Microbial Mechanisms of Pathogenicity

TABLE 15.1 Portals of Entry for the Pathogens of Some Common Diseases

Portal of Entry	Pathogen*	Disease	Incubation Period
Mucous Membranes			
Respiratory tract	*Streptococcus pneumoniae*	Pneumococcal pneumonia	Variable
	Mycobacterium tuberculosis†	Tuberculosis	Variable
	Bordetella pertussis	Whooping cough (pertussis)	12–20 days
	Influenza virus *(Influenzavirus)*	Influenza	18–36 hours
	Measles virus *(Morbillivirus)*	Measles (rubeola)	11–14 days
	Rubella virus *(Rubivirus)*	German measles (rubella)	2–3 weeks
	Epstein-Barr virus *(Lymphocryptovirus)*	Infectious mononucleosis	2–6 weeks
	Varicella-zoster virus *(Varicellovirus)*	Chickenpox (varicella) (primary infection)	14–16 days
	Histoplasma capsulatum (fungus)	Histoplasmosis	5–18 days
Gastrointestinal tract	*Shigella* spp.	Shigellosis (bacillary dysentery)	1–2 days
	Brucella spp.	Brucellosis (undulant fever)	6–14 days
	Vibrio cholerae	Cholera	1–3 days
	Salmonella enterica	Salmonellosis	7–22 hours
	Salmonella typhi	Typhoid fever	14 days
	Hepatitis A virus *(Hepatovirus)*	Hepatitis A	15–50 days
	Mumps virus *(Rubulavirus)*	Mumps	2–3 weeks
	Trichinella spiralis (helminth)	Trichinellosis	2–28 days
Genitourinary tract	*Neisseria gonorrhoeae*	Gonorrhea	3–8 days
	Treponema pallidum	Syphilis	9–90 days
	Chlamydia trachomatis	Nongonococcal urethritis	1–3 weeks
	Herpes simplex virus type 2	Herpes virus infections	4–10 days
	Human immunodeficiency virus (HIV)‡	AIDS	10 years
	Candida albicans (fungus)	Candidiasis	2–5 days
Skin or Parenteral Route			
	Clostridium perfringens	Gas gangrene	1–5 days
	Clostridium tetani	Tetanus	3–21 days
	Rickettsia rickettsii	Rocky Mountain spotted fever	3–12 days
	Hepatitis B virus *(Hepadnavirus)*‡	Hepatitis B	6 weeks–6 months
	Rabiesvirus *(Lyssavirus)*	Rabies	10 days–1 year
	Plasmodium spp. (protozoan)	Malaria	2 weeks

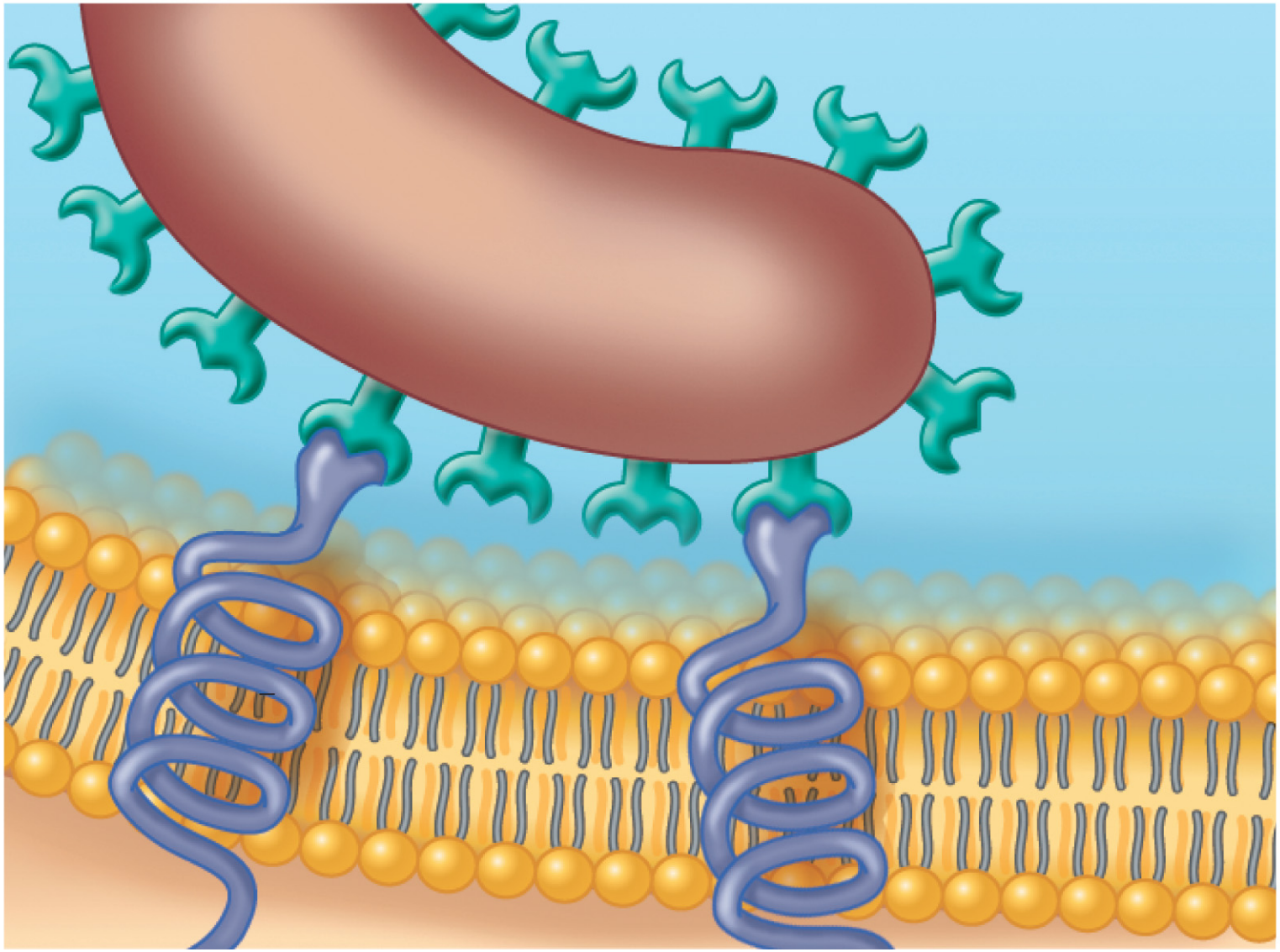
Bacillus anthracis

Portal of Entry	**ID_{50}**
Skin	10–50 endospores
Inhalation	10,000–20,000 endospores
Ingestion	250,000–1,000,000 endospores

