MIKROBIOLOGI KESEHATAN

By: KUSNADI,MSI.

Figure 15-12 Microbiology, 6/e

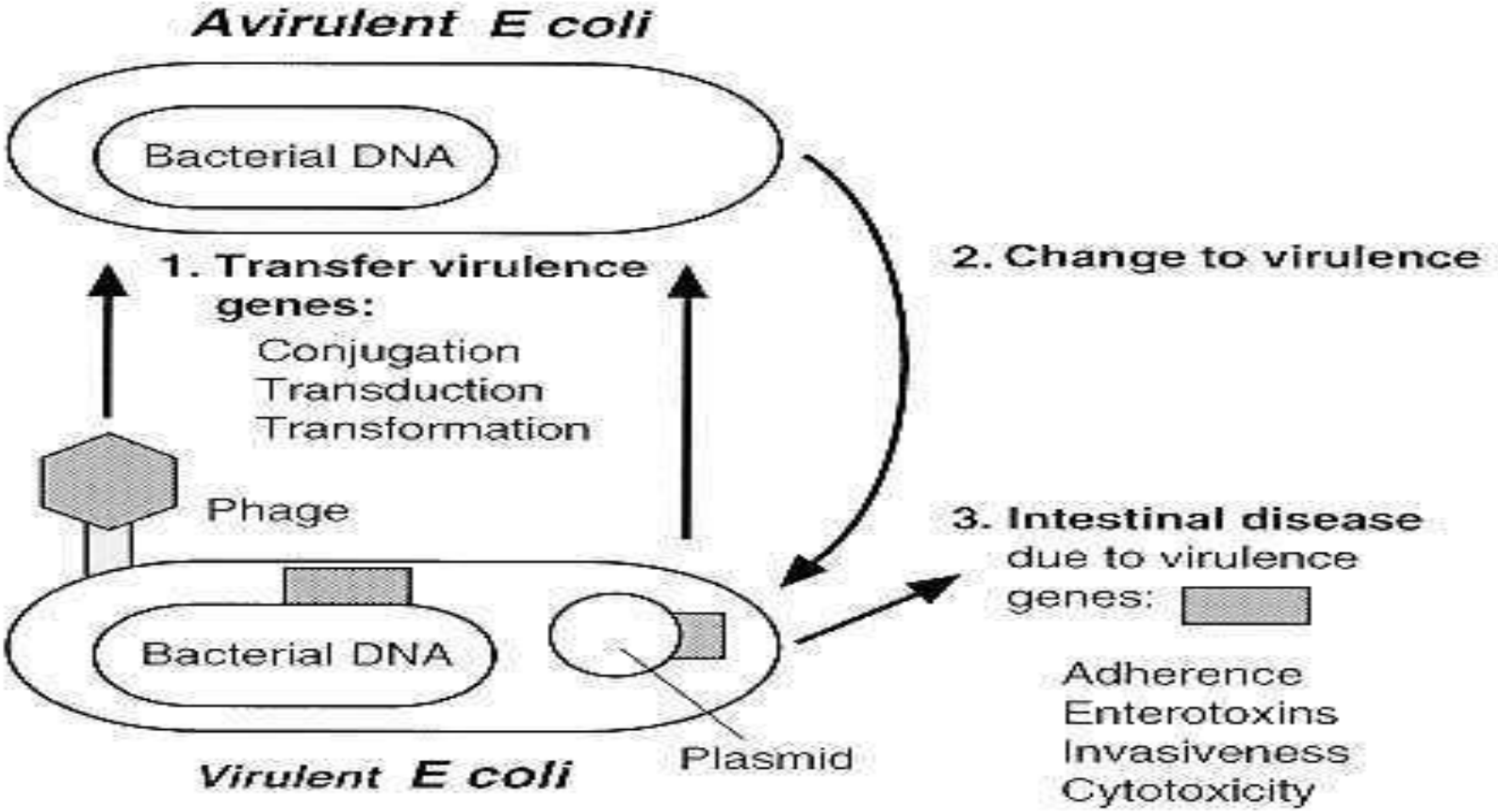
Mikroba bagi manusia

- Menguntungkan------ mikroflora normal sebagai komensal

  mikroflora normal ⟹ patogen oportunistik

- Merugikan ------agen kausatif berbagai penyakit infeksi-----sebagai parasit

Oportunistik : *E.coli*



Mikroorganisme secara alami senantiasa kontak dengan tubuh organisme, yang dapat terjadi secara :

1. **Transien** : mikroorganisme kontak dengan organisme tanpa memperbanyak diri
2. **Residen** : mikroorganisme kontak dengan organisme dan melakukan perbanyakan diri

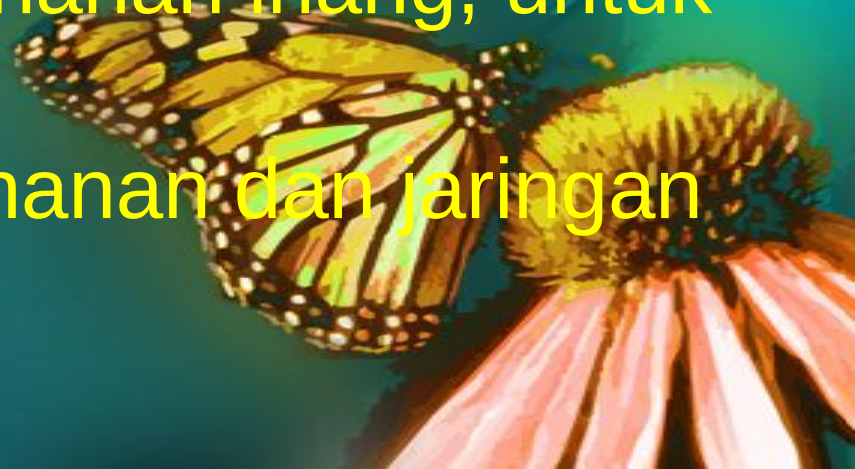
- Mikroorganisme yang membuat kerusakan atau kerugian terhadap tubuh inang disebut Patogen.
- Kemampuan mikroorganisme untuk menimbulkan penyakit disebut Patogenisitas (patogenesis)
- Derajat patogenisitas suatu parasit dalam menimbulkan penyakit disebut Virulensi.
- Kemampuan organisme melawan suatu infeksi penyakit disebut Resistensi.

Mikroba sebagai patogen

- Mikroba sebagai patogen tergantung pada:
  - virulensi mikroba
  - jumlah mikroba mencapai quorum
  - imunitas tubuh inang

Tahapan mikroba menyebabkan penyakit infeksi

- Harus menginfeksi inang (suatu patogen primer harus memasuki jaringan inang).
- Harus melakukan metabolisme dan memperbanyak diri dalam jaringan inang (kolonisasi).
- Harus melawan pertahanan inang, untuk sementara.
- Harus merusak pertahanan dan jaringan inang.



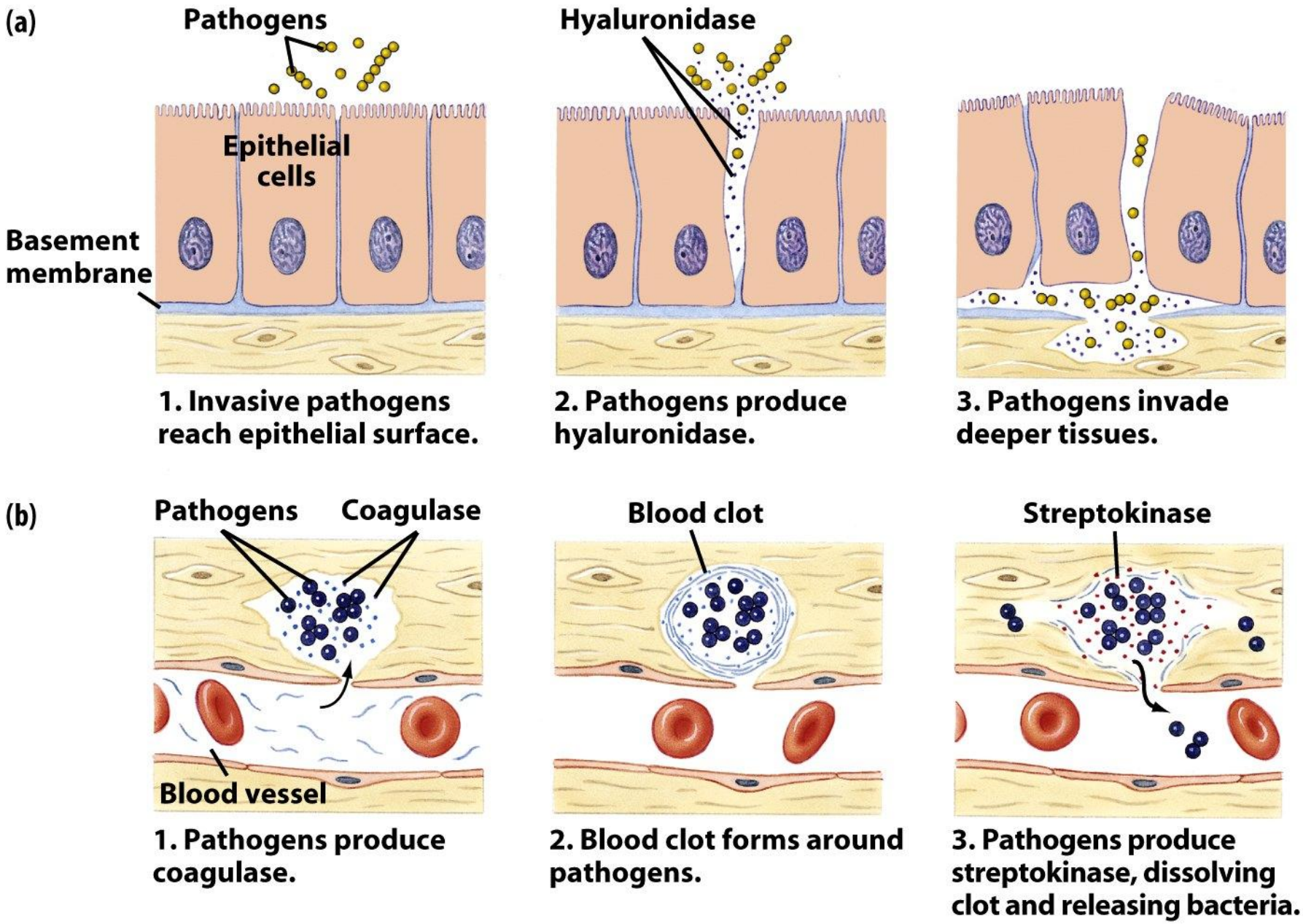
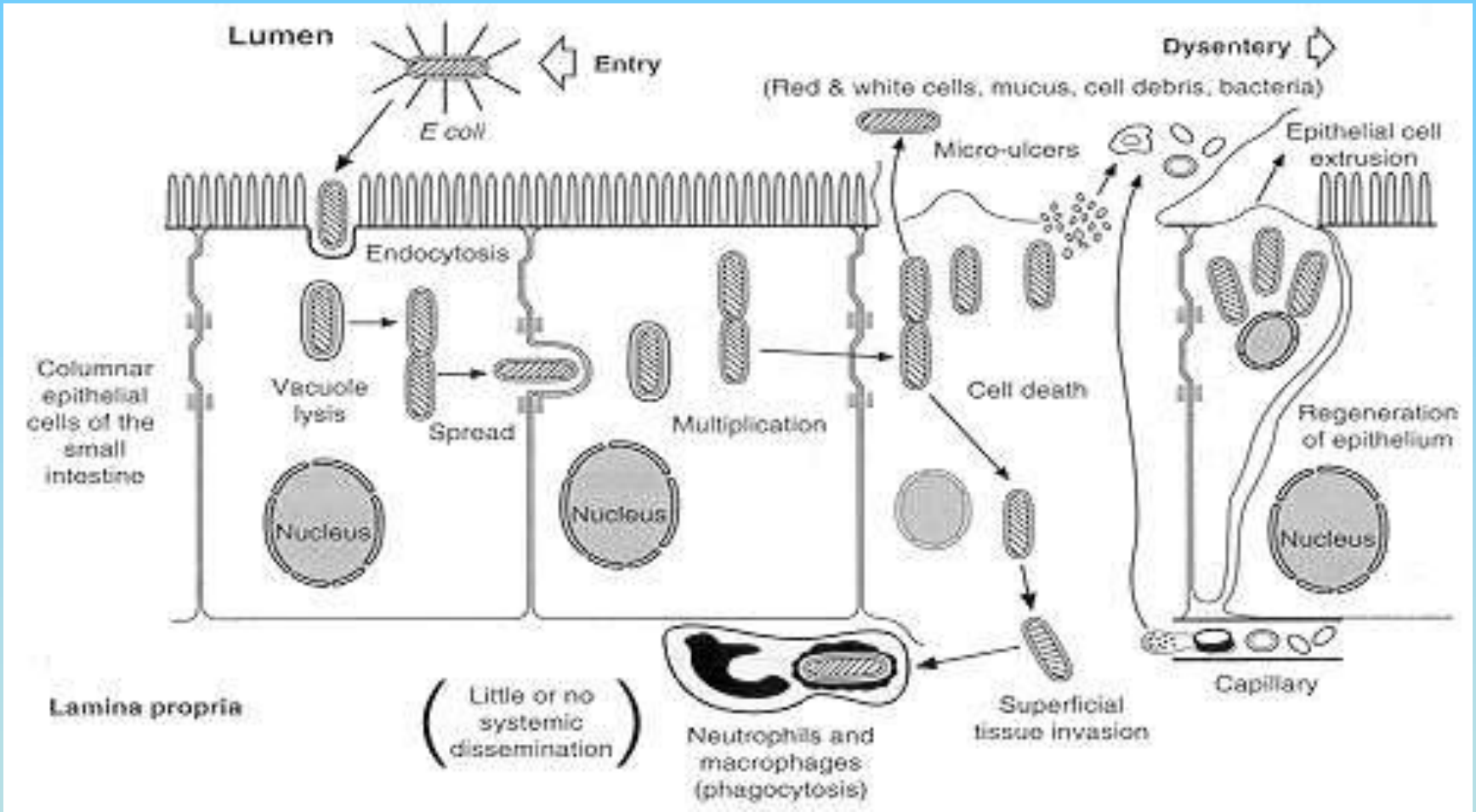


