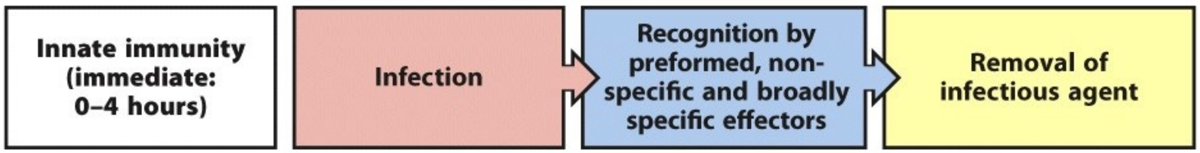



conceito importante!


| | | | | |
|---|---|---|---|---|
| Early induced innate response (early: 4–96 hours) | Infection → | Recruitment of effector cells → | Recognition of PAMPS. Activation of effector cells and inflammation → | Removal of infectious agent |

conceito importante!

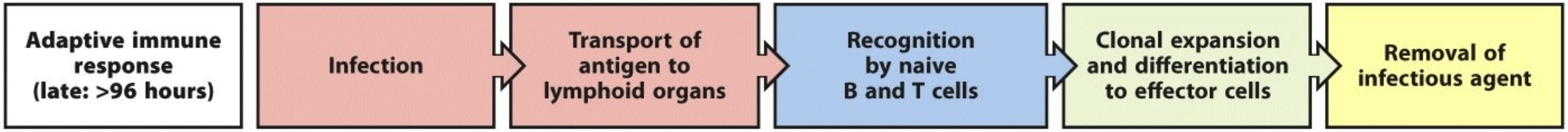




Figure 2.1 Janeway's Immunobiology, 8ed. (© Garland Science 2012)


conceito importante!

