CHAPTER 18 THE COCCI OF MEDICAL IMPORTANCE

Gram-positive and gram-negative cocci that cause infection are presented. The difference between commensal and pathogenic strains is explained, because many of the cocci are part of the normal flora or commensals. The general characteristics of *Staphylococcus*, *Streptococcus*, *Enterococcus*, and *Neisseria* are reviewed. The clinical presentations of diseases caused by members of these genera are presented, as well as host defenses, treatment, prevention, and laboratory cultivation and diagnosis.

Learning Objectives

A student should learn the following concepts:
1. Infections with gram-positive and gram-negative cocci tend to stimulate pus formation (pyogenic cocci).
2. The genus *Staphylococcus* inhabits the skin and mucous membranes, is typically gram-positive, and does not contain spores or flagella.
3. *Staphylococcus aureus* is a facultative anaerobe that can withstand a range of temperatures, salt, and pH.
4. The pathogenicity of *Staphylococcus aureus* arises from a combination of different virulence factors.
5. *Staphylococcus aureus* is a common intimate associate of humans; the pathogen is harbored intermittently rather than chronically.
6. *Staphylococcus aureus* infections can range from localized to systemic depending on the degree of invasion or toxin production.
7. Staphylococci produce biofilms on inanimate objects placed into the body.
8. *Staphylococcus aureus* coagulates plasma.
9. Staphylococci are notorious in their acquisition of resistance to new drugs.
10. The increasing incidence of nosocomial outbreaks of staphylococcal infections requires consistent practice of universal precautions by hospital staff.
11. Streptococci are non-spore-forming, non-motile, facultative anaerobic, gram-positive organisms.
12. *Streptococcus pyogenes* is the most serious streptococcal pathogen of humans; this species can generate a substantial array of surface antigens, toxins, and enzymes.
13. Group A streptococcal infections that resolve may occasionally result in complications such as rheumatic fever or acute glomerulonephritis.
14. Most strains of *Streptococcus pyogenes* continue to be sensitive to penicillin.
15. Subacute endocarditis is a serious complication of streptococcal infections caused by the Viridans group; these blood-borne bacteria settle on abnormal areas of the heart and form a biofilm.
16. *Streptococcus pneumoniae* is responsible for 60–70% of all bacterial pneumonias, as well as meningitis and otitis media.
17. Pneumonia occurs when mucus containing a load of bacterial cells is aspirated from the pharynx into the lungs of susceptible individuals.
18. *Neisseria* are typically gram-negative, non-spore forming, aerobic, capsular organisms.
19. Gonococcus virulence is attributed to pili and other surface molecules that slow phagocytosis and promote attachment.
20. Gonococci tend to be phagocytosed by phagocytes and remain viable; therefore, the presence of gram-negative diplococci in neutrophils often suggests gonococci infection.
21. *Neisseria gonorrhoeae* causes infections of the male and female reproductive tracts.
22. *Neisseria meningitidis* is carried in the nasopharynx and can cause meningitis.
23. The genera *Branhamella*, *Moraxella*, and *Acinetobacter* are emerging species in causing diseases in hosts with compromised immune systems.

**Chapter Outline (also see Chapter Summary with Key Terms p. 560)**

18.1 General Characteristics of the Staphylococci
   - Growth and Physiological Characteristics of *Staphylococcus aureus*
     - Virulence Factors of *Staphylococcus aureus*
       - Enzymes
         - Coagulase
         - Staphylokinase
         - DNAse
         - Lipase
       - Toxins
         - Hemolysin
         - Leukocidin
         - Enterotoxins
         - Exfoliative Toxin
         - Toxic Shock Syndrome Toxin
     - Epidemiology and Pathogenesis of *Staphylococcus aureus*
       - MRSA
   - The Scope of Clinical Staphylococcal Disease
     - Localized Cutaneous Infections
       - Folliculitis
       - Furuncle
       - Carbuncles
       - Bullous Impetigo
     - Miscellaneous Systemic Infections
       - Osteomyelitis
       - Pneumonia
       - Bacteremia
     - Toxigenic Staphylococcal Disease
       - Toxic Shock Syndrome
       - Food Poisoning
       - Scalded Skin Syndrome
• Host Defenses Against *Staphylococcus aureus*