Figure 14-5 Microbiology, 6/e

Infeksi E.coli



Infeksi kulit

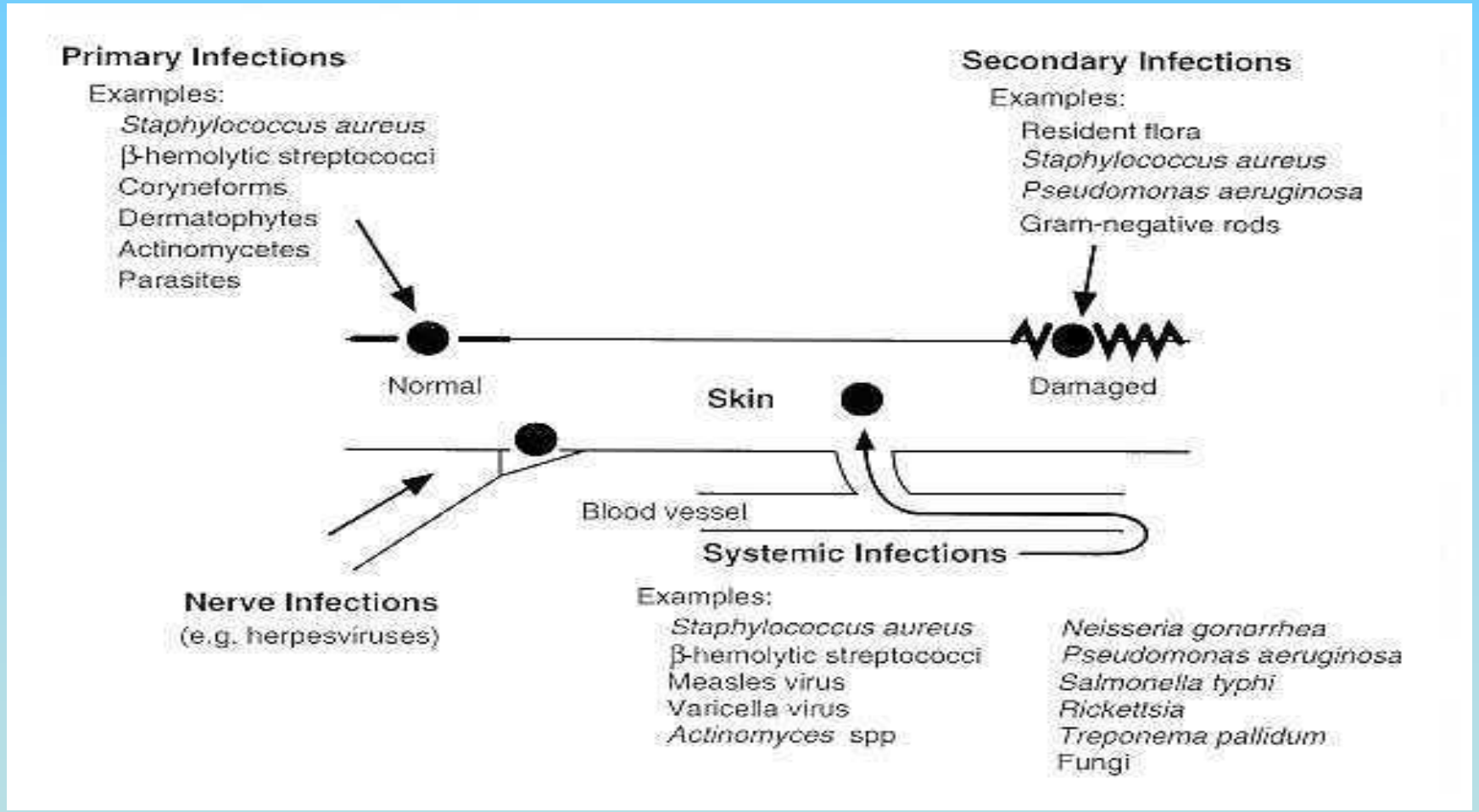


TABLE 7-1 Types of Bacterial Infections

Type of Infection	Description	Examples
Inapparent (subclinical)	No detectable clinical symptoms of infection	Asymptomic gonorrhea in women and men
Dormant (latent)	Carrier state	Typhoid carrier
Accidental	Zoonosis or environmental or inadvertent exposures	Anthrax, cryptococcal infection, and laboratory exposure, respectively
Opportunistic	Infection caused by normal flora or transient bacteria when normal host defenses are compromised	*Serratl* or *Candida* infection of the genitourinary tract
Primary	Clinically apparent (e.g., invasion and multiplication of microbes in body tissues, causing local tissue injury)	*Shigella* dysentery
Secondary	Microbial invasion subsequent to primary infection	Bacterial pneumonia following viral lung infection
Mixed	Two or more microbes infecting the same tissue	Anaerobic abscess (*E coli* and *Bacteroides fragilis*)
Acute	Rapid onset (hours or days); brief duration (days or weeks)	Diphtheria
Chronic	Prolonged duration (months or years)	Mycobacterial diseases (tuberculosis and leprosy)
Localized	Confined to a small area or to an organ	Staphylococcal boil
Generalized	Disseminated to many body regions (gonococcemia)	Gram-negative bacteremia
Pyogenic	Pus-forming	Staphyloccal and streptococcal infection
Retrograde	Microbes ascending in a duct or tube against the flow of secretions or excretions	*E coli* urinary tract infection
Fulminant	Infections that occur suddenly and intensely	Airborne *Yersinia pestis* (pneumonic plague)

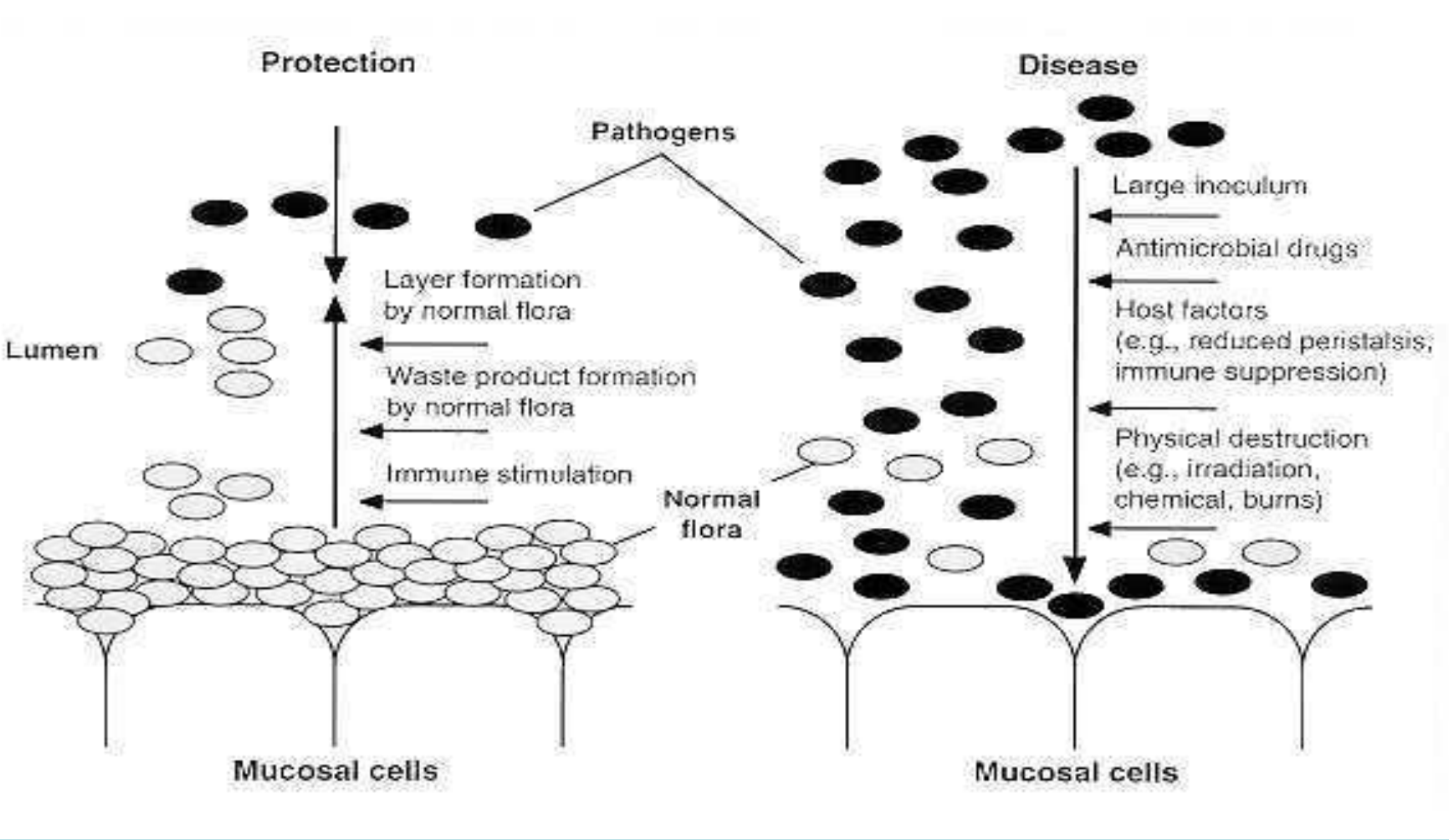
Mikroflora normal dalam tubuh

- Flora normal hidup pada organ tubuh, seperti: kulit, saluran pernafasan, saluran pencernaan, saluran urogenital dsb.
- Jumlah dan komposisi flora normal dipengaruhi oleh faktor: Usia, jenis kelamin dan nutrisi, Jumlah flora normal pada kulit sekitar 10^{12} , pada mulut 10^{10} dan 10^{14} pada saluran pencernaan
- Keberadaannya Flora normal menguntungkan bagi tubuh

Beberapa Keuntungan Flora normal dalam tubuh Inang

1. Flora normal mensintesis beberapa vitamin, seperti: Vitamin K dan B12
2. Flora normal mencegah timbulnya kolonisasi mikroba patogen
3. Flora normal bersifat antagonis dengan bakteri lain yang merugikan
4. Flora normal menstimulus perkembangan jaringan tertentu (sperti jaringan limpatik)
5. Flora normal menstimulus produksi antibodi