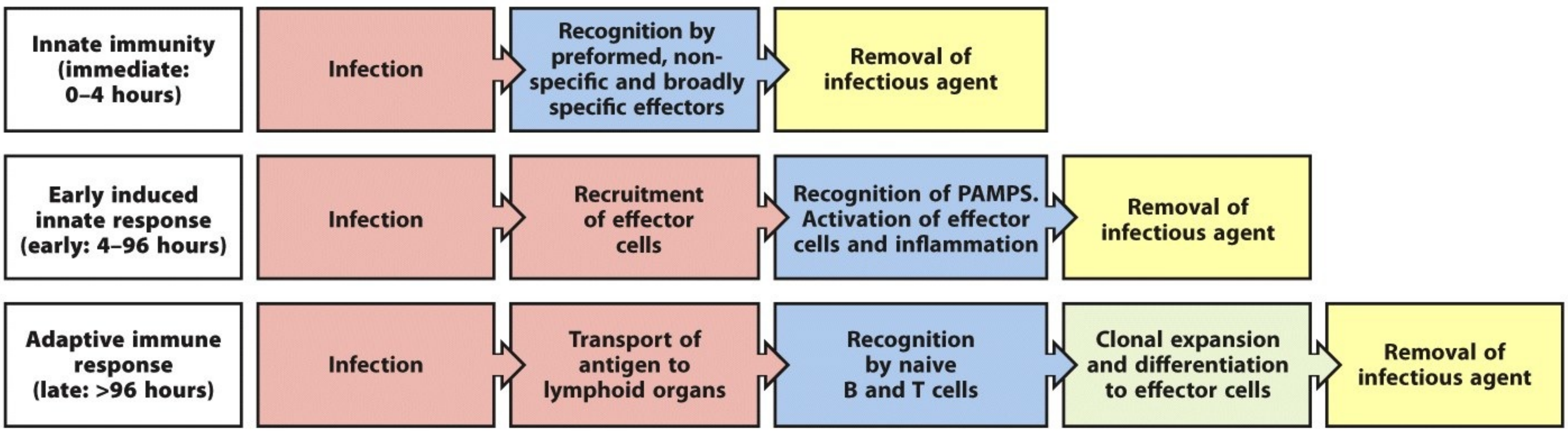
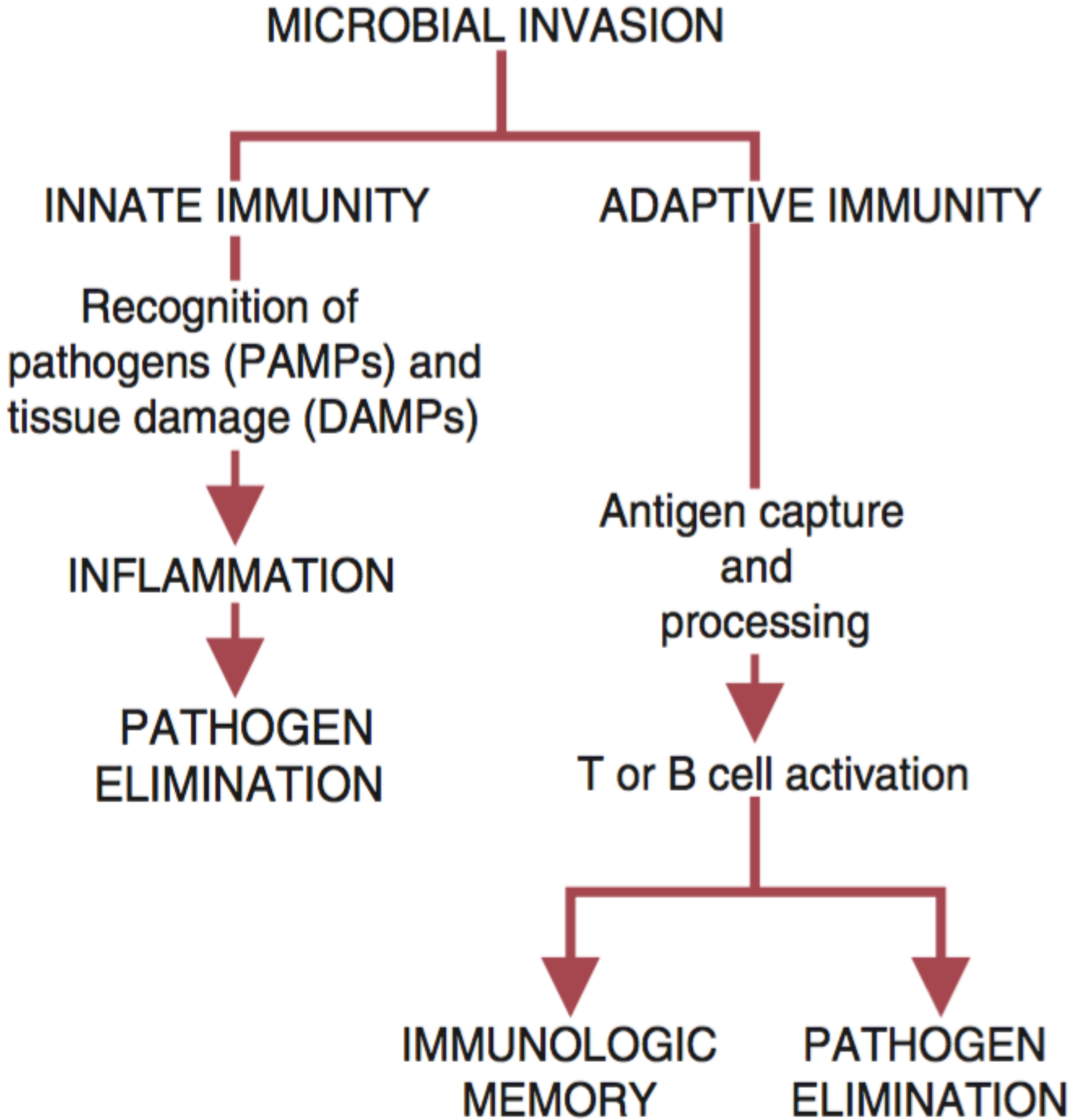
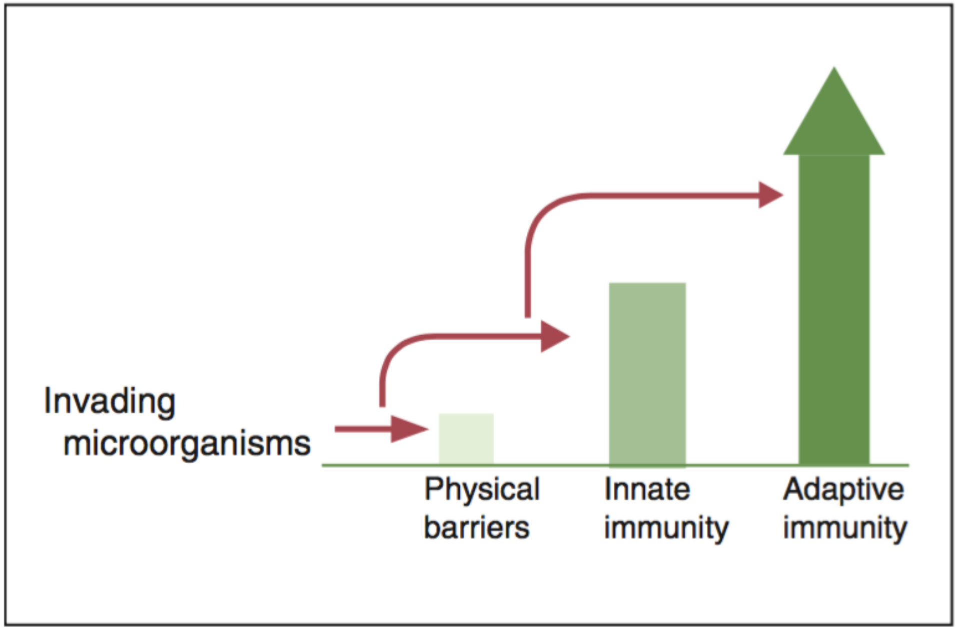
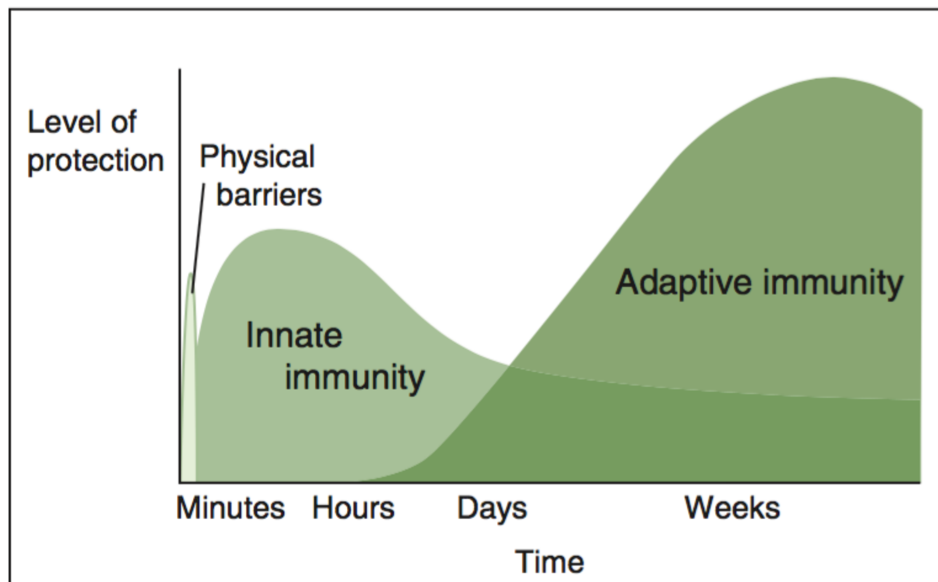


