

Diseases of the skin and eyes

Lecture 19 - Chapter 18

Topics

- Skin Defenses
- Eye Defenses
- Skin Diseases
- Eye Diseases

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Skin Defenses

- Skin
- Normal flora

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Skin

- Epidermis
 - Stratum corneum (dead cells are sloughed off)
 - Keratin (protein)
 - Waterproof the skin
 - Protects from microbial invasion
 - Replaced every 25-45 days
 - No nerve endings or blood vessels

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Skin continued

- Dermis
 - Source for epidermis cells
 - Connective tissue (fibers)
 - Nerves, blood vessels, lymphatic
 - Hair follicles, glands (sebum, lysozyme)
- Subcutaneous layer

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The different layers of the skin are important defenses of the skin.

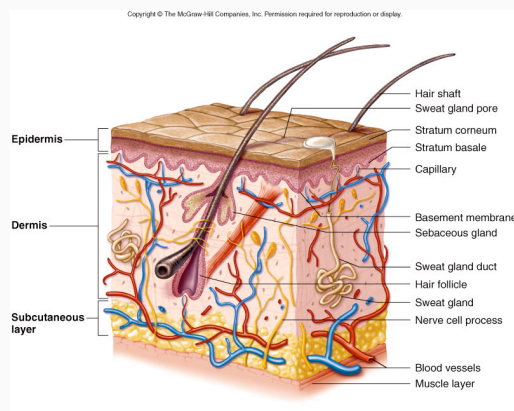


Fig. 18.1 A cross-section of skin

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Normal flora of the skin

- Must survive dry and salty conditions
- Dense populations in the skin folds
- Major organism groups
 - Diphtheroids (*Propionibacterium acnes*)
 - Micrococci (*Staphylococcus epidermidis*)
 - Yeast (*Candida albicans*)

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Eye defenses

- Eye
- Normal flora

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Eye

- Conjunctiva
 - Thin membrane that covers the eye, except the cornea
 - Secretes oil & mucous-containing fluids => best defense
- Cornea
 - Covers the iris
 - Several layers of epithelial cells
 - Epithelial cells can regenerate if damaged
- No lymphocytes, no inflammation
 - Immunologically privileged

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The main parts of the eye that are important eye defenses.

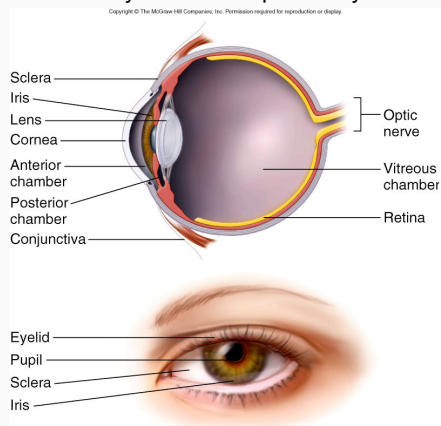


Fig. 18.2 The anatomy of the eye.

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The best defense of the eye is the film of tears, which originates from the lacrimal apparatus of the eye.

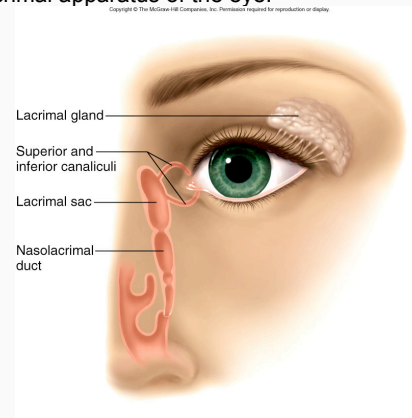


Fig. 18.3 The lacrimal apparatus of the eye.

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Normal flora

- Very few present
- Resemble skin normal flora
 - Diphtheroids
 - Staphylococci
 - *Micrococcus*
 - Streptococci
 - Yeast

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Skin Diseases

- Acne
- Impetigo
- Cellulitis
- Staphylococcal Scalded Skin Syndrome (SSSS)
- Gas gangrene
- Skin rashes
- Warts
- Large skin lesions

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Acne

- Bacterial infection
- Follicle-associated lesion
- Types
 - Comedo
 - Whitehead
 - Blackhead
 - Pustule
 - Cystic

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Features of acne.