Peran flora normal dalam tubuh



Mikroflora normal dalam tubuh

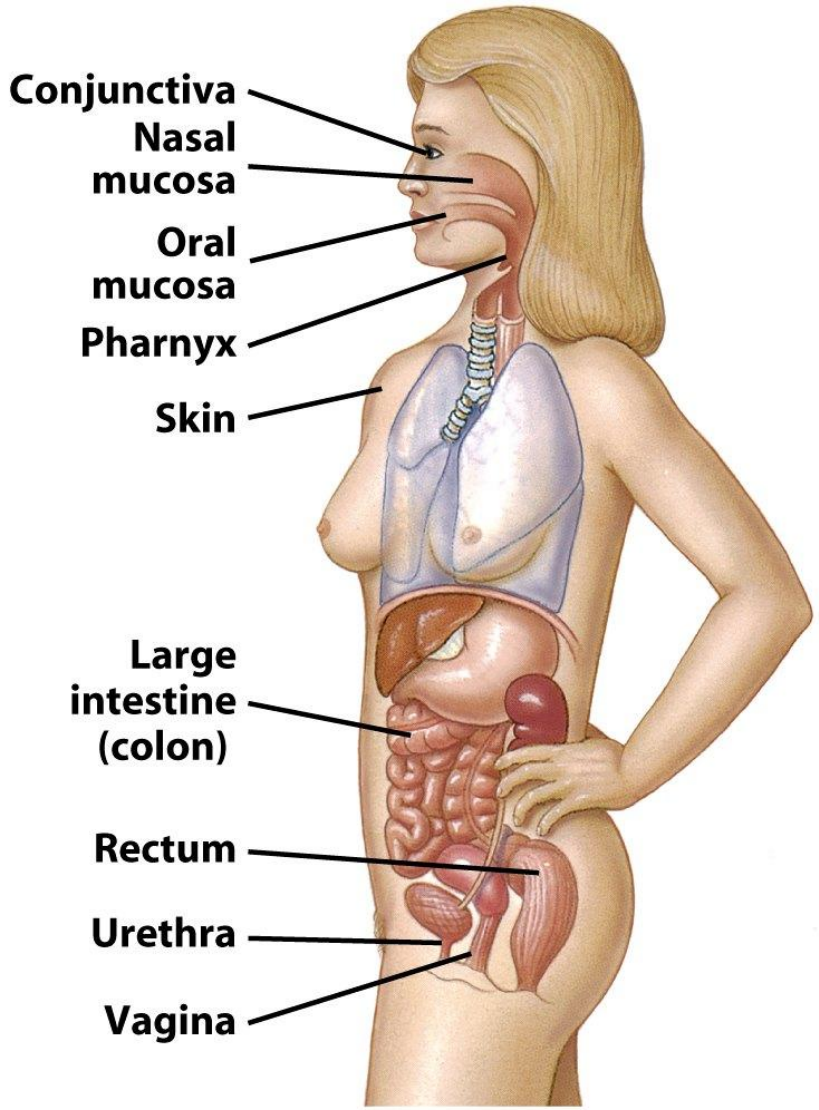
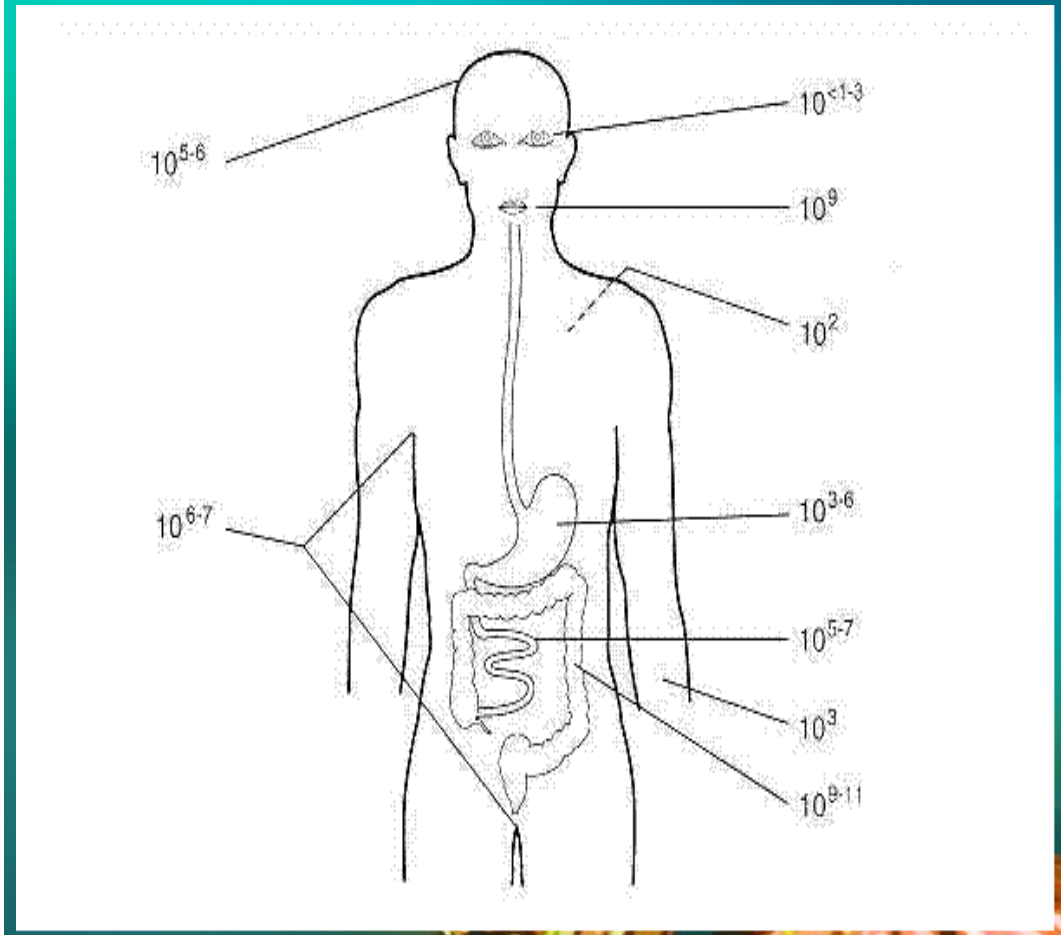


Figure 14-3 Microbiology, 6/e
© 2005 John Wiley & Sons



Flora normal dalam tubuh

TABLE 14.2

Major Normal Microflora (Unless Otherwise Noted, Bacteria) of the Human Body

Skin

*Staphylococcus epidermidis**
Staphylococcus aureus
Lactobacillus species
*Propionibacterium acnes**
Pityrosporon ovale (fungus)*

Mouth

*Streptococcus salivarius**
Streptococcus pneumoniae
*Streptococcus mitis**
Streptococcus sanguis
Streptococcus mutans
*Staphylococcus epidermidis**
Staphylococcus aureus
Moraxella catarrhalis
*Veillonella alcalescens**
Lactobacillus species*
Klebsiella species
*Haemophilus influenzae**
*Fusobacterium nucleatum**
*Treponema denticola**
Candida albicans (fungus)*
Entamoeba gingivalis (protozoan)*
Trichomonas tenax (protozoan)*

Upper Respiratory Tract

*Staphylococcus epidermidis**
Staphylococcus aureus
*Streptococcus mitis**
Streptococcus pneumoniae
Moraxella catarrhalis
Lactobacillus species
Haemophilus influenzae

Intestine

*Staphylococcus epidermidis**
Staphylococcus aureus
*Streptococcus mitis**
Enterococcus species*
Lactobacillus species*
Clostridium species*
*Eubacterium limosum**
*Bifidobacterium bifidum**
Actinomyces bifidus
*Escherichia coli**
Enterobacter species*
Klebsiella species
Proteus species
Pseudomonas aeruginosa
Bacteroides species*
Fusobacterium species
Treponema denticola
Endolimax nana (protozoan)
Giardia intestinalis (protozoan)

Urogenital Tract

*Streptococcus mitis**
Streptococcus species*
*Staphylococcus epidermidis**
Lactobacillus species*
Clostridium species
Actinomyces bifidus
Candida albicans (fungus)*
Trichomonas vaginalis (protozoan)

*Well-established associations.

Table 14-2 Microbiology, 6/e

BACTERIUM	Skin	Conjunctiva	Nose	Pharynx	Mouth	Lower Intestine	Anterior urethra	Vagina
Staphylococcus epidermidis (1)	++	+	++	++	++	+	++	++
*Staphylococcus aureus** (2)	+	+/-	+	+	+	++	+/-	+
Streptococcus mitis				+	++	+/-	+	+
Streptococcus salivarius				++	++			
*Streptococcus mutans** (3)				+	++			
*Enterococcus faecalis** (4)				+/-	+	++	+	+
*Streptococcus pneumoniae** (5)		+/-	+/-	+	+			+/-
*Streptococcus pyogenes** (6)	+/-	+/-		+	+	+/-		+/-
Neisseria sp. (7)		+	+	++	+		+	+
*Neisseria meningitidis** (8)			+	++	+			+
Veillonellae sp.					+	+/-		
Enterobacteriaceae (Escherichia coli)* (9)		+/-	+/-	+/-	+	++	+	+
Proteus sp.		+/-	+	+	+	+	+	+
*Pseudomonas aeruginosa** (10)				+/-	+/-	+	+/-	
*Haemophilus influenzae** (11)		+/-	+	+	+			
*Bacteroides sp.**						++	+	+/-
Bifidobacterium bifidum (12)						++		
Lactobacillus sp. (13)				+	++	++		++
*Clostridium sp.** (14)					+/-	++		
Clostridium tetani (15)						+/-		
Corynebacteria (16)	++	+	++	+	+	+	+	+
Mycobacteria	+		+/-	+/-		+	+	
Actinomycetes				+	+			
Spirochetes				+	++	++		
Mycoplasmas				+	+	+	+/-	+

++ = nearly 100 percent + = common +/- = rare * = potential pathogen

TABLE. BACTERIA FOUND IN THE LARGE INTESTINE OF HUMANS

BACTERIUM	Range of Incidence
Bacteroides fragilis	100
Bacteroides melaninogenicus	100
Bacteroides oralis	100
Lactobacillus	20-60
Clostridium perfringens	25-35
Clostridium septicum	5-25
Clostridium tetani	1-35
Bifidobacterium bifidum	30-70
Staphylococcus aureus	30-50
Enterococcus faecalis	100
Escherichia coli	100
Salmonella enteritidis	3-7
Salmonella typhi	0.00001
Klebsiella sp.	40-80
Enterobacter sp.	40-80
Proteus mirabilis	5-55
Pseudomonas aeruginosa	3-11
Peptostreptococcus sp.	common
Peptococcus sp.	moderate
Methanogens (Archaea)	common

TABLE. FREQUENTLY ENCOUNTERED BACTERIA IN PLAQUE, DENTAL CARIES, GINGIVITIS AND PERIODONTITIS

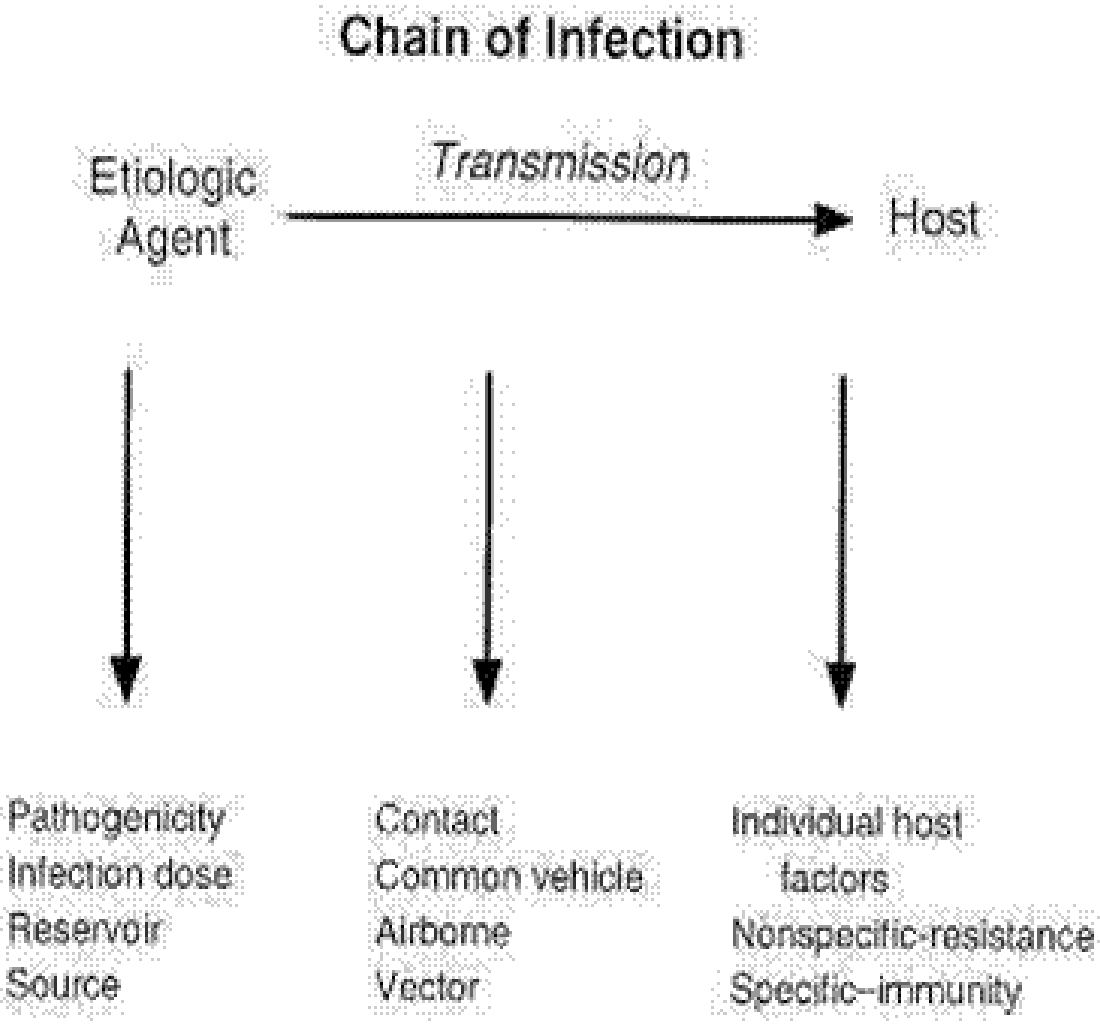
BACTERIUM	Plaque	Dental caries	Gingivitis	Periodontitis
Streptococcus sanguis	++	++	++	+
S. mutans	++	++	0	0
S. salivarius	0	0	0	0
Actinomyces viscosis	+	+	++	+
A. israelii	+	+	++	++
Lactobacillus sp.	+	+	0	0
Propionibacterium acnes	0	+	+	++
Bacteroides sp.	0	0	+	++
Selenomonas sputagena	0	0	+	++
Large spirochetes	0	0	0	++

++ = Frequently encountered in high proportions; + = Frequently encountered in low to moderate proportions; 0 = Sometimes encountered in low proportions or not detectable. Modified from Davis, et al.: Microbiology. 4th ed. J. B. Lippincott. Philadelphia, 1990.