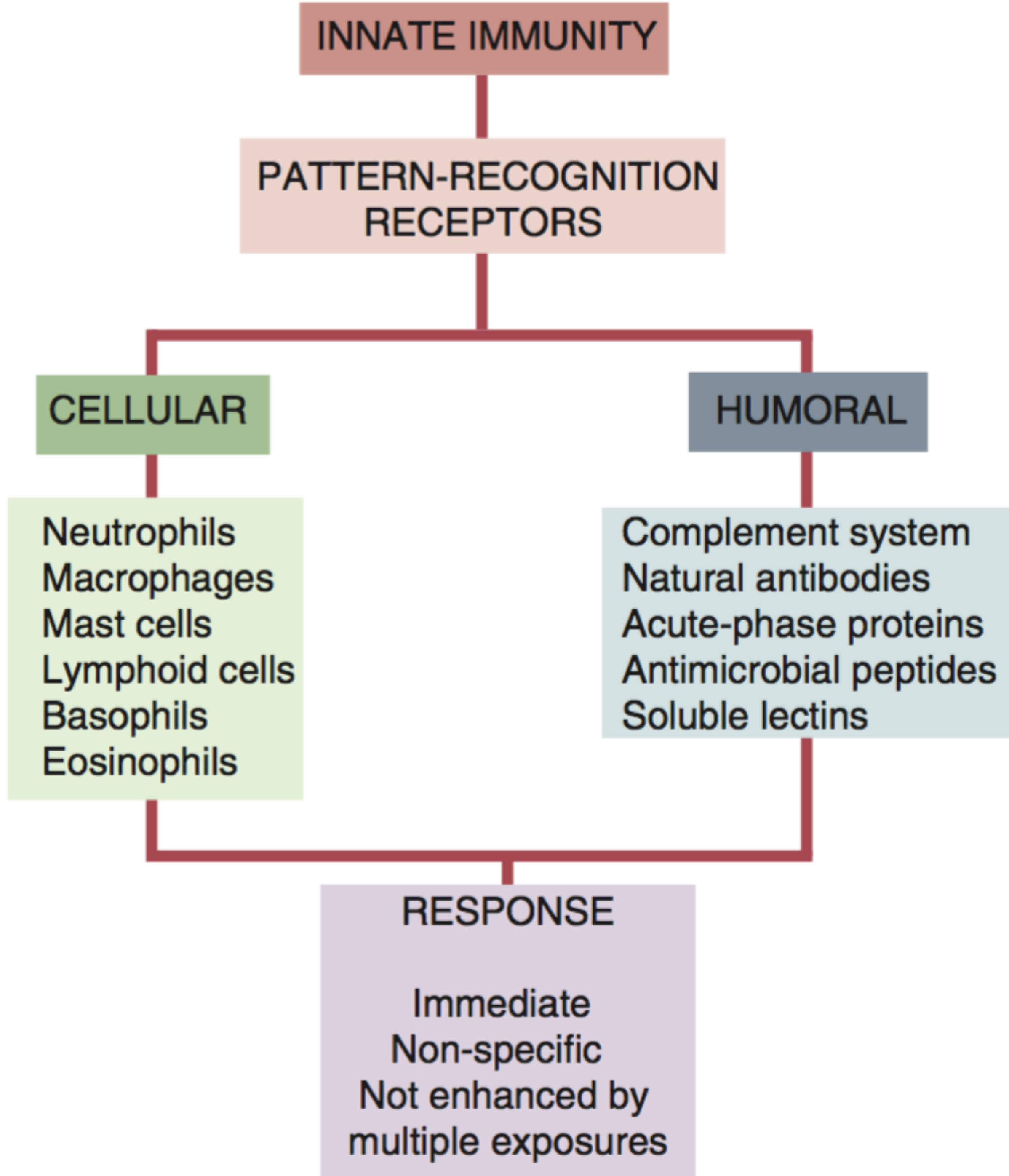

Figure 2.1 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

conceito importante!


MICROBIAL INVASION
INNATE IMMUNITY
ADAPTIVE IMMUNITY
Recognition of pathogens (PAMPs) and tissue damage (DAMPs)
INFLAMMATION
PATHOGEN ELIMINATION
Antigen capture and processing
T or B cell activation
IMMUNOLOGIC MEMORY
PATHOGEN ELIMINATION


Invading
microorganisms
Physical
barriers
Innate
immunity
Adaptive
immunity


Level of
protection
Physical
barriers
Innate
immunity
Adaptive immunity
Minutes
Hours
Days
Weeks
Time


INNATE IMMUNITY
PATTERN-RECOGNITION RECEPTORS
CELLULAR
HUMORAL
Neutrophils
Macrophages
Mast cells
Lymphoid cells
Basophils
Eosinophils
Complement system
Natural antibodies
Acute-phase proteins
Antimicrobial peptides
Soluble lectins
RESPONSE
Immediate
Non-specific
Not enhanced by
multiple exposures

| Routes of infection for pathogens | | | | |
|---|---|---|---|---|
| **Route of entry** | **Mode of transmission** | **Pathogen** | **Disease** | **Type of pathogen** |
| **Mucosal surfaces** | | | | |
| Mouth and respiratory tract | Inhalation or ingestion of infective material (e.g. saliva droplets) | Measles virus | Measles | Paramyxovirus |
| | | Influenza virus | Influenza | Orthomyxovirus |
| | | Varicella-zoster | Chickenpox | Herpesvirus |
| | | Epstein-Barr virus | Mononucleosis | Herpesvirus |
| | | *Streptococcus pyogenes* | Tonsilitis | Gram-positive bacterium |
| | | *Haemophilus influenzae* | Pneumonia, meningitis | Gram-negative bacterium |
| | Spores | *Neisseria meningitidis* | Meningococcal meningitis | Gram-negative bacterium |
| | | *Bacillus anthracis* | Inhalation anthrax | Gram-positive bacterium |
| Gastrointestinal tract | Contaminated water or food | Rotavirus | Diarrhea | Rotavirus |
| | | Hepatitis A | Jaundice | Picornavirus |
| | | *Salmonella enteritidis, S. typhimurium* | Food poisoning | Gram-negative bacterium |
| | | *Vibrio cholerae* | Cholera | Gram-negative bacterium |
| | | *Salmonella typhi* | Typhoid fever | Gram-negative bacterium |
| Reproductive tract and other routes | Sexual transmission/ infected blood | Hepatitis B virus | Hepatitis B | Hepadnavirus |
| | | Human immunodeficiency virus (HIV) | Acquired immunodeficiency syndrome (AIDS) | Retrovirus |
| | Sexual transmission | *Neisseria gonorrhoeae* | Gonorrhea | Gram-negative bacterium |
| | | *Treponema pallidum* | Syphilis | Bacterium (spirochete) |
| **Opportunistic pathogens** | | | | |
| | Resident microbiota | *Candida albicans* | Candidiasis, thrush | Fungus |
| | Resident lung microbiota | *Pneumocystis jirovecii* | Pneumonia | Fungus |
| **External epithelia** | | | | |
| External surface | Physical contact | *Trichophyton* | Athlete's foot | Fungus |
| Wounds and abrasions | Minor skin abrasions | *Bacillus anthracis* | Cutaneous anthrax | Gram-positive bacterium |
| | Puncture wounds | *Clostridium tetani* | Tetanus | Gram-positive bacterium |
| | Handling infected animals | *Francisella tularensis* | Tularemia | Gram-negative bacterium |
| Insect bites | Mosquito bites (*Aedes aegypti*) | Flavivirus | Yellow fever | Virus |
| | Deer tick bites | *Borrelia burgdorferi* | Lyme disease | Bacterium (spirochete) |
| | Mosquito bites (*Anopheles*) | *Plasmodium* spp. | Malaria | Protozoan |