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✓ CHECKPOINT 18.1 Acne	
Causative Organism(s)	<i>Propionibacterium acnes</i>
Most Common Mode(s) of Transmission	Endogenous
Virulence Factors	Lipase, inflammatory mediator, other enzymes
Culture/Diagnosis	Based on clinical picture
Prevention	None
Treatment	Antibiotics (topical or oral), isotretinoin

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Checkpoint 18.1 Acne

Impetigo

- Types
 - *Staphylococcus aureus*
 - *Streptococcus pyogenes*
- Peeling skin

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An example of impetigo.

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Fig. 18.4 Impetigo lesions on the face

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Major agent of skin diseases: *Staphylococcus aureus*

- Associated with a number of diseases, including impetigo
- Enzymes
 - Coagulase
 - Hyaluronidase
 - Staphylokinase
 - Lipases
- Most studied non-spore forming pathogen

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Scanning Electron Micrograph (SEM) and colonies of *Staphylococcus aureus*, the causative agent of impetigo.

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Fig. 18.5 *Staphylococcus aureus*

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A positive coagulase test is a primary method of identifying *Staphylococcus aureus*.

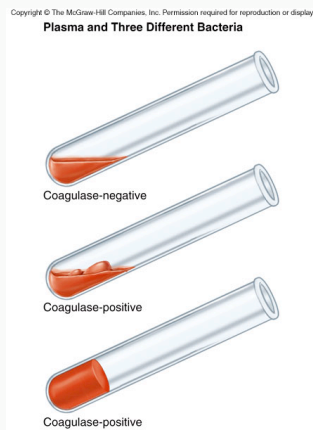


Fig. 18.6 The coagulase test.

Streptococcus pyogenes

- Associated with a number of diseases, including impetigo
- Beta-hemolytic
- M protein

Pathogenesis of *S. pyogenes* involves the conversion of plasminogen to plasmin, which can degrade host tissue.

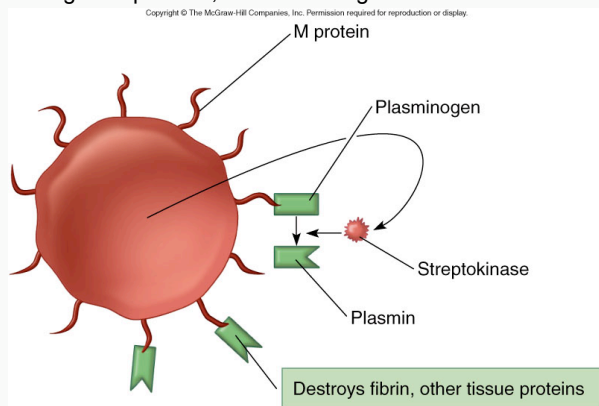


Fig. 18.8 Plasmin activation by *S. pyogenes*

Features of impetigo caused by either *Streptococcus pyogenes* or *Staphylococcus aureus*.

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CHECKPOINT 18.2 Impetigo		
Causative Organism(s)	<i>Staphylococcus aureus</i>	<i>Streptococcus pyogenes</i>
Most Common Modes of Transmission	Direct contact, indirect contact	Direct contact, indirect contact
Virulence Factors	Exfoliative toxin A, coagulase, other enzymes	Streptokinase, plasminogen-binding ability, hyaluronidase, M protein
Culture/Diagnosis	Routinely based on clinical signs, when necessary, culture and Gram stain, coagulase and catalase tests, multitest systems, PCR	Routinely based on clinical signs, when necessary, culture and Gram stain, coagulase and catalase tests, multitest systems, PCR
Prevention	Hygiene practices	Hygiene practices
Treatment	Topical mupirocin, oral cephalosporin	Topical mupirocin, oral cephalosporin
Distinguishing Features	Seen more often in older children, adults	Seen more often in newborns; may have some involvement in all impetigo (preceding <i>S. aureus</i> in staphylococcal impetigo)

Checkpoint 18.2 Impetigo

Cellulitis

- Bacterial infection
- Fungal infection
- Infection of the dermis and subcutaneous tissues
- Lymphangitis
- Immunocompromised individuals are at risk

Features associated with cellulitis.