• Other Important Staphylococci
  o Coagulase-Negative Staphylococci
    ▪ *Staphylococcus epidermidis*
    ▪ *Staphylococcus saprophyticus*

• Identification of *Staphylococcus* Isolates in Clinical Samples

• Clinical Concerns in Staphylococcal Infections
  o Treatment of Staph Infections
  o Prevention of Staph Infections

18.2 General Characteristics of the Streptococci and Related Genera

• β-Hemolytic Streptococci: *Streptococcus pyogenes*
  o Cell Surface Antigens and Virulence Factors
    ▪ Major Extracellular Toxins
      • Streptolysin
      • Erythrogenic Toxin
      • Superantigens
    ▪ Major Extracellular Enzymes
      • Streptokinase
      • Hyaluronidase
  o Epidemiology and Pathogenesis of *Streptococcus pyogenes*
    ▪ Skin Infections
      • Streptococcal Impetigo
      • Erysipelas
      • Streptococcal Pharyngitis
      • Necrotizing Fasciitis
    ▪ Systemic Infections
      • Scarlet Fever
      • Streptococcal Pneumonia
      • Streptococcal Toxic Shock Syndrome
  o Long-Term Complications of Group A Infections
    ▪ Rheumatic Fever
    ▪ Acute Glomerulonephritis

• Group B: *Streptococcus agalactiae*
  o Neonatal Infections
  o Skin and Wound Infections
  o Endocarditis

• Group D Enterococci and Groups C and G Streptococci

• Laboratory Identification Techniques

• Treatment and Prevention of Group A, B, and D Streptococcal Infections

• α-Hemolytic Streptococci: The Viridans Group
  o Subacute Endocarditis

• *Streptococcus pneumoniae*: The Pneumococcus
Epidemiology and Pathology of the Pneumococcus
  - The Pathology of *Streptococcus pneumoniae*
    - Lobar Pneumonia
      - Consolidation
    - Otitis Media
  - Laboratory Cultivation and Diagnosis
  - Treatment and Prevention of Pneumococcal Infections
    - Pneumovax
    - Prevnar

18.3 The Family Neisseriaceae: Gram-Negative Cocci
  - *Neisseria gonorrhoeae*: The Gonococcus
    - Factors Contributing to Gonococcal Pathogenicity
    - Epidemiology and Pathology of Gonorrhea
      - Genital Gonorrhea in the Male
      - Genitourinary Gonorrhea in the Female
      - Extragenital Gonococcal Infections in Adults
      - Gonococcal Infections in Children
    - Clinical Diagnosis and Control of Gonococcal Infections
  - *Neisseria meningitidis*: The Meningococcus
    - Epidemiology and Pathogenesis of Meningococcal Disease
    - Clinical Diagnosis of Meningococcal Disease
    - Immunity, Treatment, and Prevention of Meningococcal Infection
  - Differentiating Pathogenic from Nonpathogenic *Neisseria*
  - Other Genera of Gram-Negative Cocci and Coccobacilli

**Student Activities**

1. Ask students to research the universal precautions that should be taken in a hospital to reduce the rate of nosocomial infections. Then give students the choice of writing an essay on these precautions or putting on a skit to demonstrate the precautions that should be taken. Have students determine how they would handle soiled linen and sharps. Ask students which body fluids they would be concerned about, and what types of protective clothing/eyewear they would use. Also request that students include in their presentations when they would wash their hands. The following websites are of use: [http://www.cdc.gov/handhygiene/](http://www.cdc.gov/handhygiene/) and [http://www.thebody.com/content/whatis/art5982.html](http://www.thebody.com/content/whatis/art5982.html).

