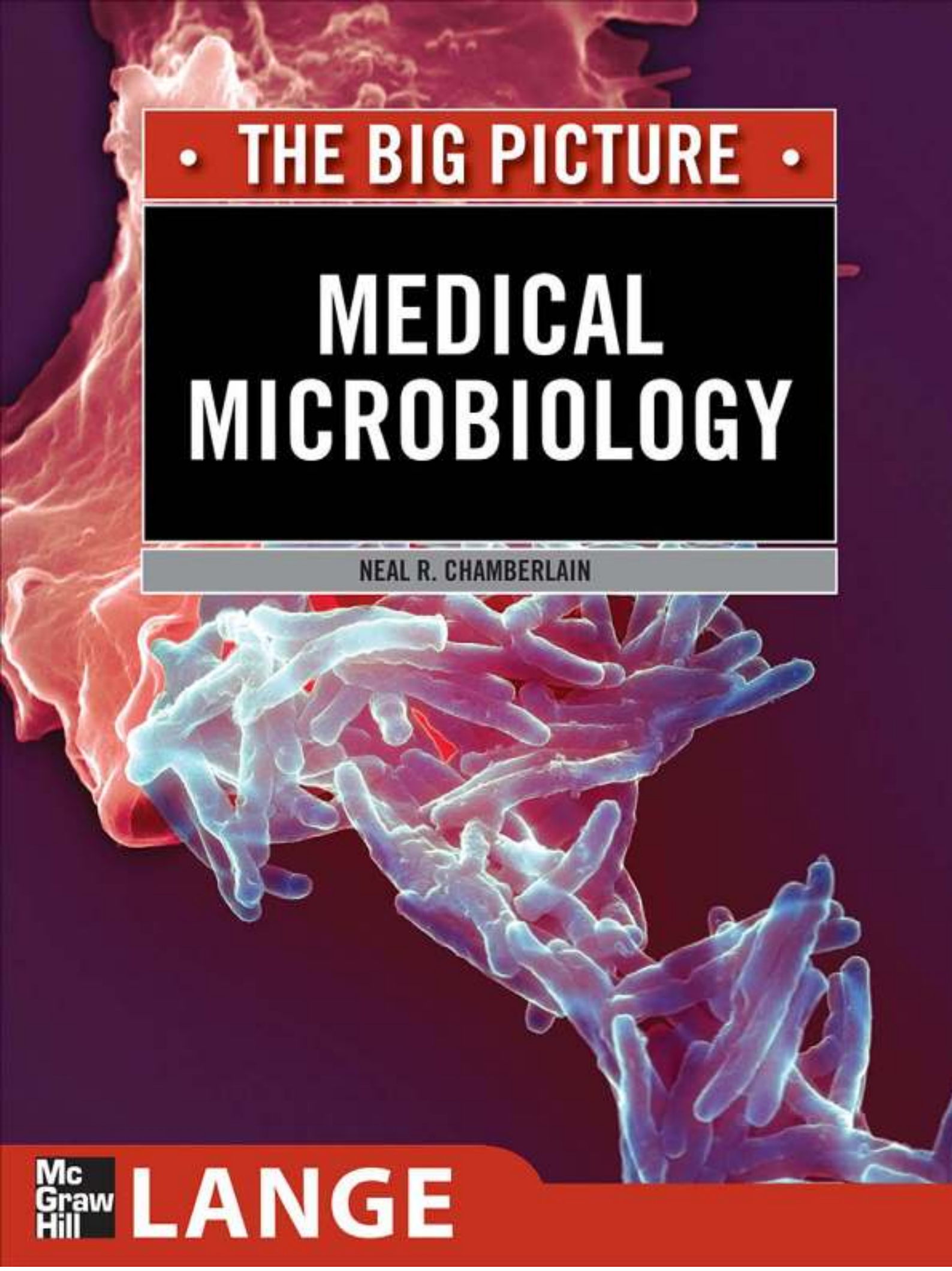
• THE BIG PICTURE •
MEDICAL MICROBIOLOGY
NEAL R. CHAMBERLAIN
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THE BIG PICTURE

MEDICAL MICROBIOLOGY

NOTICE

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THE BIG PICTURE

MEDICAL MICROBIOLOGY

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Cover image caption: Macrophage engulfing tuberculosis vaccine. Colored scanning electron micrograph (SEM) of a macrophage white blood cell engulfing (red) Mycobacterium bovis bacteria (blue). This is the BCG (bacillus of Calmette-Guerin) strain of the bacteria, used in the vaccination for tuberculosis (TB). The bacteria is live but attenuated (weakened). The macrophage engulfs (phagocytoses) the bacteria and destroys them. The vaccine primes the immune system, without causing disease, so that it responds more rapidly if infected with TB bacteria. Magnification: 3,500 when printed 10 centimeters tall. Credit: SPL / Photo Researchers, Inc.

DEDICATION

To my loving wife, wonderful children, and to the one who gave up everything so that I might live this dream.

—Neal Chamberlain

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CONTENTS

PREFACE

Patients don't walk into their physician's office and say, "I think I have typical pneumonia due to *Streptococcus pneumoniae*." Instead, patients describe their symptoms and how they feel. Physicians then complete a physical examination and draw upon their knowledge about pneumonia and the likely causes of the disease to develop a treatment plan. In other words, physicians must recognize disease and then determine causes.

Most microbiology professors don't live in a clinical world. Microbiologists know a lot about the microorganisms that cause human disease, and they go into great detail talking about them. They will then briefly tell you what diseases these pathogens cause. You then learn all these microbial characteristics and organize them by bacterial shape and Gram stain, viral family, fungal classification, or parasitic class, leaving the diseases to hang at the end of each microbial knowledge set.

This approach to teaching medical microbiology creates a dilemma when an attending physician says to his student physicians, "Dr. Chamberlain's chest radiograph indicates that he has pneumonia. Tell me the most common cause of typical pneumonia in this middle-aged male?" The way you learned microbiology requires that you recall a catalog of organism by organism to see which ones cause pneumonia. This takes several minutes and before you can answer, your attending says, "Don't you know that bacteria are the most likely cause of typical pneumonia and that the most common cause of this pneumonia is *Streptococcus pneumoniae*?" You walk away saying to yourself, "How did my attending get the answer so quick?" What the attending physician did was relearn medical microbiology. Instead of learning the microorganisms and recalling the diseases they cause, this physician learned how to identify a disease and then created lists of the microorganisms that caused that disease.

