
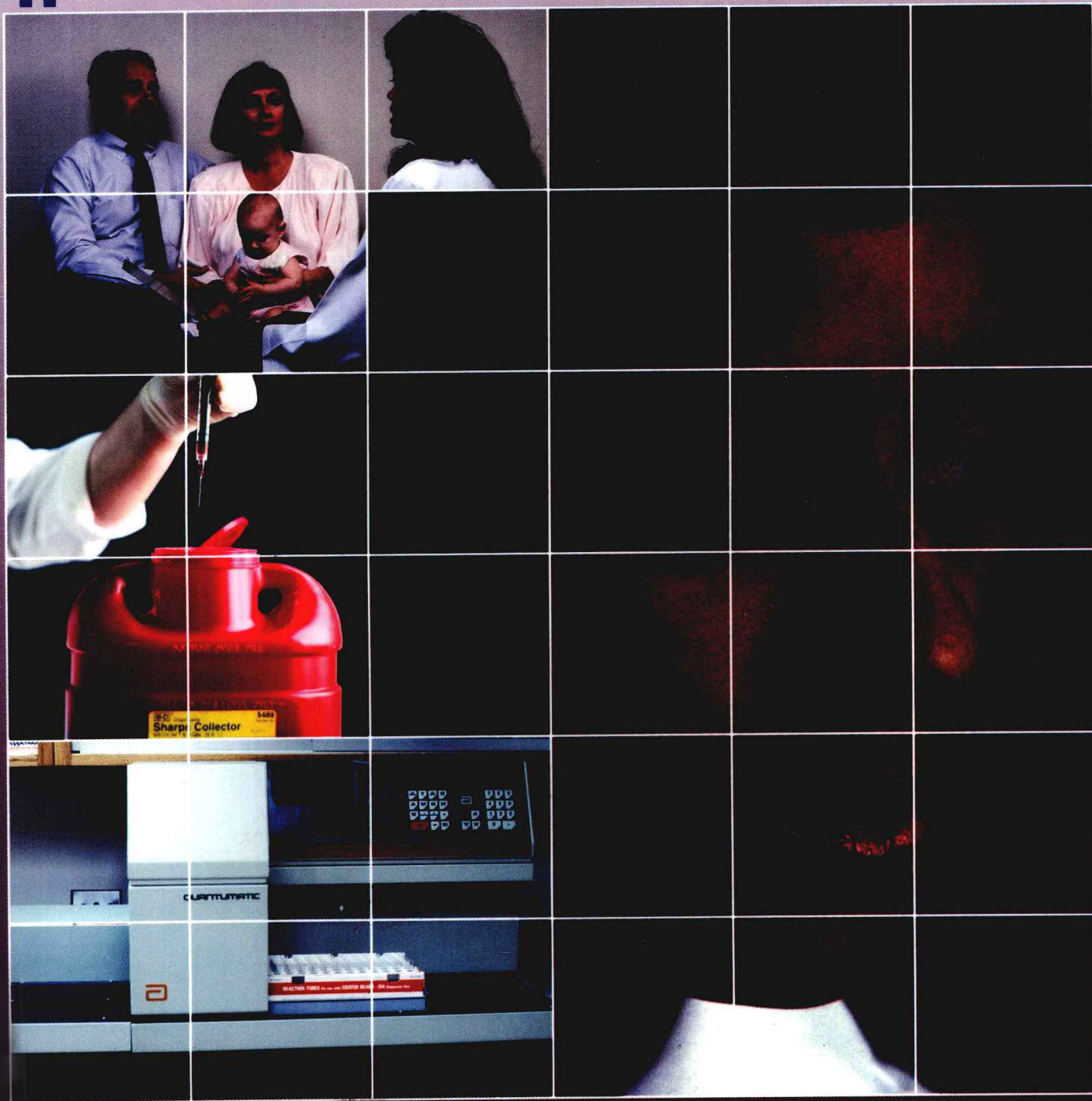


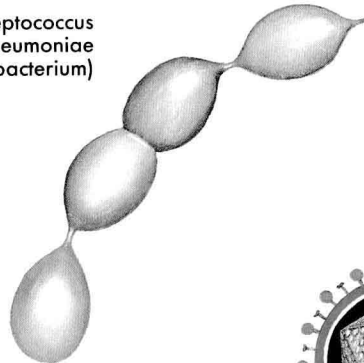
INFECTIOUS DISEASES

 Mosby's Clinical Nursing Series

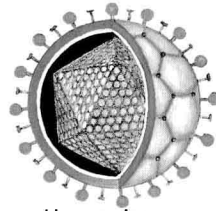
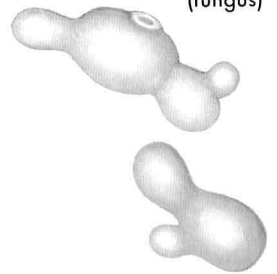


INFECTIOUS DISEASES

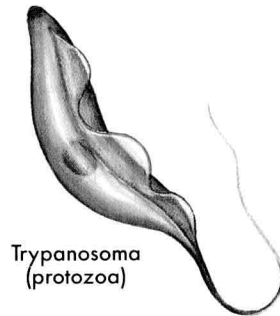
Streptococcus pneumoniae
(bacterium)



