



**Ministry of Higher education and scientific research**

**University of Tikrit**

**College of science**

**Department of Biology**

## **Lectures of Pathogenic Bacteria**

**For Diploma students – Pathological analyses - 2025-2026**

**Assistant professor Dr. Bushra Ali Kadhim**

**[bushraa.ali@tu.edu.iq](mailto:bushraa.ali@tu.edu.iq)**



**Pathogenic bacteria:** are bacteria that cause bacterial infection. This subject deals with human pathogenic bacteria. Although most bacteria are harmless or often beneficial, several are pathogenic. One of the bacterial diseases is tuberculosis, caused by the bacterium *Mycobacterium tuberculosis* discovered by Robert Koch (1843-1910).

**Koch's postulates** are four criteria designed to establish a causative relationship between a microbe and a disease (scientific basis which provides the evidence for microorganism to be the causative agent of an infectious disease). The postulates were formulated by Robert Koch and Friedrich Loeffler in 1884.

**Koch's postulates are the following:**

1. The microorganism must be found in abundance in all organisms suffering from the disease, but should not be found in healthy organisms.
2. The microorganism must be isolated from a diseased organism and grown in pure culture.
3. The cultured microorganism should cause disease when introduced into a healthy organism.
4. The microorganism must be re isolated from the inoculated, diseased experimental host and identified as being identical to the original specific causative agent.

In (1865-1869) Louis Pasteur ( French Scientist) discovered the parasite infects the silk worm. Joseph Lister (British scientist ) in 1867 used phenol as disinfectant.

**Host parasite relationship:**

**Symbiosis** - long-term interactions between different biological species, which can be mutualistic, commensal or parasitic

**Parasitism** the two (both) organisms of different species exist in a relationship in which the parasite where benefits while the another is harmed.

**Mutualism** is individual benefits, in which both organisms benefit (such as individual) and bacteria within their intestines (normal flora) .

**Commensalism** is a class of relationship between two organisms where one organism benefits without affecting the other.

**Amensalism**, where one is harmed while the another is unaffected.

**Antagonism** refers to the action of any organism that suppresses or interfere the normal growth and activity of a pathogen, such as the main parts of bacteria or fungi.

### **NORMAL FLORA**

The microorganisms are found normally in the tissues such as intestine, skin , and other mucous membranes . But the blood, brain, muscle, urine etc., are normally free of microorganisms. However, the surface tissues, i.e., skin and mucous membranes, are constantly in contact with environmental

organisms and become readily colonized by various microbial species. The mixture of organisms regularly found at any anatomical site is referred to as the normal flora. The normal flora of humans consists of a few eukaryotic fungi and protists, but bacteria are the most numerous and obvious microbial components of the normal flora.

### Thus we can classify normal flora in to

#### 1-TRANSIENT FLORA

Some of These organisms may be Pathogens (more frequently among the transient flora group ).Some among the normal flora may be opportunists (may be found for hours. Days , weeks) .

#### 2- Resident flora :

Regularly found inside the body (endosymbionts) or on its surfaces (ectosymbionts) usually not pathogenic but may be opportunistic.

**Table 1. Bacteria commonly found on the surfaces of the human body.**

BACTERIUMS	C	N	Ph	M G	Ur	V
<i>Staphylococcus epidermidis</i>	++	+	++	++	++	++
<i>Staphylococcus aureus</i> *	+	+/-	+	+	++	+/-
<i>Streptococcus mitis</i>				+++	+/-	+
<i>Streptococcus salivarius</i>			++	++		
<i>Streptococcus mutans</i> *			+	++		
<i>Enterococcus faecalis</i> *			+/-	+	++	+
<i>Streptococcus pneumoniae</i> *	+/-	+/-	+	+		+/-
<i>Streptococcus pyogenes</i> *	+/-	+/-	+	+	+/-	+/-
<i>Neisseria sp.</i>	+	+	++	+	+	+
<i>Neisseria meningitidis</i> *		+	++	+		+
<i>Enterobacteriaceae</i> *( <i>Escherichia coli</i> )	+/-	+/-	+/-	+	++	+
<i>Proteus sp.</i>	+/-	+	+	+	+	+

S:skin C:conjunctiva N: Nose ph: pharynx M: mouth G: GIT Ur: urethra V: vagina ++ = nearly 100 % + = common (about 25 %) +/- = rare (less than 5%)

### What are the bacterial virulence factors?

Virulence factors can most simply be defined as the character(s) that are directly involved in the development of disease. The term virulence is used to grade the ability of an organism to cause disease.

The measurement of virulence is made by comparing the numbers of organisms necessary to cause disease in a suitable model.

- 1- Adherence to host cells
- 2- Invasiveness
- 3- Iron sequestering
- 4- Virulence factors that inhibits phagocytosis
- 5- Bacterial toxins include ( Exotoxin , Endotoxin )
- 6- Antibiotic resistance
- 7- Super antigen

### • Infectivity:

Describes the ability of an organism to establish itself in a new host and this is defined by the LD50.

The LD50 is used in the measurement of virulence between two strains of the same organism.

**1. Colonization** The first stage of microbial infection is colonization: the establishment of the pathogen at the appropriate portal of entry. Pathogens usually colonize host tissues that are in contact with the external environment. Sites of entry in human hosts include skin and mucous membrane, such as (the urogenital tract, the digestive tract, the respiratory tract and the conjunctiva). In its simplest form, bacterial adherence or attachment to a eukaryotic cell or tissue surface requires the participation of two factors: a receptor and a ligand. The receptors so far defined are usually specific carbohydrate or peptide residues on the eukaryotic cell surface. The bacterial ligand, called an adhesin, is typically a macromolecular component of the bacterial cell surface which interacts with the host cell receptor. There are several terms used to describe adherence factors in microbiology such as (Adhesin, Receptor, Fimbriae, Biofilm, Capsule..etc)

### 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**

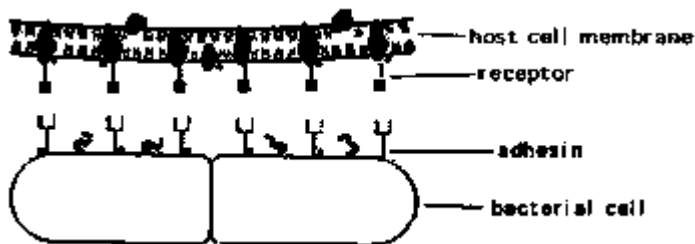
The mechanisms for adherence may involve two steps:

**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 by

- (a) mechanisms for colonization (adherence and initial multiplication)
- (b) production of extracellular substances ("invasins"), that promote the immediate invasion of tissues
- (c) 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.

**How we can differentiate between exotoxin and endotoxin for G+ and some G-**

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 for animals

### Microbial epidemiology

• **Epidemiology** is the science that studies the patterns, causes, and effects of health and disease conditions in defined populations.

The distinction between

- **Epidemic diseases** that are visited upon a population , from
- **Endemic disease** that reside within a population (endemic).

Epidemiologists also study the interaction of diseases in a population, a condition known as a **Syndemic**, is the aggregation of two or more diseases in a population.