**Figure 2.2 Janeway's Immunobiology, 8ed. (© Garland Science 2012)**

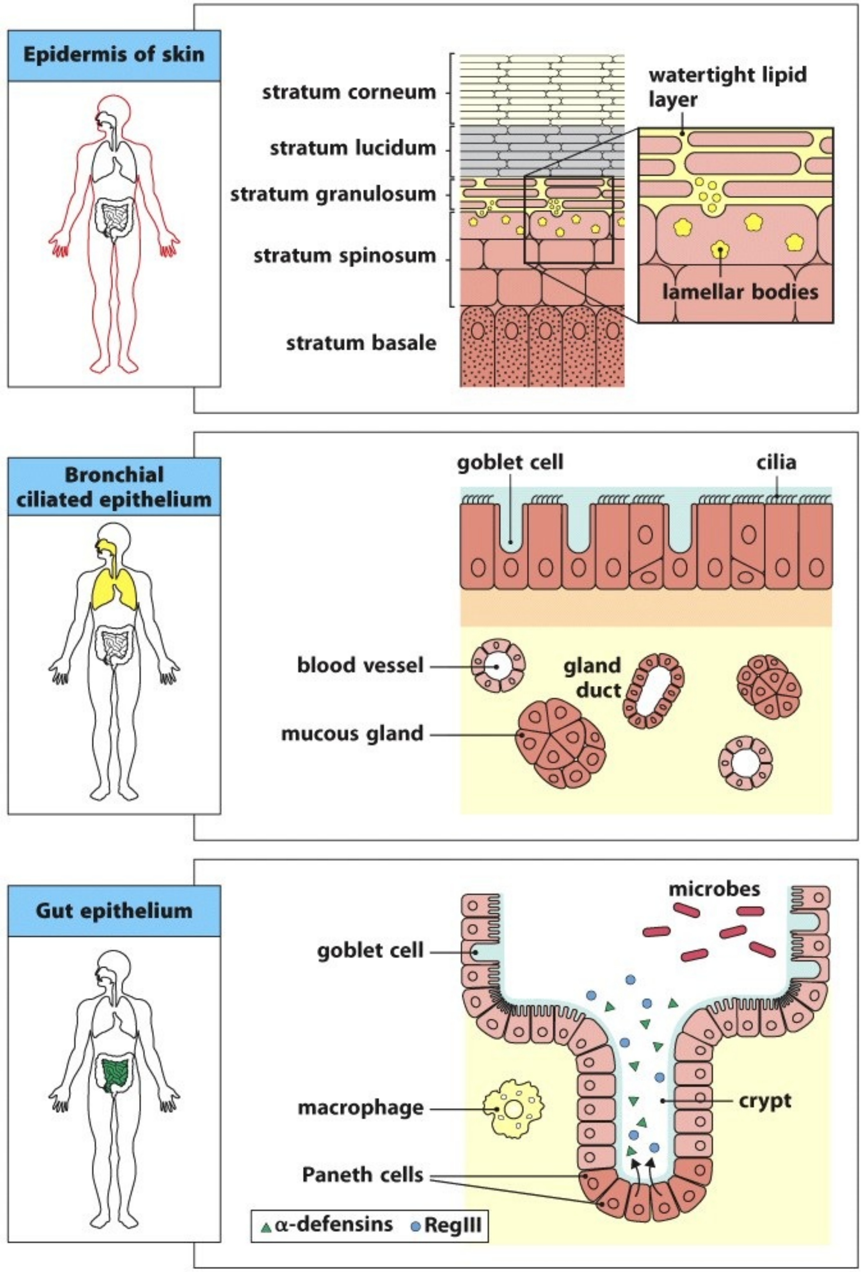



Figure 2.10 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

$1.5 - 2\ m^2$

$30 - 100\ m^2$

~ 1/2 quadra de tênis

$30 - 40\ m^2$

~ studio

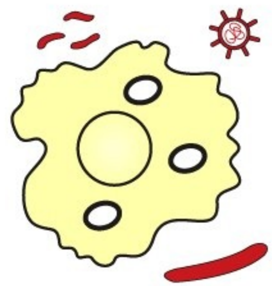
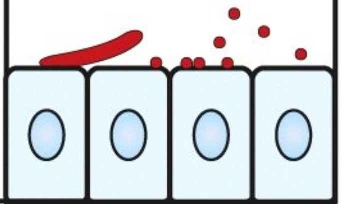
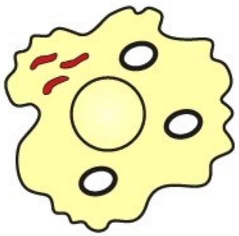
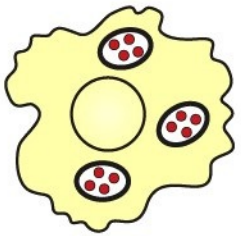
| | **Extracellular** | | **Intracellular** | |
|---|---|---|---|---|
| | **Interstitial spaces, blood, lymph** | **Epithelial surfaces** | **Cytoplasmic** | **Vesicular** |
| **Site of infection** |  |  |  |  |
| **Organisms** | Viruses<br>Bacteria<br>Protozoa<br>Fungi<br>Worms | *Neisseria gonorrhoeae*<br>*Streptococcus pneumoniae*<br>*Vibrio cholerae*<br>*Helicobacter pylori*<br>*Candida albicans*<br>Worms | Viruses<br>*Chlamydia* spp.<br>*Rickettsia* spp.<br>Protozoa | *Mycobacterium* spp.<br>*Yersinia pestis*<br>*Legionella pneumophila*<br>*Cryptococcus neoformans*<br>*Leishmania* spp. |
| **Protective immunity** | Complement<br>Phagocytosis<br>Antibodies | Antimicrobial peptides<br>Antibodies, especially IgA | NK cells<br>Cytotoxic T cells | T-cell and NK-cell dependent macrophage activation |