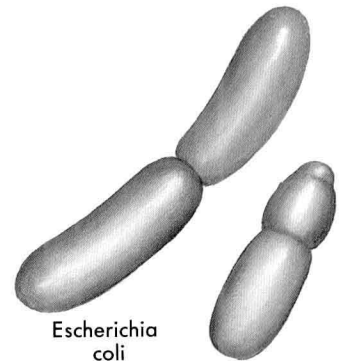
Yeast
(fungus)



Herpesvirus
(virus)



Trypanosoma
(protozoa)



Escherichia coli
(bacterium)



Mosby's Clinical Nursing Series

西文书

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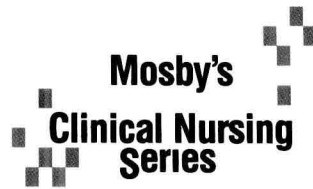
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INFECTIOUS DISEASES

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PREFACE

Infectious Diseases is the third volume in *Mosby's Clinical Nursing Series*, a new kind of resource for practicing nurses.

The *Series* is the result of the most elaborate market research ever undertaken by Mosby–Year Book, Inc. We first surveyed hundreds of working nurses to determine what kind of resources practicing nurses want in order to meet their advanced information needs. We then approached clinical specialists—proven authors and experts in 10 practice areas, from cardiovascular to ENT—and asked them to develop a common format that would meet the needs of nurses in practice, as specified by the survey respondents. This plan was then presented to 9 focus groups composed of working nurses over a period of 18 months. The plan was refined between each group, and in the later stages we published a 32-page full-color sample so that detailed changes could be made to improve the physical layout and appearance of the book, section by section and page by page.

The result is a new genre of professional books for nursing professionals.

Infectious Diseases begins with an innovative Color Atlas of Infectious Disease Pathophysiology. This is a complete and up-to-date review of the physiology of human defenses and immune system responses to infection. Color drawings enhance the presentation. Because of the increasing concern with prevention and control of infectious disease, the first chapter addresses transmission and control of infection.

Chapter 2 is a concise but comprehensive overview of nursing assessment for infection risks and signs of infection in all body systems. It includes detailed photographs of assessment techniques and drawings of skin le-

sions. Color plates of manifestations of specific infectious diseases on pages x to xvi complement this chapter.

Chapter 3, Diagnostic Procedures, is divided into two parts. The first describes the procedures for collection and handling of specimens and patient teaching for each procedure. Again, color photographs show proper equipment and technique in sharp detail. Part two describes the full range of diagnostic tests used to identify pathogens and antibodies against the pathogens.

Chapters 4 through 11 discuss various categories of infectious disease. Over 70 infectious diseases are grouped by chapter, either according to the body system where signs and symptoms are manifest or by mode of transmission or control. These chapters cover central nervous system infectious diseases, gastrointestinal infectious diseases, hepatitis and hematolymphatic infections, respiratory infectious diseases, AIDS, sexually transmitted diseases, vector-transmitted fevers, and childhood and vaccine-preventable diseases. With the exception of Chapter 11, which discusses the childhood and vaccine-preventable diseases, all other chapters combine diseases with similarities for nursing care.

Each disease chapter is presented in a similar format. Unique overview tables summarize the epidemiology for groups of similar diseases. Essential information is provided in these tables to enable nurses to quickly identify transmission risks and participate in control of infectious diseases. Pathophysiology is presented to answer the question that practicing nurses frequently ask. Disease complications are highlighted to alert nurses to prevent, observe, respond to, and report changes in the patient's condition. Definitive diagnostic tests and the physician's treatment plan are reviewed to facilitate interdisciplinary collaboration.

The nursing care for groups of diseases is presented according to the nursing process in easy-to-use tables. These pages are bordered in red to make them easy to find. Prevention and patient teaching are strongly emphasized nursing interventions. The nursing care is structured to integrate the five steps of the nursing process, centered around appropriate nursing diagnoses accepted by the North American Nursing Diagnosis Association (NANDA). The material can be used to develop individualized care plans quickly and accurately, and it meets the standards of nursing care required by the Joint Commission on the Accreditation of Hospitals (JCAH). By facilitating the development of individualized and authoritative care plans, this book can actually save you time to spend on direct patient care.

The format for Chapter 12, Nosocomial Infections, is different. The intent of this chapter is to provide nurses with recent information to prevent these hospital-acquired infections. Chapter 13, Therapeutic Procedures, presents the latest recommendations from the Centers for Disease Control on immunizations and isolation procedures.

In response to requests from scores of nurses participating in our research, a distinctive feature of this book is its use in patient teaching. Background information on diseases and medical interventions enables nurses to answer with authority questions patients often ask. The illustrations in the book, particularly those in the Color Atlas and the chapter on Diagnostic Procedures, are specif-

ically designed to support patient teaching. Chapter 14 consists of 13 Patient Teaching Guides written at a ninth-grade level so they can be copied, distributed to patients and their families, and used for self-care after discharge. Patient teaching sections in each care plan provide nurses with checklists of concepts to teach, promoting this increasingly vital aspect of nursing care.

The book concludes with a complete guide to antineoplastic drugs. The inside front cover contains frequently used information such as Universal Precautions, Immunization Schedules, and important reference phone numbers. Inside the back cover is a complete presentation on the nursing diagnosis for hyperthermia.

