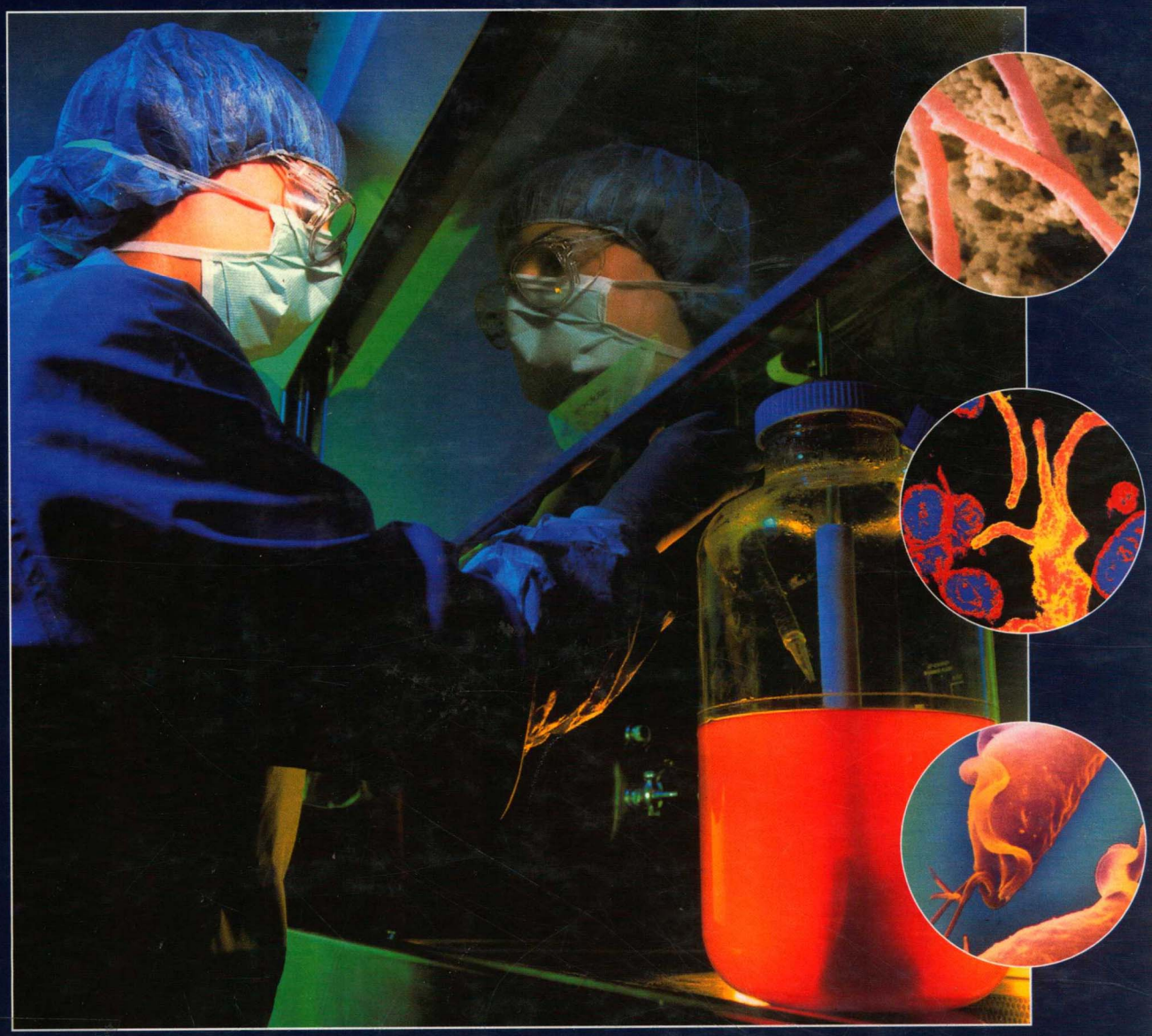
MICROBIOLOGY for the Health Sciences



FOURTH EDITION

Jensen Wright Robison

FOURTH EDITION

Microbiology for the Health Sciences

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BACTERIAL DISEASES

Disease	Organism	Shape/Staining
acne	*Propionibacterium acnes*	R, +
actinomycosis	*Actinomyces israelii*	I, +
anthrax	*Bacillus anthracis*	R, +
bacterial meningitis	*Haemophilus influenzae*	R, –
	Neisseria meningitidis	C, –
	Streptococcus pneumoniae	C, +
	Listeria monocytogenes	R, +
bacterial vaginitis	*Gardnerella vaginalis*	R, –
botulism	*Clostridium botulinum*	R, +
brucellosis (undulant fever, Malta fever)	*Brucella* sp.†	CB, –
cat scratch fever	*Afipia felis, Bartonella henselae*	R, – CB, NA
chancroid	*Haemophilus ducreyi*	R, –
cholera (Asiatic cholera)	*Vibrio cholerae*	vibrio, –
conjunctivitis	*Haemophilus aegyptius*	CB, –
dental caries	*Viridans Streptococci*	C, +
diphtheria	*Corynebacterium diphtheriae*	R, +
ehrlichiosis	*Ehrlichia* sp.	R, NA
endocarditis	*Enterococcus* sp.	C, +
food poisoning	*Staphylococcus aureus*	C, +
	Streptococcus pyogenes	C, +
	Clostridium perfringens	R, +
	Clostridium botulinum	R, +
	Bacillus cereus	R, +
	Listeria monocytogenes	R, +
	Campylobacter sp.	R, –
	Shigella sp.	R, –
	Salmonella sp.	R, –
	Vibrio parahaemolyticus	R, –
gas gangrene	*Clostridium perfringens* and others	R, –
gonorrhea	*Neisseria gonorrhoeae*	C, –
granuloma inguinale (donovanosis)	*Calymmatobacterium granulomatis*	R, –
Hansen's disease (leprosy)	*Mycobacterium leprae*	R, A-F
Legionnaires' disease (legionellosis)	*Legionella pneumophilia*	R, –
leptospirosis	*Leptospira interrogans*	S, –
listeriosis	*Listeria monocytogenes*	R, +
Lyme disease	*Borrelia burgdorferi*	S, –
lymphogranuloma venereum	*Chlamydia trachomatis*	coccoid, NA
Madura foot (maduromycosis)	*Actinomyces, Streptomyces, Nocardia*	I, +, some A-F
nongonococcal urethritis (NGU)	*Chlamydia trachomatis*	R, VAR
	Ureaplasma urealyticum	I, NA