Figure 2.3 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

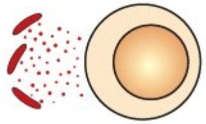
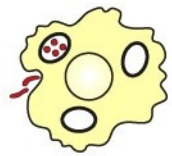
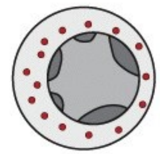
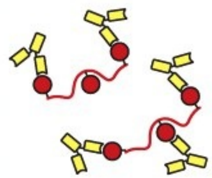
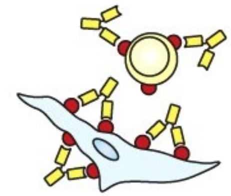
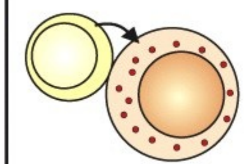
| | Direct mechanisms of tissue damage by pathogens | | | Indirect mechanisms of tissue damage by pathogens | | |
|---|---|---|---|---|---|---|
| | Exotoxin production | Endotoxin | Direct cytopathic effect | Immune complexes | Anti-host antibody | Cell-mediated immunity |
| **Pathogenic mechanism** |  |  |  |  |  |  |
| **Infectious agent** | *Streptococcus pyogenes*<br>*Staphylococcus aureus*<br>*Corynebacterium diphtheriae*<br>*Clostridium tetani*<br>*Vibrio cholerae* | *Escherichia coli*<br>*Haemophilus influenzae*<br>*Salmonella typhi*<br>*Shigella*<br>*Pseudomonas aeruginosa*<br>*Yersinia pestis* | Variola<br>Varicella-zoster<br>Hepatitis B virus<br>Polio virus<br>Measles virus<br>Influenza virus<br>Herpes simplex virus<br>Human herpes virus 8 (HHV8) | Hepatitis B virus<br>Malaria<br>*Streptococcus pyogenes*<br>*Treponema pallidum*<br>Most acute infections | *Streptococcus pyogenes*<br>*Mycoplasma pneumoniae* | Lymphocytic choriomeningitis virus<br>Herpes simplex virus<br>*Mycobacterium tuberculosis*<br>*Mycobacterium leprae*<br>*Borrelia burgdorferi*<br>*Schistosoma mansoni* |
| **Disease** | Tonsilitis, scarlet fever<br>Boils, toxic shock syndrome, food poisoning<br>Diphtheria<br>Tetanus<br>Cholera | Gram-negative sepsis<br>Meningitis, pneumonia<br>Typhoid fever<br>Bacillary dysentery<br>Wound infection<br>Plague | Smallpox<br>Chickenpox, shingles<br>Hepatitis<br>Poliomyelitis<br>Measles, subacute sclerosing panencephalitis<br>Influenza<br>Cold sores<br>Kaposi's sarcoma | Kidney disease<br>Vascular deposits<br>Glomerulonephritis<br>Kidney damage in secondary syphilis<br>Transient renal deposits | Rheumatic fever<br>Hemolytic anemia | Aseptic meningitis<br>Herpes stromal keratitis<br>Tuberculosis<br>Tuberculoid leprosy<br>Lyme arthritis<br>Schistosomiasis |

**Figure 2.4 Janeway's Immunobiology, 8ed. (© Garland Science 2012)**

# Barreiras não-imunológicas

| | Skin | Gut | Lungs | Eyes/nose/ oral cavity |
|---|---|---|---|---|
| Mechanical | Epithelial cells joined by tight junctions | | | |
| | Longitudinal flow of air or fluid | | Movement of mucus by cilia | Tears<br>Nasal cilia |
| Chemical | Fatty acids | Low pH<br>Enzymes (pepsin) | Pulmonary surfactant | Enzymes in tears and saliva (lysozyme) |
| | β-defensins<br>Lamellar bodies<br>Cathelicidin | α-defensins (cryptdins)<br>RegIII (lecticidins)<br>Cathelicidin | α-defensins<br>Cathelicidin | Histatins<br>β-defensins |
| Microbiological | Normal microbiota | | | |

Figure 2.6 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

conceito importante!