In the past, this reorienting of the students' medical microbiology knowledge was occurring while medical students were completing their clinical rotations. Unfortunately, case-based questions on USMLE and COMLEX, clinically oriented medical school course work, and recently revised medical school curricula require most medical students to reorient or organize their microbiology knowledge in a clinically relevant way even before they begin their clinical years. *The Big Picture Medical Microbiology* book was written to help you reorient or obtain medical microbiology knowledge in a clinically oriented way and to help you in your clinical rotations.

ORGANIZATION OF THE BOOK

- *The parade of microorganisms does not exist in this book.* This book is organized by organ systems and the infectious diseases caused by microorganisms in that particular system.
- The first chapter in each section presents a "Big Picture" overview that explains the organization of the organ system, some immunologic responses that the system uses to ward off infection, and the diseases as well as the common causes of the diseases that are discussed in chapters that follow within each section. Information in each chapter is discussed using similar headings of etiology, manifestations, epidemiology, pathogenesis, diagnosis, and therapy and prevention.
- About 280 color images are included to help you visualize many of the diseases; some of the images illustrate the results of laboratory tests that are used to identify certain pathogens.
- About 120 tables compare and contrast the various types of a particular disease, summarize the signs and symptoms of a disease, and quickly compare the causes of a disease.
- The last section of the book contains 100 case-based examination questions. Over 30 of the questions contain an image that is necessary for you to examine to correctly answer the question. The questions are in random order to better simulate actual board-type examinations.
- The Appendix contains 19 summary tables and 2 flow charts, which contain a variety of information about microorganisms that will help refresh your memory.

I hope you find this book helpful while studying for your medical school courses and examinations, when preparing to talk with attending physicians about patients in your clinics, and when preparing for USMLE and COMLEX.

—Neal Chamberlain

ACKNOWLEDGMENTS

I thank my father for advice he gave me when I was in college and was anxious about what I should do for a living. He said, "Son, do what you want to do, not what you have to do." Writing this book was something I have dreamed about for a long time. His words stayed with me and helped me through the times when I got stuck looking at a blank page.

I also thank Susan Kelly for all of her hard work as my editor. Her eye for detail, her expertise, and her problem-solving ability were essential to the successful completion of the book.

Thank you to Andi Lynch, a former graduate student and current medical student, class of 2008, who reviewed every chapter and provided invaluable comments while I continued to write and revise the manuscript.

My thanks go to Scott Anderson, DO, and my medical students for helping me reorient the ATSU infectious disease course to make it more useful in the clinics.

Lastly, I thank Gary Schilt, Robert Shaffer, and James Rider for many helpful discussions and suggestions while I was dreaming and thinking about how to write this book.

—Neal Chamberlain

SECTION 1

INTEGUMENT AND SOFT TISSUES

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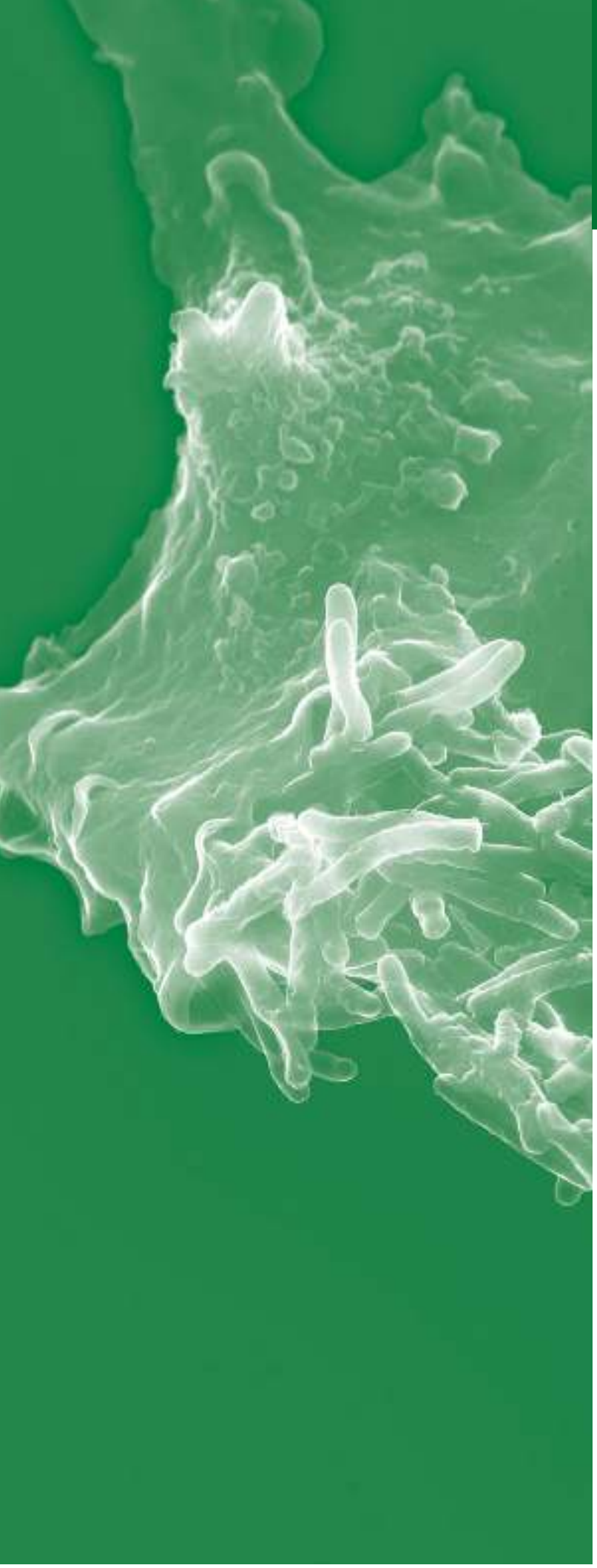

CHAPTER 1

THE BIG PICTURE: INFECTIONS OF THE INTEGUMENT AND SOFT TISSUES

OVERVIEW

The skin, the mucous membranes (e.g., gastrointestinal, respiratory, and urogenital tracts), and other membranous surfaces (e.g., eye) form a barrier that protects the body daily from microbial infections. The skin is the largest of these barriers and is the largest organ of the body. It provides a physical, chemical, and mechanical barrier that protects the body from dehydration, helps maintain proper body temperature, and protects the body from infectious agents.

The skin consists of two layers, the epidermis and the dermis. The epidermis consists of an outer layer of cornified keratinocytes called the stratum corneum. The most common fungal infections of the skin, the dermatophytic infections (e.g., tinea [ringworm]), are seen on and in this layer.