ornithosis (psittacosis)	*Chlamydia psittaci*	coccoid, NA
peptic ulcer	*Helicobacter pylori*	R, –
pharyngitis (strep throat)	*Streptococcus pyogenes*	C, +
plague (black death) bubonic plague pneumonic plague	*Yersinia pestis*	R, –
pneumonia	*Streptococcus pneumoniae*	C, +
	Klebsiella pneumoniae	R, –
pneumonia, atypical (walking pneumonia)	*Mycoplasma pneumoniae*	I, NA
pseudomembranous colitis	*Clostridium difficile*	R, +
puerperal fever (childbed fever)	*Streptococcus pyogenes*	C, +
Q fever	*Coxiella burnetti*	CB, NA
relapsing fever	*Borrelia* sp.	S, –
rheumatic fever	*Streptococcus pyogenes*	C, +
rickettsialpox	*Rickettsia akari*	CB, NA
Rocky Mountain spotted fever	*Rickettsia rickettsii*	CB, NA
salmonellosis	*Salmonella* sp.	R, –
shigellosis (bacillary dysentery)	*Shigella* sp.	R, –
skin and wound infections (scalded skin syndrome, scarlet fever, erysipelas, impetigo, etc.)	*Staphylococcus aureus*	C, +
	Staphylococcus epidermidis	C, +
	Streptococcus sp.	C, +
	Providencia stuartii	R, –
	Pseudomonas aeruginosa	R, –
	Serratia marcescens	R, –
syphilis	*Treponema pallidum*	S, –
tetanus	*Clostridium tetani*	R, +
toxic shock syndrome	*Staphylococcus aureus*	C, +
trachoma	*Chlamydia trachomatis*	coccoid, NA
trench fever	*Rochalimaea quintana*	CB, NA
tuberculosis	*Mycobacterium tuberculosis*	R, A-F
tuberculosis, avian	*Mycobacterium avium*	R, A-F
tularemia	*Francisella tularensis*	R, –
typhoid fever	*Salmonella typhi*	R, –
typhus, epidemic	*Rickettsia prowazekii*	CB, NA
typhus, endemic (murine typhus)	*Rickettsia typhi*	CB, NA
typhus, recrudescent (Brill-Zinsser disease)	*Rickettsia prowazekii*	CB, NA
typhus, scrub (tsutsugamushi disease)	*Rickettsia tsutsugamushi*	CB, NA
vibriosis	*Vibrio parahaemolyticus*	R, –
whooping cough (pertussis)	*Bordetella pertussis*	CB, –
yersiniosis	*Yersinia enterocolitica*	R, –

***Key:**

Shape	Staining
C = coccus	– = gram-negative
CB = coccobacillus	+ = gram-positive
R = rod	VAR = gram-variable
S = spiral	A-F = acid-fast
I = irregular	NA = not applicable