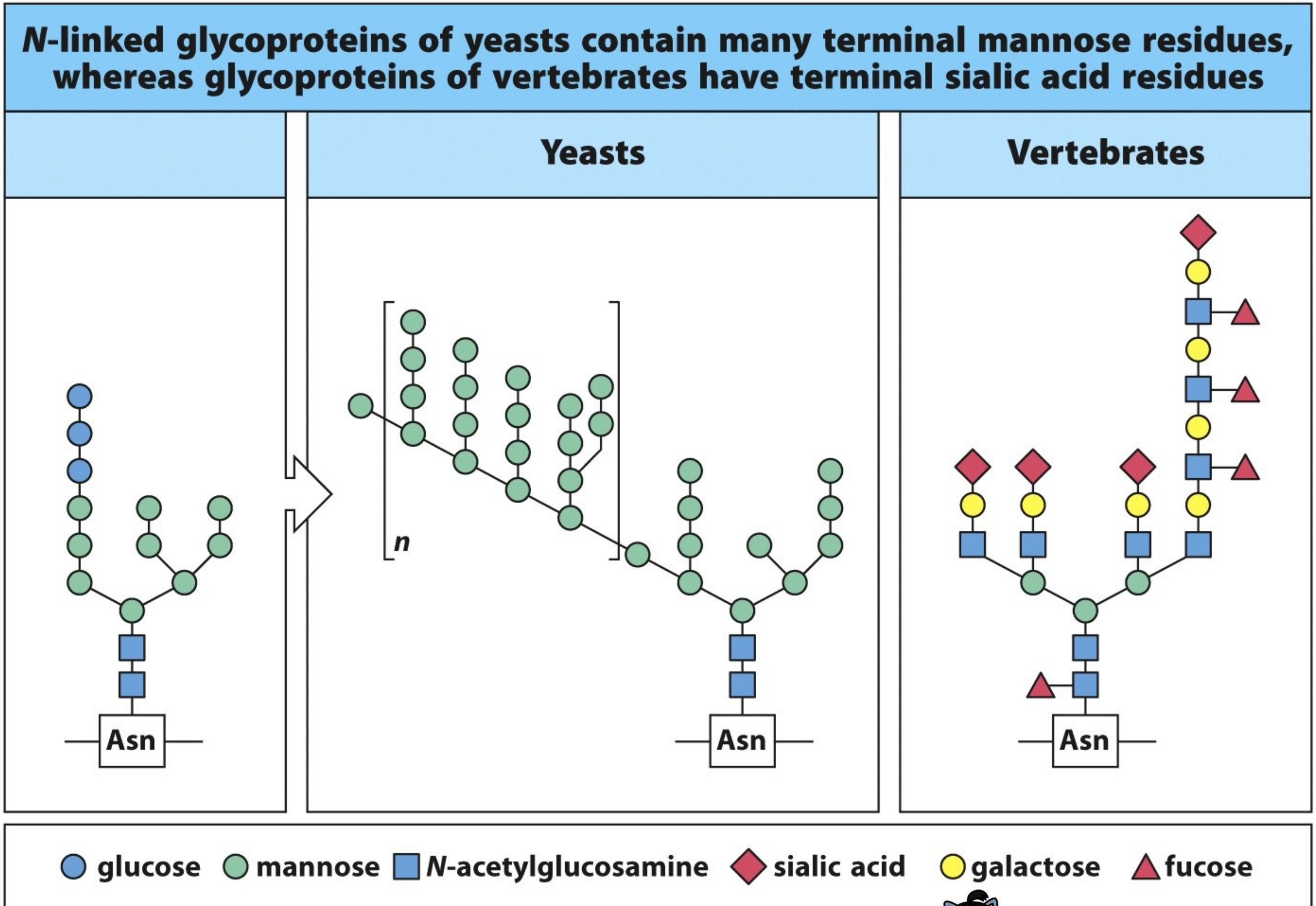



Figure 2.14 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

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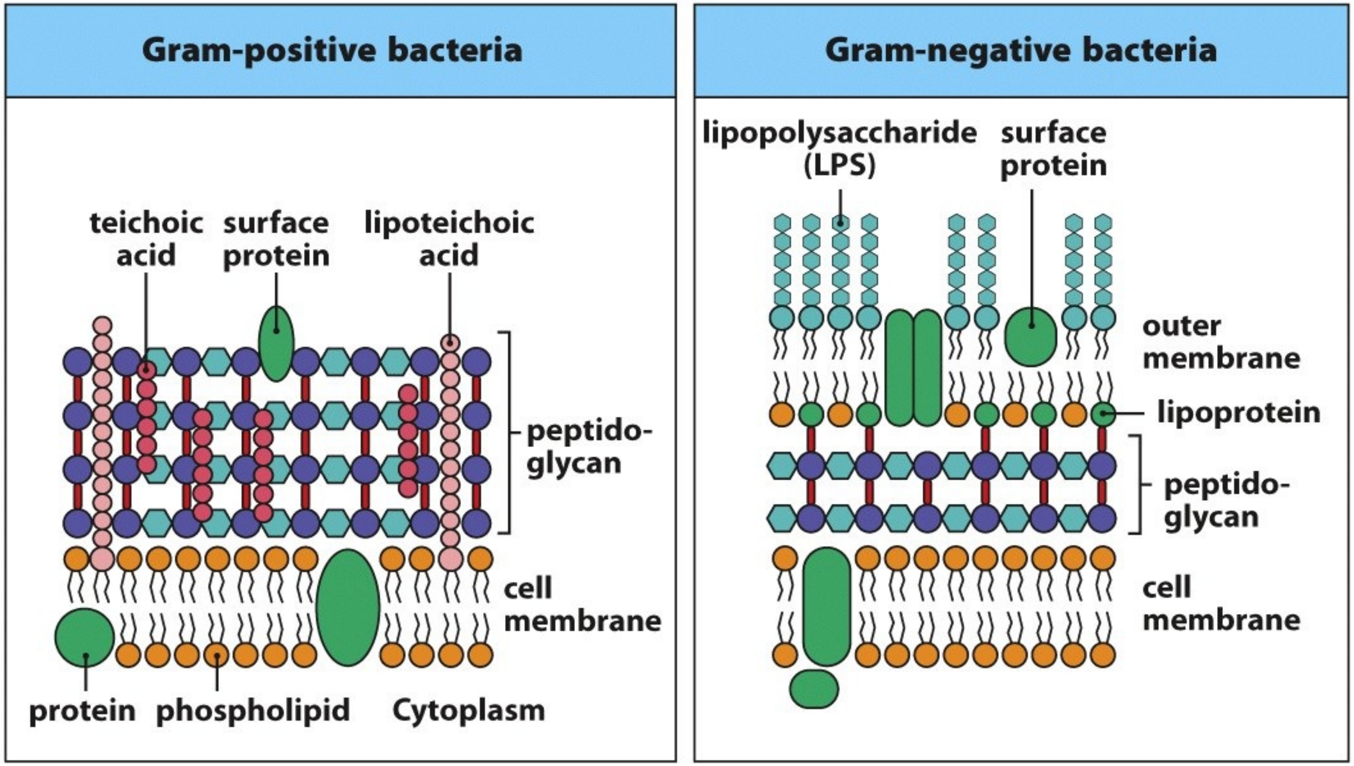


PAMPS


conceito importante!