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CHECKPOINT 18.3 Cellulitis			
Causative Organism(s)	<i>Staphylococcus aureus</i>	<i>Streptococcus pyogenes</i>	Other bacteria or fungi
Most Common Modes of Transmission	Parenteral implantation	Parenteral implantation	Parenteral implantation
Virulence Factors	Exfoliative toxin A, coagulase, other enzymes	Streptokinase, plasminogen-binding ability, hyaluronidase, M protein	–
Culture/Diagnosis	Based on clinical signs	Based on clinical signs	Based on clinical signs
Prevention	–	–	–
Treatment	Aggressive treatment with oral or IV antibiotic (cephalexin); surgery sometimes necessary	Aggressive treatment with oral or IV antibiotic (cephalexin); surgery sometimes necessary	Aggressive treatment with oral or IV antibiotic (cephalexin); surgery sometimes necessary
Distinguishing Features	–	–	More common in immunocompromised

Checkpoint 18.3 Cellulitis

Staphylococcus scalded skin syndrome **SSSS**

- Bacterial infection
- Affects mostly newborns and babies
- Bullous lesions
- Desquamation (lose of protective keratinized layer)

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Exfoliative toxin causes the major signs and symptoms of SSSS.

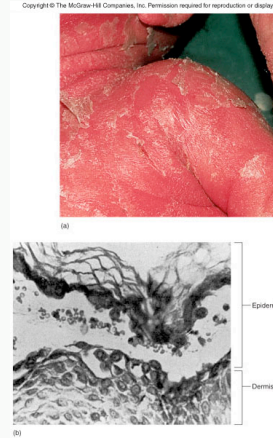


Fig. 18.9 Staphylococcus scalded skin syndrome in a newborn

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Features of SSSS or desquamation diseases.

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✓ CHECKPOINT 18.4 Scalded Skin Syndrome

Causative Organism(s)	<i>Staphylococcus aureus</i>
Most Common Modes of Transmission	Direct contact, droplet contact
Virulence Factors	Exfoliative toxins A and B
Culture/Diagnosis	Histological sections; culture performed but false negatives common
Prevention	Eliminate carriers in contact with neonates
Treatment	Immediate systemic antibiotics (cloxacillin or cephalixin)
Distinguishing Features	Split in skin occurs <i>within</i> epidermis

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Checkpoint 18.4 Major Desquamation Diseases

Gas Gangrene

- Bacterial infection
- Anaerobic
- Toxins
- Gas formation
- Two forms
 - Localized
 - Diffused (myonecrosis)

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Myonecrosis, which has spread to other areas of the body.

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Fig. 18.10 The clinical appearance of myonecrosis

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Features of gas gangrene.

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✓ CHECKPOINT 18.5 Gas Gangrene

Causative Organism(s)	<i>Clostridium perfringens</i> , other species
Most Common Modes of Transmission	Vehicle (soil), endogenous transfer from skin, GI tract, reproductive tract
Virulence Factors	Alpha toxin, other exotoxins, enzymes, gas formation
Culture/Diagnosis	Gram stain, CT scans (abdominal infections), X ray, clinical picture
Prevention	Clean wounds, debride dead tissue
Treatment	Cephalosporin, surgical removal, oxygen therapy

Checkpoint 18.5 Gas gangrene

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Lepromatous leprosy is a more severe lesion, and is associated with disfigurement (lepromas).



Fig. 18.13 A clinical picture of lepromatous leprosy.

Features of leprosy.

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✓ CHECKPOINT 18.6 Leprosy	
Causative Organism(s)	<i>Mycobacterium leprae</i>
Most Common Modes of Transmission	Not clear, possibly direct or droplet contact, mechanical vector
Virulence Factors	Binding to Schwann cells, ability to survive within macrophages
Culture/Diagnosis	Clinical signs, microscopy, biopsy, PCR, patient history
Prevention	Isolation of infected people, chemoprophylaxis of contacts
Treatment	Multidrug treatment including rifampin and dapsone; varies with form of leprosy

Checkpoint 18.6 Leprosy

Skin rashes

- Vesicular or pustular rash diseases
- Maculopapular rash diseases

Vesicular rash diseases

- ==> Elevated lesions filled with fluid
- Viral infection
 - Chickenpox
 - Smallpox

Chickenpox

- Common
- Benign
- Life-threatening for immunocompromised individuals
- Recuperation can result in **Varicella-zoster virus** infection
 - Reemerge as shingles (skin lesion)

Chicken pox reemerges as shingles, due to stress including X-ray treatments, drug therapy, or a developing malignancy.