This book is intended for nurses practicing in both inpatient and outpatient care settings. We expect it to be a helpful reference for hospital medical-surgical, emergency room, and infection control nurses, and for nurses in ambulatory care clinics, school and college health programs, occupational health, home health, and public health care. We also anticipate that the book will be a valuable adjunct to medical-surgical and community health nursing texts for students learning nursing in a variety of settings.

We hope this book contributes to the advancement of professional nursing and the quality of patient care by serving to provide professional literature for nurses to call their own.

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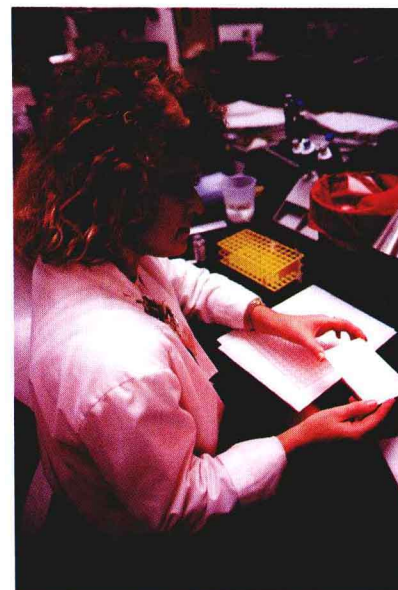
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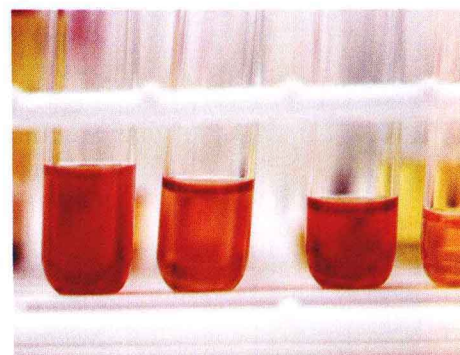
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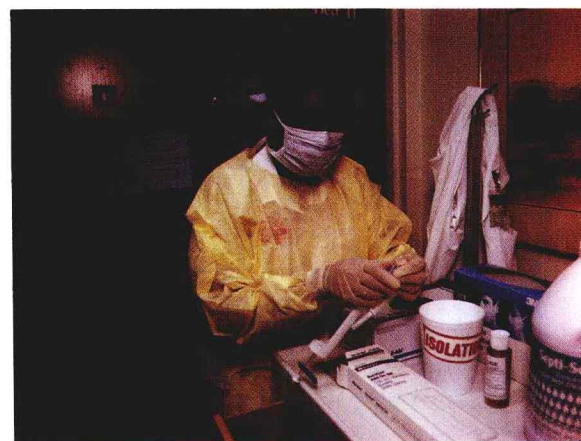
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Color Atlas of Infectious Disease Pathophysiology

Of all life-forms on earth, microorganisms compose the largest population. They are everywhere, living on and in water, soil, plants, animals, and even minerals—and, of course, humans. As a group, microbes are remarkably adaptable. That we are unaware of these invisible life-forms, at least most of the time, is a testament to their success. Many have evolved a friendly, mutually beneficial relationship with humans by performing a needed service in exchange for nutritional support.

Nonetheless, numerous organisms are pathogenic to humans. Infectious diseases rank as the fifth most common cause of death in the United States. They are an important complication of other disorders and cause a significant amount of morbidity even in healthy populations.

A susceptible host, an infectious agent, and an environment that permits transmission of the agent are not enough to produce an infectious disease. The infectious agent must also elicit symptoms of disease in the host, either from the action of the pathogen or its products on body cells or from physiologic responses elicited to eradicate the agent (Figure 1-1).

Each step in the infection process has more than one possible outcome. *Contamination*, the presence of an infectious agent on a body surface or object, is only a first step. *Infection* is implantation and successful reproduction of an infectious agent in a living host, but if no physiologic response occurs, then the agent has merely *colonized* the host. A colonized host who also sheds the agent, transmitting it to others, is a *carrier* but is not necessarily ill. A physiologic response that occurs without producing clinical symptoms is termed *subclinical infection*. Only if tissue injury or physiologic response produces clinical symptoms of illness is an *infectious disease* present.

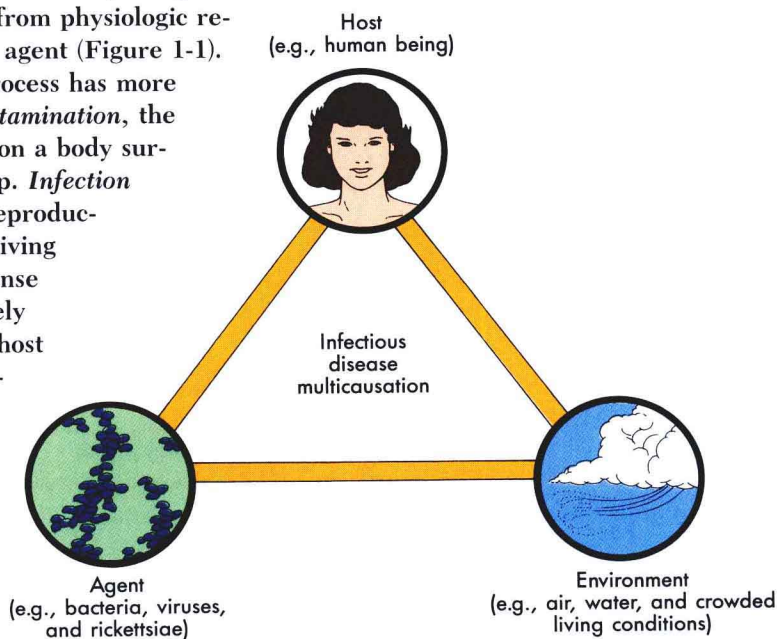


FIGURE 1-1
Multicausation of infectious disease: agent, host, and environmental conditions interact to produce infectious disease in the host.