Toxins

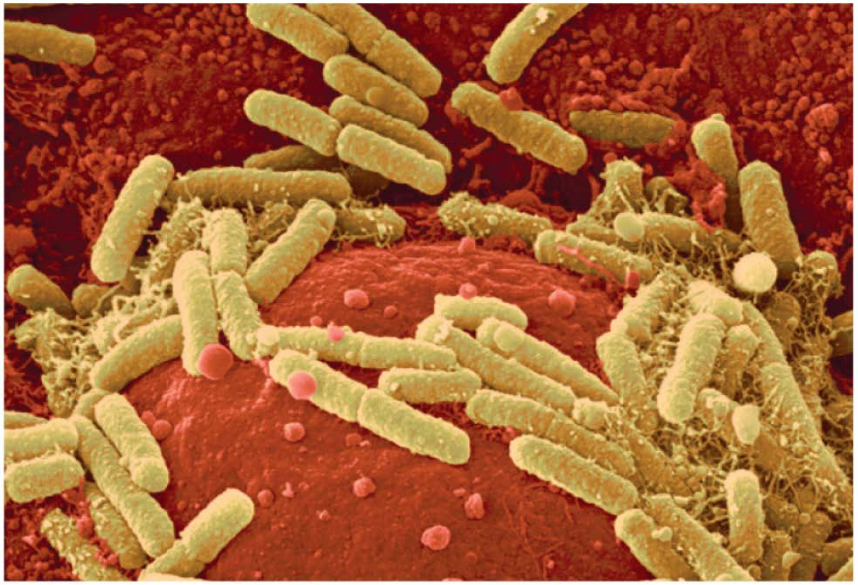
Portal of Entry	**ID_{50}**
Botulinum	0.03 ng/kg
Shiga toxin	250 ng/kg
Staphylococcal enterotoxin	1350 ng/kg

Figure 15.1a Adherence.



(a)

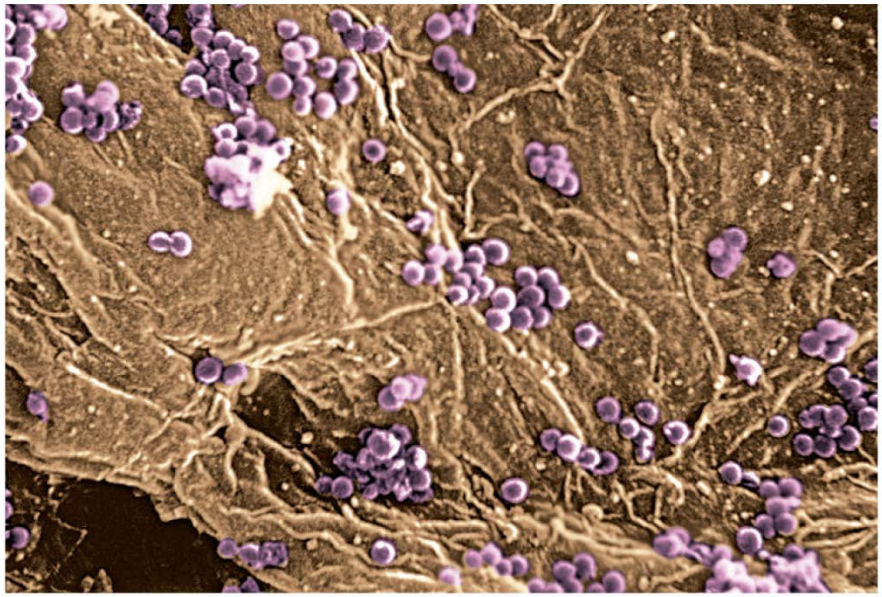
Figure 15.1b-c Adherence.