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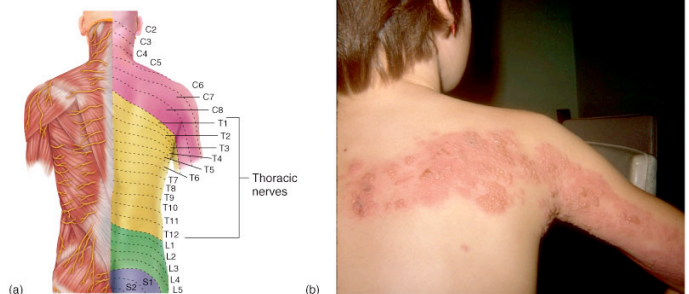


Fig. 18.15 Varicella-zoster virus reemergence as shingles

Smallpox

- Only pathogen ever completely eliminated due to world-wide vaccine program
- Very infectious viral disease (epidemic)
- Considered a bioterrorism agent

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Examples of the rashes associated with chickenpox and smallpox in humans.





Fig. 18.14 Images of chickenpox and small pox.

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Features of chicken pox and small pox.

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CHECKPOINT 18.7 Vesicular/Pustular Rash Diseases		
Disease	Chickenpox	Smallpox
Causative Organism(s)	Human herpesvirus 3 (varicella-zoster virus)	Variola virus
Most Common Modes of Transmission	Droplet contact, inhalation of aerosolized lesion fluid	Droplet contact, indirect contact
Virulence Factors	Ability to fuse cells, ability to remain latent in ganglia	Ability to dampen, avoid immune response
Culture/Diagnosis	Based largely on clinical appearance	Based largely on clinical appearance
Prevention	Live attenuated vaccine	Live virus vaccine (vaccinia virus)
Treatment	None in uncomplicated cases; acyclovir for high risk	–
Distinguishing Features	No fever prodrome; lesions are superficial, in centripetal distribution (more in center of body)	Fever precedes rash, lesions are deep and in centrifugal distribution (more on extremities)
Appearance of Lesion		

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Checkpoint 18.7 Vesicular/pustular rash

Maculopapular rash diseases

- Flat to slightly raised colored bump
 - Measles or Rubeola (Syncytia formation)
 - Rubella (Disrupts fetus development)
- => Vaccine available: MMR-measles, mumps, rubella
- Fifth disease (Erythema infectiosum, human parvovirus B19)
- Roseola (HHV6 or HHV7; mononucleosis- or hepatitis-like symptoms)

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An example of the rash of measles.

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Fig. 18.16 The rash of measles.

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Fetal injury varies based on the time of infection.

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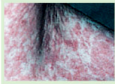





Fig. 18.17 An infant born with congenital rubella

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Features for measles, rubella, fifth disease, and roseola.

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CHECKPOINT 18.8 Maculopapular Rash Diseases				
Disease	Measles	Rubella	Fifth Disease	Roseola
Causative Organism(s)	Measles virus	Rubella virus	Parvovirus B19	Human herpesvirus 6 or 7
Most Common Modes of Transmission	Droplet contact	Droplet contact	Droplet contact, direct contact	?
Virulence Factors	Syncytium formation, ability to suppress CMI	In fetuses: inhibition of mitosis, induction of apoptosis, and damage to vascular endothelium	-	Ability to remain latent
Culture/Diagnosis	ELISA for IgM, acute/convalescent IgG	Acute IgM, acute/convalescent IgG	Usually diagnosed clinically	Usually diagnosed clinically
Prevention	Live attenuated vaccine (MMR)	Live attenuated vaccine (MMR)	-	-
Treatment	No antivirals; vitamin A, antibiotics for secondary bacterial infections	-	-	-
Distinguishing Features of the Rashes	Starts on head, spreads to whole body, lasts over a week	Milder red rash, lasts approximately 3 days	"Slapped-face" rash first; spreads to limbs and trunk, tends to be confluent rather than distinct bumps	High fever precedes rash stage—rash not always present
Appearance of Lesions				

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Checkpoint 18.8 Maculopapular rash diseases



Warts

- Papillomas - different HPV virus types (Nearly everyone is infected)
 - Plantar warts (HPV-1)
 - Flat warts (HPV-3,10,28,49)
- Molluscum contagiosum (Distributed world-wide (endemic))

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Features of papillomas and molluscum contagiosum.