The dermis is composed of dense connective tissue with many white collagenous and elastic fibers. It is much thicker than the epidermis and contains many blood vessels, nerve endings, sebaceous glands, and hair follicles. Furuncles, carbuncles, and erysipelas are examples of diseases that can penetrate this region of the skin following a break in the protective epidermis.

HOW THE SKIN PREVENTS MICROBIAL INFECTIONS

The arid nature of skin prevents many microorganisms from colonizing on it (e.g., gram-negative bacteria). The continuous sloughing off of keratinocytes from the surface of the epidermis does not allow colonizers to overgrow and cause disease. The keratinocytes also provide a waterproof barrier that prevents entry of infectious agents into the body.

The sebaceous glands and the sweat glands secrete substances that inhibit the growth of many organisms. Sebaceous glands in the dermis secrete sebum, which contains a variety of fatty acids and lactic acid. Fatty acids kill most gram-positive bacteria and gram-negative cocci (e.g., *Neisseria* sp.). Lactic acid in the sebum reduces the pH of the skin surface and inhibits the growth of many microorganisms. The sweat glands secrete a

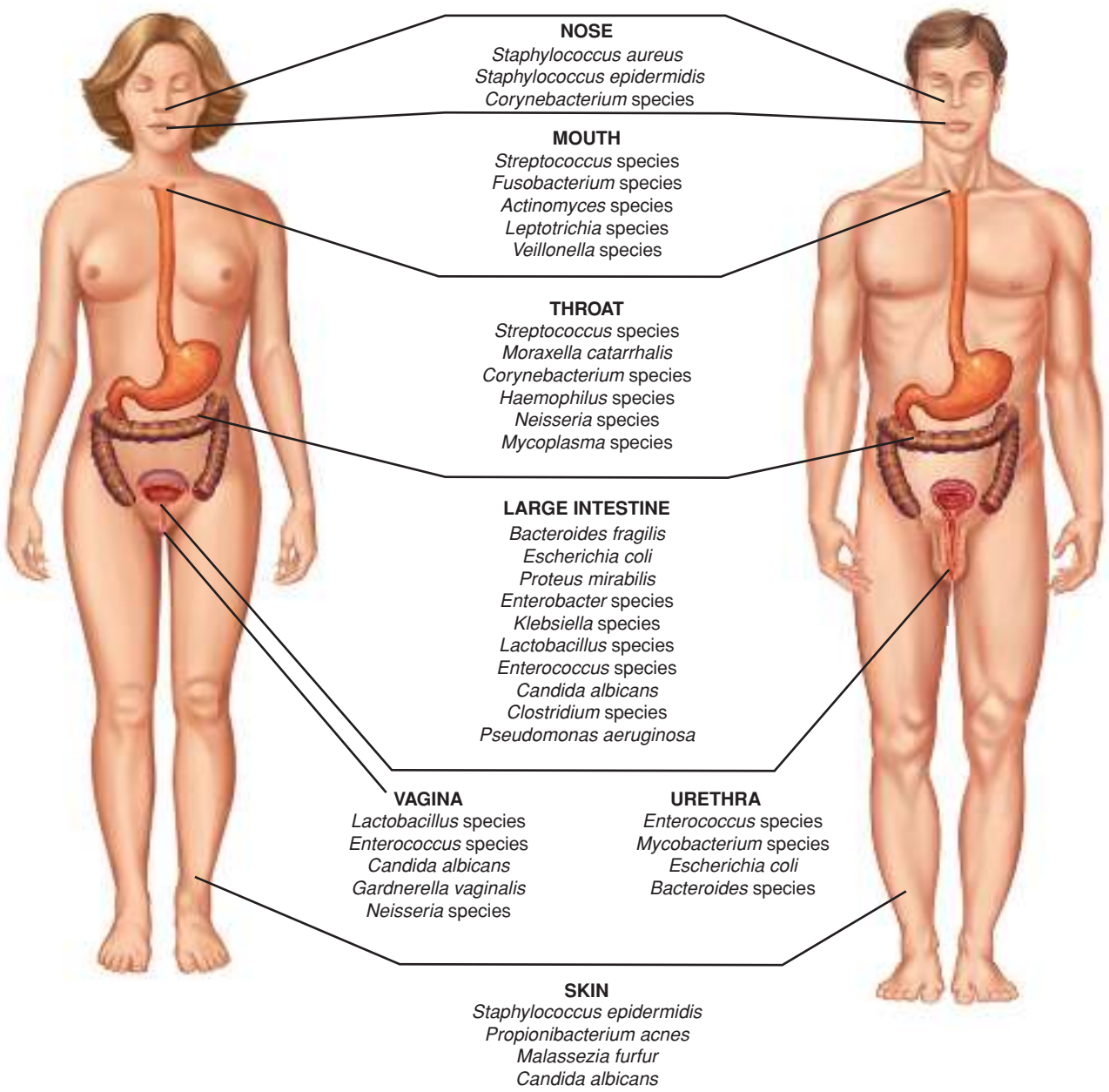

Figure 1-1. Normal flora of the human body. Notice the organisms dwelling on the skin near the bottom of the figure. This is not a comprehensive list but rather the common normal flora organisms.

substance that contains lysozyme and high concentrations of sodium chloride. Lysozyme can catalyze the degradation of bacterial cell walls of certain bacteria, and the high content of sodium chloride in sweat can inhibit the growth of many bacteria. Skin secretions also contain microcidal peptides called beta defensins, which kill microorganisms by disrupting their membranes.

NORMAL FLORA OF THE SKIN

In spite of being a hostile residence for microorganisms, skin is still colonized by a number of microorganisms (Figure 1-1).