(b) *E. coli* bacteria (yellow-green) on human urinary bladder cells

SEM

1 μm

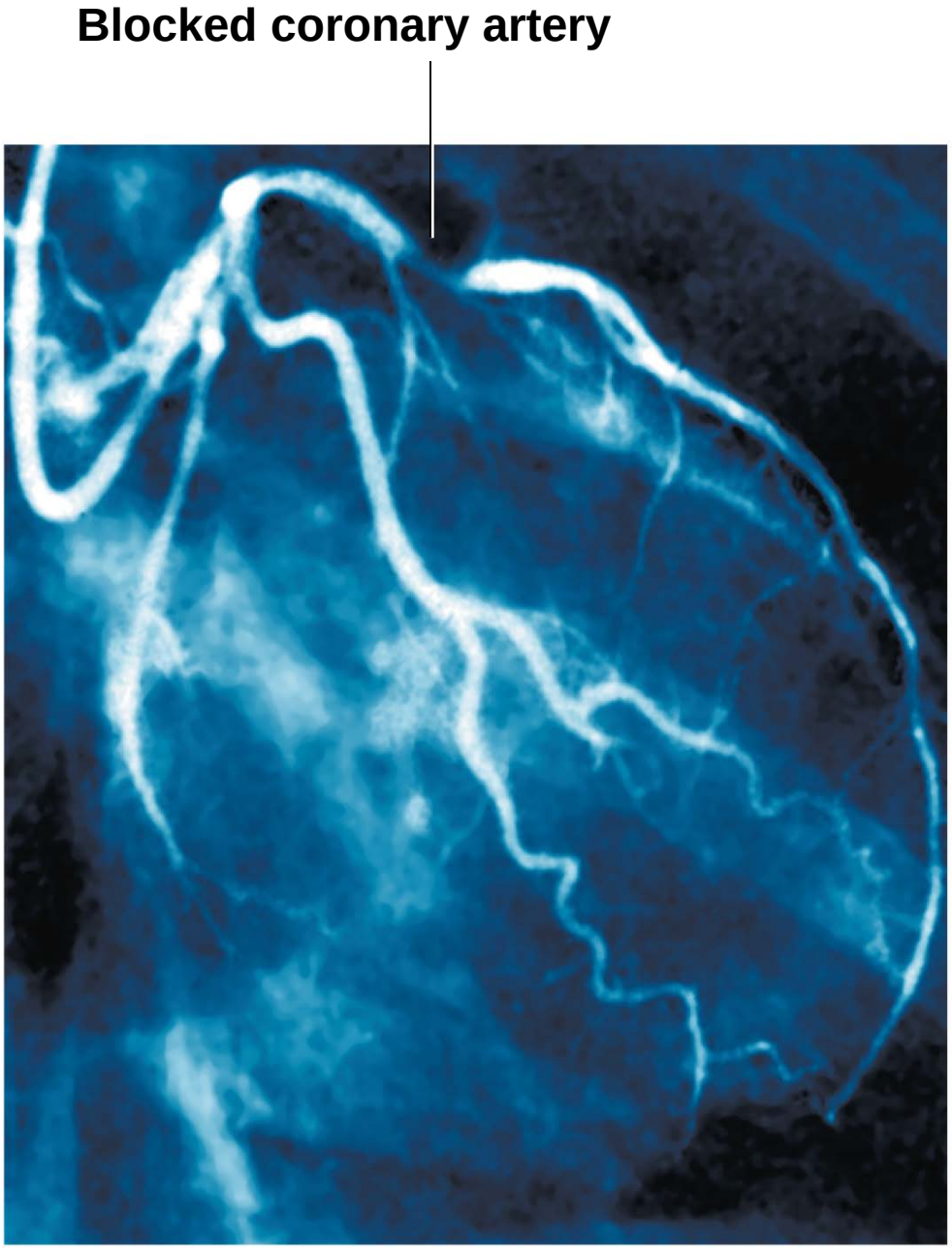


(c) Bacteria (purple) adhering to human skin

SEM

9 μm

Chapter 15, unnumbered figure A, p. 434.



Coagulase test

Chapter 15, unnumbered figure C, p. 434.

Mechanism of streptokinase

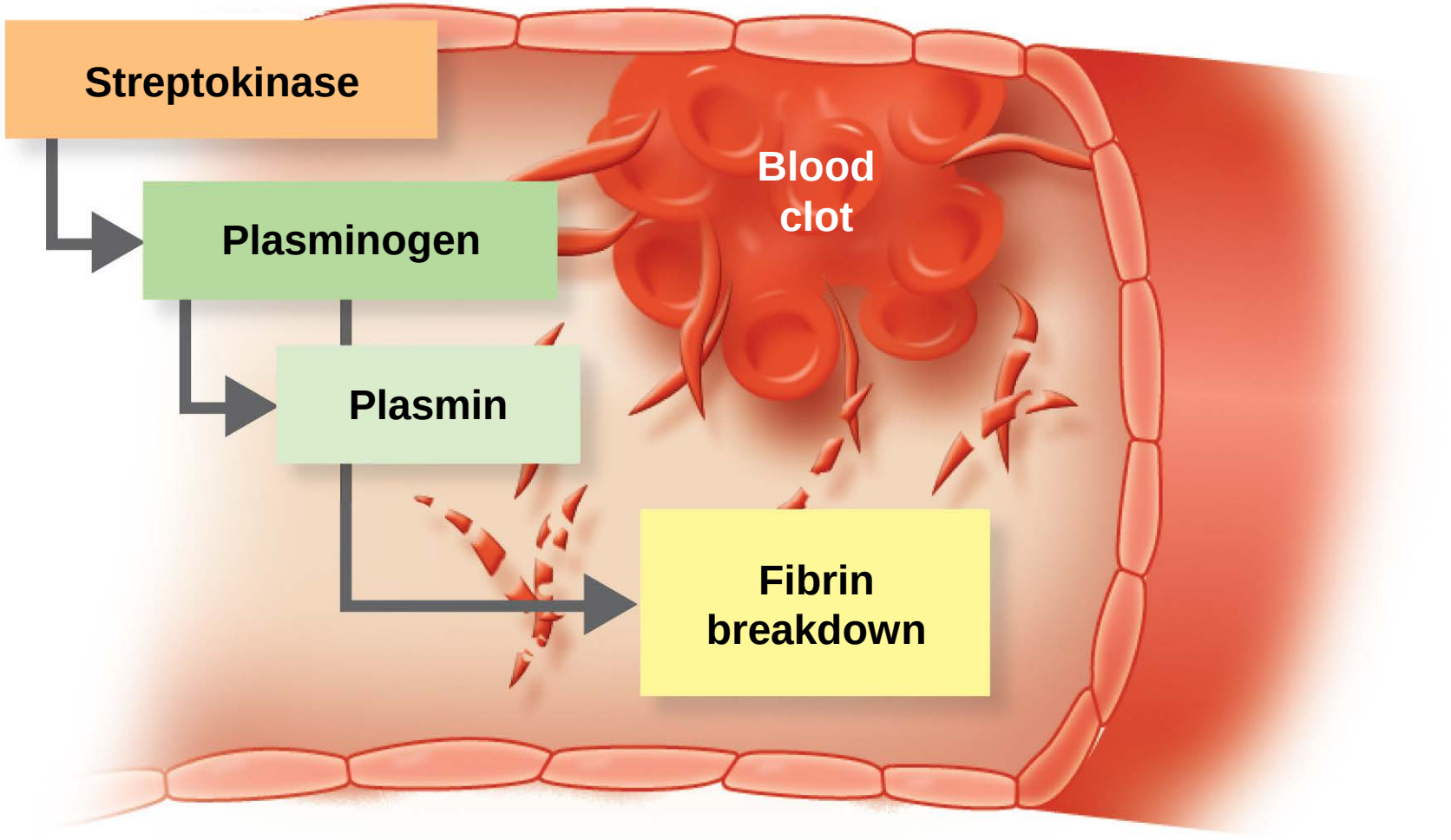


Figure 22.16 How trypanosomes evade the immune system.