† Species

VIRAL DISEASES

Disease	Virus	Reservoir
bronchitis, rhinitis	parainfluenza	humans, some other mammals
Burkitt's lymphoma	Epstein-Barr	humans
chickenpox	varicella-zoster	humans
coryza (common cold)	rhinovirus	humans
	coronavirus	humans
cytomegalic inclusion disease	cytomegalovirus	humans
Dengue fever	Dengue	humans
encephalitis	Colorado tick fever	mammals
	Eastern equine encephalitis	birds
	St. Louis encephalitis	birds
	Venezuelan equine encephalitis	rodents
	Western equine encephalitis	birds
epidemic keratoconjunctivitis	adenovirus	humans
hantavirus pulmonary syndrome	bunyavirus	rodents
hemorrhagic fever, Bolivian	arenavirus	rodents and humans
hemorrhagic fever, Korean	bunyavirus (Hantaan)	rodents
hemorrhagic fever	Ebola virus (filovirus)	humans (?)
	Marburg virus (filovirus)	humans (?)
hepatitis A (infectious hepatitis)	hepatitis A	humans
hepatitis B (serum hepatitis)	hepatitis B	humans
hepatitis C (non-A, non-B)	hepatitis C	humans
hepatitis D (delta hepatitis)	hepatitis D	humans
hepatitis E (enterically transmitted non-A, non-B, non-C)	hepatitis E	humans
herpes, oral	usually herpes simplex type 1, sometimes type 2	humans
herpes, genital	usually herpes simplex type 2, sometimes type 1	humans
HIV disease, AIDS	human immunodeficiency virus (HIV)	humans
infectious mononucleosis	Epstein-Barr	humans
influenza	influenza	swine, humans (type A) humans (type B) humans (type C)
Lassa fever	arenavirus	rodents
measles (rubeola)	measles	humans
meningoencephalitis	herpes	humans
molluscum contagiosum	poxvirus group	humans
mumps	paramyxovirus	humans
pneumonia	adenoviruses, respiratory syncytial virus	humans
poliomyelitis	poliovirus	humans
rabies	rabies	all warm-blooded animals
respiratory infections	adenovirus paramyxoviruses	humans none
Rift Valley fever	bunyavirus (phlebovirus)	humans, sheep, cattle
rubella (German measles)	rubella	humans
shingles	varicella-zoster	humans
smallpox	variola (major and minor)	humans
viral enteritis	rotavirus	humans
warts, common (papillomas)	human papillomavirus	humans
yellow fever	yellow fever	monkeys, humans, mosquitoes

The tables of fungal and protozoal diseases appear on the back cover endpapers

Microbiology
for the
Health Sciences

Preface

Microbiology for the Health Sciences has always been written with the idea that a meaningful introduction to microbiology can be provided for the health-science oriented student in a single semester or quarter of study without demanding earlier exposure to the subject or an extensive background in chemistry and mathematics. Although the text provides the necessary background support for extended study in the discipline, the primary purpose of the text is to provide a useful and basic understanding of the microbe in its role as a disease-producing agent. Thus, the content has been kept at a minimum in an effort to provide only that information essential to an appreciation of microorganisms in the disease process.

Because of its narrower scope, *Microbiology for the Health Science* is intended to be neither an encyclopedic reference of general microbiology nor a detailed analysis of host responses to parasitic microorganisms. Rather, the reader will find that this text provides a succinct, easy-to-read background of the agents of infectious diseases and the human disease processes associated with microorganisms.

In reviewing the fourth edition we have tried to retain the innovative style and student-oriented nature of the first three editions. However, we recognize that advances in the discipline have been rapid and far-reaching, and we've done our best to keep pace in terms of including appropriate updates throughout the text. Our new co-author, Richard Robison, received his doctorate in immunology and has taught the introductory course for many years. His involvement has helped to ensure that the material is up-to-date and accurate in one of the most exciting and rapidly changing fields, immunology.

In a sense, an introductory text like this is in a constant state of revision, because the abundance of scientific discoveries and research milestones of our day demand that we as authors categorize, prioritize, and then integrate new facts and findings into our text—never an easy task, but always a rewarding one.

TAXONOMIC ORGANIZATION

Microbiology for the Health Sciences takes a taxonomic approach to introduce students to the pathogens of medical significance—that is, the chapters are organized according to taxonomic groups. Within each chapter, important members of the group are discussed in terms of their physical characteristics. This brief introduction to the infectious agent is always followed then by a systematic discussion of the disease(s) it causes: pathogenesis and clinical manifestations; transmission and epidemiology; diagnosis and treatment; and prevention and control. Throughout our many years of teaching, we have found that students are able to assimilate the information more readily in this kind of standardized format.

A **Graphic Sidebar**, which visually depicts the basic characteristics of the group under discussion, is included in the opening page of each organismal chapter. This serves as a quick reference to remind students of the common attributes of members of the group: its basic "cell type"—whether it is a prokaryote, eukaryote, or virus; its Gram morphology (for bacteria); its general shape(s); and its general size or size range.

AN EMPHASIS ON CLINICAL ASPECTS

Designed for students entering nursing or other allied-healthcare professions, this text has always had a clinical focus. However we've introduced several new clinical elements that will help students remember this important aspect of their microbiology course:

Affected Body Systems. This new chapter feature highlights the systems of the body affected by the organism(s) discussed in the chapter. Found near the beginning of each organismal chapter, this provides a quick overview of the clinical discussion ahead.