| Receptor characteristic | Innate immunity | Adaptive immunity |
| --- | --- | --- |
| Specificity inherited in the genome | Yes | No |
| Expressed by all cells of a particular type (e.g. macrophages) | Yes | No |
| Triggers immediate response | Yes | No |
| Recognizes broad classes of pathogens | Yes | No |
| Interacts with a range of molecular structures of a given type | Yes | No |
| Encoded in multiple gene segments | No | Yes |
| Requires gene rearrangement | No | Yes |
| Clonal distribution | No | Yes |
| Able to discriminate between even closely related molecular structures | No | Yes |

Figure 3.1 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

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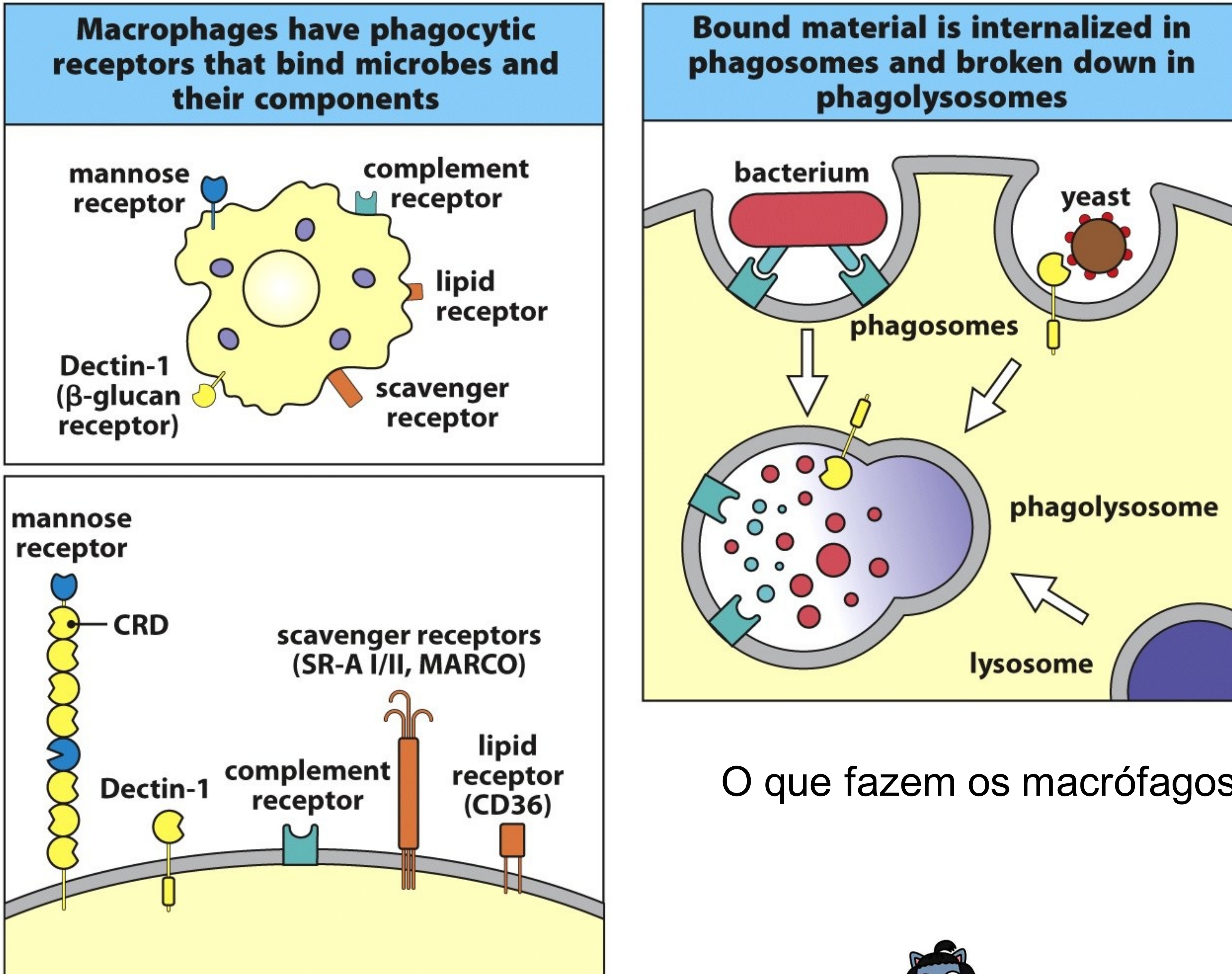



Figure 3.2 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

O que fazem os macrófagos?

conceito importante!

<table>
<tr><th colspan="3">Antimicrobial mechanisms of phagocytes</th></tr>
<tr><th>Class of mechanism</th><th>Macrophage products</th><th>Neutrophil products</th></tr>
<tr><td>Acidification</td><td colspan="2">pH=~3.5–4.0, bacteriostatic or bactericidal</td></tr>
<tr><td>Toxic oxygen-derived products</td><td colspan="2">Superoxide $O_2^-$, hydrogen peroxide $H_2O_2$, singlet oxygen $^1O_2^{\bullet}$, hydroxyl radical $^{\bullet}OH$, hypohalite $OCl^-$</td></tr>
<tr><td>Toxic nitrogen oxides</td><td colspan="2">Nitric oxide NO</td></tr>
<tr><td>Antimicrobial peptides</td><td>Cathelicidin, macrophage elastase-derived peptide</td><td>α-Defensins (HNP1–4), β-defensin HBD4, cathelicidin, azurocidin, bacterial permeability inducing protein (BPI), lactoferricin</td></tr>
<tr><td>Enzymes</td><td colspan="2">Lysozyme: digests cell walls of some Gram-positive bacteria<br>Acid hydrolases (e.g. elastase and other proteases): break down ingested microbes</td></tr>
<tr><td>Competitors</td><td></td><td>Lactoferrin (sequesters $Fe^{2+}$), vitamin $B_{12}$-binding protein</td></tr>
</table>

