enzymes and acidic pH. In some cases, bacteria kill phagocyte or multiply in macrophages, escaping from hostile ... lymphocytes, play an important role in innate immunity to bacterial infections. During bacterial cell interactions with

the growth of bacteria that penetrate the skin and mucous membranes is competition for iron. Usually, the amount of free ... binds to almost all iron in the blood. Similarly, hemoglobin in erythrocytes binds to iron. Without free iron, bacterial

addition, mucus contains secretion immunoglobins (predominantly sIgA) that are synthesized by plasma cells that ... local antibodies for various intestinal bacteria that colonize the surface of the mucosa. Another mechanism limiting

colonized by normal bacterial flora, which ensures competition for pathogens whose skin is exposed. Similarly, mucus ...

Protective levels of lysozyme, lactoferrin, and lactoperoxidase in mucus either kill bacteria or limit their growth. In

shed. The entire process reportedly takes only 36-48 hours for complete replacement of the epithelium, which reduces the ...

membranes of the respiratory, gastrointestinal, and urogenital systems represent other portals through which bacteria can ...

response are susceptible to frequent recurrent infections with even the least virulent bacteria. The most famous example ...

pathogenicity of an organism. The degree of virulence is directly related to the ability of the organism to cause disease in the host; it is affected by many variables such as the number of bacteria entering the host, the innate immune system, the specific and nonspecific host defense mechanisms, and the virulence factors produced by the pathogen. Several factors affect virulence factors, including: (1) the ability of the pathogen to compete for nutrients, (2) the ability to avoid host defenses, and (3) the ability to cause disease.

The following are the types of virulence factors: Compliance Factor: Many pathogenic bacteria use adherence mechanisms to attach to host cells and then use a variety of enzymes and proteins to escape from the protective mechanisms of the host. These enzymes and proteins include: (a) hemolysins, which lyse red blood cells; (b) neuraminidase, which cleaves sialic acid from glycoproteins and glycolipids and allows bacteria to enter the host; (c) proteases, which destroy proteinacious host defenses; (d) adhesins, which are proteins that bind to specific receptors on host cells; (e) fimbriae, which are hair-like appendages that allow bacteria to attach to host cells; and (f) pili, which are bacterial appendages that allow bacteria to attach to host cells. Other virulence factors include: (a) antigenic variation, which allows bacteria to change their surface antigens in order to escape from the immune system; (b) biofilm formation, which allows bacteria to form a protective matrix that shields them from the immune system; and (c) quorum sensing, which allows bacteria to communicate with each other and coordinate their behavior.

Dedicated to the memory of H. Williams ('Willie') Smith. Your password has been changed Please check your email for instructions on recovering your password. If you don't receive or receive within 10 minutes, your email address may not be registered, and you may need to create a new Wiley Online Library account. Can't get it? Forget your username? Enter your email address below and we will send you your username if the address matches an existing account. You will receive an email containing instructions to retrieve your username and resistance to bacterial infections enhanced by phagocytic cells and a intact immune system. Initial resistance is provided by non-specific mechanisms. Specific immunity develops over time. Susceptibility to some infections is higher in very young and very old patients and immunocompromised patients. Bacterial infection also impairs lymphocytes between bacterial infections and immunocompromised patients. The lack of bacteria in multiplicity and the rapid response to bacterial infections in the host depend on the rapid response to bacterial infections in the host. The rapid response to bacterial infections in the host depends on immunocompetent lymphocytes. Once the body's surface is penetrated, bacteria first enter the body and then they reach the systemic circulation. Bacteria in the body can be eradicated by immune responses. In certain infections (for example, tuberculosis), tissue damage results from toxic mediators released by lymphocytic cells rather than from bacterial toxins. Some bacteria (for example, Staphylococcus aureus) that cause cell death do not need the host's death. Most pathogenic bacteria multiply in tissue fluids and in host cells. Viability factors help bacteria (1) attach to the host's cells, (2) cause disease, and (3) resist host defense. The following are the types of virulence factors: Compliance Factor. Most pathogenic bacteria multiply in tissue fluids, and some do not need the host's death. Most pathogenic bacteria multiply in tissue fluids and in host cells. Viability factors help bacteria (1) attach to the host's cells, (2) cause disease, and (3) resist host defense. The following are the types of virulence factors: Compliance Factor.
Endotoxin gives the host a profound biological effect and may be deadly. Because it is omnipresent in the outer membrane of Gram-negative bacteria, it is a common factor in many infections. Endotoxin is a complex carbohydrate-lipid molecule that is released into the bloodstream when the cell wall of a Gram-negative bacterium is broken down, typically as a result of the host's immune response. Endotoxin can be measured in serum and can be used as a marker for the severity of an infection.

The biological effects of endotoxin have been studied extensively. Purified Lipid A (conjugated to beef serum albumin) is a potent endotoxin that can induce fever, shock, and disseminated intravascular coagulation. More toxic effects include pyrogenicity, leukopenia followed by leukocytosis, complement activation, depression of blood pressure, and anaphylactic shock. These effects can be produced in animals by injection of endotoxin or by incubating blood with endotoxin.

Endotoxins consist of toxic lipopolysaccharide components of the outer membrane of Gram-negative bacteria (see Table 7-2). The most toxic endotoxins are those from Salmonella spp., E. coli, and other enteric bacteria. Endotoxins from these bacteria are more toxic than those from other Gram-negative bacteria. Endotoxins from these bacteria are more toxic than those from other Gram-negative bacteria.

endotoxin, since it is not eliminated either by autolysis or by dialysis. After endotoxin is injected into the bloodstream, it is not cleared from the body by the kidneys. Endotoxin can be measured in serum and can be used as a marker for the severity of an infection.

The toxicity of endotoxin is greatly reduced after hydrolysis of the phosphate group or deacylation of one or more fatty acids. The molecular structure of endotoxin from Salmonella spp. and E. coli is known to be a complex carbohydrate-lipid molecule that is released into the bloodstream when the cell wall of a Gram-negative bacterium is broken down, typically as a result of the host's immune response. Endotoxin can be measured in serum and can be used as a marker for the severity of an infection.

The protective effect of endotoxin is mediated by the production of antibodies that neutralize its toxic effects. Antibodies to endotoxin are produced in response to infection with Gram-negative bacteria. Antibodies to endotoxin are produced in response to infection with Gram-negative bacteria.

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