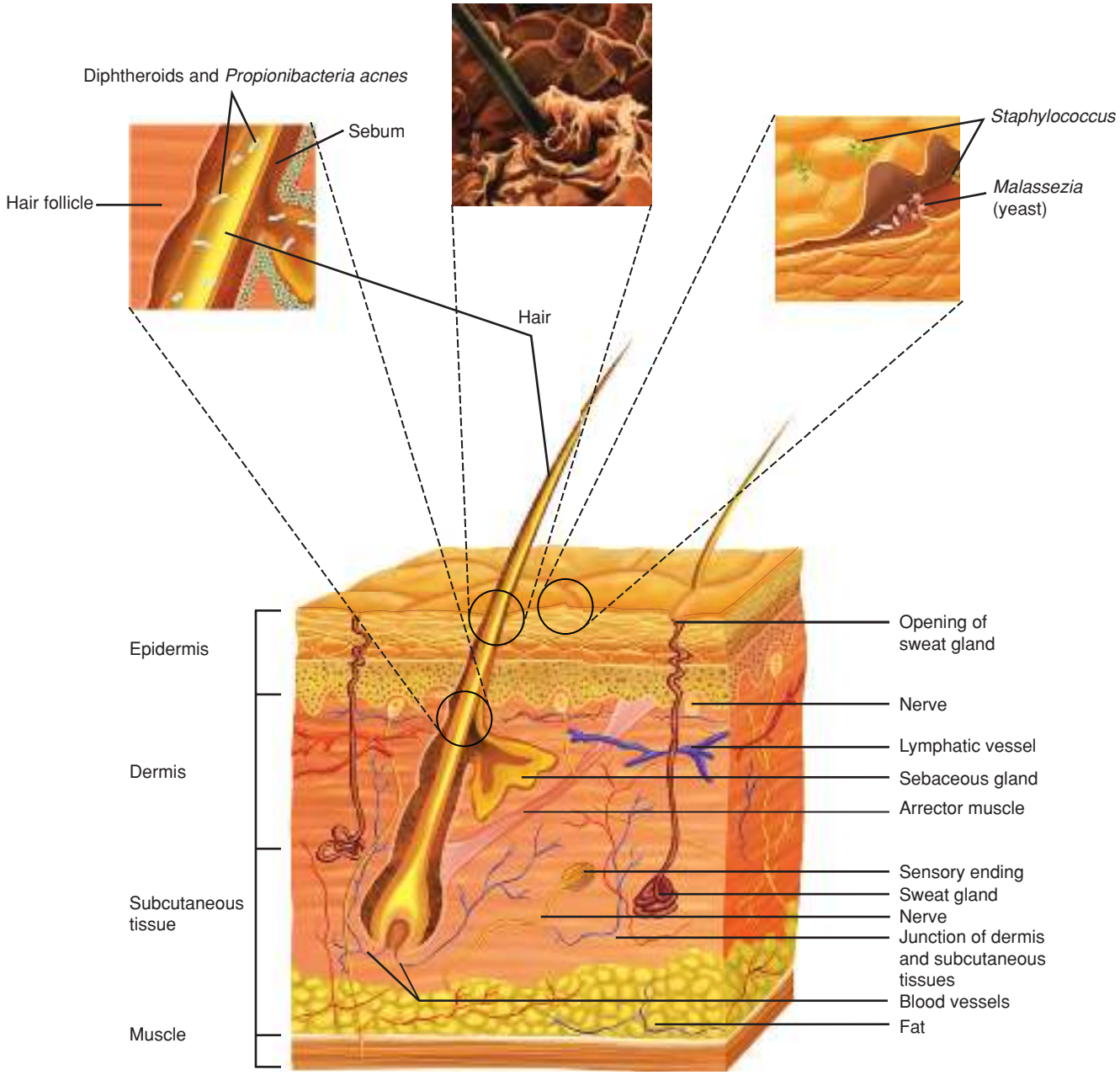

Figure 1-2. Anatomy of skin and sites where the microbes can dwell.

The normal flora (microbes) have the ability to prevent most pathogens from colonizing by preventing their attachment to the surface of the skin or by producing toxins that inhibit the growth of other microorganisms. Most microorganisms live in the superficial layers of the stratum corneum and in the upper parts of the hair follicles (Figure 1-2). Some areas of the skin are colonized more heavily than others (Figure 1-3). For example, moist areas such as the scalp, axilla, and perineum are more heavily colonized than drier areas such as the arms, legs, chest, and back.

Only a few microorganisms are capable of surviving in this hostile, moist environment. Gram-positive bacteria such as coagulase-negative *Staphylococcus*, *Corynebacterium*, and *Propionibacterium* are the most abundant colonizers of the skin.

Staphylococcus epidermidis is the most abundant inhabitant of the skin. *Candida* and *Malassezia* are the fungi that are commonly found colonizing the skin surface. Gram-negative bacilli such as *Enterobacter*, *Pseudomonas*, *Klebsiella*, *Escherichia coli*, and *Proteus* only inhabit the moister regions of the skin.

COMMON BACTERIAL CAUSES OF SKIN INFECTIONS

The most common bacterial causes of skin infection are *Staphylococcus aureus*, *Streptococcus pyogenes,* and *Propionibacterium acne*. *S aureus* can cause bullous impetigo, folliculitis, furuncles, carbuncles, cellulitis, myositis, scalded skin syndrome, and toxic shock syndrome (TSS). Most of these staphylococcal diseases result from invasion and destruction of the skin. Scalded skin syndrome and TSS are examples of bacterial diseases due primarily to toxins such as exfoliative or epidermolytic toxins and TSS toxin, respectively.

S pyogenes can cause impetigo, scarlet fever, erysipelas, necrotizing fasciitis, and streptococcal TSS. Impetigo, erysipelas, and necrotizing fasciitis are caused by invasion or colonization of the skin, whereas scarlet fever and streptococcal TSS are primarily the result of toxin (streptococcal pyogenic exotoxin [SPE] or erythrogenic toxin) production. *Propionibacterium acne* colonizes the hair follicles and is important in contributing to the formation of acne.

COMMON VIRAL INFECTIONS

Oral and genital herpes are caused by either herpes simplex virus type 1 (HSV-1) or HSV-2. Several types of wart-causing papillomaviruses (warts) infect millions of people each year worldwide. The common childhood exanthems are caused by viruses and are discussed in detail in Chapter 2 and are listed in Table 2-1.

COMMON FUNGAL INFECTIONS

Malassezia furfur, the dermatophytes *Microsporum*, *Trichophyton,* and *Epidermophyton,* and *Candida albicans* are the most common causes of skin infections, most of which are limited to the epidermis. *M furfur* and the dermatophytes are only able to infect the superficial keratinized layers of the epidermis. The dermatophytes can also infect the hair and nails. *C albicans* infections are usually restricted to the epidermis and cause intertrigo, folliculitis, paronychia, and onychomycosis.

HOW MICROORGANISMS INFECT THE SKIN

There are numerous infectious agents that cause skin lesions, which can occur via several modes: infection of the skin by the microbe, production of toxins by the microbe, or as an inflammatory response to a microbial infection. Most microorganisms infect the skin through breaks in the protective outer layers of the epidermidis or by infecting hair follicles. Breaks in the skin usually occur following trauma such as bites from insects, animals, or humans, needle sticks, scratches, or burns. If the hair follicles are clogged, they are more likely to become infected. These infections can penetrate deeper into the dermis and in some cases into the subcutaneous fat, fascia, and muscles under the skin causing severe disease (e.g., necrotizing fasciitis, myositis, and gas gangrene).