PATHOGENIC AGENTS

Pathogens are parasites that create pathologic process in their human host. Because they cannot synthesize their own amino acids, they must rely on a host to supply their nutritional requirements. Some pathogens are metabolically structured so that they can survive outside a host. By contrast, viruses depend on a host for all sustenance because they lack the ability to perform any metabolic function on their own and can only survive in the human cell.

Clearly, pathogens are a remarkably successful life-form. One key to their success is viability, an organism's ability to survive in an adverse environment. This quality is determined by the morphology and chemical composition of the agent. The more viable the pathogen, the better able it is to resist an adverse environment. Many organisms are adaptable enough to evade physical, chemical, and thermal insults. For example, tetanus bacilli form spores that enable the bacilli to survive until they reach more welcoming environments. Genetic mutation creates antibiotic-resistant strains of bacteria and viruses that can parasitize previously immune hosts.

The Variability of Pathogenicity

The likelihood of a pathogen causing infectious disease is influenced by the organism's mode of action, infectivity, pathogenicity, virulence, antigenicity and toxicogenicity.

Mode of action

Pathogens have various modes of action for invading and reproducing in a host. They may directly damage cells by causing hyperplasia, necrosis, and cell death. Intracellular pathogens, such as viruses, interfere with cellular metabolism, rendering the host cell dysfunctional by the accumulation of pathogens and their products inside the cell (inclusion bodies). Another

mode of action is the production of toxins that cause local or systemic reactions (Figure 1-2).

Infectivity

Infectivity of the agent is its ability to invade and multiply in the host. It is affected by host defenses and pathogen-produced enzymes that facilitate invasiveness. Coagulase, an extracellular enzyme of some cells that causes coagulation, enables organisms, such as staphylococci, to clot plasma and form a sticky fibrin layer around themselves to protect against host defenses. Streptococci produce streptokinase to dissolve fibrin clots, allowing the organism to spread through host tissue. Hyaluronidase breaks down connective tissue and increases tissue permeability, enabling streptococci, pneumococci, and clostridia to spread throughout host tissues. Collagenase degrades collagen to facilitate deep invasion of *Clostridium perfringens* and other pathogens into tendons, cartilage, and bone.

Pathogens are graded according to their infectivity potential. For example, poliomyelitis virus is rated a highly infective agent, rubella virus has intermediate infectivity, and *Mycobacterium tuberculosis* has low infectivity.

Pathogenicity

The ability of an agent to produce disease depends on its speed of reproduction, extent of tissue damage, and production of a toxin. Agents can be graded according to pathogenicity. Examples of highly pathogenic agents are those that cause smallpox and rabies. Infection by them almost invariably results in disease. The rubella virus has intermediate pathogenicity, and the poliomyelitis virus has low pathogenicity.

Virulence

The potency, or virulence, of a pathogen determines the severity of disease it produces. Virulence is measured in terms of the number of microorganisms or micrograms of toxin required to kill a given host. Viru-

THE MANY RELATIONSHIPS BETWEEN HUMANS AND ORGANISMS

Symbiosis	Benefits only the human; no harm to the organism
Mutualism	Benefits the human and the organism
Commensalism	Benefits only the organism; no harm to the human
Pathogenicity	Benefits the organism; harms the human (Opportunism is the situation when benign organisms become pathogenic because of decreased human host resistance)

Some organisms that have a symbiotic or commensal relationship on one part of the body can become pathogenic when transferred to another area. For example, alpha-streptococci are part of the normal flora in the nasopharynx but become pathogenic when transferred to heart valves.