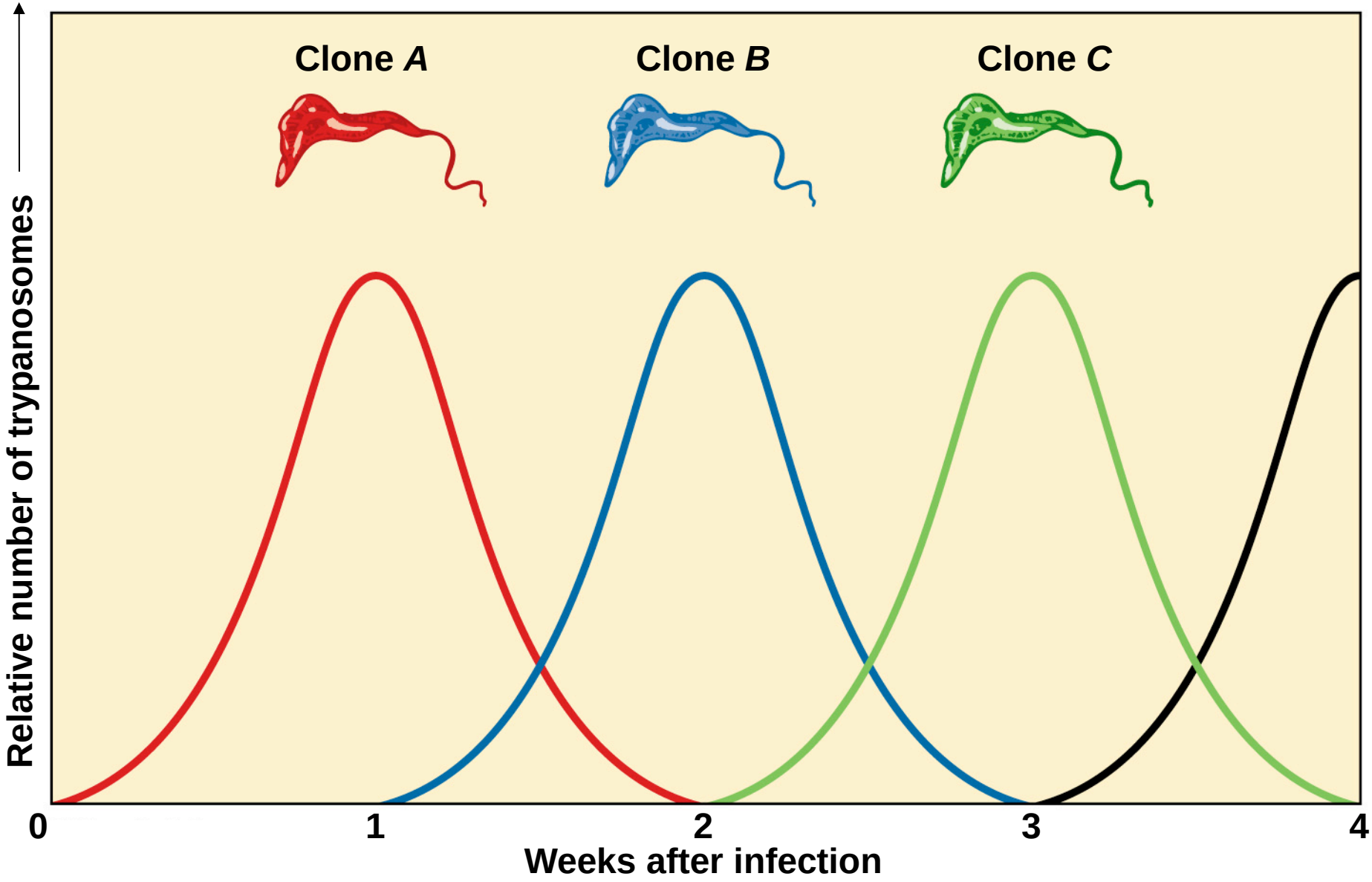


Figure 15.2 *Salmonella* entering intestinal epithelial cells as a result of ruffling.

Figure 15.3 Structure of enterobactin, one type of bacterial siderophore.