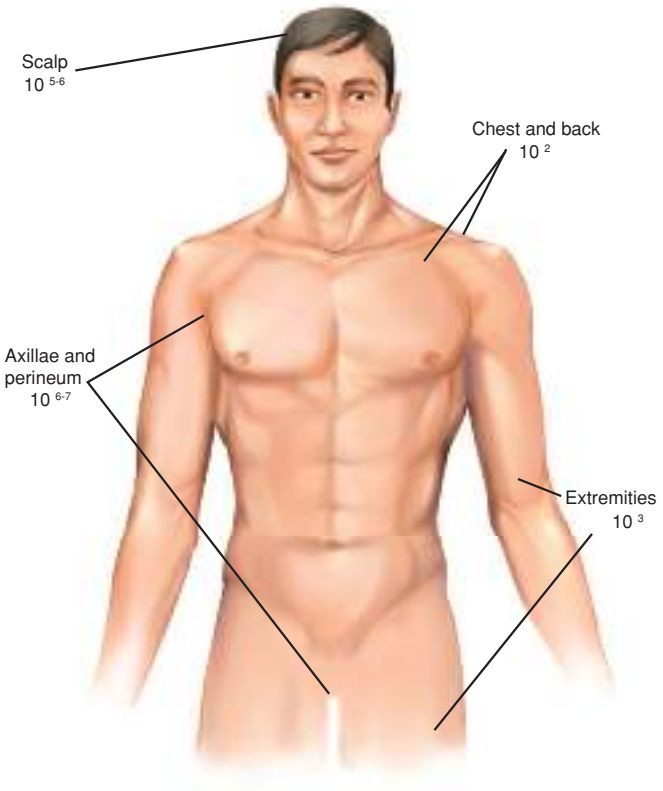

Figure 1-3. Number of normal flora commensals per gram of skin.

TABLE 1-1. Types of Skin Lesions

Depressed Skin Lesions	Flat Skin Lesions	Elevated Skin Lesions	
Gangrene	Macule	Papule	Bulla(e)
Sinus	Patch	Plaque	Exudates (crusts)
Ulcer	Petechiae Purpura Ecchymosis Gangrene	Nodule Vesicle	Abscess Pustule Furuncle Carbuncle

Several different types of skin lesions can develop following a skin infection (Table 1-1). Macules are flat lesions that occur in the plane of the skin (Figure 1-4); ulcers are depressed lesions below the plane of the skin; and papules are elevated above the plane of the skin (Figure 1-5). Some skin lesions are characteristic of the infecting microorganism such as anthrax, which produces a black eschar, whereas other skin lesions such as childhood exanthems and erythematous rashes can be the result of several different organisms.

Not all skin lesions occur following infection of the skin. Some lesions occur following exposure to one or more toxins, and others occur as the result of damage to the capillaries. For example, leakage of small amounts of blood from the capillaries results in the formation of small nonblanching petechiae. If more capillary leakage occurs, purpura and ecchymosis can develop. Still other skin lesions, such as erythema nodosum, follow an inflammatory response to an infection occurring elsewhere in the body.

GROUPING INFECTIONS OF THE INTEGUMENT AND SOFT TISSUES

Infections of the integument and soft tissues can be grouped in several ways: by the microorganisms that cause the disease; by the depth of the infection or damage in the skin; or by the types of lesions produced by the microorganisms. Because most physicians must initially identify infections of the integument and soft tissues by lesions that are visible on a patient, these diseases will be presented based on the type of lesion produced. Chapters 2 through 5 will discuss infections of the integument and soft tissues; each of the chapters will discuss a different set of skin lesions and the microbes that cause them. Maculopapular rashes are discussed in Chapter 2. Chapter 3 discusses papules, plaques, and patches; Chapter 4 discusses vesicular, bullous, and purulent lesions; and Chapter 5 discusses petechial, hemorrhagic, ulcerative, and necrotic lesions.

Figure 1-4. A schematic of a macule, which is a flat lesion usually less than 1 cm in diameter that can be brown, blue, red, or hypopigmented. Note the change in the color of the skin. The lesion cannot be felt but must be seen to be detected.

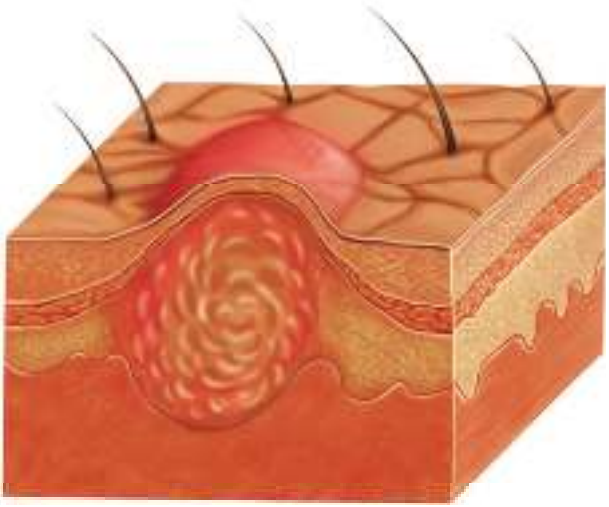

Figure 1-5. A schematic of a papule, a small (<0.5 cm in diameter) solid, elevated skin lesion. The top of a papule can be flat, pointed, or rounded.

