Host parasite relationship

Symbiosis is defined as "life together", i.e., that two organisms live in an association with one another.

Types of Symbiotic Associations:-
1. Mutualism:- Both members of the association benefit. For humans, one classic mutualistic association is that of the lactic acid bacteria that live on the vaginal epithelium of a woman
2. Commensalism:- The relationship in which parasite driving nourishment from a host without causing any harm or benefit to the host.
3- Parasitism:- the relationship may be in which one organism damage the other. Parasitism that is the condition is take place when human are invaded by infectious microorganism.

* Saprophyte:- The microorganism that found in nature, not parasite and living on dead organic matter.
* Carrier:- A person with asymptomatic infection that can be transmitted to another susceptible person.
* Infection:- Multiplication of infectious agent within the body. Multiplication of the bacteria that are part of the normal flora of the gastrointestinal tract, skin, etc, is generally not considered an infection, while the multiplication of pathogenic bacteria (eg. Salmonella) even if the person is asymptomatic is deemed an infection.
* Virulence:- Degree of pathogenicity of microorganism gent to cause disease. Virulent agent cause disease when introduce into the host in small number. Virulence involves adherence, invasion and toxigenicity.

   Adherence:- The process by which bacteria stick to the surface cell. Adherence is a major initial step in the infection process.
   Invasion:- The process whereby bacteria, parasite, fungi, and viruses enter host cells and spread in the body.
   Toxiginicity:- The ability of microorganism to produce toxin that contributes to the development of disease.

Bacterial Pathogenesis:-
Pathogen:- is a microorganism that is able to produce disease.
Pathogenicity:- is the ability of a microorganism to cause disease in another organism, namely the host for the pathogen.
Potential pathogens:- are pathogens that live in a commensal or parasitic relationship without producing disease. They do not cause disease in their host unless they have an opportunity brought on by some compromise or weakness in the host's anatomical barriers, tissue resistance or immunity.
Obligate pathogens: are pathogens that don’t associate with their host except in the cause of causing disease.

Opportunistic Pathogens: Bacteria which cause a disease in a immunocompromised host which typically would not occur in a healthy (noncompromised) host are acting as opportunistic pathogens. A member of the normal flora can such as *Staphylococcus aureus* or *E. coli* can cause an opportunistic infection.

Mechanisms of Bacterial Pathogenicity:

Two broad qualities of pathogenic bacteria underlie the means by which they cause disease:

1. The ability to invade tissues: Invasiveness, which encompasses mechanisms for
   a- colonization (adherence and initial multiplication),
   b- ability to bypass or overcome host defense mechanisms, and
   c- the production of extracellular substances which facilitate invasion.
2. The ability to produce toxins: Toxigenesis. Bacteria produce two types of toxins called exotoxins and endotoxins.

* Bacterial Invasions:

1- 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.

a- Hyaluronidase: is the original spreading factor. It is produced by *streptococci*, *staphylococci*, and *clostridia*. The enzyme attacks the interstitial cement ("ground substance") of connective tissue by depolymerizing hyaluronic acid.

b- Collagenase: is produced by *Clostridium histolyticum* and *Clostridium perfringens*. It breaks down collagen, the framework of muscles, which facilitates gas gangrene due to these organisms.

c- Neuraminidase: is produced by intestinal pathogens such as *Vibrio cholerae* and *Shigella dysenteriae*. It degrades neuraminic acid (also called sialic acid), an intercellular cement of the epithelial cells of the intestinal mucosa.

d- Streptokinase and Staphylokinase: 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.

2- Enzymes that Cause Hemolysis and/or Leucolysis:

   These enzymes usually act on the animal cell membrane by insertion into the membrane (forming a pore that results in cell lysis), or by enzymatic attack on phospholipids, which destabilizes the membrane. They may be referred to as lecithinases or phospholipases, and if they lyse red blood cells they are sometimes called hemolysins. Leukocidins, produced by *staphylococci* and streptolysin produced by *streptococci* specifically lyse phagocytes and their granules. These latter two
enzymes are also considered to be bacterial exotoxins. Phospholipases, produced by *Clostridium perfringens* (i.e., alpha toxin), hydrolyze phospholipids in cell membranes by removal of polar head groups. Lecithinases, also produced by *Clostridium perfringens*, destroy lecithin (phosphatidylcholine) in cell membranes.