Disease/Causative Organism Reference and Notifiable Diseases Summary. The printed endpapers of the text feature a quick-reference alphabetical listing of diseases and the organisms that cause them. On the back endpapers, notifiable diseases also summarized by year and numbers of reported cases.

Clinical Notes. One of the most popular chapter features from earlier editions of this text, these

real-life accounts—based on the Morbidity and Mortality Weekly Reports (MMWR) issued by the Centers for Disease Control—have been updated and expanded. These valuable boxes focus on pertinent on-going concerns in infectious disease microbiology and provide the student with an opportunity to see the study of microbiology from an applied perspective.

Clinical Summary Tables. Found in the end-of-chapter Material for Review, this new feature provides a succinct review of the clinical content regarding each of the major microbes discussed in the chapter: its virulence mechanisms, the diseases it causes, its mode of transmission, and methods of treatment and prevention.

VOCABULARY DEVELOPMENT

We recognize that building vocabulary is an important task in any scientific discipline. Therefore, we have included the following aids for students:

Key Terms/New Words. The most important words for the chapter are boldfaced where they are defined in the text, while additional terms with which the student might not be familiar (but which are not necessarily "key terms") are italicized. All boldfaced and italicized terms are included in the **Glossary** at the end of the text.

Running Glossary. A running glossary is provided to expand the student's understanding of terms *as they are used.* These bottom-of-the-page entries reduce the time required to read the text and later provide excellent chapter review material for the student

END-OF-CHAPTER REVIEW ELEMENTS

Concept Summary. This numbered list of concepts, written in sentence form, recaps the essential points of the chapter.

Clinical Summary Table. This table provides a quick review of the organisms covered in the chapter and some specifics of the diseases they cause.

Study Questions. These 5–10 questions are designed for students to recall the important facts of the chapter.

Challenge Questions. These 2–3 questions ask the student to go beyond mere memorization of the facts to apply their knowledge to a specific problem.

SUPPLEMENTS

This text is accompanied by a variety of supplements for the student and the instructor.

Combined Instructor's Manual and Test Item File. Each chapter in this resource, developed by Jeffrey Pommerville of Glendale Community College, contains a chapter overview, teaching tips, instructor goals, and student learning objectives—plus answers to the Study and Challenge Questions found in the main text. Additionally, the test item portion contains approximately 30–50 questions for each of the 40 chapters in the book.

Transparency Masters. Transparency masters featuring all of the illustrations from the text is available to qualified adopters of the text.

Student Study Guide. The Study Guide, also developed by Jeffrey Pommerville of Glendale Community College, contains chapter outlines, objectives, vocabulary exercises, and numerous questions and exercises to help students master the material.

Prentice Hall Microbiology Laserdisc. Prentice Hall Microbiology Laser Disc contains more than 2000 images, including full-color micrographs, photographs, illustrations, and animations for use in either a lecture or lab setting. All images are indexed and accessible with or without a bar code scanner. Free to adopters with a minimum of 150 copies.

Prentice Hall/New York Times Themes of the Times. *The New York Times* Themes of the Times consists of selected articles from *The New York Times* dealing with topics related to microbiology. This supplement is updated annually and is available free to adopters, who can order as many copies as the number of new texts purchased.

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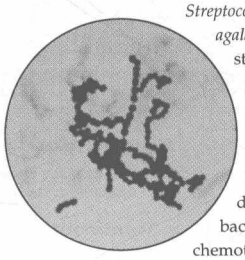
Marcus M. Jensen
Donald N. Wright
Richard A. Robison

CHAPTER

15 STREPTOCOCCI

OUTLINE

Both pathogenic and nonpathogenic species of streptococci are commonly associated with humans and animals. A wide variety of species are present as normal flora on skin and mucous membranes of all humans. Three species, *Streptococcus pyogenes*, *Streptococcus pneumoniae*, and *Streptococcus agalactiae*, are responsible for most of the streptococcal infections in humans. The pathogenic species produce a wide variety of toxins and cause a wide variety of lesions and diseases. Historically, some streptococcal diseases have been among the most serious diseases of humans. Fortunately, these bacteria are usually easily destroyed by chemotherapeutic agents, and as a result, even though streptococcal infections are still common, their impact on illness and death today is only a small fraction of what it was prior to the 1930s.