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CHECKPOINT 18.9 Wart and Wartlike Eruptions		
Disease	Warts	Molluscum contagiosum
Causative Organism(s)	Human papillomaviruses	Molluscum contagiosum viruses
Most Common Modes of Transmission	Direct contact, autoinoculation, indirect contact	Direct contact, including sexual contact, autoinoculation
Virulence Factors	-	-
Culture/Diagnosis	Clinical diagnosis, also histology, microscopy, PCR	Clinical diagnosis, also histology, microscopy, PCR
Prevention	Avoid contact	Avoid contact
Treatment	Home treatments, cryosurgery (virus not eliminated)	Usually none, although mechanical removal can be performed (virus not eliminated)
Appearance of Lesions		

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Checkpoint 18.9 Wart and wart-like eruptions.

Large skin lesions

- Cutaneous anthrax
- Ringworm
- Superficial mycoses

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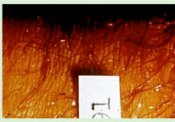

Cutaneous anthrax

- Bacterial infection (*Bacillus anthracis*)
- Endospore formation and germination
- Untreated cases can be fatal
- Vaccine available

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Features of leishmaniasis and cutaneous anthrax.

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CHECKPOINT 18.10 Large Pustular Skin Lesions		
Disease	Leishmaniasis	Cutaneous Anthrax
Causative Organism(s)	<i>Leishmania</i> spp.	<i>Bacillus anthracis</i>
Most Common Modes of Transmission	Biological vector	Direct contact with endospores
Virulence Factors	Multiplication within macrophages	Endospore formation; capsule, lethal factor, edema factor (see chapter 20)
Culture/Diagnosis	Culture of protozoa, microscopic visualization	Culture on blood agar; serology, PCR performed by CDC
Prevention	Avoiding sand fly	Avoid contact; vaccine available but not widely used
Treatment	Pentamist	Ciprofloxacin, doxycycline, penicillin
Distinguishing Features	Mucocutaneous and systemic forms	Can be fatal
Appearance of Lesions		

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Checkpoint 18.10 large pustula skin lesions

Ringworm

- Infection by dermatophytic fungus (mycosis)
- Names of infectious conditions— tinea
 - Scalp (tinea capitis)
 - Beard (tinea barbae)
 - Body (tinea corporis)
 - Groin (tinea cruris)
 - Foot (tinea pedis)
 - Hand (tinea poris)
 - Nail (tinea unguium)

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Ringworm of the scalp (Fig. 18.18) and body (Fig. 18.19).

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An example of ringworm of the feet and fingernail.



Fig. 18.20 Ringworm of the extremities

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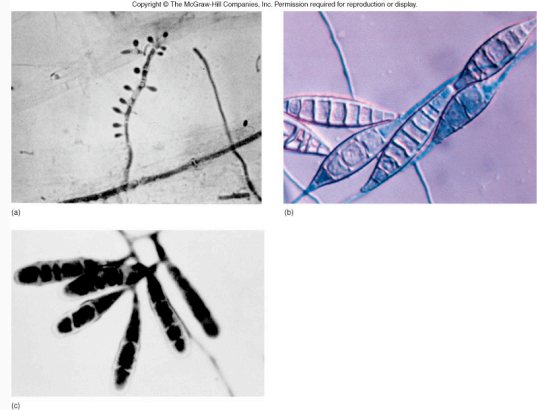


Fig. 18.21 Dermatophyte spores associated with ringworm

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Superficial mycosis

- Fungal infection
 - Yeast infection
 - *Trichophyton* infection (Athlete's foot)
- Cosmetic effects with no inflammation
 - Tinea versicolor

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Features of cutaneous and superficial mycoses.

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CHECKPOINT 18.11 Cutaneous and Superficial Mycoses		
Disease	Cutaneous Infections	Superficial Infections (Tinea Versicolor)
Causative Organism(s)	<i>Trichophyton</i> , <i>Microsporum</i> , <i>Epidermophyton</i>	<i>Malassezia furfur</i>
Most Common Modes of Transmission	Direct and indirect contact, vehicle (soil)	Endogenous "normal flora"
Virulence Factors	Ability to degrade keratin, invoke hypersensitivity	–
Culture/Diagnosis	Microscopic examination, KOH staining, culture	Usually clinical, KOH can be used
Prevention	Avoid contact	None
Treatment	Topical tolnaftate, itraconazole, terbinafine, miconazole, thiabendazine	Topical antifungals

Checkpoint 18.11 Cutaneous and superficial mycoses.