2. Provide students with two “unknown” cultures of *Streptococcus* (preferably one beta-hemolytic and one alpha-hemolytic), and ask students to identify the cultures. Review Figure 18.9 on p. 544. If reagents are available, review Table 18.4 on p. 550 and ask students to further characterize their unknown β-Hemolytic Streptococci into either Group A, Group B, Groups C/G, or Group D.

3. To determine if one is a carrier of *Staphylococcus* or *Streptococcus*, a nasopharyngeal swab is taken and cultured. This test produces some discomfort and is usually only performed by medical personnel. To loosely replicate this
procedure, ask students to do a nasal swab (insert a sterile swab into the nasal cavity, less than one inch, and rotate swab a few times against the nasal cavity) and then streak a blood agar plate. Have students perform a Gram stain, catalase tests, and coagulase tests (reviewed in Figure 18.6 on p. 541) to determine if they are carriers of Staphylococcus or Streptococcus.


Classroom Discussion

1. The textbook discusses the well-developed human resistance to staphylococcal infections. This human resistance is often based on the skin being intact. As discussed in Chapter 14, the skin as a barrier provides a fantastic nonspecific host defense. Talk with students about wound healing and infection, highlight why a wound is needed not only for the entry of staph into the body, but also for the dissemination of the disease. When a wound is introduced into the skin, the body is repairing the wound as well as trying to protect the exposed region from microbes. Even non-infected wounds create stress, which can also cause the immune system to be compromised.

2. Superantigens are mentioned on p. 546: “Some of the streptococcal toxins contribute to increased tissue injury by acting as superantigens.” Discuss with students the definition of a superantigen: molecules that can stimulate an entire subset of T cells, not binding to the specific TCR discussed in Chapter 15. Superantigens bind to the outer surface of a TCR and the MHC II receptor of an antigen-presenting cell and stimulate anywhere from 2 to 20% of all T cells in a process referred to as polyclonal T cell activation. Because this is nonspecific stimulation of the immune system, it does not lead to adaptive immunity. Superantigens cause an enormous release of cytokines that suppress the immune response and contribute to toxicity. The following website provides an animation demonstrating how superantigens circumvent the normal mechanisms of T cell activation: http://public-1.cryst.bbk.ac.uk/sagdb/intro.html.

3. Bacteria develop resistance to antimicrobial agents by acquiring new genes or by mutations in their genomes. Ask students to think about why staphylococci are resistant to numerous antibiotics, while streptococci generally do not have a wide resistance to antibiotics. Does it have to do with the ability of staph to acquire genes, or its rate of mutation? Ask students how they think staph might have acquired genes encoding antimicrobial resistance (plasmids, transposons, bacteriophages). Many antibiotic resistance genes encode enzymes which breakdown the antibiotic, or encode altered cell surface receptors or proteins that prevent binding of the antibiotic.
Applicable Online Quizzes

www.mhhe.com/talaro7

Superantigens
Chapter Quiz

Key Terms and Phrases

Staphylococcus  Staphylococcus  otitis media
pyogenic cocci  saprophyticus  pneumococcus
Streptococcus
coagulase  Viridans  lobar pneumonia
hemolysins  Viridans streptococci  consolidation
leukocidin  streptolysins  prevnar
enterotoxins  erythrogenic toxin  Neisseria
exfoliative  superantigens  gonorrhea
toxic shock syndrome  impetigo  gonorococcus
toxin  pyoderma  salpingitis
abscess  erysipelas  PPNG
furuncle  streptococcal  MRSA
 carbuncle  pharyngitis  meningococcus
impetigo  purulent  cerebrospinal meningitis
impetigo  scarlet fever  petechiae
osteomyelitis  rheumatic fever  ecchymoses
staphylococcal scalded  acute glomerulonephritis  endocarditis
skin syndrome  subacute endocarditis  meningococcus
coagulase-negative  Branhamella catarrhalis
Staphylococcus  Moraxella
Staphylococcus  Pneumoniae  Acinetobacter
epidermidis

Instructors are encouraged to visit the Foundations in Microbiology ARIS (Assessment, Review, Instruction System) site at www.mhhe.com/talaro7 for animations of key processes, online quizzing, case presentations, and more.