STREPTOCOCCI: GENERAL CHARACTERISTICS

Streptococci are gram-positive, coccal-shaped bacteria that usually appear in chains of various lengths (Figure 15-1; see Plate 5). These bacteria are moderately resistant to environmental factors; that is, they may remain living for days to weeks after being expelled from
readily killed
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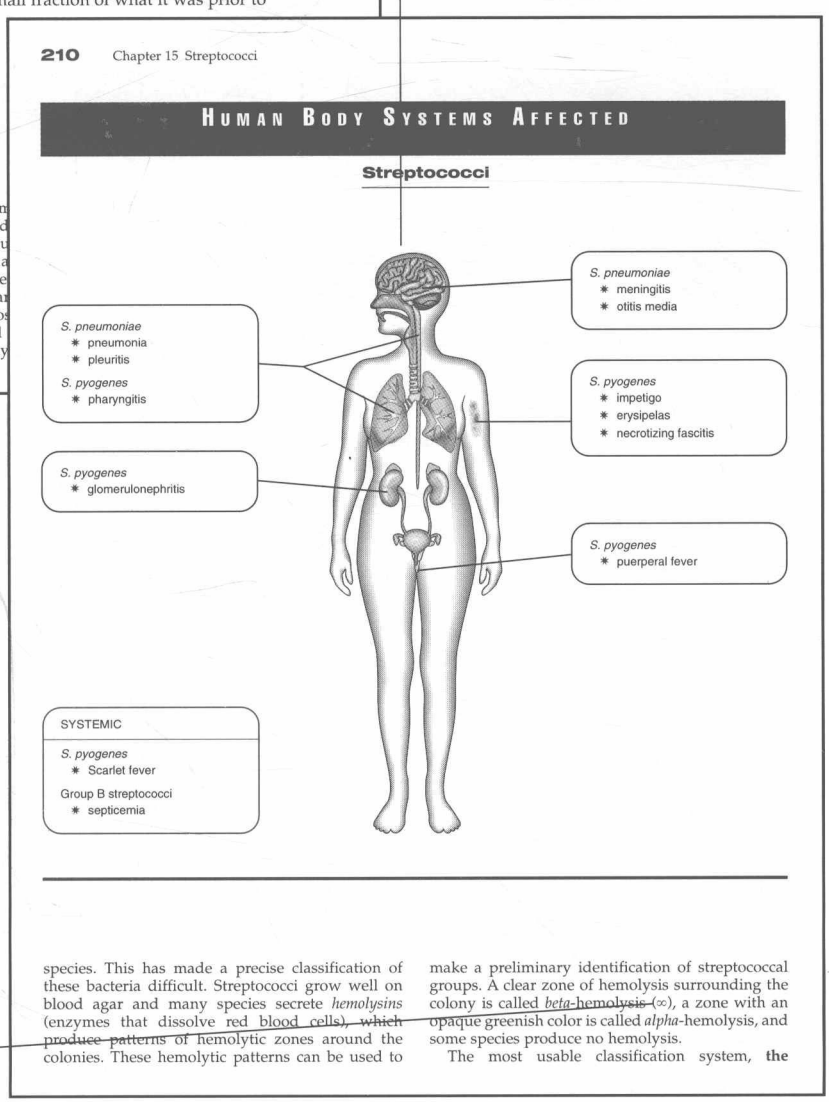
Chapter Outline
Lists the topics to be covered in the chapter.



Graphic Sidebar
Provides visual quick-reference of the common attributes of members of the taxonomic group of the chapter: its basic "cell type"—whether it is a prokaryote, eukaryote, or virus; its Gram morphology (for bacteria); its general shape(s); and its general size or size range.

Affected Body Systems
Highlights the systems of the body affected by the organism(s) discussed in the chapter. Found near the beginning of each organismal chapter, this provides a quick overview of the clinical discussion ahead.

Concept Links
Cue the student that new material is related to or builds on an earlier discussion.

species. This has made a precise classification of these bacteria difficult. Streptococci grow well on blood agar and many species secrete *hemolysins* (enzymes that dissolve red blood cells), which produce patterns of hemolytic zones around the colonies. These hemolytic patterns can be used to make a preliminary identification of streptococcal groups. A clear zone of hemolysis surrounding the colony is called *beta*-hemolysis (∞), a zone with an opaque greenish color is called *alpha*-hemolysis, and some species produce no hemolysis.

The most usable classification system, **the**

Clinical Notes
Provide an applied perspective by focusing on pertinent on-going concerns in infectious disease microbiology. Based on the Morbidity and Mortality Weekly Reports (MMWR) issued by the Centers for Disease Control.

New Artwork
Makes understanding basic concepts of cell biology, genetics and immunology easier. Organismal chapters use art consistently to show pathogenesis of various diseases and graph the incidence of disease occurrence.

CLINICAL NOTE

Outbreak of *Shigella flexneri* 2a Infections on a Cruise Ship

During 29 August–1 September 1994, an outbreak of gastrointestinal illness occurred on the cruise ship *Viking Serenade* (Royal Caribbean Cruises, Ltd.) during its roundtrip voyage from San Pedro, California, to Ensenada, Mexico. A total of 586 (37%) of 1589 passengers and 24 (4%) of 594 crew who completed a survey questionnaire reported having diarrhea or vomiting during the cruise. One death occurred in a 78-year-old man who was hospitalized in Mexico with diarrhea. *Shigella flexneri* 2a has been isolated from fecal specimens from at least 12 ill passengers. Antimicrobial susceptibility testing of representative isolates indicated resistance to tetracycline and susceptibility to ampicillin and trimethoprim sulfamethoxazole. The subsequent two cruises of the ship were canceled. Investigation of the mode of transmission is under way (*MMWR* 43:657, 1994).