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Eye Diseases

- Conjunctivitis
- Trachoma
- Keratitis
- River blindness

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Conjunctivitis

- Bacterial infection
- Viral infection
- Neonates are at risk during birth
- Inflammation

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An example of neonatal conjunctivitis.

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Fig. 18.23 Conjunctivitis

Features of neonatal, bacterial and viral conjunctivitis.

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CHECKPOINT 18.12 Conjunctivitis			
Disease	Neonatal Conjunctivitis	Bacterial Conjunctivitis	Viral Conjunctivitis
Causative Organism(s)	<i>Chlamydia trachomatis</i> or <i>Neisseria gonorrhoeae</i>	<i>Streptococcus pyogenes</i> , <i>Streptococcus pneumoniae</i> , <i>Staphylococcus aureus</i> , <i>Haemophilus influenzae</i> , <i>Moraxella</i> , and also <i>Neisseria gonorrhoeae</i> , <i>Chlamydia trachomatis</i>	Adenoviruses and others
Most Common Modes of Transmission	Vertical	Direct, indirect contact	Direct, indirect contact
Virulence Factors	–	–	–
Culture/Diagnosis	Gram stain and culture	Clinical diagnosis	Clinical diagnosis
Prevention	Screen mothers, apply antibiotic or silver nitrate to newborn eyes	Hygiene	Hygiene
Treatment	Topical and oral antibiotics	Broad-spectrum topical antibiotic, often ciprofloxacin	None, although antibiotics often given because type of infection not distinguished
Distinguishing Features	In babies <28 days old	Mucopurulent discharge	Serous (clear) discharge

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Checkpoint 18.12 Conjunctivitis

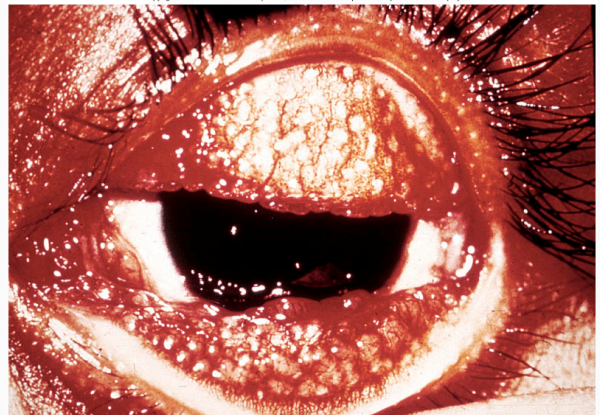
Trachoma

- Bacterial infection (*Chlamydia trachomatis*)
- Endemic
- Pannus - immune-mediated corneal flap
- Blindness – chronic and secondary infections

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An example of ocular trachoma.

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Fig. 18.24 Ocular trachoma.

Features of trachoma.

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CHECKPOINT 18.13 Trachoma

Causative Organism(s)	<i>C. trachomatis</i> serovars A–C
Most Common Modes of Transmission	Indirect contact, mechanical vector
Virulence Factors	Intracellular growth
Culture/Diagnosis	Detection of inclusion bodies in stained preparations
Prevention	Hygiene, vector control, prompt treatment of initial infection
Treatment	Oral doxycycline or topical erythromycin

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Checkpoint 18.13 Trachoma

Keratitis

- HSV infection of the Cornea
- Serious infection – complete corneal destruction

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Features of keratitis.

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CHECKPOINT 18.14 Keratitis

Causative Organism(s)	Herpes simplex virus	Miscellaneous microorganisms
Most Common Modes of Transmission	Reactivation of latent virus, although primary infections can occur in the eye	Often traumatic introduction (parenteral)
Virulence Factors	Latency	Various
Culture/Diagnosis	Usually clinical diagnosis; viral culture or PCR if needed	Various
Prevention	–	–
Treatment	Topical vidarabine and/or oral acyclovir	Specific antimicrobials

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Checkpoint 18.14 keratitis

River blindness

- Parasite infection
 - *Onchocerca volvulus* nematode contains endophytic bacterium *Wolbachia* sp. (mutualism) and both contribute towards the infection
- Chronic
- Endemic

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Features of river blindness.