TABLE 3. EXAMPLES OF SPECIFIC ATTACHMENTS OF BACTERIA TO HOST CELL OR TISSUE SURFACES

Bacterium	Bacterial ligand for attachment	Host cell or tissue receptor	Attachment site
Streptococcus pyogenes	Protein F	Amino terminus of fibronectin	Pharyngeal epithelium
Streptococcus mutans	Glycosyl transferase	Salivary glycoprotein	Pellicle of tooth
Streptococcus salivarius	Lipoteichoic acid	Unknown	Buccal epithelium of tongue
Streptococcus pneumoniae	Cell-bound protein	N-acetylhexosamine-galactose disaccharide	Mucosal epithelium
Staphylococcus aureus	Cell-bound protein	Amino terminus of fibronectin	Mucosal epithelium
Neisseria gonorrhoeae	N-methylphenyl-alanine pili	Glucosamine-galactose carbohydrate	Urethral/cervical epithelium
Enterotoxigenic *E. coli*	Type-1 fimbriae	Species-specific carbohydrate(s) (e.g. mannose)	Intestinal epithelium
Uropathogenic *E. coli*	Type 1 fimbriae	Complex carbohydrate	Urethral epithelium
Uropathogenic *E. coli*	P-pili (pap)	Globobiose linked to ceramide lipid	Upper urinary tract
Bordetella pertussis	Fimbriae ("filamentous hemagglutinin")	Galactose on sulfated glycolipids	Respiratory epithelium
Vibrio cholerae	N-methylphenylalanine pili	Fucose and mannose carbohydrate	Intestinal epithelium
Treponema pallidum	Peptide in outer membrane	Surface protein (fibronectin)	Mucosal epithelium
Mycoplasma	Membrane protein	Sialic acid	Respiratory epithelium
Chlamydia	Unknown	Sialic acid	Conjunctival or urethral epithelium

Mata rantai infeksi



Beberapa cara penularan bakteri patogen yang dapat menyebabkan terjadinya infeksi, antara lain:

1). Kontak langsung melalui hubungan seksual (sifilis, gonorhoe, trakoma)

2). Udara pernapasan -----airborne diseases (influenza, tuberkulosis, cacar, campak, gondongan)

3). Melalui mulut : waterborne diseaser (kolera, disentri), makanan beracun---foodborne disease (*Clostridium botulinum*).

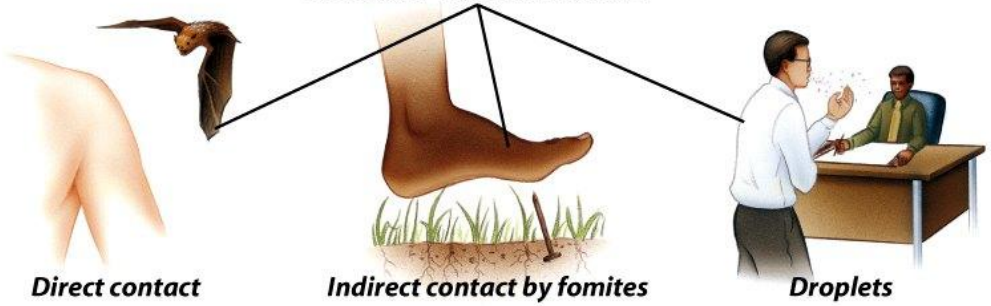
4). Melalui tusukan benda tajam : tetanus, rabies (gigitan anjing gila), hepatitis, AIDS (jarum untuk transfusi darah).

5). Serangga : serangga bekerja sebagai vektor mekanik (disentri dan demam tifoid oleh lalat rumah) atau vektor biologi (malaria dan demam berdarah oleh nyamuk).

6). Infeksi melalui laboratorium klinik : infeksi dapat terjadi melalui kelalaian pekerja medis di rumah sakit atau tempat praktek, sebagai contoh, dalam penyuntikan, fungsi lumbal, katerterisasi, dan lain-lain, yang dilakukan tidak menurut ketentuan kesehatan.

7) Infeksi nosokomial: penularan penyakit di rumah sakit

Contact transmission



Direct contact

Rabies, rat bite fever, syphilis, gonorrhea, herpes, staphylococcal infections, cutaneous anthrax, genital warts

Indirect contact by fomites

Tetanus, common cold, enterovirus infections, ringworm

Droplets

Common cold, influenza, measles,Q fever, pneumonia, whooping cough

Vehicle transmission

Waterborne

Cholera, shigellosis, leptospirosis, *Campylobacter* infections

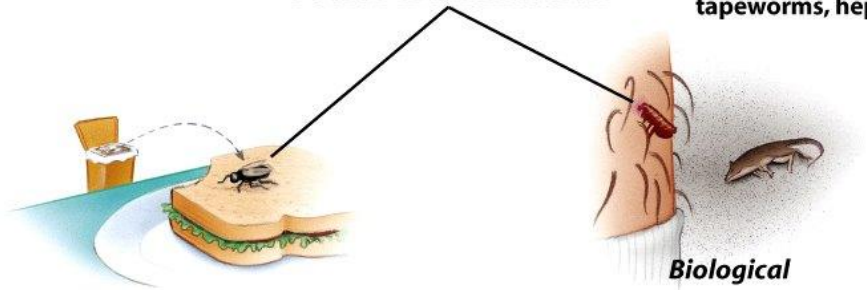
Airborne, including dust particles

Chickenpox, tuberculosis, coccidioidomycosis, histoplasmosis, influenza, measles

Foodborne

Intoxication with aflatoxins and botulinum toxin, paralytic shellfish poisoning, staphylococcal food poisoning, typhoid fever, salmonellosis, listeriosis, toxoplasmosis, tapeworms, hepatitis A

Vector transmission



Mechanical (on insect bodies)

E. coli diarrhea, salmonellosis, trachoma

Biological

Plague, malaria, yellow fever, typhus fever, Rocky Mountain spotted fever, Chagas' disease, Lyme disease

Figure 15-11 Microbiology, 6/e

TABLE 15.1

Selected Zoonoses (with Emphasis on Those That Occur in Pets)

Disease	Animals Infected	Modes of Transmission
Bacterial Diseases		
Avian tuberculosis	Birds	Respiratory aerosols
Anthrax	Domestic animals, including dogs and cats	Direct contact with animals, contaminated soil, and hides; ingestion of contaminated milk or meat; inhalation of spores
Brucellosis (undulant fever)	Domestic animals	Direct contact with infected tissues; ingestion of milk from infected animals
Bubonic plague	Rodents	Fleas
Lyme disease	Deer, field mice	Ticks
Leptospirosis	Primarily dogs; also pigs, cows, sheep, rodents, and other wild animals	Direct contact with urine, infected tissues, and contaminated water
Cat scratch fever	Cats	Scratches, bites, and licking
Psittacosis	Parrots, parakeets, and other birds	Respiratory aerosols
Relapsing fever	Rodents	Ticks and lice
Rocky Mountain spotted fever	Dogs, rodents, and other wild animals	Ticks
Salmonellosis	Dogs, cats, poultry, turtles, and rats	Ingestion of contaminated food or water
Viral Diseases		
Equine encephalitis (several varieties)	Horses, birds, and other domestic animals	Mosquitoes
Rabies	Dogs, cats, bats, skunks, and wolves	Bites, infectious saliva in wounds, and aerosols
Lassa fever, hantavirus pulmonary syndrome, hemorrhagic fevers	Rodents	Urine
Fungal Diseases		
Histoplasmosis	Birds	Aerosols of dried infected feces
Ringworm (several varieties)	Cats, dogs, and other domestic animals	Direct contact
Parasitic Diseases		
African sleeping sickness	Wild game animals	Tsetse flies
Tapeworms	Cattle, swine, rodents	Ingestion of cysts in meat or via proglottids in feces
Toxoplasmosis	Cats, birds, rodents, and domestic animals	Aerosols, contaminated food and water, and placental transfer

Table 15-1 Microbiology, 6/e

Faktor virulensi patogen

1. Faktor Virulensi yang Berperan dalam Kolonisasi: struktur permukaan bakteri (fimbria, flagela, antigen, kapsul, enzim, dan komponen membran luar).

a) Pergerakan Bakteri:*Proteus* sp. *Vibrio* spp., *Serratia* spp

b) Perlekatan Bakteri: fimbria,adhesin

2. Enzim:

Protease: *N. gonorrhoea, N. meningitidis, H. influenzae*, dan *Streptococcus pneumoniae,* memproduksi enzim proteolitik

Neuraminidase: menyerang komponen glikoprotein dan glikolipid membran sel

- Faktor Virulensi
- Kolonisasi
- Alam

movement.exe

- Proteus
- Bacillus
- Clostridium
- Vibrio Serratia

. \. . .

. . \. . : *r ot eus* (. . \. . .)

. . . : *or det el l a per t usi s* (. \. . . . .)

t r ept ococcus pneumor i i ae (. . \. . .)

. ol i (. \. . .)

. . . . \. . : *r ot eus mi r abi l i s*

. . . . (. . . . .) : *or det el l a per t ussi s*

ecoli.exe

Faktor Virulensi Yang Merusak Inang

1). **Asam Teikoat:*Staphylococcus aureus* adalah asam teikoat ribitol grup-spesifik dinding sel**

2) **Asam Lipoteikoat (LTA)** *Streptococcus pyogenes* ---perlekatan pada sel epitel

3). **Kapsul Polisakarida**

Pneumococcus penghasil kapsul ekstraseluler, yang merusak jaringan inang

4). Protein A

Protein A merupakan suatu antigen khusus kelompok-spesifik *Staphylococcus aureus*.

5) Enzim

a. **Asam amino deaminase**

b. Urease

c. lipase

sam ami no . eami nase

. emecah asam ami no → sam α ket o

↓

er f ungsi sebagai i dephor e

↓

engi kat esi dari . i ngkungan

rease

. mirabilis, . penneri (. . .)

rea → monia + . (p. meningkat)

engendapan komponen urin

a +, g +

atu struvit (g .)

atu apatite karbonat ([])

elicobacter pylori : infeksi lambung akut

. ur eus : ber kol oni sasi pada kel enj ar sebasea

nvasi kel enj ar j ar . ut anea dan
subkut an bi sul

nzi m ekst r asel ul er

i al ur oni dase : hi dr ol i si s asam hyal ur onat (semen
j ar i ngan esensi al pengi kat sel hi dup)

TABLE 14.4

Examples of Adhesive Virulence Factors

Bacterium	Disease	Adhesion Mechanism
Upper Respiratory Tract		
Mycoplasma pneumoniae	Atypical pneumonia	Adhesin on cell surface adheres to receptor in respiratory lining
Neisseria meningitidis	Meningitis	Adhesin on pili
Streptococcus pneumoniae	Pneumonia	Surface adhesins attach to carbohydrate on respiratory lining
Mouth		
Streptococcus mutans	Dental caries	Capsule attaches to tooth enamel
Intestinal Tract		
Shigella species	Dysentery	Unknown mechanism for attachment to intestinal lining
Escherichia coli	Diarrhea	Adhesins on pili attach to receptor on intestinal lining to intestinal lining
Escherichia coli	Diarrhea	Adhesins on pili attach to receptor on intestinal lining
Campylobacter jejuni	Diarrhea	Adhesins on flagella attach to intestinal lining
Vibrio cholerae	Cholera	Adhesins on flagella bind to receptors on intestinal lining
Urogenital Tract		
Treponema pallidum	Syphilis	Bacterial protein attaches to cells
Neissera gonorrhoeae	Gonorrhea	Adhesins on pili attach to lining of genital tract

Table 14-4 Microbiology, 6/e

Toksin : bakteri patogen

1). Eksotoksin. Eksotoksin dikeluarkan dari sel mikroorganisme ke suatu medium biakan atau ke dalam jaringan inang. *Clostridium botulinum*, mengandung toksin botulinum menghasilkan makanan beracun yang disebut botulism

2). **Enterotoksin.** Enterotoksin merupakan eksotoksin yang beraksi dalam usus halus, umumnya menyebabkan pengeluaran cairan secara besar-besaran ke dalam lumen usus, menimbulkan symptom diare. Enterotoksin dihasilkan oleh bermacam bakteri termasuk organisme peracun-makanan *Staphylococcus aureus,*

3). **Endotoxin**. Beberapa mikroorganisme, khususnya bakteri Gram-negatif, tidak mengeluarkan suatu toksin terlarut, tetapi membuat suatu endotoksin yang dibebaskan ketika sel mengalami pembelahan, lisis dan mati. Endotoksin dari bakteri Gram-negatif merupakan komponen struktural membran luar dari dinding sel bakteri Gram-negatif. Komponen ini merupakan polisakarida (lipid A)

4). **Hemolisin**

Hemolisin merupakan enzim ekstraseluler yang bersifat toksik. Toksin ini merupakan bahan yang menghancurkan sel darah merah dan melepaskan hemoglobin.

hemolysis.htm

Beberapa Penyakit yang disebabkan oleh Bakteri yang menghasilkan Eksotoksin

Penyakit	Spesies Bakteri	Aktivitas Toksin in vivo
Botulism	*Clostridium botulinum*	Neurotoksin; paralisis otot
Kolera	*Vibrio cholerae*	Hilangnya cairan dari usus Kecil, muntah.
Keracunan makanan	*Staphylococcus aureus*	Nausea, muntah, diare
Difteria	*Corynebacterium diphtheriae*	Paralysis saraf, rusak jantung
Disenteri basiler	*Shigella dysenteriae*	Gangguan nerurologi, diare
Tetanus	*Clostridium tetani*	Neurotoksin, kontraksi otot Spamodik
Demam Scarlet	*Streptococcus pyogenes*	Ruam
Batuk Rejan	*Bordetella pertussis*	Batuk paroksimal; muntah.