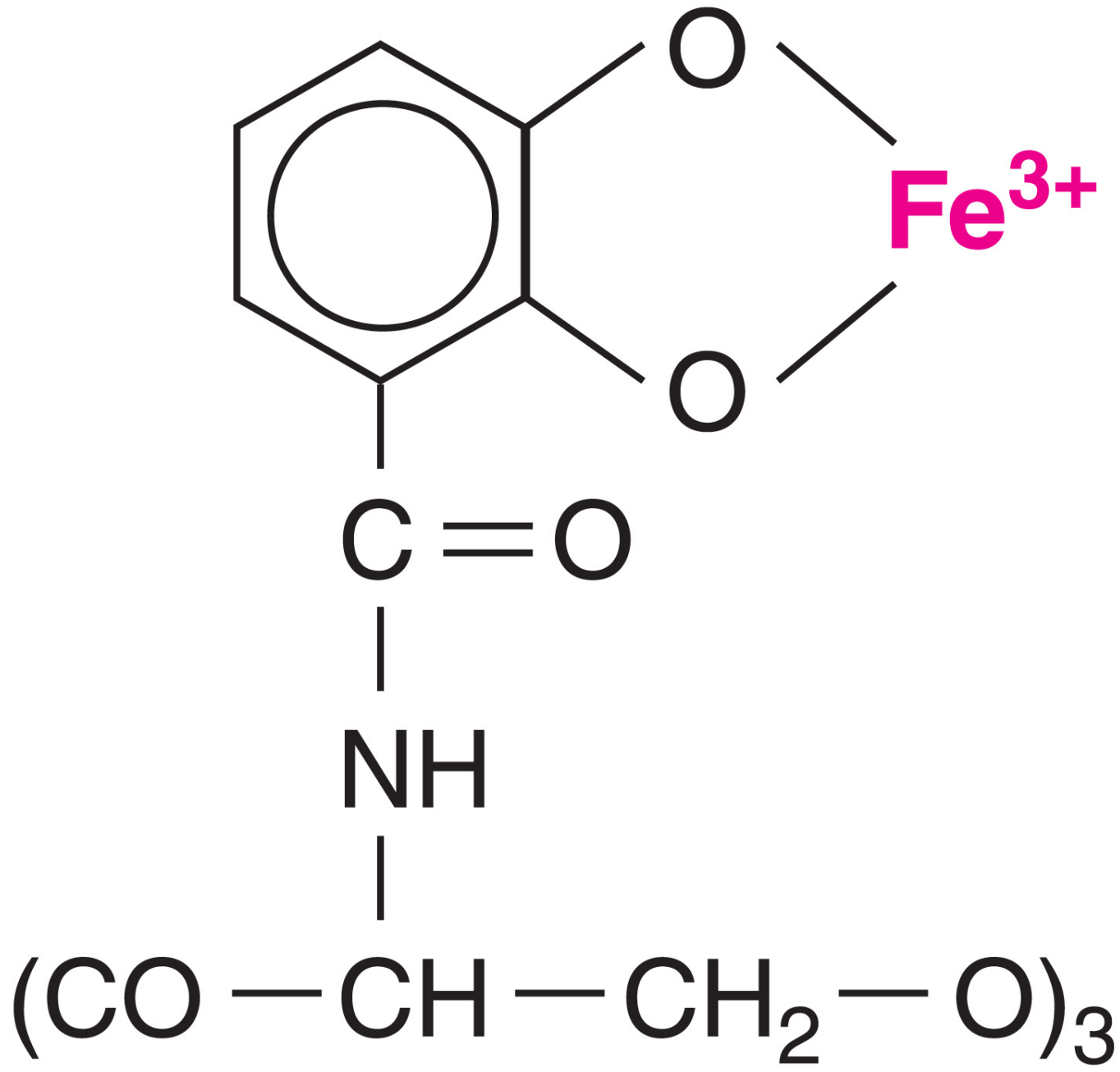


Figure 4.13c Bacterial cell walls.

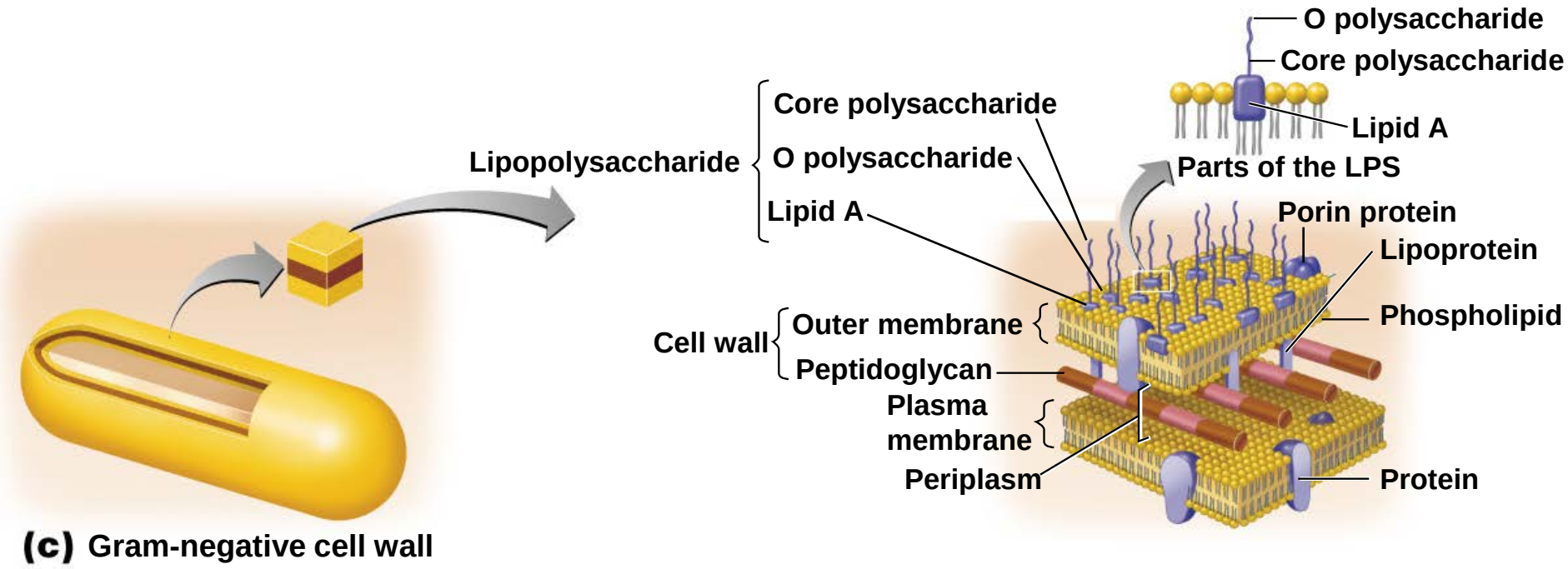
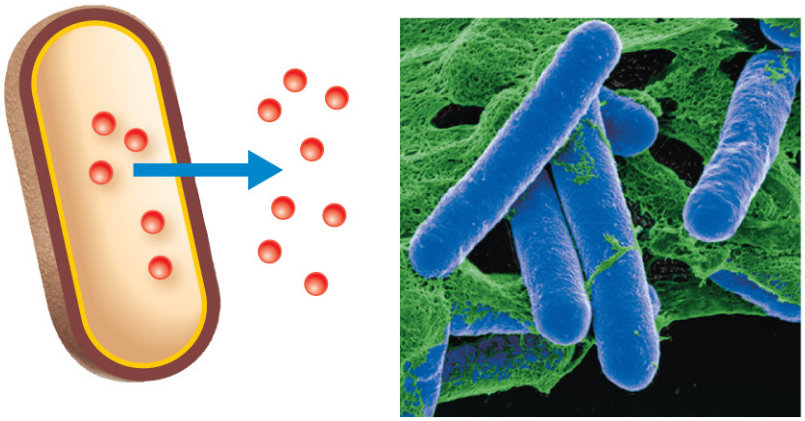


Figure 15.4 Exotoxins and Endotoxins.