Shigella species is commonly associated with poor or crowded living conditions. Most of the approximately 20,000 cases occurring annually in the United States (Figure 21-7) are associated with institutionalized individuals, where hygienic conditions may be difficult to maintain because of crowding and lack of individual capabilities. This relationship between an ability to maintain personal hygiene and the frequency of shigella infection is reflected in the age distribution of the disease in the United States, where it is seen most frequently in the pediatric population.

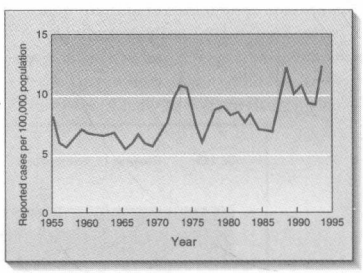


FIGURE 21-7 Reported cases of shigellosis in the United States, 1955–1994. (Courtesy Centers for Disease Control, Atlanta)

Shigellosis is endemic in underdeveloped countries. Historically, dysentery has been a problem in military populations and entire armies have become temporarily disabled when living under unsanitary conditions that commonly exist during wartime. People traveling from countries like the United States often contract bacillary dysentery within a short period after entering a country where dysentery is endemic.

✳ Diagnosis

Diagnosis is made by isolating shigellae from the feces or intestinal tract.

✳ Treatment

In contrast with *Salmonella* gastroenteritis, most cases of shigellosis are improved by chemotherapy. The recent development of *multiresistant* strains of *S. sonnei* (resistant to ampicillin, tetracycline, and trimethoprimsulfamethoxazole) has complicated the approach to therapy, but several available antibiotics remain effective. Oral rehydration and maintaining proper electrolyte balance is an essential component of treatment.

✳ Prevention and Control

Prevention of person-to-person transmission by following good sanitary practices is the most effective means of avoiding shigellosis. Patients with the disease should be isolated.

Multiresistant Bacteria that are resistant to a variety of antibiotics with different mechanisms of antimicrobial action.

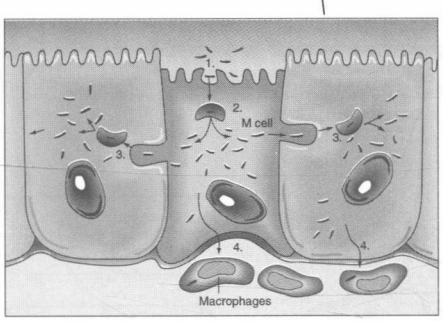


FIGURE 21-6 Invasion of intestinal epithelial cells by *Shigella*. 1. *Shigella* attach to M cells and induce their own phagocytosis. 2. *Shigella* escape the phagocytic vacuole and multiply. 3. Penetration of adjacent epithelial cells with multiplication and cell destruction. 4. *Shigella* released from infected cells are phagocytized by macrophages, thus preventing the spread to deeper tissues.

S. DYSENTERIAE (AND OTHERS): BACILLARY DYSENTERY (SHIGELLOSIS)

✳ Pathogenesis and Clinical Diseases

Following ingestion, the shigellae usually penetrate the large intestine by stimulating the endothelial cells, called M cells, that line the intestine to phagocytize them. However, these "nonprofessional" phagocytes are unable to kill the ingested bacteria and the bacteria then multiply and invade neighboring cells (Figure 21-6). Generally, penetration is not deeper than the submucosal cells. Inflammation, together with sloughing of the epithelial cells, results in ulcerative lesions. After 1 to 3 days of incubation the patient experiences a sudden onset of symptoms—abdominal cramps, fever, and diarrhea. The diarrheal stool frequently contains mucus and blood. Significant loss of water and salts may occur and in young and/or debilitated patients this dehydration and electrolyte imbalance may cause death. In otherwise healthy persons the disease is usually self-limiting and recovery occurs in 3 to 7 days. The death rate from dysentery in young children is significant in countries with poor sanitation and nutrition.

Infections due to *S. dysenteriae* are always potentially more serious than those due to other species. This organism produces a very powerful exotoxin (*shiga toxin*) that greatly increases its virulence. During a recent epidemic in Central and South America the mortality rate among those infected with this organism was between 8 and 10%. Although not endemic in the United States, this species has recently been introduced by tourists returning from Central America and Mexico. Most residents in areas where dysentery is endemic develop some immunity to the disease either through clinical or subclinical cases. Many such persons, however, remain carriers of the organism and serve as a source of infection for new susceptibles, such as visitors or newborns entering the population.

✳ Transmission and Epidemiology

Transmission is from human to human via the fecal–oral route by "fingers, food, feces, fomites, or flies." Infection can occur with as few as 10 or 10^2 bacteria (this is in contrast to 10^5–10^7 bacteria necessary to cause salmonellosis). Transmission by

Shiga toxin A powerful toxin produced by *S. dysenteriae* that acts on tissues of the central nervous system.