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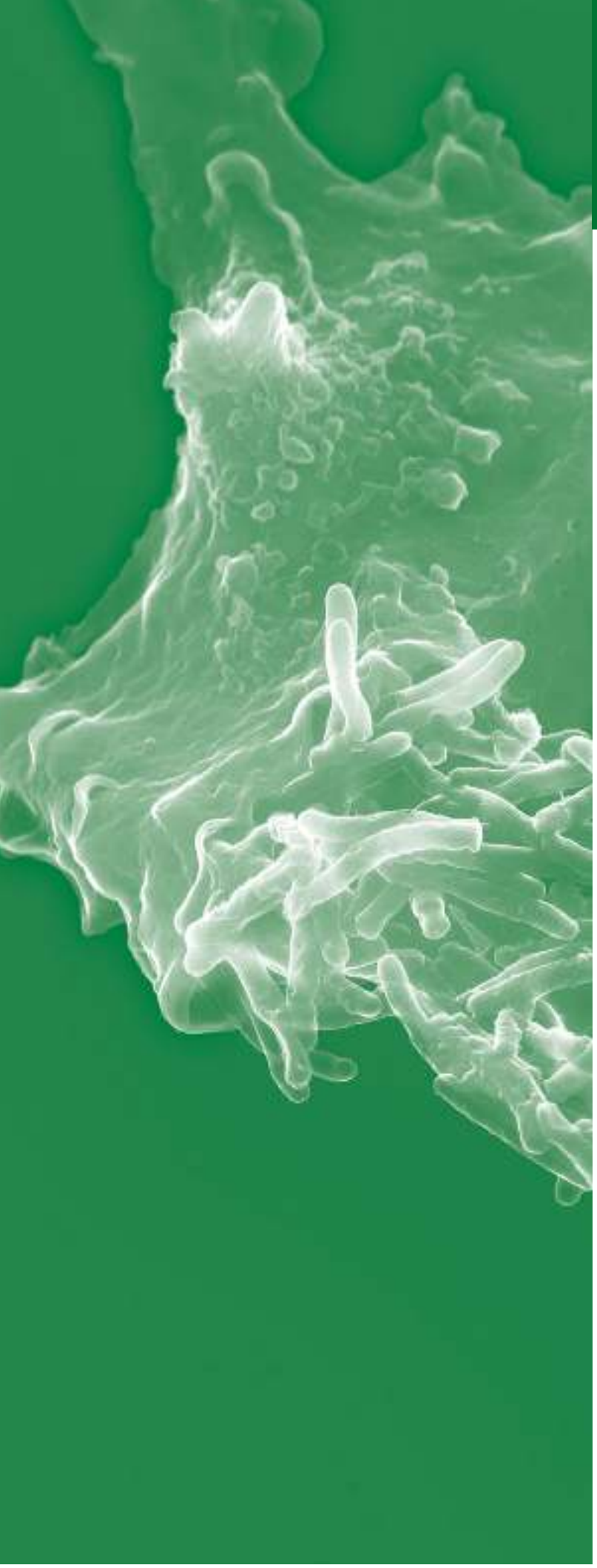

CHAPTER 2

MACULOPAPULAR RASHES

OVERVIEW

A macule is a flat lesion, usually less than 1 cm in diameter, and is a change in the color of the skin. Macules can be brown, blue, red, or hypopigmented; they cannot be felt but must be seen to be detected (Figure 2-1). A papule is a solid raised skin lesion that has distinct borders and is less than 1 cm in diameter (Figure 2-2). Maculopapular rashes are red or erythematous skin lesions with flat or slightly elevated areas in the rash lesion (Figure 2-3).

Table 2-1 lists several different diseases discussed in this chapter. Six of the diseases—erythema infectiosum, scarlet fever, exanthem subitum, rubeola, rubella, and enteroviral rashes—are frequently seen in children and have widespread lesions. Because the diseases are often difficult to distinguish from each other, they are discussed together in the following section. The other maculopapular diseases will be discussed separately, after the discussion of childhood exanthems.

CHILDHOOD EXANTHEMS

Exanthems are widespread rashes that are usually accompanied by systemic symptoms of fever, malaise, and headache. They can occur following a reaction to a microbial toxin, by microbial skin damage, or as an immune response to a microorganism. The common childhood exanthems are listed in Table 2-2.

ETIOLOGY

Viruses are the most common cause of exanthems. With the exception of scarlet fever, which is the only exanthem that is caused by a bacterium, all other childhood exanthems are caused by viruses (Table 2-3).

MANIFESTATIONS

Exanthems

The diseases classified as exanthems are quite similar in appearance and are easily misdiagnosed. Fortunately, most childhood

exanthems have unique characteristics that aid in determining a diagnosis (Table 2-4).

Stages of erythema infectiosum

Erythema infectiosum skin lesions can appear in three stages. Immediately before the rash appears, there is a mild prodromal period that includes headache, coryza, low-grade fever, pharyngitis, and malaise.

- **Stage 1:** The exanthem begins with the classic slapped-cheek appearance when a bright red erythema appears abruptly over the cheeks (Figure 2-4*A*). Usually no erythema is seen on the nose or around the mouth or eyes. The exanthem usually has a sunburn-like appearance and fades within 2–4 days.
- **Stage 2:** Within 1–4 days after the appearance of the "slapped-cheek" rash, an erythematous macular-to-morbilliform eruption occurs primarily on the extremities. Morbilliform lesions are macules 2–20 mm in diameter, and can be confluent in certain regions (see Figure 2-4*B*). The rash is more commonly found on the extensor surfaces and can occasionally involve the palms and soles. Pruritus is rare.
- **Stage 3:** After several days, most of the eruptions that occurred in the second stage fade into a lacy pattern, which is most apparent on the proximal extremities. This reticulate (net-like) pattern is distinctly characteristic of erythema infectiosum and may be the only manifestation seen in some patients. The third stage lasts from 3 days to 3 weeks. Even after the rash begins to fade, it may recur intermittently over several weeks following exercise, sun exposure, friction, bathing in hot water, or a stressful event.

Roseola infantum

Roseola infantum is characterized by a history of high fever (40°C) followed by rapid defervescence. A rash appears after the patient no longer has a fever. This nonpruritic rash is an erythematous macular exanthem or a maculopapular exanthem beginning on the trunk and spreading to the extremities (Figure 2-5). The skin lesions are usually discrete and do not coalesce. The rash will blanch on pressure and will fade within a few hours to 2 days. A prodrome can occur and includes listlessness and irritability. Other symptoms include seizures, diarrhea, and cough.

Scarlet fever

Scarlet fever begins with a prodrome that includes pharyngitis, vomiting, fever, headache, and abdominal pain that precedes the rash from 1 to 2 days. The rash typically begins on the neck and extends to the trunk and extremities (Figure 2-6). This erythematous rash (scarlatiniform) is sandpaper-like in appearance (Figure 2-7) and blanches on pressure. The rash can be pruritic, but it is not painful. The patient's face is usually flushed with perioral pallor (Figure 2-8). After the rash has extended to the trunk and extremities, it becomes more intense along skin folds and produces lines of confluent petechiae, known as Pastia sign. During the first 2 days of the disease, the tongue has a

Figure 2-1. A macule showing the flat lesion with changes in the color of the skin. Macules can be brown, blue, red, or hypopigmented.