Tabel Beberapa Perbedaan Sifat Eksotoksin dan Endotoksin

Ciri-ciri	Eksotoksin	Endotoksin
Sumber	Dikeluarkan oleh bakteri Gram-positif dan Gram negatif tertentu	Dilepaskan dari dinding sel bakteri Gram-negatif yang hancur.
Bahan Kimia	Protein	Lipopolisakarida
Toleransi panas	Labil	Stabil
Imunologi	Mampu berubah menjadi toxoid	Tidak membentuk toxoid
Efek biologi	Khusus untuk tipe fungsi sel tertentu	Umumnya menyebabkan demam dan shock
Dosis letal	Dalam menit (toxisitas tinggi)	Lebih lama (toxisitas Rendah)

TABLE 14.5

Properties of Toxins

Property	Exotoxins	Endotoxins
Organisms producing	Almost all Gram-positive; some Gram-negative	Almost all Gram-negative
Location in cell	Extracellular, excreted into medium	Bound within bacterial cell wall; released upon death of bacterium
Chemical nature	Mostly polypeptides	Lipopolysaccharide complex
Stability	Unstable; denatured above 60°C and by ultraviolet light	Relatively stable; can withstand several hours above 60°C
Toxicity	Among the most powerful toxins known (some are 100 to 1 million times as strong as strychnine)	Weak, but can be fatal in relatively large doses
Effect on tissues	Highly specific; some act as neurotoxins or cardiac muscle toxins	Nonspecific; ache-all-over systemic effects or local site reactions
Fever production	Little or no fever	Rapid rise in temperature to high fever
Antigenicity	Strong; stimulates antibody production and immunity	Weak; recovery from disease often does not produce immunity
Toxoid conversion and use	By treatment with heat or chemicals; toxoid used to immunize against toxin	Cannot be converted to toxoid; cannot be used to immunize
Examples	Botulism, gas gangrene, tetanus, diphtheria, staphylococcal food poisoning, cholera, enterotoxins, plague	Salmonellosis, tularemia, endotoxic shock

Table 14-5 Microbiology, 6/e

TABLE 14.6

Effects of Exotoxins

Bacterium	Name of Toxin or Disease	Action of Toxin	Host Symptoms
Bacillus anthracis	Anthrax (cytotoxin)	Increases vascular permeability	Hemorrhage and pulmonary edema
Bacillus cereus	Enterotoxin	Causes excessive loss of water and electrolytes	Diarrhea
Clostridium botulinum	Botulism (eight serological types; neurotoxins)	Blocks release of acetylcholine at nerve endings	Respiratory paralysis, double vision
Clostridium perfringens	Gas gangrene (α-toxin, a hemolysin)	Breaks down lecithin in cell membranes	Cell and tissue destruction
	Food poisoning (enterotoxin)	Causes excessive loss of water and electrolytes	Diarrhea
Clostridium tetani	Tetanus (lockjaw) (neurotoxin)	Inhibits antagonists of motor neurons of brain; 1 nanogram can kill 2 tons of cells	Violent skeletal muscle spasms, respiratory failure
Corynebacterium diphtheriae	Diphtheria; produced by virus-infected (cytotoxin) bacteria	Inhibits protein synthesis	Heart damage can cause death weeks after apparent recovery
Escherichia coli	Traveler's diarrhea (enterotoxin)	Causes excessive loss of water and electrolytes	Diarrhea
Escherichia coli	O157:H7 (enterotoxin)	Hemolytic uremic syndrome	Destroys intestinal lining and causes hemorrhages in kidney
			Bleeding and kidney hemorrhage and failure
Pseudomonas aeruginosa	Various infections (exotoxin A)	Inhibits protein synthesis	Lethal, necrotizing lesions
Shigella dysenteriae	Bacillary dysentery (enterotoxin)	Cytotoxic effects; as potent as botulinum toxin	Diarrhea, causes paralysis in rabbits from spinal cord hemorrhage and edema
Staphylococcus aureus	Food poisoning (enterotoxin)	Stimulates brain center that causes vomiting	Vomiting
	Scalded skin syndrome (exfoliatin)	Causes intradermal separation of cells	Redness and sloughing of skin
Streptococcus pyogenes	Scarlet fever (erythrogenic, or red-producing toxin)	Causes vasodilation	Maculopapular (slightly raised, discolored) lesions
Vibrio cholerae	Cholera (enterotoxin)	Causes excessive loss of water (up to 30 liters/day) and electrolytes	Diarrhea; can kill within hours

Table 14-6 Microbiology, 6/e

Tabel Beberapa Enzim Ekstraseluler yang Mendukung Virulensi Bakteri

Enzim	Aksi	Contoh Bakteri Penghasil-enzim
Hyaluronidase	Hidrolisis asam hialuronat pada jaringan; faktor invasif	*Staphylococcus, Streptococcus, Clostridium*.
Coagulase	Membekukan plasma, Digunakan untuk identifikasi	*Staphylococcus aureus*
Lechitinase	Hidrolisis lipid membran	*Clostridium perfringens*
Collagenase	Hidrolisis kolagen pada Jaringan, faktor invasif	*Clostridium perfringens*
Leucocidin	Membunuh Leukosit	*Staphylococcus aureus*
Hemolysin	Lisis sel darah merah, Faktor invasif	*Staphylococcus, streprtococcus, Clostridium*.

Faktor yang mempengaruhi infeksi

- Usia
- Nutrisi: gizi
- Stress
- Jenis kelamin
- Trauma
- Kemoterapi penyakit

TABLE 14.8

Terms Used to Describe Infections

Term	Characteristic of Infection
Acute disease	Disease in which symptoms develop rapidly and that runs its course quickly
Chronic disease	Disease in which symptoms develop slowly and disease is slow to disappear
Subacute disease	Disease with symptoms intermediate between acute and chronic
Latent disease	Disease in which symptoms appear and/or reappear long after infection
Local infection	Infection confined to a small region of the body, such as a boil or bladder infection
Focal infection	Infection in a confined region from which pathogens travel to other regions of the body, such as an abscessed tooth or infected sinuses
Systemic infection	Infection in which the pathogen is spread throughout the body, often by traveling through blood or lymph
Septicemia	Presence and multiplication of pathogens in blood
Bacteremia	Presence but not multiplication of bacteria in blood
Viremia	Presence but not multiplication of viruses in blood
Toxemia	Presence of toxins in blood
Sapremia	Presence of metabolic products of saprophytes in blood
Primary infection	Infection in a previously healthy person
Secondary infection	Infection that immediately follows a primary infection
Superinfection	Secondary infection that is usually caused by an agent resistant to the treatment for the primary infection
Mixed infection	Infection caused by two or more pathogens
Inapparent infection	Infection that fails to produce full set of signs and symptoms

Table 14-8 Microbiology, 6/e

Pertahanan tubuh terhadap infeksi : fisik/kimia/biologi

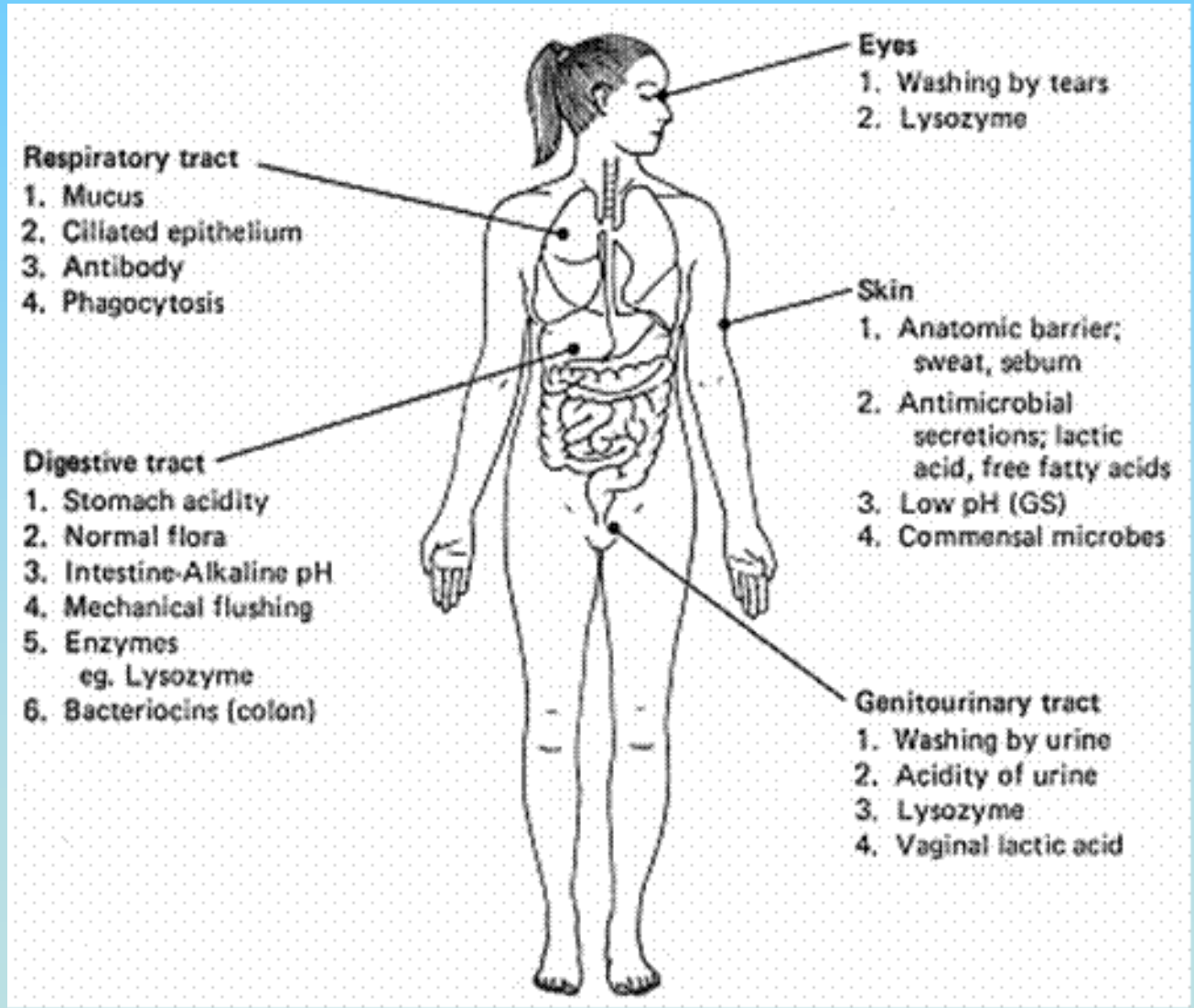


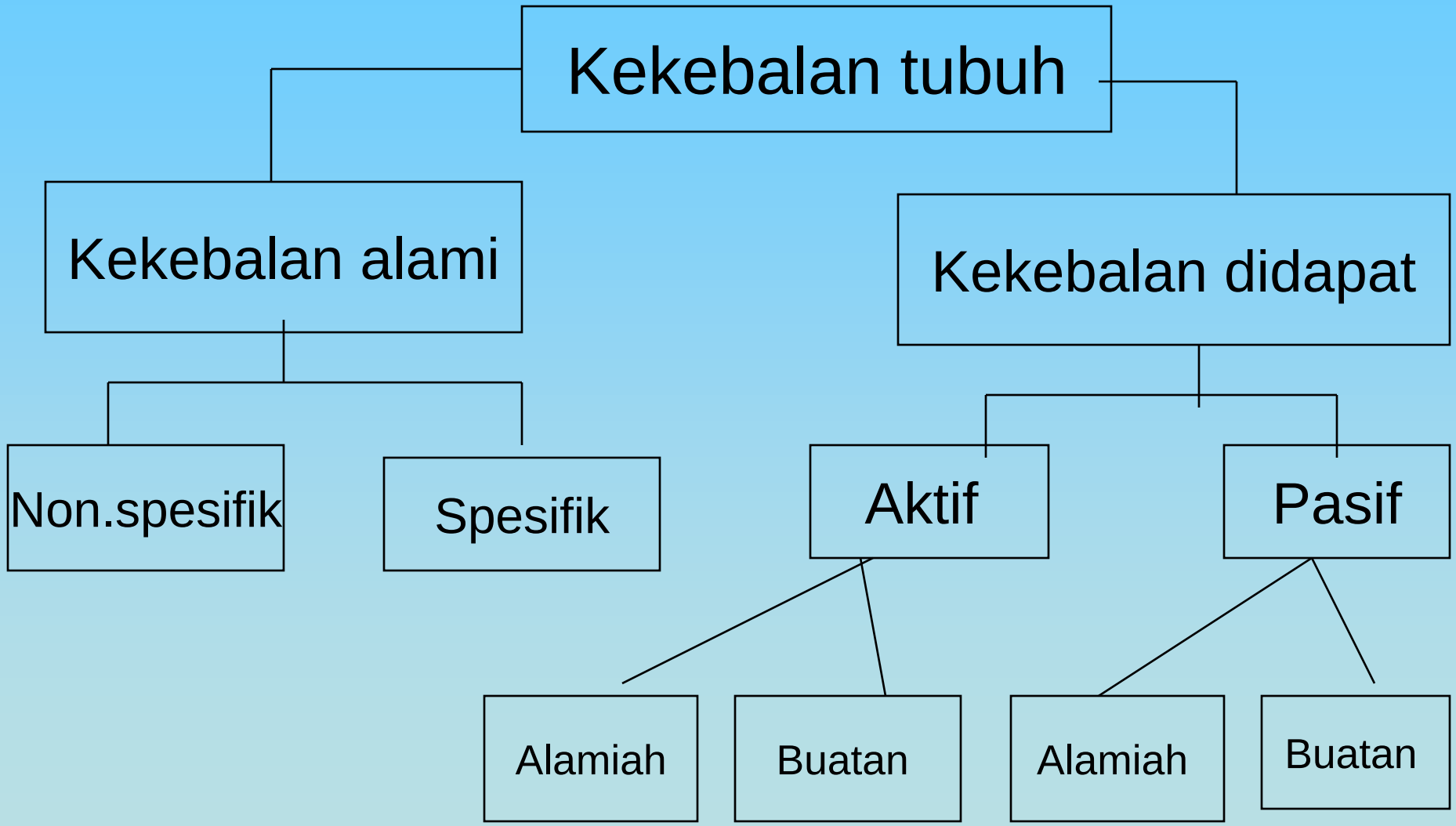
TABLE. ANTIMICROBIAL SUBSTANCES OF HOST ORIGIN PRESENT IN BODY FLUIDS AND ORGANIZED TISSUES