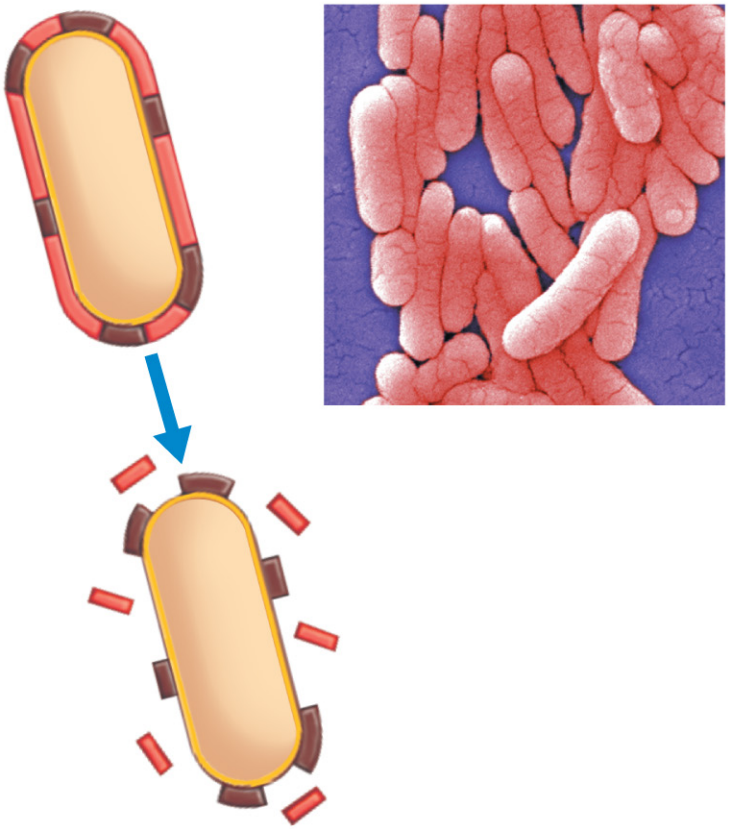


Figure 15.5 The action of an A-B exotoxin.

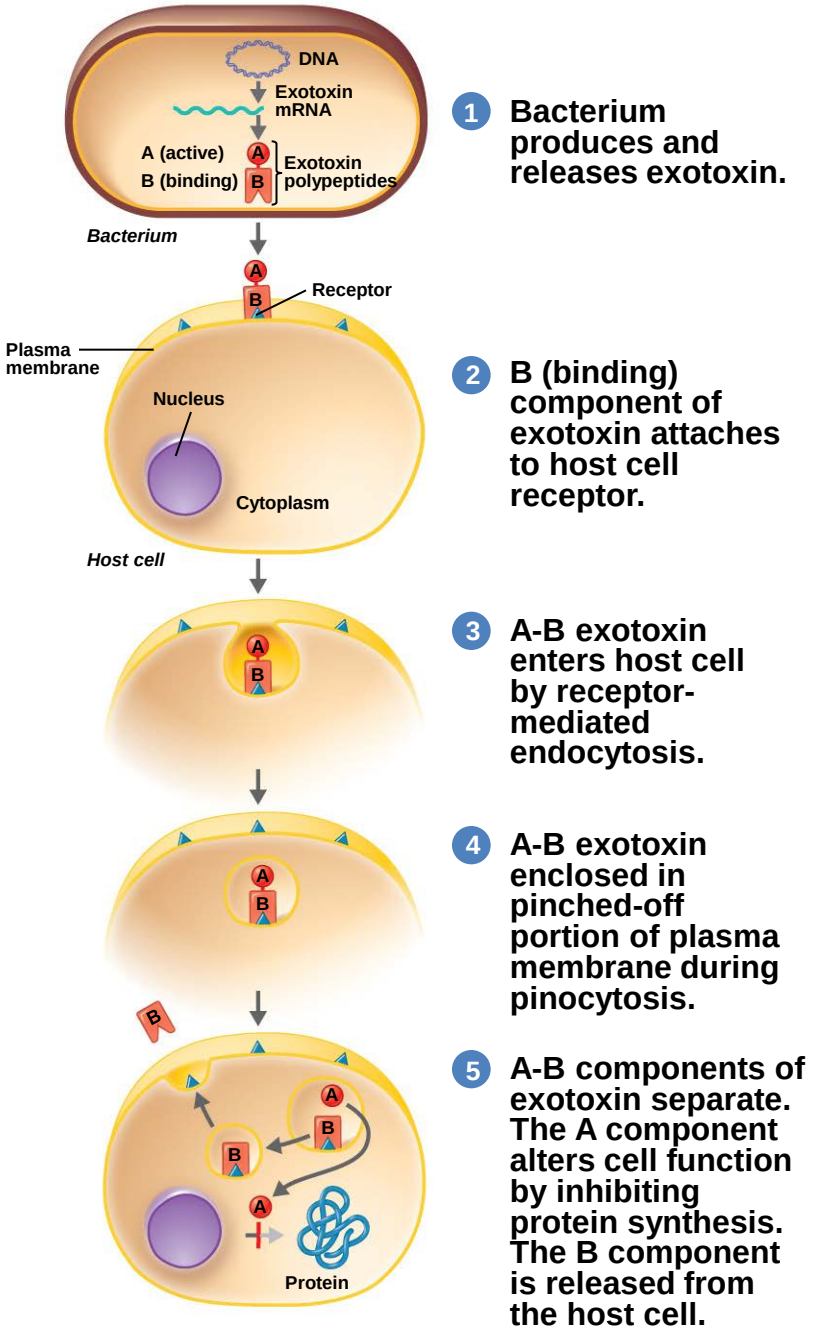
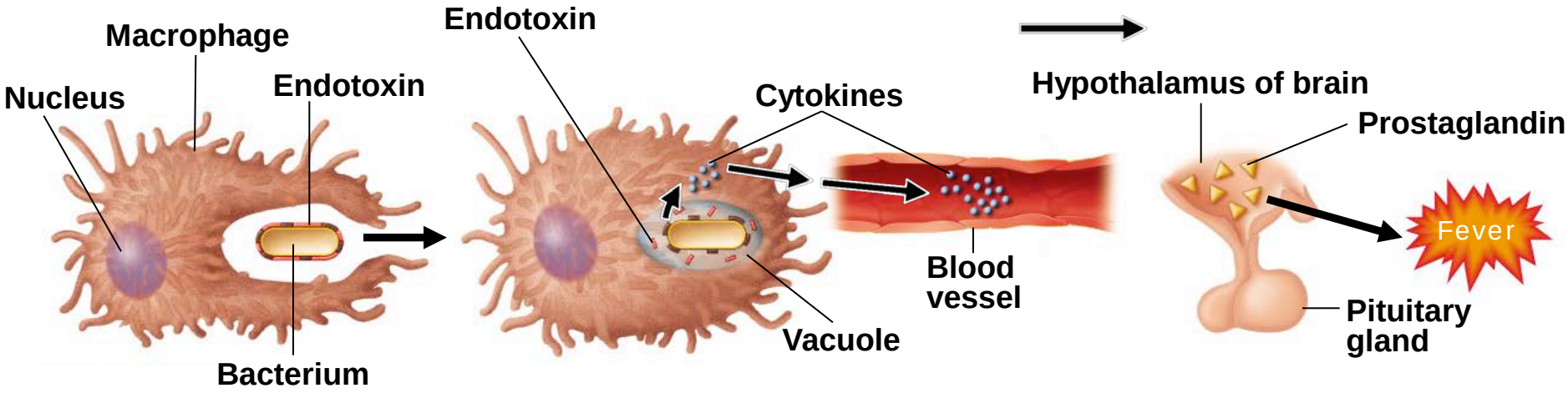
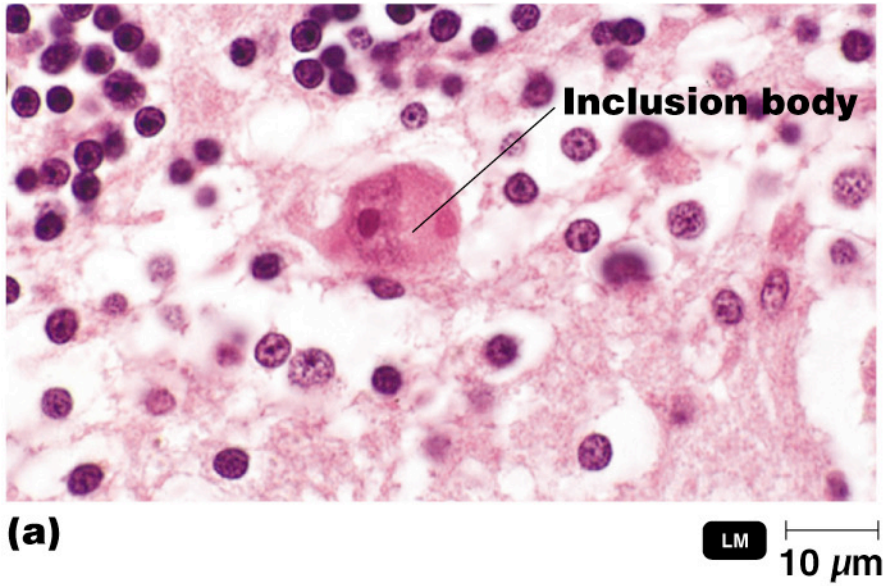


