Chapter 19 Host-Microbe Interactions

Summary Outline

19.1 The skin and mucous membranes constitute an anatomical barrier to invading microorganisms and supply the foundation a complex **ecosystem**.

19.2 **Normal flora** is comprised of the microorganisms that grow in or on the body without producing obvious harmful effects.

- A. **Symbiosis** describes the living together of two dissimilar organisms.
  1. **Commensalism**: One partner benefits, the other is unaffected.
  2. **Mutualism**: Both partners benefit in mutualism.
  3. **Parasitism**: The parasite benefits at the expense of the host.
- B. The **normal flora** is **acquired at birth** and **changes** in response to **variations in the environment**, such as changes in diet, acidity, or antibiotic intake.
- C. **Normal flora inhibits potentially harmful organisms** by **preventing attachment**, **competing for essential nutrients**, **producing antimicrobial substances, stimulating the immune system** and **inducing** the production of **antibodies** that cross-react with potential pathogens.

19.3 Principles of infectious disease

- A. Colonization of a host
  1. **Infection** occurs when parasitic organisms grow in or on the host.
  2. **Disease** occurs if the infection causes damage.
  3. **Symptoms** are the effects of the disease reported by the patient.
  4. **Signs** are effects of the disease that can be observed.
  5. **A primary infection** is the initial infection.
  6. **A secondary infection** occurs as a result of the primary infection.
- B. Pathogenicity
  1. **Pathogens** are **disease-producing organisms.**
  2. **Opportunist** cause disease when the body's defense systems are compromised
  3. **Virulence**, the degree of pathogenicity, describes the properties (**virulence factors**) of an infectious agent that promote its pathogenicity.
- C. **Infectious diseases** are **communicable** or **contagious** and spread between people or animals.
- D. **Stages** of infectious diseases
  1. **Incubation period**
  2. **Period of illness**
  3. **Convalescence**
- E. **Infections** may be **acute** or **chronic,** or **latent.** Infections may be **localized** or **systemic**.

19.4 **Establishing the cause** of an infectious disease

- A. **Koch's postulates** are used with organisms that can be cultured in the laboratory to demonstrate the cause of an infectious disease.
- B. **Molecular postulates** use genes to identify virulence factors in pathogenic strains of organisms.

19.5 Establishment of infection

- A. **Adherence**: Microorganisms adhere to host cell receptors by means of adhesins.
- B. **Colonization**: Microorganisms colonize the host in order to become established.
- C. Type III secretion systems of Gram-negative bacteria allow them to deliver compounds directly to the host cells.

19.6 **Invasion of tissues**: A pathogen may enter through a break in the skin or mucous membranes, destroy the basement membrane underlying epithelial cells, or go between or through the cells of the membrane. Some may be endocytosed or phagocytized.

19.7 Avoiding the host defenses

A. **Pathogens** make a variety of substances that **inhibit phagocytosis** and **complement** activities.
B. Some bacteria can evade host defenses by remaining inside of host cells.
C. Some **serum-resistant** bacteria can interfere with the activation of the complement system via the alternative pathway, thus postponing the formation of the membrane attack complex.
D. Bacterial mechanisms can prevent encounters with phagocytes and destroy phagocytic cells.
E. Bacterial means of avoiding recognition and attachment by phagocytes include **capsules, M protein, Mac proteins,** and **Fc receptors.**
F. Mechanisms for the bacteria to survive within the phagocyte include:
    1. Escape from the phagocyte
    2. Preventing phagosome-lysosome fusion
    3. Surviving within the phagosome
D. Mechanisms to avoid antibodies include **IgA protease, antigenic variation**, and mimicking the host.

19.8 Damage to the host

A. **Exotoxins** are powerful toxic proteins with specific damaging effects. They may be classified as **neurotoxins, enterotoxins,** or **cytotoxins** based on the kinds of host cells that they damage.
B. **Superantigens** bind directly to many T cells at sites distinct from the regular antigen receptor sites. This causes the **release of** huge amounts of **cytokines** that have **toxic effects** in such large quantities.
C. **Endotoxins of Gram-negative cell walls** consist of lipopolysaccharides that contain a **toxic lipid A portion** and an **O polysaccharide antigen**. **Lipid A** is responsible for the toxic properties that include **fever** and sometimes **shock**.
D. **Peptidoglycan** and some other bacterial components induce various cells to produce proinflammatory **cytokines**.
F. **Cytolytic toxins** and **toxic enzymes** break down cells or tissue components.
G. **Antigen-antibody complexes** can cause damage to the kidneys and joints; **cross-reactive antibodies** can promote an autoimmune response.

19.9 Mechanisms of viral pathogenesis

A. Binding to host cells and invasion
    1. Some viruses bind to and infect mucous membrane cells; others enter at sites that are damaged or penetrated.
    2. Viruses attach to specific receptors on the target cells.
B. Avoiding immune responses
    1. Some viruses can avoid the effects of interferon.
    2. Some viruses can regulate **apoptosis** of the host cell.
    3. Some viruses transfer directly from cell to cell in order to avoid antibodies.
    4. The surface antigens of some viruses change more quickly than the production of complimentary antibodies.
C. Viruses and host damage: Viruses can damage the host by:
    1. Direct damage
    2. Virally induced apoptosis
    3. Altering the immune response to the infection

19.10 Mechanisms of eukaryotic pathogenesis

A. Fungi: Saprophytic fungi are usually opportunistic; dermatophytes cause superficial infections of the skin, hair, and nails. The dimorphic fungi cause the most serious fungal infections.
B. Eukaryotic parasites attach to specific receptors on host cells. They use a variety of mechanisms to avoid antibodies; the damage varies considerably.