Substance	Common Sources	Chemical Composition	Activity
Lysozyme	Serum, saliva, sweat, tears	Protein	Bacterial cell lysis
Complement	Serum	Protein-carbohydrate lipoprotein complex	Cell death or lysis of bacteria; participates in inflammation
Basic proteins and polypeptides (histones, ß-lysins and other cationic proteins, tissue polypeptides)	Serum or organized tissues	Proteins or basic peptides	Disruption of bacterial plasma membrane
Lactoferrin and transferrin	Body secretions, serum, organized tissue spaces	Glycoprotein	Inhibit microbial growth by binding (withholding) iron
Peroxidase	Saliva, tissues, cells (neutrophils)	Protein	Act with peroxide to cause lethal oxidations of cells
Fibronectin	Serum and mucosal surfaces	Glycoprotein	Clearance of bacteria (opsonization)
Interferons	Virus-infected cells, lymphocytes	Protein	Resistance to virus infections
Interleukins	Macrophages, lymphocytes	Protein	Cause fever; promote activation of immune system

MEKANISME PERTAHANAN EKSTERNAL

1. **Kulit dan Membran Mukosa**
2. **Sekresi Senyawa Kimia**

Kekebalan tubuh



Macam Kekebalan alamiah

- **kekebalan spesies**: Individu dari spesies yang sama menunjukkan pola kepekaan yang sama terhadp suatu infeksi kuman. Misalnya penyakit campak, sifilis, lepra hanya timbul pada manusia.
- **Kekebalan ras**: di dalam sautu spesies perbedaan ras dapat menyebabkan perbedaan kepekaan terhadap infeksi, misalnya orang-orang Negro yang berkulit hitam kebal terhadap penyakit malaria dan kuning.
- **Kekebalan individu**: individu dalam sautu populasi dapat menunjukkan variasi responnya terhadap infeksi mikroorganisme patogen.

Faktor-faktor yang mempengaruhi kekebalan alamiah

- **Umur** : Kelompok janin dan orang lanjut usia menunjukkan kepekaaan yang tinggi terhadap penyakit infeksi
- **Pengaruh hormon:** penderita DM,rentan dari penyakit infeksi
- **Gizi** : gizi yang buruk akan menekan semua jenis respon kekebalan, sehingga meningkatkan resiko infeksi.
- **Stress:**,peningatan hormon adrenalin/efinefrin menekan fungsi sel pertahanan tubuh
- **Pemukiman** : pemukiman yang padat, resiko penyakit lebih tinggi

FAKTOR LINGKUNGAN

EPINEFRIN / ADRENALIN

MENEKAN FUNGSI BEBERAPAKELOMPOK SEL PERTAHANANN DAN MENGURANGI LUAS DAERAH MAKANISME PERTAHANAN

RENTAN TERHADAP INFEKSI

UMUR INANG

ANAK-ANAK	**SISTEM IMUN BELUM BERKEMBANG**
ORANG TUA	**EFISIENSI SISTEM IMUN MENURUN**

CONTOH : KERENTANAN TERHADAP PNEUMOCOCCUS PNEUMONIAE

TEKANAN FISIK DAN EMOSI

EPINEFRIN / ADRENALIN

MENEKAN FUNGSI BEBRAPA KELOMPOK SEL PERTAHANAN DAN MENGURANGI LUAS DAERAH MEKANISME PERTAHANAN

RENTAN TERHADAP PENYAKIT

er t ahanan nang on - pesi f i k

akt or yang empengar uhi \esi st ensi nang

- akt or i ngkungan
- \esi st ensi pesi es

Respon Imun

1. Respon Imun humoral (imunitas humoral): respon primer primer terhadap mikroba—berhubungan dengan pembentukan antibodi
2. Respon imun seluler: respon imun khas yang tidak berkaitan dengan antibodi, biasanya dengan Limfokin dari limfosit yang mempengaruhi fagositosis macrofag

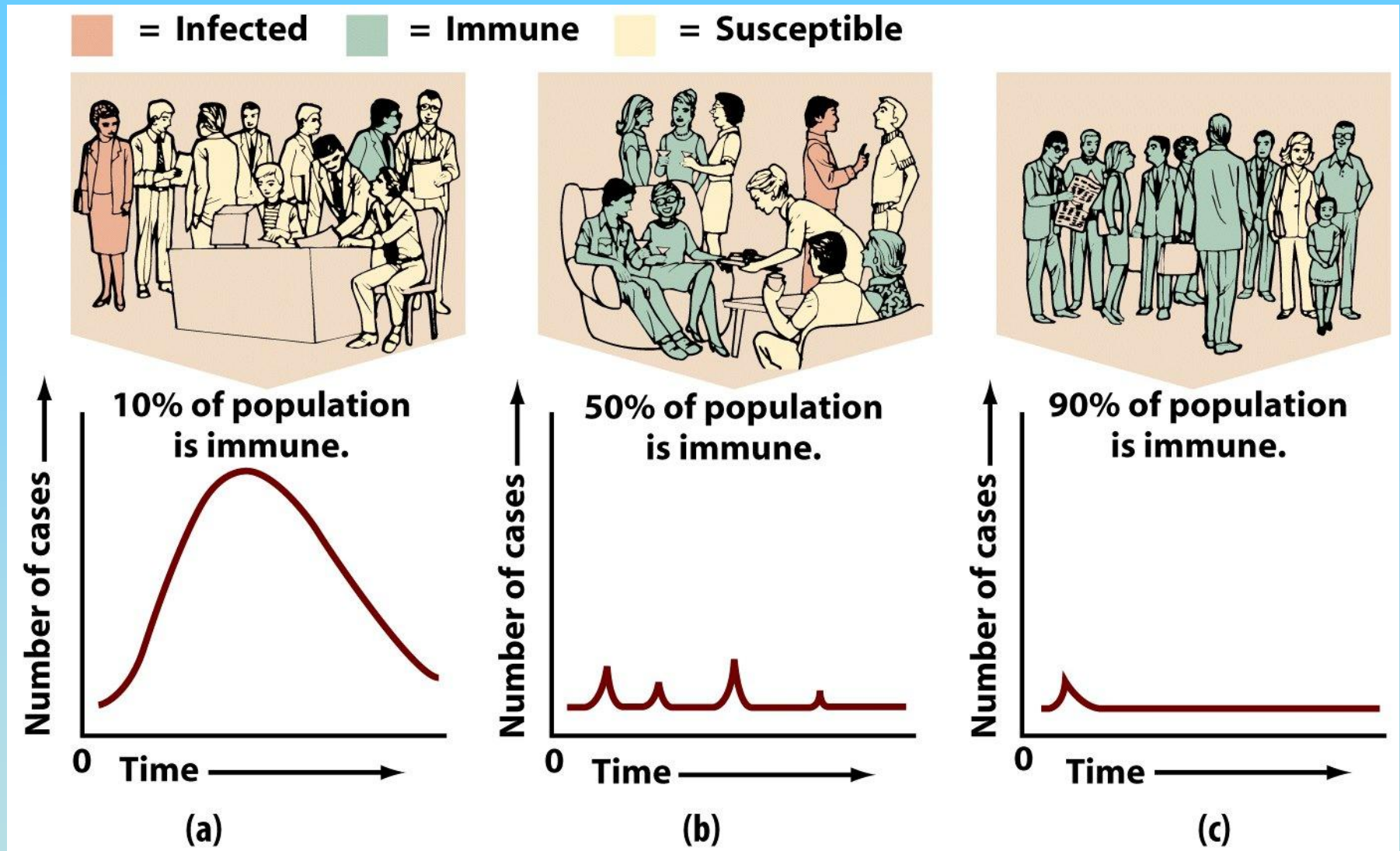


Figure 15-13 Microbiology, 6/e

Perkembangan antibodi

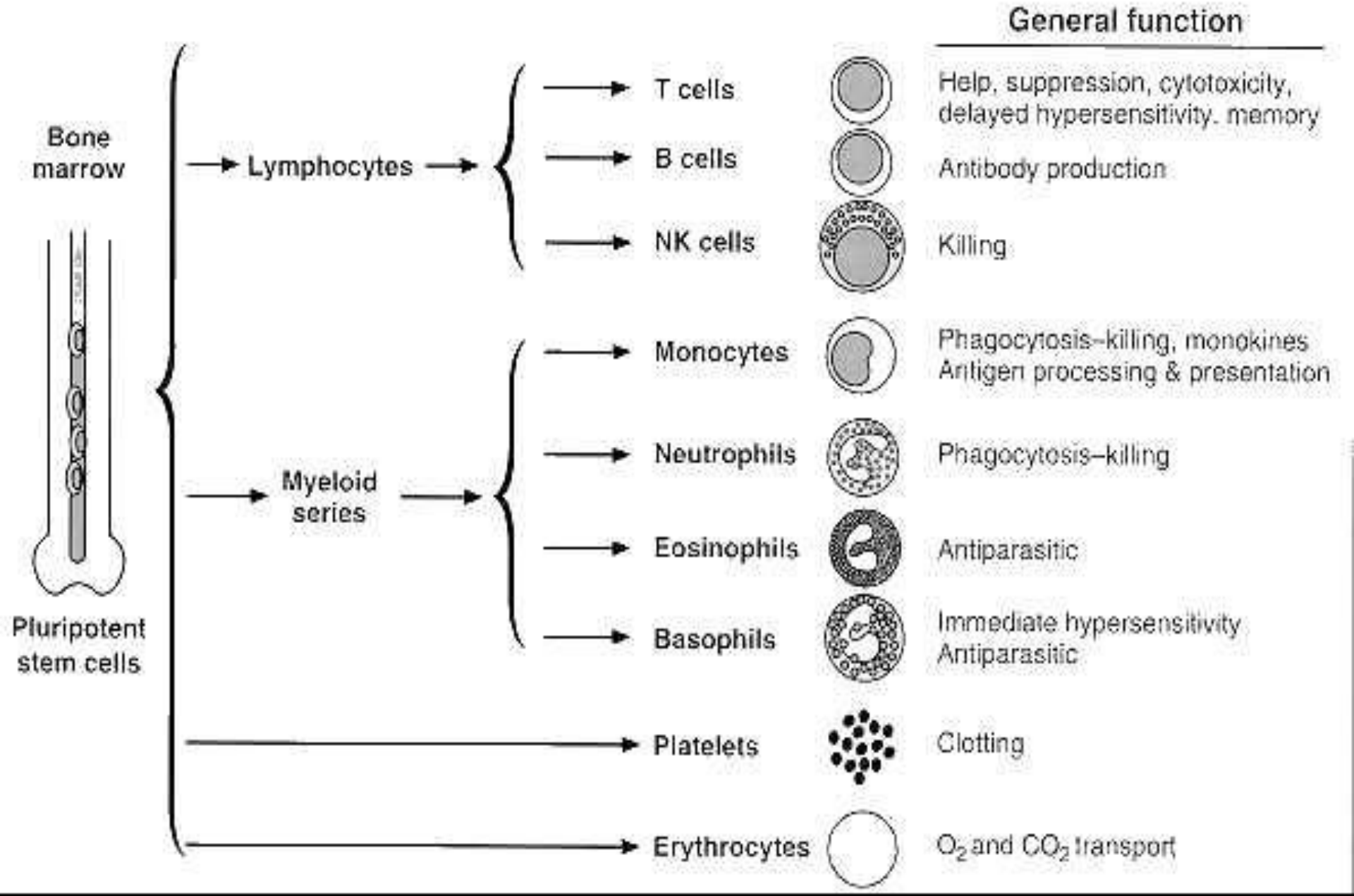


TABLE 17.4

Properties of Antibodies

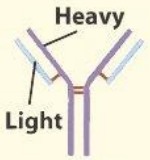
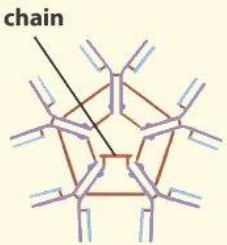
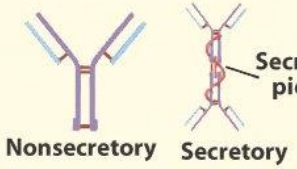
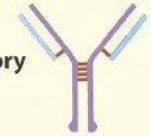
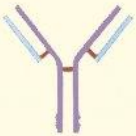
Property	Class of Immunoglobulin				
	IgG	IgM	IgA	IgE	IgD
					