Consistent Chapter Format
Highlights—from a clinical perspective—the *essential* information for each taxonomic group: Pathogenesis and Clinical Disease, Transmission and Epidemiology, Diagnosis, Treatment, Prevention and Control.

Micrographs and Clinical Photos

Show disease organisms and pathological conditions associated with infection.

Pathogenesis Illustrations

Are consistently used in the organismal chapters to depict the means by which organisms cause disease in the body.

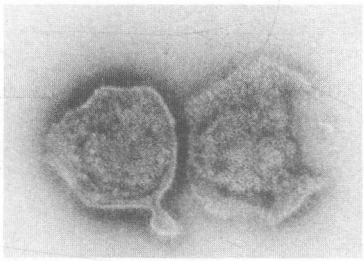


FIGURE 31-1 Transmission electron micrograph of herpesviruses showing icosahedral capsids (partially disrupted) surrounded by envelopes (magnified 160,000×). (Courtesy Robley C. Williams, University of California, Berkeley)

of cancer in lower animals are associated with these viruses and several lines of evidence associate herpesviruses, particularly the Epstein–Barr virus, with certain forms of cancer in humans. Women who have cervical herpes infection have a significantly greater incidence of cervical cancer than uninfected women.

HERPES SIMPLEX VIRUSES

Two serotypes of herpes simplex viruses (HSV) have been identified. Type 1 is generally associated with infections of the upper half of the body and type 2 with infections of the genitourinary tract and surrounding tissues. Primary infection usually occurs on the mucosal–epithelial surfaces of the body. Following initial infection, the neurons that innervate the area become infected. The primary site of infection is characterized by a lesion, while infection of the neurons leads to latent infection. Both types may cause disseminated infections in infants and compromised patients.

* Pathogenesis and Clinical Diseases

Cold Sores or Fever Blisters. Among the most common of all human infections are *cold sores* or *fever blisters* (herpes labialis), which are usually caused by the type 1 HSV (Figure 31-2). The recurring lesions on the lips are the clinical manifestation of a complex chronic interaction between the virus and the host. Most newborn infants are not readily infected, possibly as a result of passive immunity that offers some protection against primary infection. Once the passive immunity is gone, the infant is highly susceptible to primary infection. Susceptibility tends to decrease somewhat as the child gets older. However, in conditions of poor sanitation as many as 90% of the population has been infected before adulthood. Persons living under conditions of improved sanitation experience about a 50% infectivity rate. The primary infection is often asymptomatic or is not diagnosed as herpes. Symptoms are seen in 10 to 15% of the cases from 2 to 12 days after being exposed to the virus. The primary lesions may appear as small **vesicles** in the throat, mouth, or nose and go relatively unnoticed. The most noticeable form of primary infection involves the lips, mouth, and gums (*gingivostomatitis*), in which the vesicles rupture and develop into ulcerative lesions. Fever, pain, and irritability usually persist for about 1 week, followed by gradual healing during the second week.

Recovery is associated with a rise in antibodies against the virus. During the primary infection, however, the virus passes along nerve fibers to regional **ganglia**. In the case of gingivostomatitis,

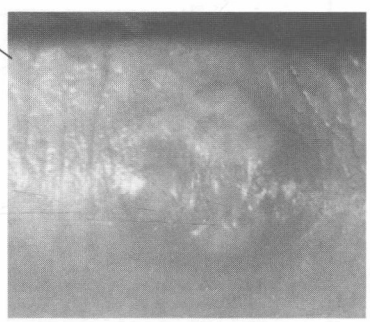


FIGURE 31-2 Herpes simplex fever blister on lower lip 2 days after onset. (Centers for Disease Control, Atlanta)

Vesicle A blister-like structure that contains a clear serous fluid.
Ganglion Major nerve trunks connecting the peripheral nerves to the CNS.

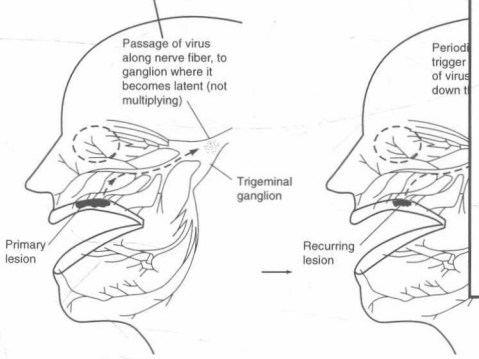


FIGURE 31-3 Aspects of the pathogenesis of primary and recurring herpes infections of the lips.

the *trigeminal ganglion* is commonly involved and the virus becomes sequestered in a latent form in this tissue. While in this latent form, the virus cannot be detected by ordinary means. It causes no symptoms and is not affected by antibodies. Periodically, in from 20 to 30% of the general population, these latent viruses become activated and move down the nerve fiber to cause recurring skin lesions at the site of the original infection (Figure 31-3). The frequency of these recurring lesions varies from person to person, ranging from once every few years to about once a month. Various stressful stimuli, such as excessive sunlight, fever, cold winds, emotional stress, and hormonal changes, apparently trigger the reactivation of the virus. As the virus moves down the nerve fibers, it passes directly into the skin cells without becoming exposed to the host's antibodies. The antibodies usually prevent the virus from spreading systemically to other tissues of the body but are unable to prevent the recurring lesions. Recurrent infections are generally less severe than primary infection. Primary and recurring infections may also occur in the eyes, causing a disease known as *herpetic keratoconjunctivitis*. Lesions on the cornea are most serious, for the accumulating scar tissue may lead to vision impairment.