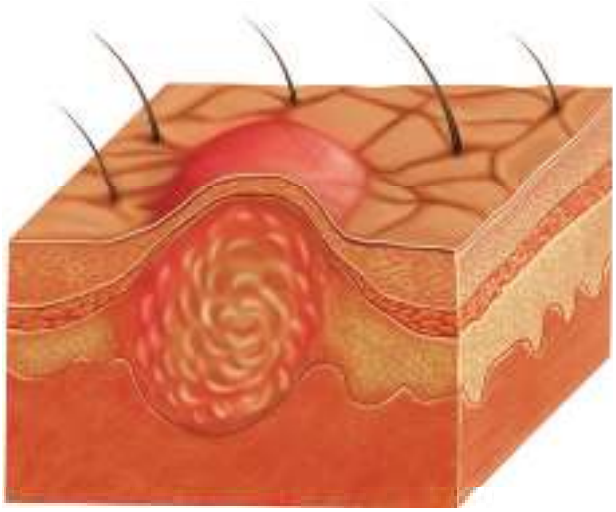

Figure 2-2. A papule showing the small, solid elevated skin lesion. The top of the papule can be flat, pointed, or rounded.

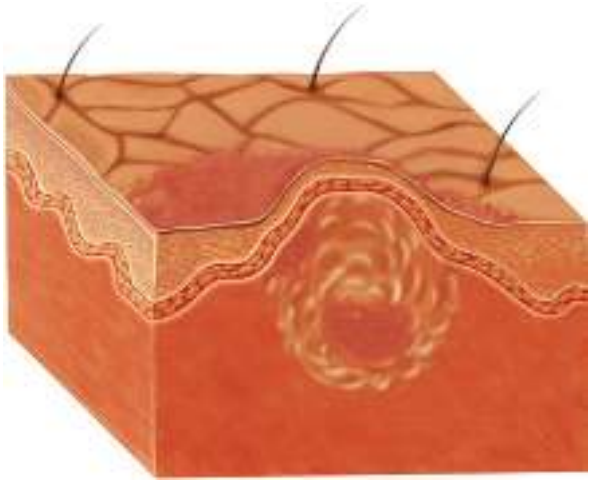

Figure 2-3. A maculopapular rash, an erythematous skin lesion with flat or slightly elevated areas in the rash lesion.

white coating through which the red and edematous papillae can be seen (white strawberry tongue).

The rash begins to fade 3–4 days after onset, and desquamation begins. This phase begins with flakes peeling from the face. Two days after the rash appears, the tongue also desquamates, causing a red tongue with prominent papillae (strawberry tongue) (Figure 2-9). Peeling from the palms and around the fingers occurs about 1 week later and can last up to 1 month.

Rubeola

Rubeola begins with a prodromal phase of coryza, conjunctivitis, nonproductive cough (known as the three Cs of rubeola), and fever. The prodrome is usually followed within 2–3 days by the pathognomonic Koplik spots—blue-gray macules 1–2 mm in diameter on an erythematous base that typically arises on the buccal, gingival, and labial mucosae (Figure 2-10). The maculopapular erythematous rash begins at the hairline and spreads to the trunk and extremities. The rash lesion concentration is highest above the shoulders and often coalesces (morbilliform rash) in this region of the body (Figure 2-11). The rash lasts about 4–6 days and then fades from the head downward. Patients usually recover fully within 7–10 days. Desquamation may occur, but it is usually not severe.

Rubella

Rubella begins with a prodrome, which includes fever, malaise, headache, coryza, and mild conjunctivitis (*patients do not have a cough*). The nonblanching rash appears 1–5 days after the beginning of the prodromal period (Figure 2-12). The faint pink maculopapular rash begins on the forehead and face and spreads caudally to the trunk and extremities. The rash may coalesce, resulting in a scarlatiniform (sandpaper-like) eruption (see Figure 2-7). Within 3 days, the rash fades, beginning at the forehead and face and fading caudally. Petechial lesions on the soft palate, called Forchheimer spots, may also be present. The patient usually has postauricular and suboccipital lymphadenopathy.

Enteroviral rashes

Enteroviral viruses can cause a large variety of rashes, and many patients will have fever, malaise, and headache. The rash may appear at the time the fever begins or it may appear near the end of the fever. The type of rash depends on the type of virus causing the exanthem. Echoviruses can cause a rubella-, measles-, or roseola-like rash. Roseola-like rashes typically appear after the temperature returns to normal, as is seen with roseola infantum. Echovirus 16 causes a roseola-type rash that has been named Boston exanthem. Coxsackie A viruses can cause pustular stomatitis and widespread vesicular lesions.

EPIDEMIOLOGY

- All exanthems are found worldwide and are usually seen in children between the ages of 3 and 15 years.

TABLE 2-1. Maculopapular Rashes

Childhood exanthems
- Enteroviral rashes
- Erythema infectiosum (fifth disease, or slapped-cheek syndrome)
- Roseola infantum (exanthem subitum)
- Scarlet fever
- Rubeola (measles)
- Rubella

Infectious mononucleosis
Secondary syphilis
Rocky Mountain spotted fever
Toxic shock syndrome

TABLE 2-2. Common Viral Childhood Exanthems

Virus	Disease
Coxsackie viruses	Enteroviral rash
Echoviruses	Enteroviral rash
Erythrovirus B19 (formerly called parvovirus B19)	Erythema infectiosum ("slapped-cheek syndrome," or fifth disease)
Human herpesvirus 6B (HHV-6B) and HHV-7	Roseola or exanthem subitum
Varicella-zoster virus (HHV-3)	Chickenpox and zoster (shingles)
Measles virus	Rubeola ("hard measles")
Rubella virus	Rubella (German measles)

TABLE 2-3. Causes of Childhood Exanthems

Causes of the Disease	Disease(s)
Erythrovirus B19	Erythema infectiosum, "slapped-cheek syndrome," or fifth disease
HHV-6 and occasionally HHV-7	Roseola infantum or exanthem subitum
Streptococcus pyogenes	Scarlet fever or scarlatina
Measles virus	Rubeola
Rubella virus	Rubella
Coxsackie A viruses and Echoviruses	Enteroviral rashes or viral rash