Figure 15.6 Endotoxins and the pyrogenic response.



1. A macrophage ingests a gram-negative bacterium.
2. The bacterium is degraded in a vacuole, releasing endotoxins that induce the macrophage to produce cytokines IL-1 and TNF-α.
3. The cytokines are released into the bloodstream by the macrophages, through which they travel to the hypothalamus of the brain.
4. The cytokines induce the hypothalamus to produce prostaglandins, which reset the body's "thermostat" to a higher temperature, producing fever.

Pathogenic properties: Viruses



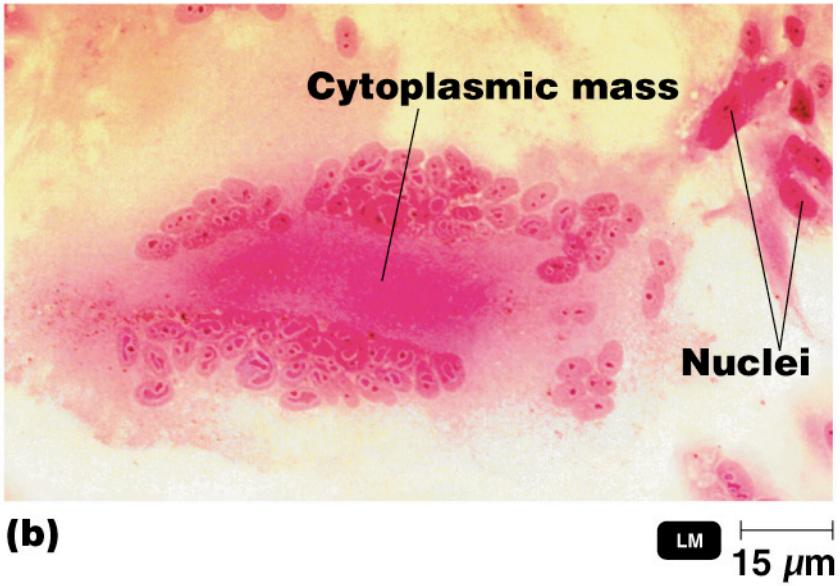


Figure 15.7 Some cytopathic effects of viruses.

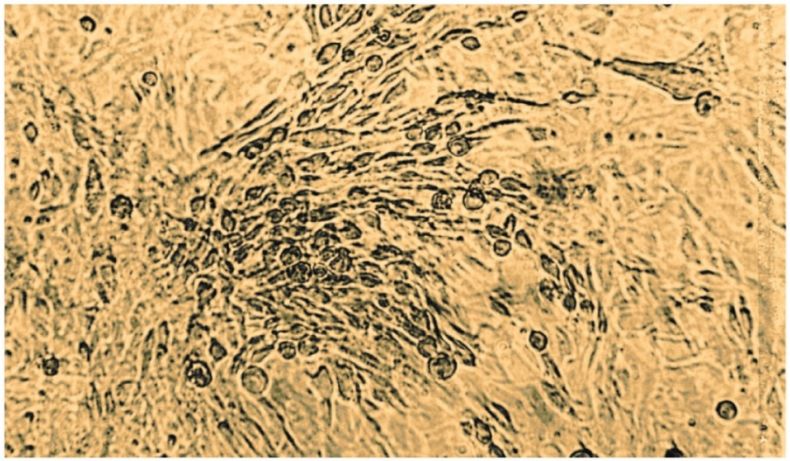


Figure 15.8 Transformed cells.