Number of units	1	5	1 or 2	1	1
Activation of complement	Yes	Yes, strongly	Yes, by alternative pathway	No	No
Crosses placenta	Yes	No	No	No	No
Binds to phagocytes	Yes	No	No	No	No
Binds to lymphocytes	Yes	Yes	Yes	Yes	No
Binds to mast cells and basophils	No	No	No	Yes	No
Half-life (days) in serum	21	5–10	6	2	3
Percentage of total blood antibodies in serum	75–85	5–10	10	0.005	0.2
Location	Serum, extra vascular, and across placenta	Serum and B cell membrane	Transport across epithelium	Serum and extra-vascular	B cell membrane

Table 17-4 Microbiology, 6/e

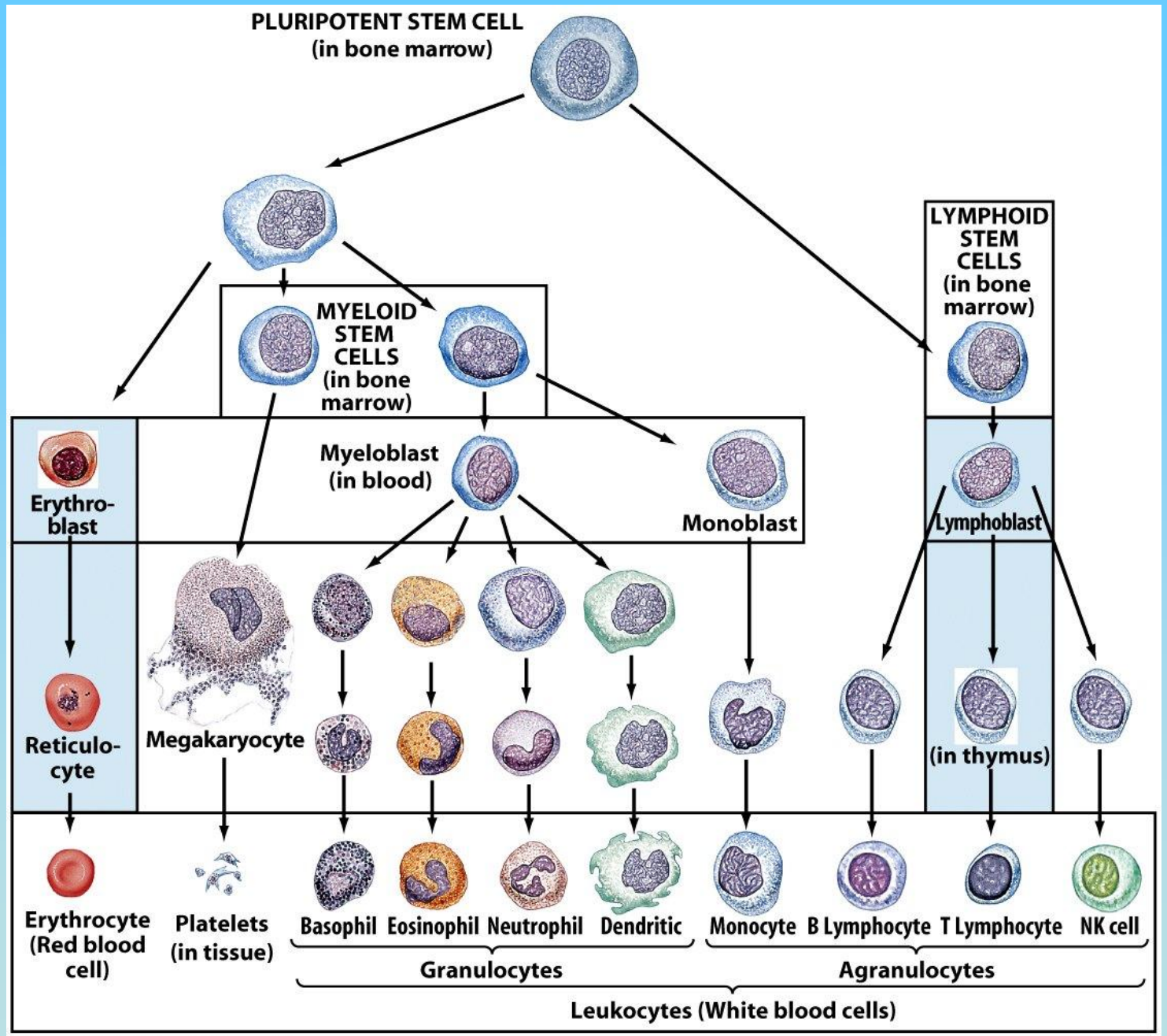


Figure 16-1 Microbiology, 6/e

Fagositosis

- Fagositosis adalah sebagai suatu mekanisme pertahanan umum untuk melindungi tubuh dari infeksi, yang pertama kali diperkenalkan oleh seorang ahli zoologi Rusia Elie Metchnikoff (1845-1916).
- Fagosit (Yunani : phagein = makan dan kytos = sel). sel pemakan partikel,
- Fagositosis digambarkan sebagai suatu cara protozoa tertentu untuk menagkap makanan.
- Dalam tubuh dilakukan oleh leukosit, terutama sel monosit yang akan berkembang menjadi macrofag

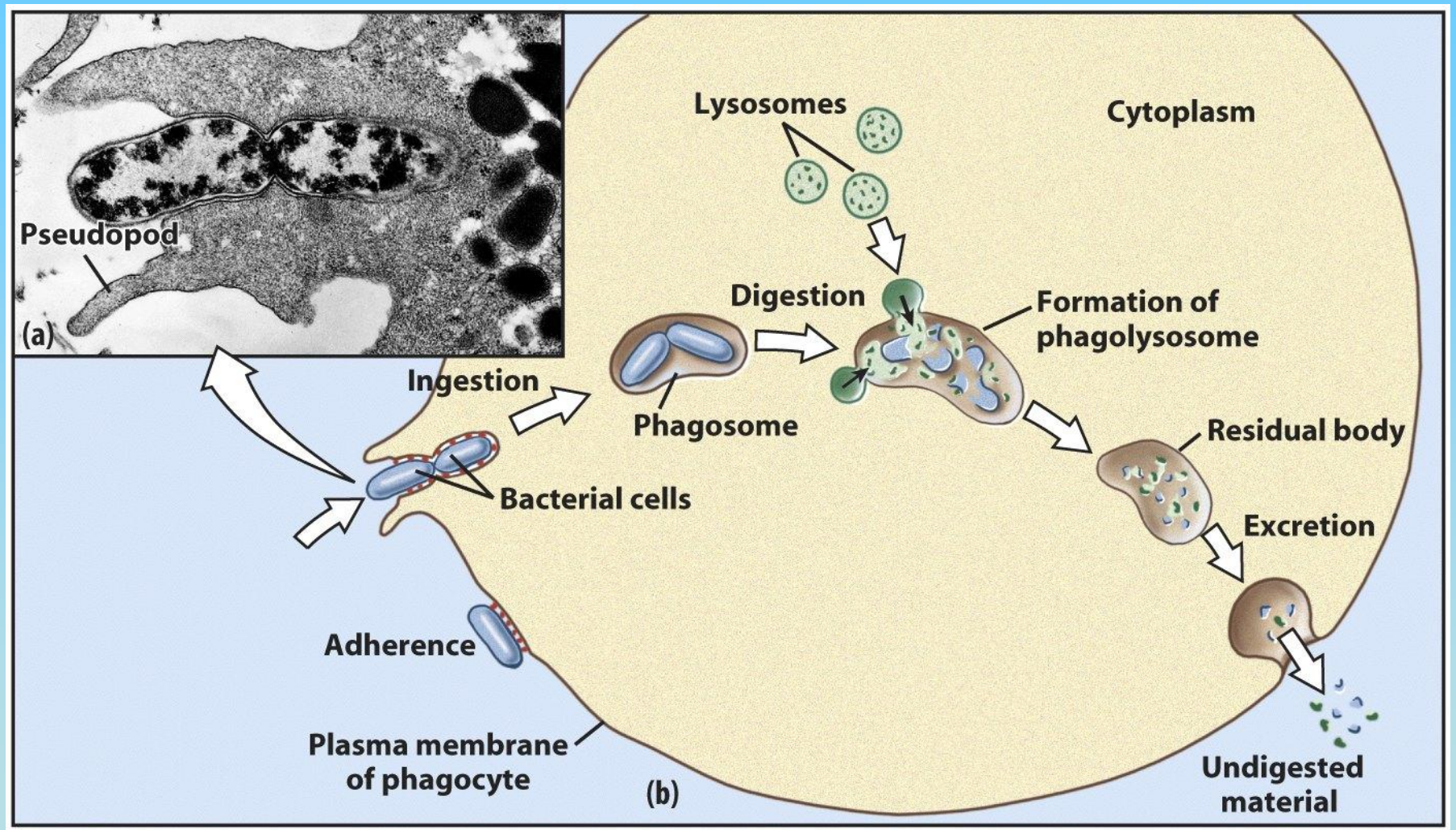


Figure 16-3 Microbiology, 6/e

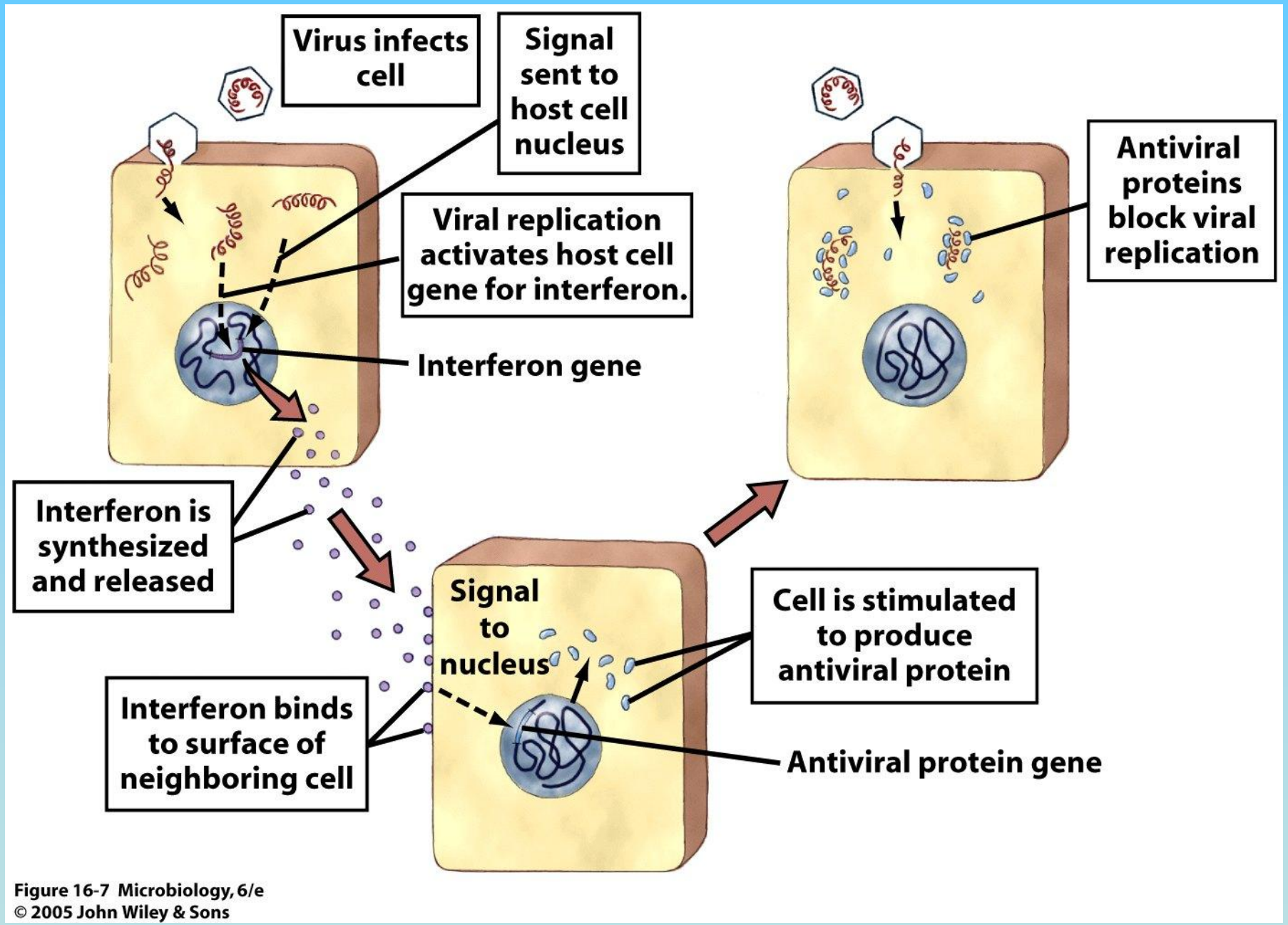


Figure 16-7 Microbiology, 6/e

Mekanisme Pertahanan Internal (Resistensi Inang nonspesifik)

