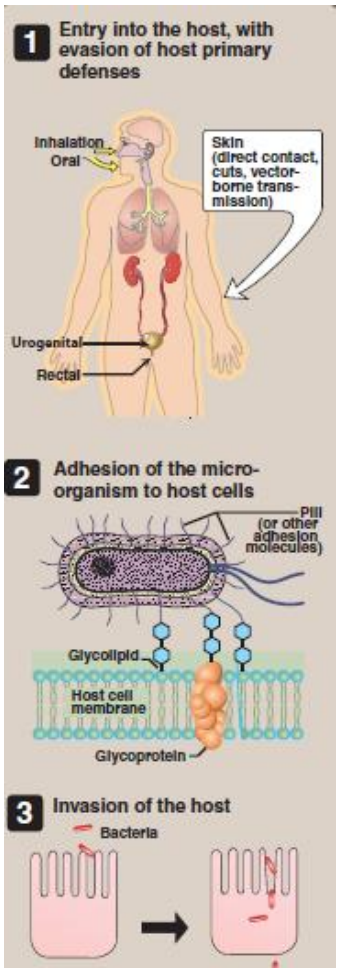
Pathogenesis and virulence:
A pathogenic microorganism is defined as one that is capable of causing disease.
The methods by which bacteria cause disease can be divided into several stages.
Pathogenicity of a microorganism depends on its success in completing some or all of these stages.
The terms "virulence" and "pathogenicity" are often used interchangeably. However, virulence can be quantified by how many organisms are required to cause disease in 50 percent of those exposed to the pathogen (ID50, where I = Infectious and D = Dose), or to kill 50 percent of test animals (LD50, where L = Lethal).
The number of organisms required to cause disease varies greatly among pathogenic bacteria. For example, less than 100 Shigella cause diarrhea by infecting the gastrointestinal (GI) tract, whereas the infectious dose of Salmonella is approximately 100,000 organisms.

The infectious dose of a bacterium depends primarily on its virulence factors. The probability that an infectious disease occurs is influenced by both the number and virulence of the infecting organisms and the strength of the host immune response opposing infection.

Virulence factors

Virulence factors are those characteristics of a bacterium that enhance its pathogenicity, that is, properties that enable a microorganism to establish itself and replicate on or within a specific host. Some of the more important steps in the infectious process are reviewed below.



Entry into the host: The first step of the infectious process
Primary host defense include:

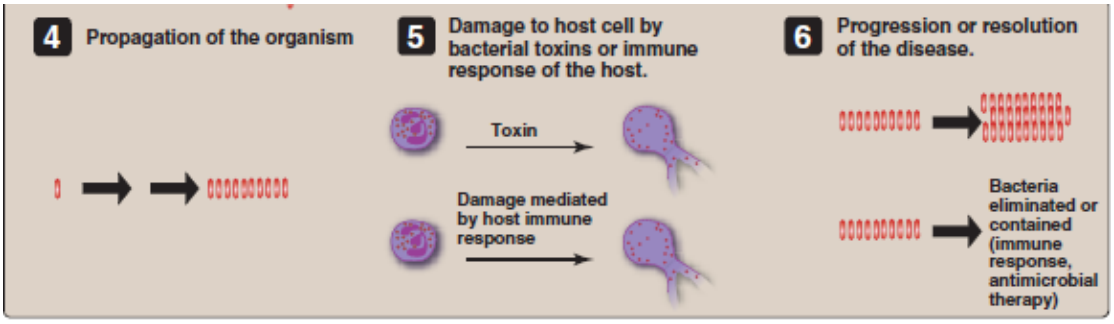
- Phagocytosis
- The acidic environments of the stomach and urogenital tract;
- hydrolytic and proteolytic enzymes found in the saliva, stomach, and small intestine.

Bacteria that have an outer polysaccharide capsule (e,g Streptococcus pneumoniae and Neisseria meningitidis) have a better chance of surviving these primary host defenses.

Adherence to host cells

Escherichia coli use pili
Group A streptococci have similar structures (fimbriae).
Neisseria gonorrhoeae in which strains that lack pili are not pathogenic.

Invasiveness: Invasive bacteria are those that can enter host cells or penetrate mucosal surfaces, spreading from the initial site of infection.
Invasiveness is facilitated by several bacterial enzymes, e,g collagenase and hyaluronidase. which degrade components of the extracellular matrix, providing the bacteria with easier access to host cell surfaces.



Invasion is followed by inflammation, which can be either pyogenic (involving pus formation) or granulomatous (having nodular inflammatory lesions), depending on the organism.
The pus of pyogenic inflammations contains mostly neutrophils, whereas granulomatous lesions contain fibroblasts, lymphocytes, and macrophages.

Bacterial toxins: Some bacteria cause disease by producing toxic substances, of which there are two general types: exotoxins and endotoxin. Exotoxins, which are proteins, are secreted by both gram-positive and gram-negative bacteria. In contrast, endotoxin, which is synonymous with lipopolysaccharide (LPS), is not secreted but instead is an integral component of the cell walls of gram-negative bacteria

Host-mediated pathogenesis
The pathogenesis of many bacterial infections is caused by the host response rather than by bacterial factors. Classic examples of host response–mediated pathogenesis are seen in diseases such as gram-negative bacterial sepsis, tuberculosis, and tuberculoid leprosy. The tissue damage in these infections is caused by various cytokines released from the lymphocytes, macrophages, and polymorphonuclear leukocytes at the site of infection or in the bloodstream. Often the host response is so intense that host tissues are destroyed, allowing remaining bacteria to proliferate.

Antigenic variation
A successful pathogen must evade the host's immune system that recognizes bacterial surface antigens. One important evasive strategy for the pathogen is to change its surface antigens. This is accomplished by several mechanisms. One mechanism, called phase variation, is the genetically reversible ability of certain bacteria to turn off and turn on the expression of genes coding for surface antigens. A second mechanism, called antigenic variation, involves the modification of the gene for an expressed surface antigen by genetic recombination with one of many variable unexpressed DNA sequences. In this manner, the expressed surface antigen can assume many different antigenic structures.