Occasionally, almost any tissue of the body may become infected with these viruses. Primary and recurring infections may occur on any cutaneous area of the body. Traumatic injury may provide a portal of entry for primary infections that may develop in both children and adults. Such infections have been seen in wrestlers (herpes gladiatorum) due to skin abrasions, or in persons following burns, or on the thumb of a thumb-sucking child or the finger of a dentist (herpetic whitlow). Children with *eczema* may acquire a serious herpes infection over large areas of the body (eczema herpeticum). Herpes simplex viruses may also infect the central nervous system, causing a severe, and often fatal, infection (herpetic encephalitis).

Genital Herpes. A very common sexually transmitted disease is *genital herpes* (see Plate 38). Over 80% of these infections are caused by HSV type 2. In females the vesicles usually occur in the mucosal tissue of the vulva, vagina, or cervix, but any of the genital or surrounding tissues may be involved. These vesicles ulcerate, producing shallow lesions. The symptoms may include malaise, urinary retention, local pain, fever, vaginal discharge, and tender, swollen inguinal lymph nodes. In males the vesicles and subsequent ulcerations

Eczema Inflammation of the skin, often associated with scaling, papules, crusting, and serous discharge.

Boldfaced and Italicized Terms

Highlight new or important words for the chapter. Key terms are boldfaced, while additional new terms are italicized. All boldfaced and italicized terms are included in the **Glossary** at the end of the text.

Color Plate References

Indicate where a color photo is available in the full-color Plate Reference at the back of the text.

Running Glossary

Helps understanding of new terms *as they are used*. These bottom-of-the-page entries reduce the time required to read the text and later provide excellent chapter review material.

has as yet not been associated with any specific clinical disease.

In late 1994, yet another human herpes virus was found, which is associated with a type of cancer called *Kaposi's sarcoma*. Kaposi's sarcoma is found in about 20% of AIDS patients. Some evidence suggests that this herpesvirus, called *Kaposi's sarcoma-associated herpesvirus*, may be the cause of Kaposi's sarcoma in AIDS patients.

MATERIAL FOR REVIEW

CONCEPT SUMMARY

1. Infection due to the DNA herpesvirus group is of considerable attention and concern today. These viruses are responsible for a wide variety of disease conditions in both humans and animals. They cause benign, latent infections, such as cold sores, and extensive life-threatening infections, such as generalized herpes.
2. Herpesviruses are widely known because of current interest in their role as agents of a sexually transmitted disease caused by herpes simplex virus type 2 and because of infectious mononucleosis due to the Epstein–Barr virus.
3. Herpes viruses are among the few viruses for which a specific antiviral therapy has been developed.
4. Although not generally well known, cytomegalovirus infection is extremely common. This agent is responsible for serious, often fatal disease in compromised patients.

CLINICAL SUMMARY TABLE

Microorganism	Virulence Mechanisms	Diseases	Transmission	Treatment	Prevention
Herpes simplex	Latency	Cold sores Conjunctivitis Genital herpes Encephalitis	Direct and Sexual contact	Acyclovir	Avoid contact
Varicella-zoster	Latency	Chickenpox Shingles	Airborne	Acyclovir	Vaccine
Epstein-Barr	Latency	Infectious mononucleosis	Oral contact	Symptomatic	None

STUDY QUESTIONS

1. Briefly describe the host–parasite relationship commonly associated with herpesvirus infections.
2. Why isn't antibody to herpesvirus type I and type II protective against recurrence of the disease?
3. What is the most common site of infection for type II herpes?
4. Why would a viral-component vaccine be particularly useful against herpesviruses?
5. What is the likely source of an outbreak of chickenpox in a community that is apparently free of the virus?
6. What is the biggest risk factor associated with cytomegalovirus infection?

CHALLENGE QUESTIONS

1. Why would there be possible opposition to approving a living vaccine against herpes simplex virus infections?
2. Chickenpox in children is usually a relatively harmless disease. Why do health officials say that chickenpox in adults can be very serious?

Concept Summary
Recaps in numbered-sentence format the essential points of the chapter.

Clinical Summary Table
Provides a quick review of the organisms covered in the chapter and some specifics of the diseases they cause.

Study Questions
Provide an opportunity for students to quiz themselves about the important facts of the chapter.

Challenge Questions
Ask the student to go beyond mere memorization of the facts to apply their knowledge to a specific problem.

Brief Contents

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