1. **Inflamasi (Peradangan)**

   Respon inflamasi atau peradangan merupakan reaksi vaskuler dan seluler terhadap adanya serbuan mikroorganisme, kerusakan, atau bahan iritan

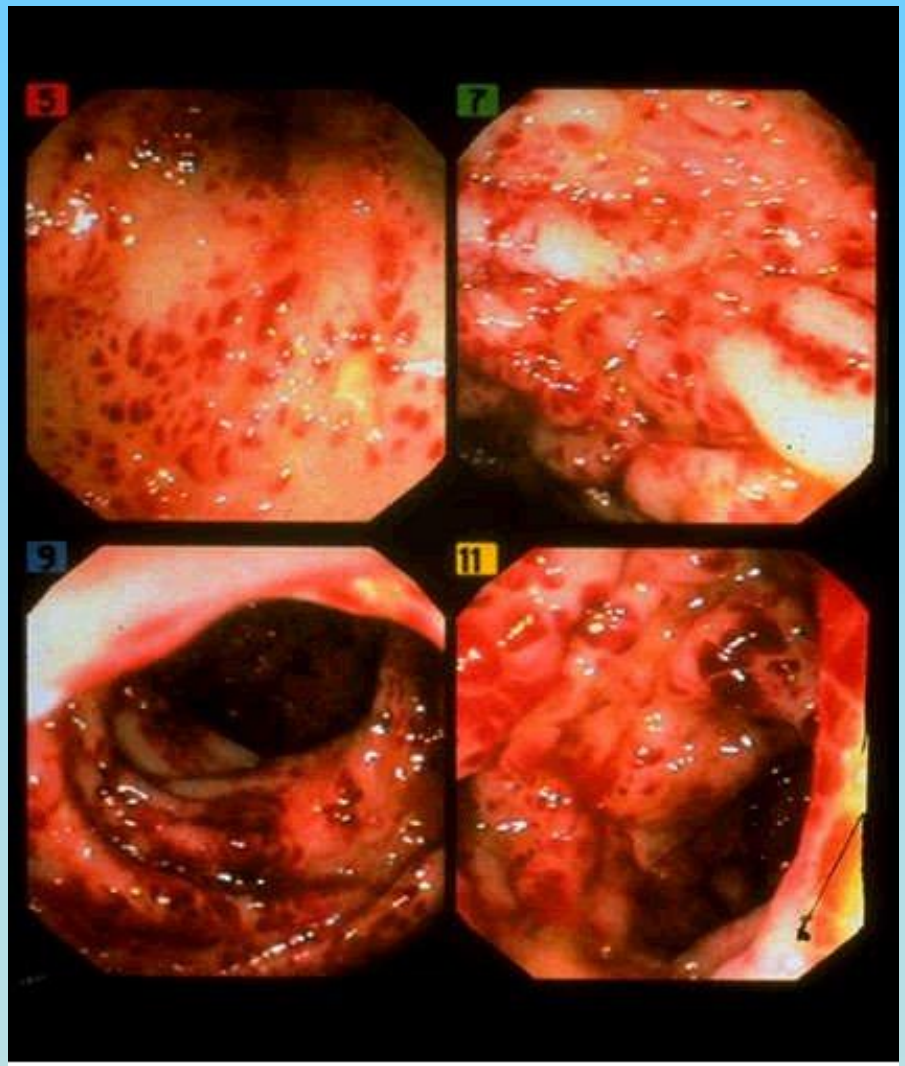
2. **Demam**

   Satu dari respon sistemik (respon tubuh secara menyeluruh) yang terpenting terhadap masuknya mikroorganisme adalah demam, suatu suhu tubuh tinggi yang tidak normal

3. **Sel "Natural Killer"**

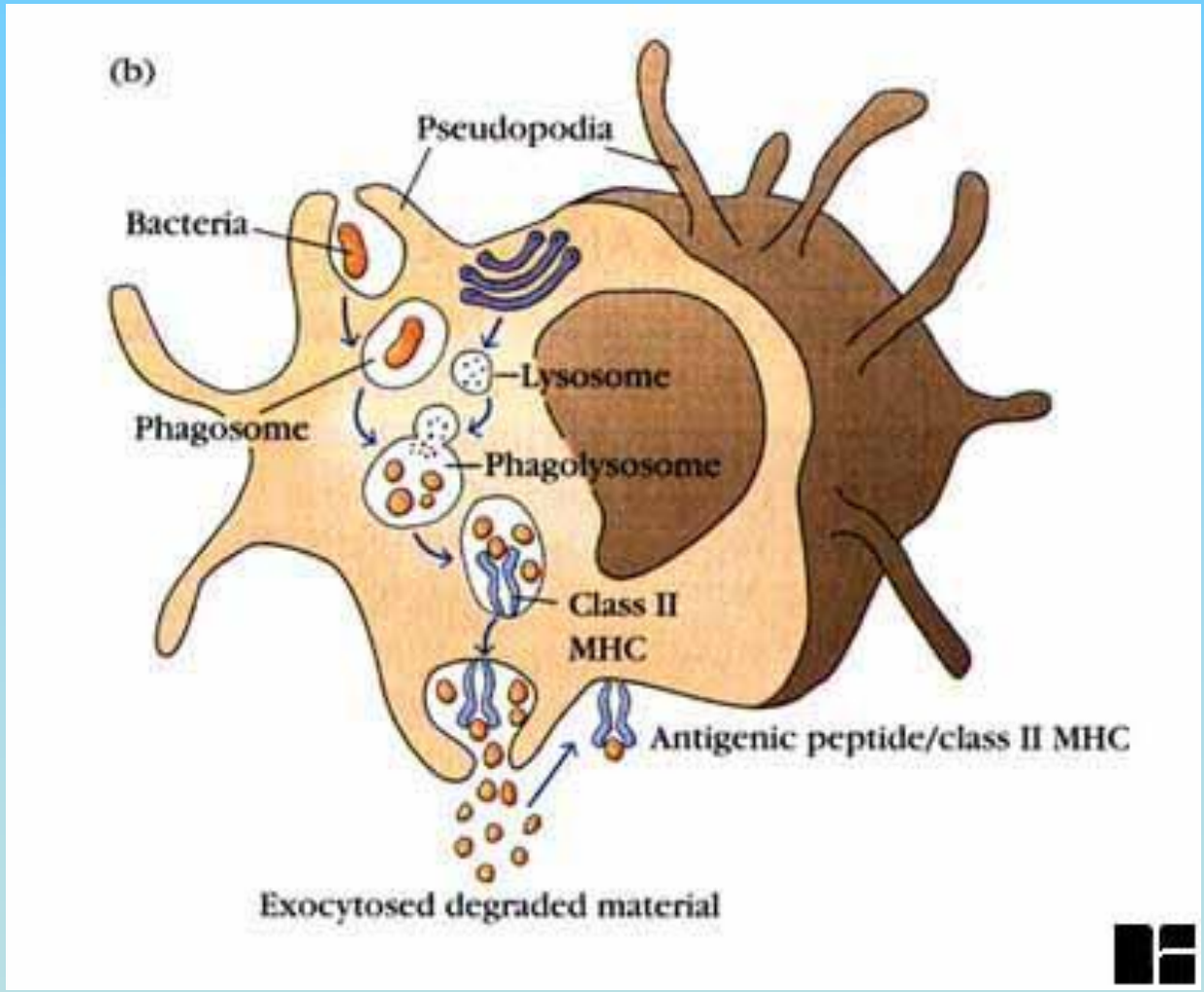
   Sel "natural killer" ("Natural killer cell") merupakan limfosit berukuran besar dengan diameter 12 – 15 mm yang berfungsi membunuh sel yang tidak diinginkan seperti sel tumor, sel yang terinfeksi-virus.

nf l amasi / er adangan



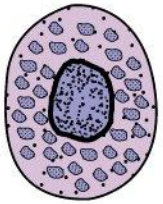
Colonoscopy View

el at ur al i l l er



INFLAMMATORY RESPONSE

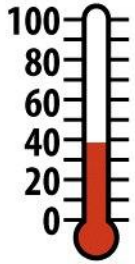
Multiple effects



1. Blood flow increased
2. Phagocytes activated
3. Capillary permeability increased
4. Complement activated
5. Clotting reaction walls off region
6. Regional temperature increased
7. Specific defenses activated

FEVER

Mobilizes defenses, accelerates repairs, inhibits pathogens



Body temperature rises above 37°C in response to pyrogens

INTERFERONS

Increase resistance of cells to infection, slow the spread of disease

Released by activated lymphocytes and macrophages and by virus-infected cells

COMPLEMENT SYSTEM

Attacks and breaks down cell walls, attracts phagocytes, stimulates inflammation

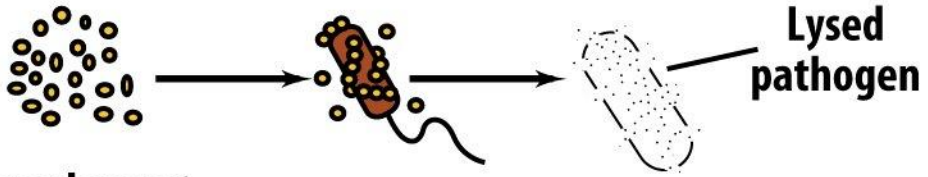
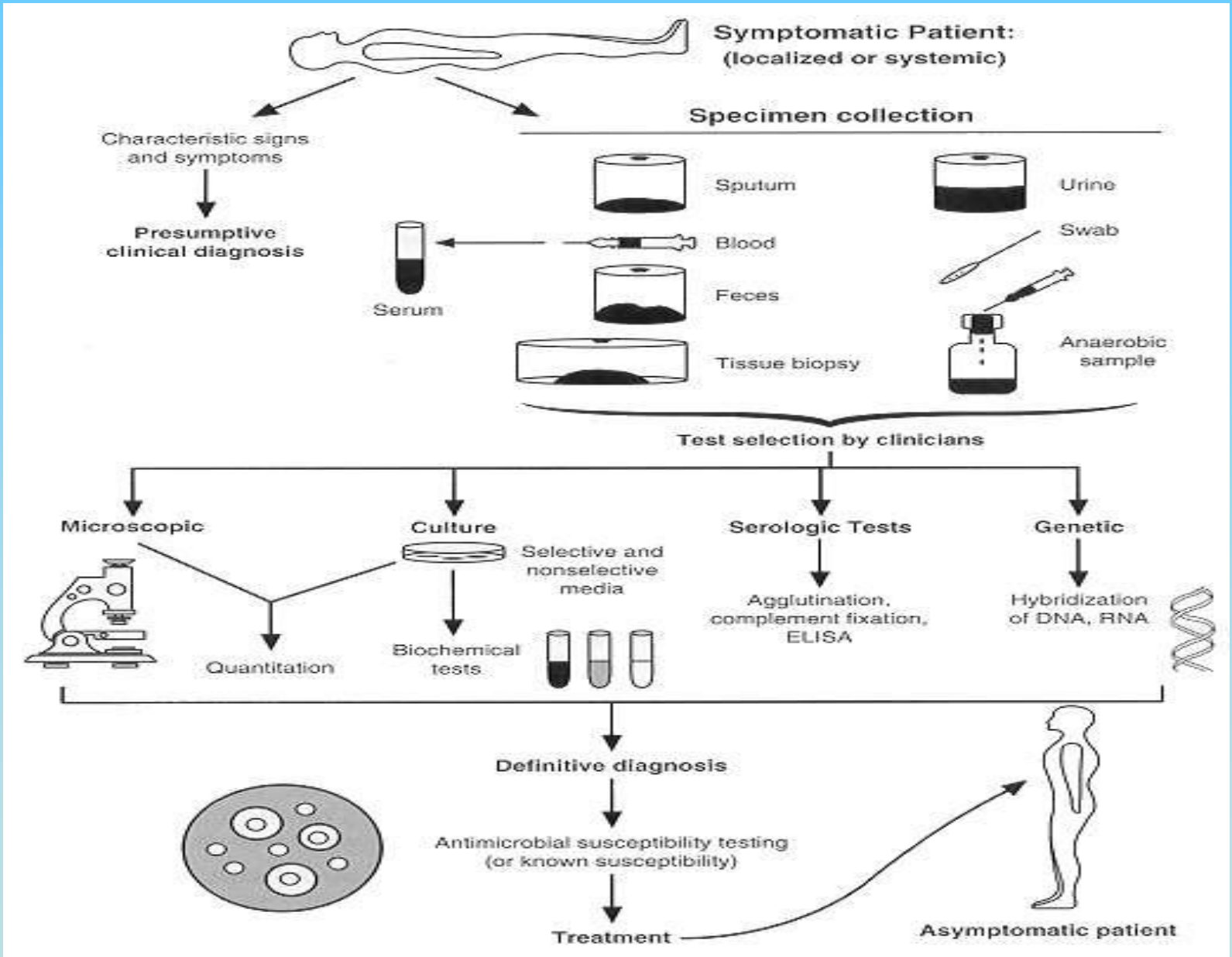


Figure 16-10 part 2 Microbiology, 6/e

Diagnosis penyakit infeksi



Terima kasih....