uomus.edu.iq
Dr. walaa Sabri Rahem walaa.Sabri.Raheem@uomus.edu.iq

## How we can differentiate between exotoxin and endotoxin for G+ and some GExotoxin

| Exotoxin | Endotoxin |
|---|---|
| Excreted by living cells, found in high conc. In fluid medium | Part of the cell wall and G- bacteria liberated up on their disintegration |
| Polypeptides , molecular weight 10000-900000 Daltons | Lipopolysaccharide complex. lipid apportion responsible for toxicity |
| Relatively unstable to temperature above 60 $^{C}$ | Relatively stable to temp. above 60C for several hours with no loss activity |
| Highly antigenic, stimulates the formation of high – titer antitoxin (neutralized toxin) | Do not stimulate formation of antitoxin, stimulate formation of antibodies to polysaccharide moiety |
| Can be converted to a toxoid | Cannot be converted to a toxoid |
| Do not produce fever in the host | produce fever in the host "pyrogenic effect" |
| Highly toxic in microgram quantities to laboratory susceptible animals | Weekly toxic, hundreds of microgram. quantities required to be lethal foranimals |