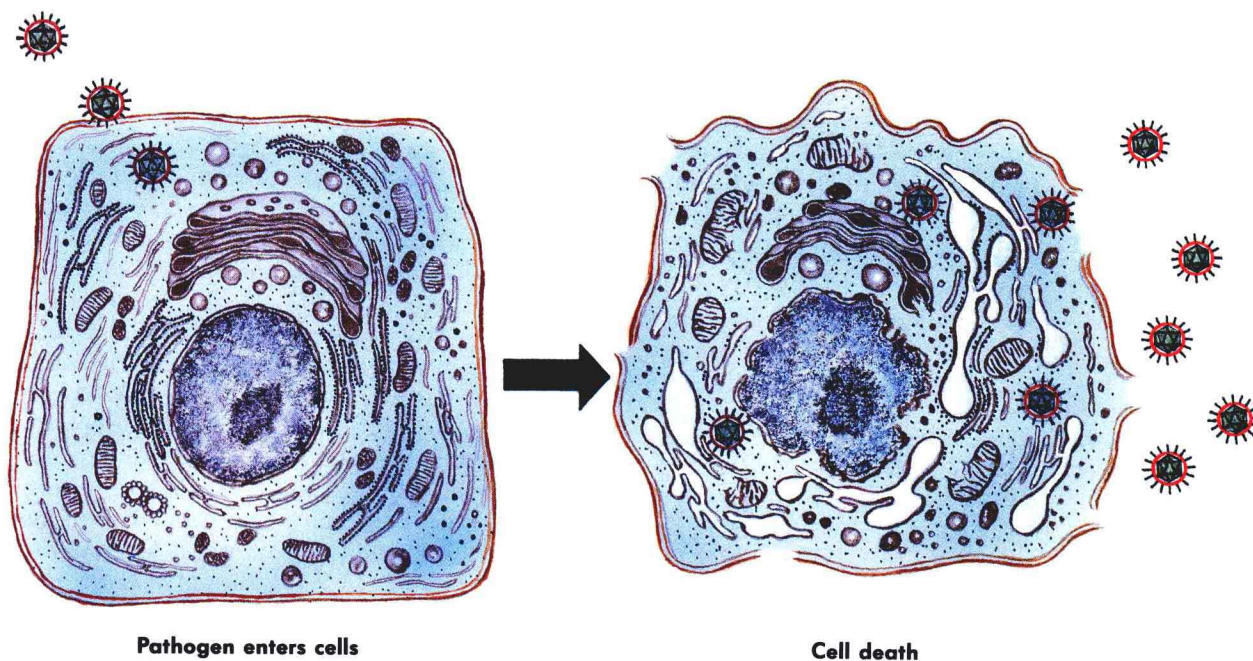


FIGURE 1-2

Mode of action of pathogenic viruses. One factor influencing pathogenicity is an organism's mode of action. Viruses can replicate only within host cells. They interfere with cellular metabolism, leading to cell death.

lence can also be graded. The rabies virus is highly virulent, the poliomyelitis virus has intermediate virulence, the measles virus is of low virulence, and the common cold has very low virulence.

Antigenicity

The ability of pathogens to induce an immune response in the host varies considerably. Some have intrinsic antigens (e.g., proteins, polypeptides, or polysaccharides) that stimulate antibody production against the antigen. Others lack antigenic structures and may be able to evade destruction for a considerable length of time.

Toxigenicity

An important factor in determining a pathogen's virulence is toxigenicity. Agent products associated with toxigenicity are hemolysin, leucocidin, and toxins. Hemolysin destroys erythrocytes, and leucocidin destroys leukocytes. Both of these products are factors in the virulence of some streptococci and staphylococci.

Some bacteria secrete water-soluble antigenic exotoxins that are distributed rapidly by the blood, causing potentially severe systemic and neurologic manifestations. Diseases associated with exotoxins are tetanus, botulism, and diphtheria.

The cell walls of some bacteria are composed of endotoxins, which cause inflammation and local de-

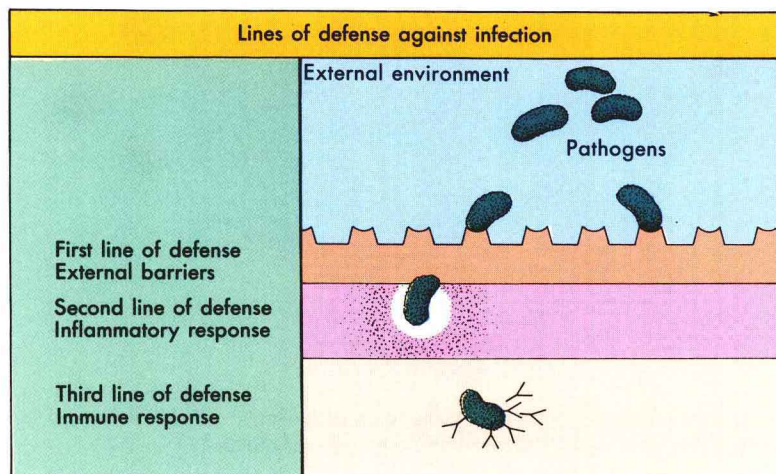
struction of tissues. Endotoxins are weakly toxic, relatively stable, and not antigenic. Diseases associated with endotoxins include staphylococcal food poisoning and cholera.

CLASSIFICATION OF PATHOGENS

Pathogenic agents are classified according to morphology, chemical composition, growth requirements, and viability. Classifications are continually changing as new organisms are identified and additional distinguishing biochemical or morphologic characteristics of known organisms are recognized. The current classification scheme used includes protozoa, fungi, bacteria, rickettsiae, chlamydiae, mycoplasmata, viruses, and helminths.

PHYSIOLOGY OF THE HUMAN RESPONSE TO INFECTION

In the course of a single day, the average person is probably exposed to thousands of pathogenic organisms, yet most persons do not succumb to infectious disease. This is possible because



the body is equipped with anatomic characteristics and physiologic processes that increase resistance to pathogens and fight the infectious process once it begins.

The first line of defense against infection is external and consists of mechanical barriers, chemical barriers, and the body's own population of microorganisms.

The two internal barriers come into play when the external line of defense is breached. Operating as the second line of defense, the inflammatory response is aimed at preventing an invading pathogen from becoming established, reproducing, and invading other tissues. The third line of defense is the immune response, which is activated after the inflammatory response. Although inflammation and immune response are two events, they cannot always be easily separated because both events involve many of the same processes and cellular components.