Hemolysins, notably produced by staphylococci (i.e., alpha toxin), streptococci (i.e., streptolysin) and various clostridia, may be channel-forming proteins or phospholipases or lecithinases that destroy red blood cells and other cells (i.e., phagocytes) by lysis.

3- Coagulase:-- formed by *Staphylococcus aureus*, is a cell-associated and diffusible enzyme that converts fibrinogen to fibrin which causes clotting. Coagulase activity is almost always associated with pathogenic *S. aureus* and almost never associated with nonpathogenic *S. epidermidis*, which has led to much speculation as to its role as a determinant of virulence. coagulase provide:--
   a- prevent phagocytosis by forming fibrin barrier around the bacteria
   b- fibrin barrier around *staphylococci* lesion which help in persist.

* Bacterial toxin:--

1- Exotoxins:--

Exotoxins are soluble proteins, which can act as enzymes. Small amount of exotoxins is needed to make toxicity. They are secreted through their exponential growth or while their cell lysis to the cell surround. So, exotoxins are considered as extra cellular component. Both gram negative and gram positive bacteria produce exotoxins.

Exotoxins are more toxic than endotoxins. Exotoxins are specific to certain bacterial strains. They produce diseases only specific to that toxin. For an example, *Clostridium tetani* produce tetanus toxin. Some times exotoxins act in very remote regions from where they originated by the growth or lysis. Exotoxins can destroy the part of host cells or inhibit their function.

There are three types of exotoxins: Enterotoxins, neurotoxins and cytotoxins. These give an indication of the site of action. Enterotoxins act on lining of GI tract. Neurotoxins act on the function of neurons. Cytotoxins damage the functioning of host cells. Cholera, Diphtheria, tetanus are the some of diseases that are caused by exotoxins. Exotoxins are highly antigenic. Exotoxins can stimulate the immune system. By stimulating the immune system, they produce antitoxins to neutralize the toxin.
2- Endotoxins:-

Endotoxins are part of the outer cell wall of bacteria. Endotoxins are invariably associated with gram negative bacteria as constituents of the outer membrane of the cell wall. Although the term endotoxin is occasionally used to refer to any "cell-associated" bacterial toxin, it should be reserved for the lipopolysaccharide complex associated with the outer envelope of Gram-negative bacteria such as E. coli, Salmonella, Shigella, Pseudomonas, Neisseria, Haemophilus, and other leading pathogens. Lipopolysaccharide (LPS) participates in a number of outer membrane functions that are essential for bacterial growth and survival, especially within the context of a host-parasite interaction.

The biological activity of endotoxin is associated with the lipopolysaccharide (LPS). Toxicity is associated with the lipid component (Lipid A) and immunogenicity (antigenicity) is associated with the polysaccharide components. The cell wall antigens (O antigens) of Gram-negative bacteria are components of LPS. For the most part, endotoxins remain associated with the cell wall until disintegration of the bacteria. Compared to the classic exotoxins of bacteria, endotoxins are less potent and less specific in their action, since they do not act enzymatically. Endotoxins are heat stable. Endotoxins, although strongly antigenic, cannot be converted to toxoids. A comparison of the properties of bacterial endotoxins compared to classic exotoxins is shown in Table.

Table:- CHARACTERISTICS OF BACTERIAL ENDOTOXINS AND EXOTOXINS

<table>
<thead>
<tr>
<th>PROPERTY</th>
<th>ENDOTOXIN</th>
<th>EXOTOXIN</th>
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<tbody>
<tr>
<td>CHEMICAL NATURE</td>
<td>Lipopolysaccharide</td>
<td>Protein</td>
</tr>
<tr>
<td>SOURCE</td>
<td>Cell wall of gram-negative bacteria</td>
<td>Certain sp. of gram-positive and gram negative bacteria</td>
</tr>
<tr>
<td>RELATIONSHIP TO CELL</td>
<td>Part of outer membrane</td>
<td>Extracellular, diffusible</td>
</tr>
<tr>
<td>SECRETED FROM CELL</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>DENATURED BY BOILING</td>
<td>No</td>
<td>Usually</td>
</tr>
<tr>
<td>ANTIGENIC</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>FORM TOXOID</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>TOXICITY</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>HEAT STABILITY</td>
<td>Stable at 100°C for 1h.</td>
<td>Destroyed rapidly at 60°C</td>
</tr>
<tr>
<td>SPECIFICITY</td>
<td>Low degree</td>
<td>High degree</td>
</tr>
<tr>
<td>PYROGENICITY</td>
<td>Yes</td>
<td>Occasionally</td>
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