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CHECKPOINT 18.15 River Blindness

Causative Organism(s)	<i>Wolbachia</i> plus <i>Onchocerca volvulus</i>
Most Common Modes of Transmission	Biological vector
Virulence Factors	Induction of inflammatory response
Culture/Diagnosis	“Skin snips”: small piece of skin in NaCl solution examined under microscope and microfilariae counted
Prevention	Avoiding black fly
Treatment	Ivermectin
Distinguishing Features	Worms often visible in eye

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Checkpoint 18.15 River blindness

Summary of diseases of the skin and eye.

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Microorganism	Disease	Chapter Location
Gram-Positive Bacteria		
<i>Propionibacterium acnes</i>	Acne	Acne, p. 543
<i>Staphylococcus aureus</i>	Impetigo, cellulitis, scalded skin syndrome, folliculitis, abscesses (furuncles and carbuncles), necrotizing fasciitis	Impetigo, p. 544 Cellulitis, p. 547 Scalded skin syndrome, p. 547 Impetigo, p. 547
<i>Streptococcus pyogenes</i>	Impetigo, cellulitis, erysipelas, necrotizing fasciitis	Impetigo, p. 545 Cellulitis, p. 547; Inguinal lymphadenitis, p. 550
<i>Clavibacterium perfringens</i>	Gas gangrene	Gas gangrene, p. 548
<i>Bacillus anthracis</i>	Cutaneous anthrax	Large pustular skin lesions, p. 544
Gram-Negative Bacteria		
<i>Mycobacterium leprae</i> *	Leprosy	Leprosy, p. 551
<i>Nisseria gonorrhoeae</i>	Neisserial conjunctivitis	Conjunctivitis, p. 548
<i>Chlamydia trachomatis</i>	Neisserial conjunctivitis, trachoma	Conjunctivitis, p. 548 Trachoma, p. 550
<i>Wolbachia</i> (in combination with <i>Chickensia</i>)	River blindness	River blindness, p. 550
DNA Viruses		
Human herpesvirus 3 (varicella) virus	Chickpox	Vesicular or pustular rash diseases, p. 554
Varicella virus	Smallpox	Vesicular or pustular rash diseases, p. 556
Herpesvirus B 19	Fifth disease	Maculopapular rash diseases, p. 562
Human herpesvirus 6 and 7	Roseola	Maculopapular rash diseases, p. 562
Human papillomavirus	Warts	Warts and wartlike eruptions, p. 562
Molluscum contagiosum virus	Molluscum contagiosum	Warts and wartlike eruptions, p. 562
Herpes simplex virus	Keratitis	Keratitis, p. 550
RNA Viruses		
Moraxella virus	Moraxella	Maculopapular rash diseases, p. 564
Rubella virus	Rubella	Maculopapular rash diseases, p. 564
Fungi		
<i>Trichophyton</i>	Ringworm	Ringworm, p. 565
<i>Microsporum</i>	Ringworm	Ringworm, p. 565
<i>Epidermophyton</i>	Ringworm	Ringworm, p. 565
<i>Microascus joffei</i>	Superficial mycosis	Superficial mycoses, p. 565
Protozoa		
<i>Leishmania</i> spp.	Leishmaniasis	Large pustular skin lesions, p. 564
Helminths		
<i>Onchocerca volvulus</i> (in combination with <i>Wolbachia</i>)	River blindness	River blindness, p. 550

*These two viruses along with the genus *Epidermophyton* is generally not considered gram positive or gram negative.

Taxonomic organization of microorganisms causing diseases of the skin and eyes.

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Infectious Diseases Affecting the Skin and Eyes.

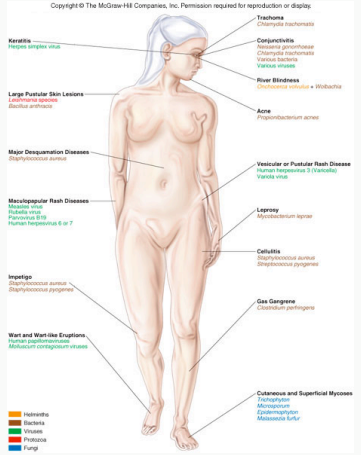


Fig. 18.p573