THE FIRST LINE OF DEFENSE: EXTERNAL BARRIERS

Every surface of the body that is exposed in any way to the environment is involved in first-line defense. The skin and mucous membranes of the digestive, respiratory, and genitourinary tract form a continuous, closed barrier between the internal organs and the environment (Figure 1-3). As long as they are intact, skin and mucous membranes are normally impervious to most pathogenic organisms. Body secretions provide an inhospitable environment by maintaining a pH level that discourages colonization or by "washing" the area to keep organisms from accumulating.

One of the most fascinating aspects of the body's first-line defense system is the normal microbial inhabitants (flora) that populate almost all of the body's surfaces. The process of colonization begins at birth. Operating on the principle of **microbial antagonism**, the presence of indigenous flora interferes with the es-

tablishment of pathogenic microorganisms in several ways. By occupying a surface, resident flora offer potential invaders stiff competition for both space and nutrients. Indigenous flora help maintain an optimal pH for their own growth, which is inhospitable to many pathogenic agents. Some of these benign organisms are known to secrete germicidal substances. There is evidence that indigenous flora also stimulate the development of the immune system.

The importance of microbial antagonism becomes evident when the normal flora is disturbed, as illustrated by the common occurrence of *Candida albicans* overgrowth following antibiotic therapy, resulting in diarrhea and vaginitis.

Some indigenous flora are themselves pathogenic under certain conditions. They can be responsible for infection when the immune system is impaired, the skin or mucous membranes are

breeched, or the flora are displaced from their natural habitat to another area of the body. This latter event is explained by the fact that the normal flora are tissue specific—that is, a particular type of bacteria normally colonizes a particular type of tissue, adhering to specific receptors on epithelial cells. As a result, the normal composition of flora varies from one part of the body to another (Table 1-1). Displacement of indigenous flora to another area is a common cause of nosocomial (hospital-acquired) infection, such as urinary tract infection from enteric bacteria following catheterization.

THE SKIN

Penetration of the skin, with its stratified and cornified epithelium, is significantly more difficult for microorganisms than penetration of mucous membrane. With extremely rare exceptions, a break in the skin is necessary for pathogens to breach this mechanical barrier. Furthermore, the skin maintains a fairly acid pH, which inhibits the growth of most pathogenic bacteria. Microbes are continually sloughed from the epidermis with dead skin cells, and oil and sweat secreted from glands in the dermis wash microorganisms from the pores. Sebaceous secretions also contain bactericidal fatty acids.

THE NOSE

Inspired air travels across coarse nasal hairs and through the turbinates, which filter out larger particles and trap some pathogens. The sticky mucosal surface moves this material toward the throat for expectoration or swallowing, and the nasal passages can be cleared by sneezing. Nasal secretions contain some antimicrobial substances, such as IgA antibody and lysozyme.

THE LUNGS

The mucous membranes lining the upper airways clear inspired debris, including microorganisms, from the respiratory tract. Mucus-producing goblet cells located in the larger airways keep these passages replenished with mucus. Particles are trapped on the sticky mucosal surface, and cilia propel mucus toward

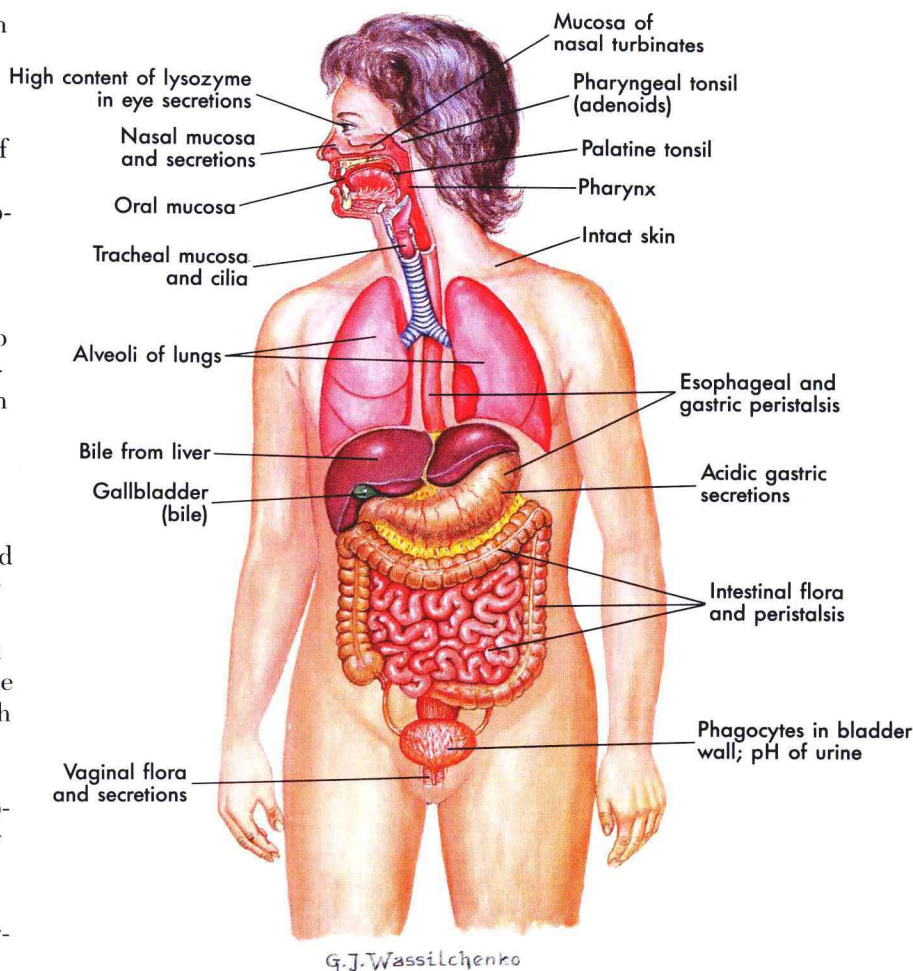


FIGURE 1-3
First line of defense—external barriers.