Pathogenic properties: Fungi

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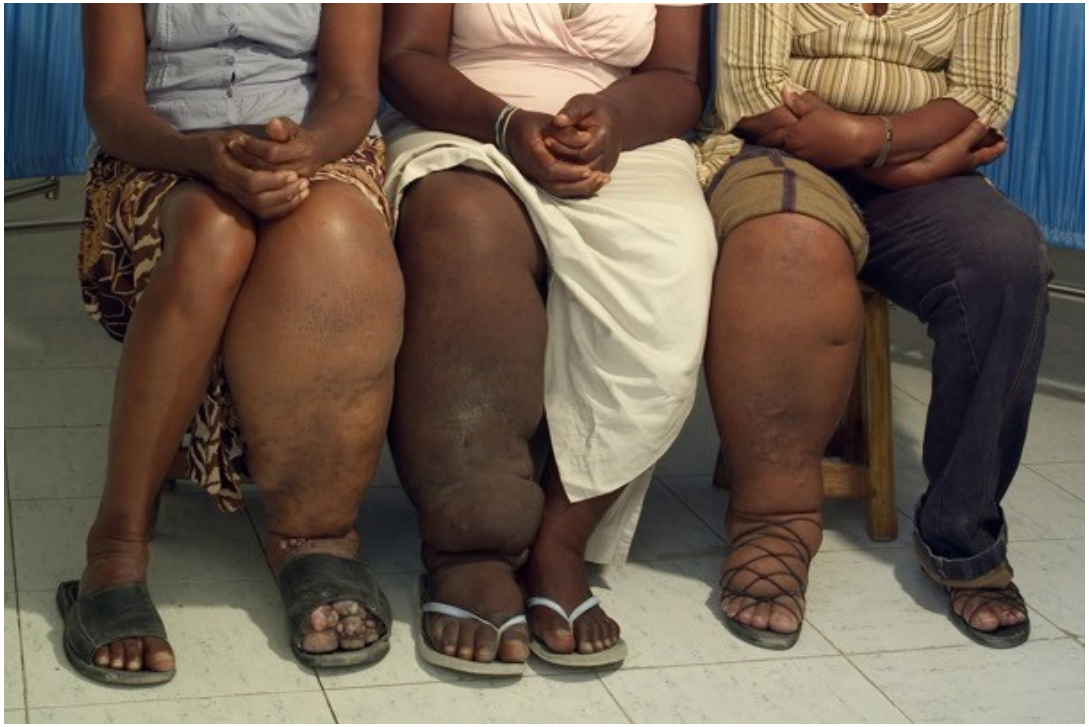
Matthias Grunewald (1500s)

Ergot

wikipedia.org

Pathogenic properties: Worms



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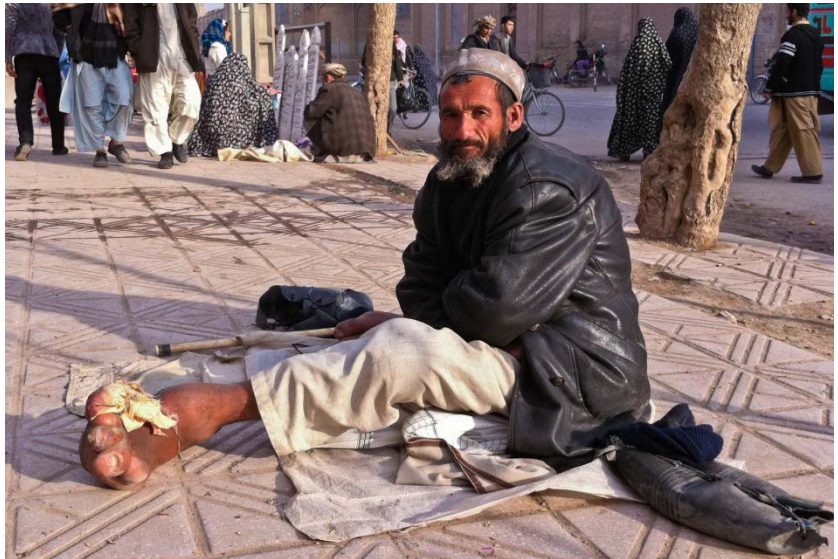

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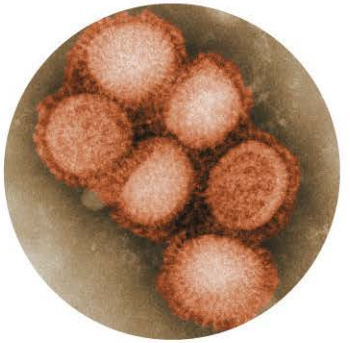
Pathogenic properties: Algae

Figure 27.13 A red tide.

Figure 15.9 Microbial Mechanisms of Pathogenicity.

When the balance between host and microbe is tipped in favor of the microbe, an infection or disease results. Learning these mechanisms of microbial pathogenicity is fundamental to understanding how pathogens are able to overcome the host's defenses.



H1N1 flu virus

portals of entry

Mucous membranes
- **Respiratory tract**
- **Gastrointestinal tract**
- **Genitourinary tract**
- **Conjunctiva**

Skin
Parenteral route

Number of invading microbes

Adherence

penetration or evasion of host defenses

Capsules
Cell wall components
Enzymes
Antigenic variation
Invasins
Intracellular growth

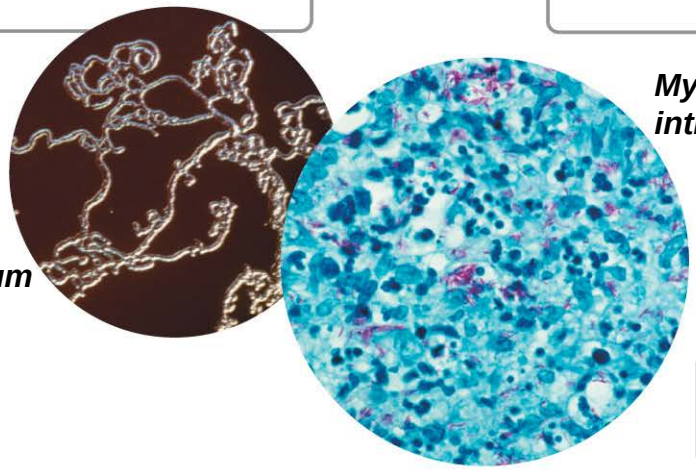
damage to host cells

Siderophores
Direct damage
Toxins
- **Exotoxins**
- **Endotoxins**

Lysogenic conversion
Cytopathic effects

portals of exit

Generally the same as the portals of entry for a given microbe:
- **Mucous membranes**
- **Skin**
- **Parenteral route**



Clostridium tetani

Mycobacterium intracellulare

Micrographs are not shown to scale.