TABLE 2-4. Manifestations of the Childhood Exanthems

Disease and Cause	Prodrome	Disease Progression	Distribution of Rash	Unique Signs and Symptoms
Erythema infectiosum and Erythrovirus B19	Headache, coryza, low grade fever, pharyngitis, malaise	**Stage 1:** slapped-cheek appearance **Stage 2:** macular to morbilliform rash on extremities **Stage 3:** reticulate or lacy pattern of rash on extremities	Slapped cheek: sunburn-like rash on cheeks but usually not on nose or around mouth or eyes Rash on extremities: tends to be on extensor surfaces	Slapped cheek rash Reticular or lacy rash
Roseola infantum and human herpes virus 6 (HHV-6) or HHV-7	High fever with rapid defervescence	After fever recedes, rash appears	Rash begins on the trunk, spreads to extremities	High fever that occurs and goes away before the rash appears
Scarlet fever and *Streptococcus pyogenes*	Pharyngitis, vomiting, fever, headache, and abdominal pain precedes rash within 1–2 days	Rash (scarlatiniform) sandpaper-like in appearance and blanches on pressure Desquamation begins when rash starts to fade	Rash begins on neck, then extends to trunk and extremities	Perioral pallor, Pastia sign, White strawberry tongue (early), Strawberry tongue (during desquamation)
Rubeola and measles virus	Coryza, conjunctivitis, nonproductive cough (the 3 Cs), and fever	Maculopapular erythematous rash Lesions usually coalesce on trunk, forming a morbilliform rash	Rash begins at hairline, then spreads to trunk and extremities Rash concentration is highest above the shoulders	The three Cs of rubeola, Koplik spots
Rubella and rubella virus	Fever, malaise, headache, coryza, and mild conjunctivitis, without cough	Nonblanching rash: a faint pink, maculopapular rash, which may coalesce, resulting in a scarlatiniform eruption (sandpaper-like eruption)	Rash begins on forehead and face, spreading caudally to trunk and extremities	Forchheimer spots
Enteroviral rashes and coxsackieviruses or echoviruses	Fever, malaise, and headache	Large variety of rashes Rash may appear at the time fever begins or near the end of fever	Type of rash depends on type of virus causing the exanthem	

- Erythema infectiosum is usually seen in patients in late winter to early spring.
- Transmission is via aerosolized respiratory droplets.

Roseola infantum

Roseola infantum is most common during the spring and fall months. Infection with these viruses is common in children from 6 months to 3 years of age. About 80% of children in the United States are seropositive between the ages of 2 and 4 years. The viruses are shed in saliva and transmitted from latently infected adults to children.

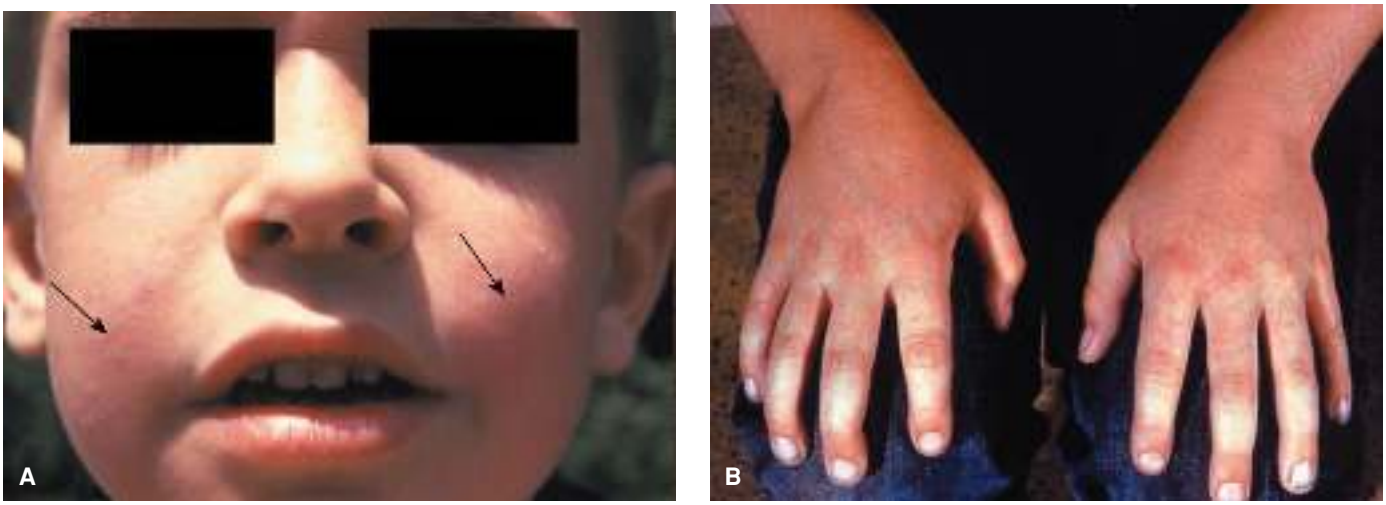

Figure 2-4. A child with erythema infectiosum (fifth disease). **A,** The "slapped-cheek" appearance. **B,** Rash on the hands. Images courtesy of the Centers for Disease Control and Prevention.

Scarlet fever
Scarlet fever is most common in children between the ages of 4 and 8 years of age, and usually occurs 12–24 hours after a pharyngeal infection caused by a pyogenic (erythrogenic) toxin-producing group A streptococcal infection. This bacterium is found in the oropharynx of 15–20% of healthy children and adults. The organism is transmitted by airborne respiratory particles from infected patients and asymptomatic carriers. Scarlet fever only occurs in 10% of patients with group A streptococcal pharyngitis.

Rubeola (Measles)
Rubeola, or measles, occurs only in humans and monkeys. The virus can be transmitted by direct contact, contaminated fomites, or droplet inhalation and is one of the most contagious exanthems, with a 90% attack rate. Due to universal vaccination, less than 100 cases of rubeola are reported in the United States annually (Figure 2-13).

Rubella
Rubella is found only in humans. Respiratory droplets transmit the virus, and it is most commonly seen in unimmunized children aged 5–9 years of age. Due to universal vaccination in the United States, less than 50 cases of rubella are reported annually (Figure 2-14).

Enteroviral rashes
Enteroviral rashes are transmitted person to person following contact with saliva or feces from infected patients. Most cases occur in infants during the summer months. About 10–15 million cases of this disease occur each year in the United States.

PATHOGENESIS

Erythema infectiosum
Erythema infectiosum is an immunologic response to infection of the virus. Acute infection leads to the production of specific

Day 1	Day 2	Day 3
Fever	Fever	Fever gone
40°C	38.9°C	Rash appears

Figure 2-5. An infant with exanthem subitum (roseola) due to human herpes virus 6 (HHV-6) or HHV-7. The infant usually has a high fever for a few days, when the fever abruptly ends and the rash appears on the face, neck, and trunk.