the throat at a rate of 10 to 20 mm/min. Coughing and sneezing expel particle-laden mucus from the respiratory tract.

The normal efficiency of the upper airways results in only the smallest particles reaching the lower respiratory tract. Microorganisms and other particles that remain in the alveoli are dispatched by alveolar macrophages. The pulmonary system is not inhabited by indigenous flora.

THE MOUTH AND PHARYNX

In addition to the mucous membrane lining these structures, lymph tissue of the tonsils and adenoids are located in the pharyngeal area. This permits a rapid lymphocytic response to pathogenic organisms entering the oral or respiratory routes.

THE DIGESTIVE SYSTEM

The stomach secretes extremely acid digestive enzymes, which neutralize or kill most bacteria. Bile de-

Table 1-1**NORMAL INDIGENOUS FLORA OF THE HUMAN BODY**

Skin	Predominantly gram-positive cocci and rods. <i>Staphylococcus epidermidis</i> , corynebacteria, mycobacteria, and streptococci are primary inhabitants; <i>S. aureus</i> in some people; also yeasts (<i>Candida</i> and <i>Pityrosporum</i>) in some areas of skin. Numerous transient microorganisms may become temporary residents. In moist areas, gram-negative bacteria. Around sebaceous glands, <i>Propionibacteria</i> and brevibacteria. The mite <i>Demodex folliculorum</i> lives in hair follicles and sebaceous glands around the face.
Nose	Predominantly gram-positive cocci and rods, especially <i>S. epidermidis</i> . Some people are nasal carriers of pathogenic bacteria, including <i>S. aureus</i> , beta-hemolytic streptococci, and <i>Corynebacterium diphtheriae</i> .
Mouth	A complex population of bacteria that includes several species of streptococci, <i>Actinomyces</i> , lactobacilli, and <i>Haemophilus</i> .
Pharynx	Anaerobic bacteria and spirochetes colonize the gingival crevices. Similar to flora in mouth plus staphylococci, <i>Neisseria</i> , and diphtheroids. Some asymptomatic persons also harbor the pathogens pneumococcus, <i>H. influenzae</i> , <i>N. meningitidis</i> , and <i>C. diphtheriae</i> .
Distal intestine Colon	Enterobacteria, streptococci, lactobacilli, anaerobic bacteria, and <i>C. albicans</i> . <i>Bacteroides</i> , lactobacilli, clostridia, <i>Salmonella</i> , <i>Shigella</i> , <i>Klebsiella</i> , <i>Proteus</i> , <i>Pseudomonas</i> , enterococci and other streptococci, bacilli, and <i>Escherichia coli</i> .
Distal urethra	Typical bacteria found on the skin, especially <i>S. epidermidis</i> and diphtheroids. Also lactobacilli and nonpathogenic streptococci.
Vagina	Birth to 1 month: similar to adult. 1 month to puberty: <i>S. epidermidis</i> , diphtheroids, <i>E. coli</i> , and streptococci. Puberty to menopause: <i>Lactobacillus acidophilus</i> , diphtheroids, staphylococci, streptococci, and a variety of anaerobes. Postmenopause: similar to prepubescence.

creases the surface tension of the cell wall in some bacteria, rendering the organisms more digestible. The esophagus and empty stomach have no resident flora, but microorganisms enter through food, saliva, and nasopharyngeal secretions. Within 1 hour of a meal, the stomach is again sterile.

Peristalsis discourages pathogenic colonization by preventing prolonged contact of fecal material with the mucous lining of the small intestine and colon. The sterile environment of the stomach extends to the upper portion of the intestinal tract, but near the terminal ileum the flora begins to resemble that of the colon.

The colon contains the largest concentration of bacteria of any area in the body. It is estimated that as much as 60% of feces, by weight, consists of bacteria and other microorganisms.

THE URINARY TRACT

Frequent flushing of the bladder and urinary tract through urination helps prevent microorganisms from colonizing the area. Urine contains urea nitrogen and ammonium, which are bacteriostatic to most pathogens. Prostatic fluid contains bactericidal substances that protect the male genitourinary tract. The bladder

wall is capable of mounting a phagocytic response within 30 minutes of pathogenic invasion. The distal portion of the urethra in both males and females is populated with bacteria found on the skin.

THE VAGINA

The microorganisms found in the vagina vary with age (Table 1-1). At birth the vagina is sterile, but within 24 hours glycogen is deposited in the vaginal mucosa under the influence of estrin, which is passively transferred from mother to infant. This establishes a pH of about 4.5, allowing flora to develop that resembles that of an adult. At about 1 month of age, glycogen secretion and the pH rises to about 7, changing the flora. At puberty, glycogen is again produced and the pH returns to about 4.5 until after menopause, when the vaginal flora again resembles that of prepubescence.

THE EYE

Tears have a high content of lysozyme, which is effective against most gram-positive bacteria. The continual flushing action of tears helps cleanse the eyes by transferring fluid through the lacrimal ducts and into the nasopharynx. No resident flora is present in the eye.

THE COMPONENTS OF INTERNAL DEFENSES

The second and third lines of defense (the inflammatory response and the immune system) share several components. These components include the lymphatic system, leukocytes, and a multitude of chemicals, proteins, and enzymes that facilitate the internal defense systems. An understanding of these components and their interactions aids in understanding inflammation and the immune response.

LYMPHATIC SYSTEM

The lymphatic system provides a network for components of the internal defenses to circulate throughout the body. It is designed to capture and destroy invading microorganisms. The lymphatic system functions in both the inflammatory response and immune reactions.

Every tissue supplied by blood vessels, except that of the brain and placenta, is invested with lymphatic vessels. The large lymphatic tissues are shown in Figure 1-4, but numerous smaller lymph nodes (varying from “pinhead” size to “lima bean” size) are grouped throughout the body. The lymphatic tissues are connected by a network of collecting ducts that parallel the vascular network, forming a closed but porous circle.

Lymph is a clear, opalescent or yellowish fluid containing a variety of white blood cells, particularly lymphocytes, and occasionally red blood cells. The fluid originates in the blood and enters the interstitial spaces, where it picks up microorganisms, cell debris, or other foreign material in the tissue. From here it passes into the profusion of microscopic lymph tubules that join to form ducts, which carry it to nearby lymph nodes.

Lymph nodes form two major functions—filtration and sensitization. Any foreign material entering a lymph node is filtered out. The “clean” lymph fluid exits the node and transfers proteins and fluids back into the circulatory system. Foreign material inside the lymph node generates one of the following events: (1) the material is destroyed by phagocytic cells inside the lymph node, or (2) a specific immune response is activated by sensitization of lymphocytes to the specific antigen of the foreign material. (See Activation of Lymphocytes in this section.)

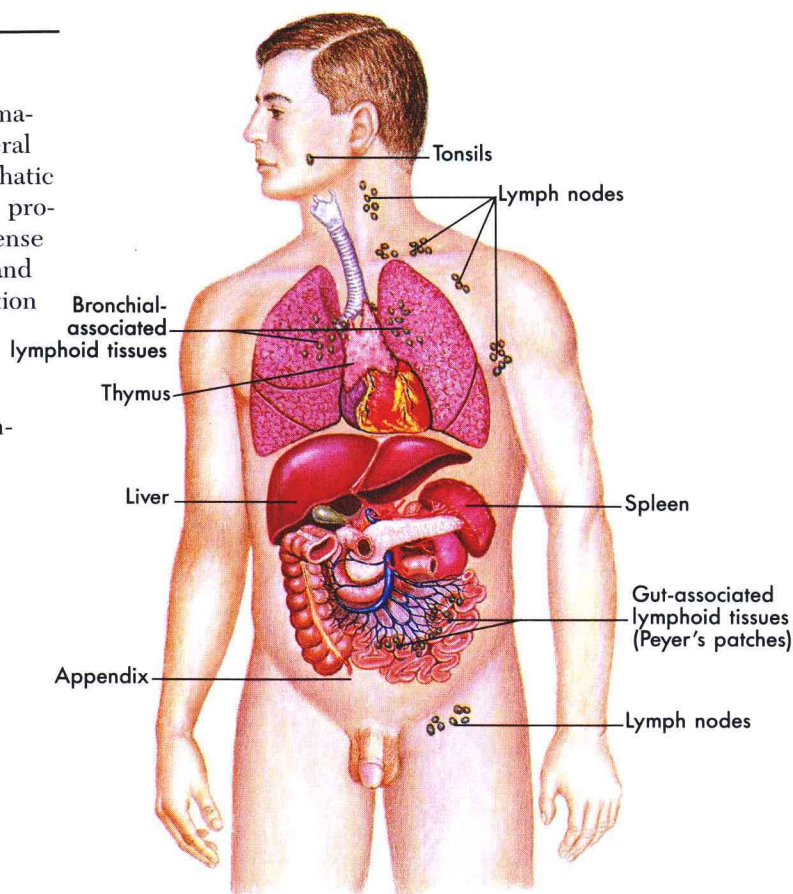


FIGURE 1-4
Components of the lymphatic system.

The thymus is critical to the maturation of T lymphocytes, a process that is influenced by thymic hormones. This gland reaches its greatest size during puberty, after which it gradually involutes as adipose tissue replaces thymic tissue. Acute disease or malnutrition can increase the involution process.

The spleen is the largest lymphatic organ. It produces leukocytes and filters venous blood. Tissue macrophages lining the splenic sinuses destroy circulating microorganisms and old red blood cells during the filtration process.

Lymphoid tissue can also be found in the tonsils and Peyer's patches. The tonsils are aggregations of lymphoid tissue, named according to their location. Those in the mouth and pharynx are termed **palatine**, **lingual**, and **pharyngeal** tonsils. The Peyer's patches are accumulations of the lymphoid tissue of the intestinal tract and the appendix.