Figure 3.4 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

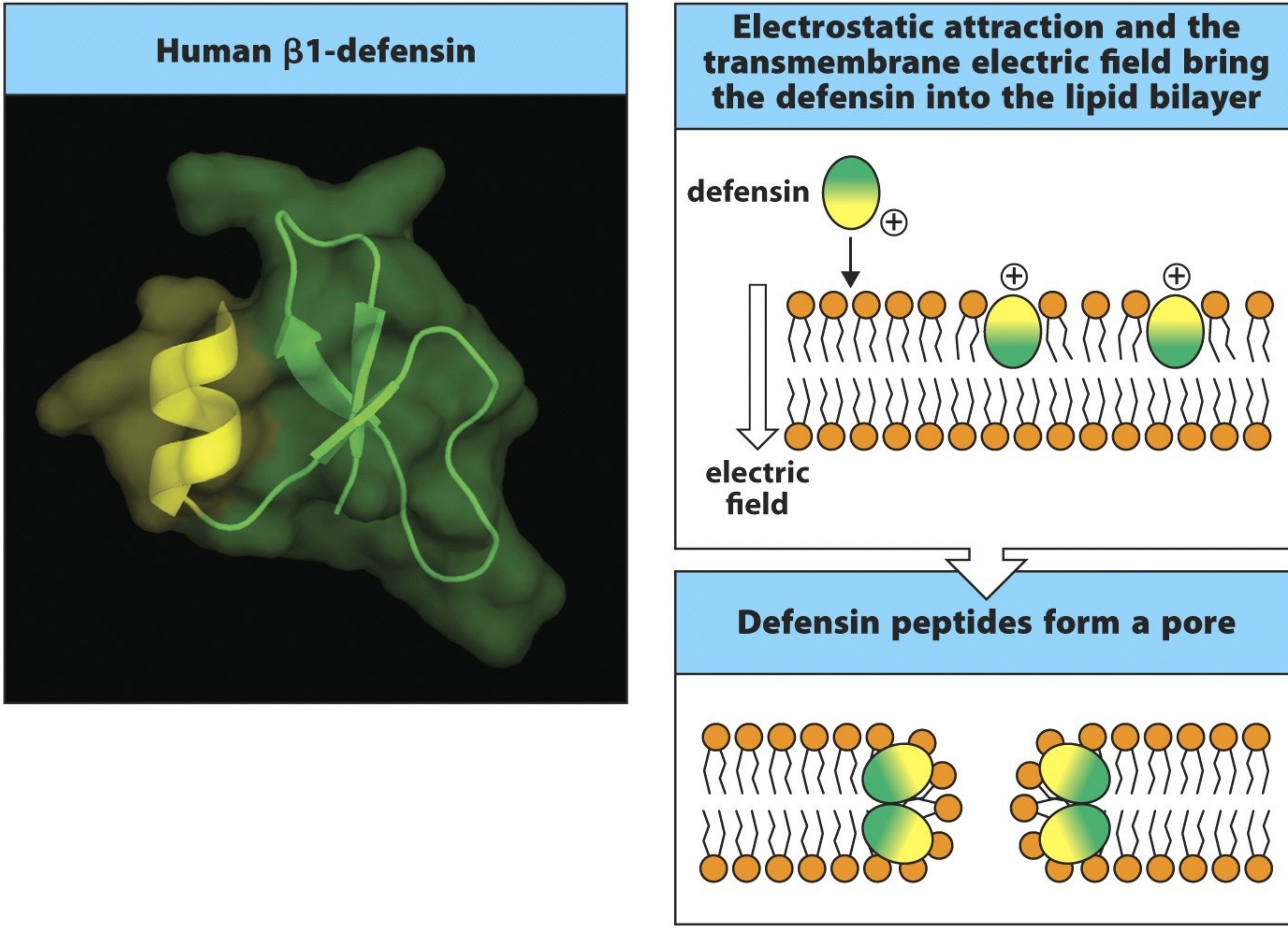



Figure 2.8 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

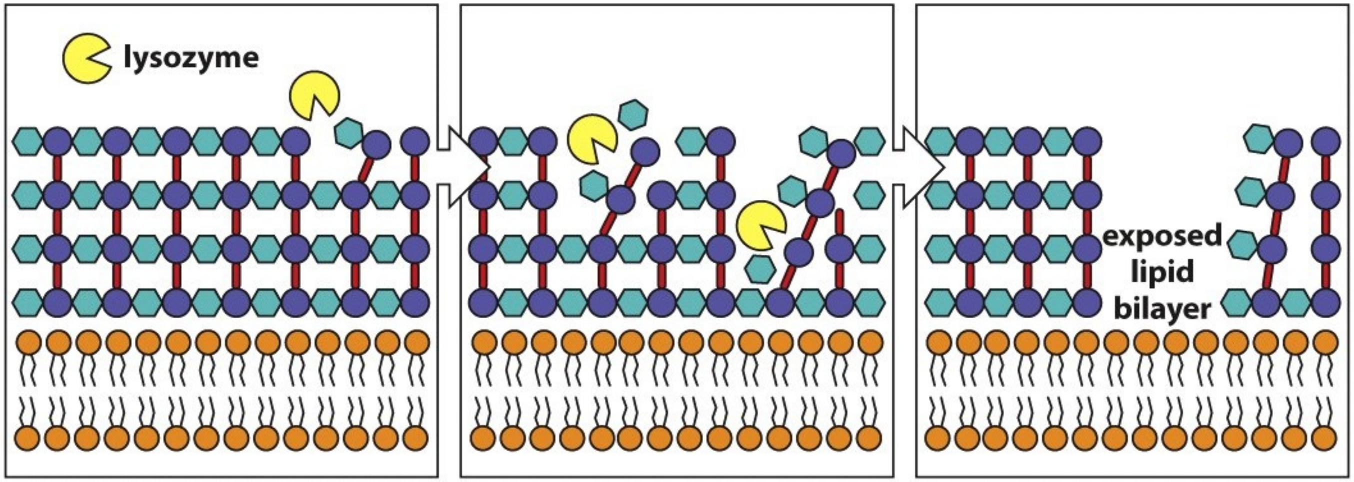



Figure 2.7 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

# Atenção aos Macrófagos:
# diferentes nomes em diferentes tecidos

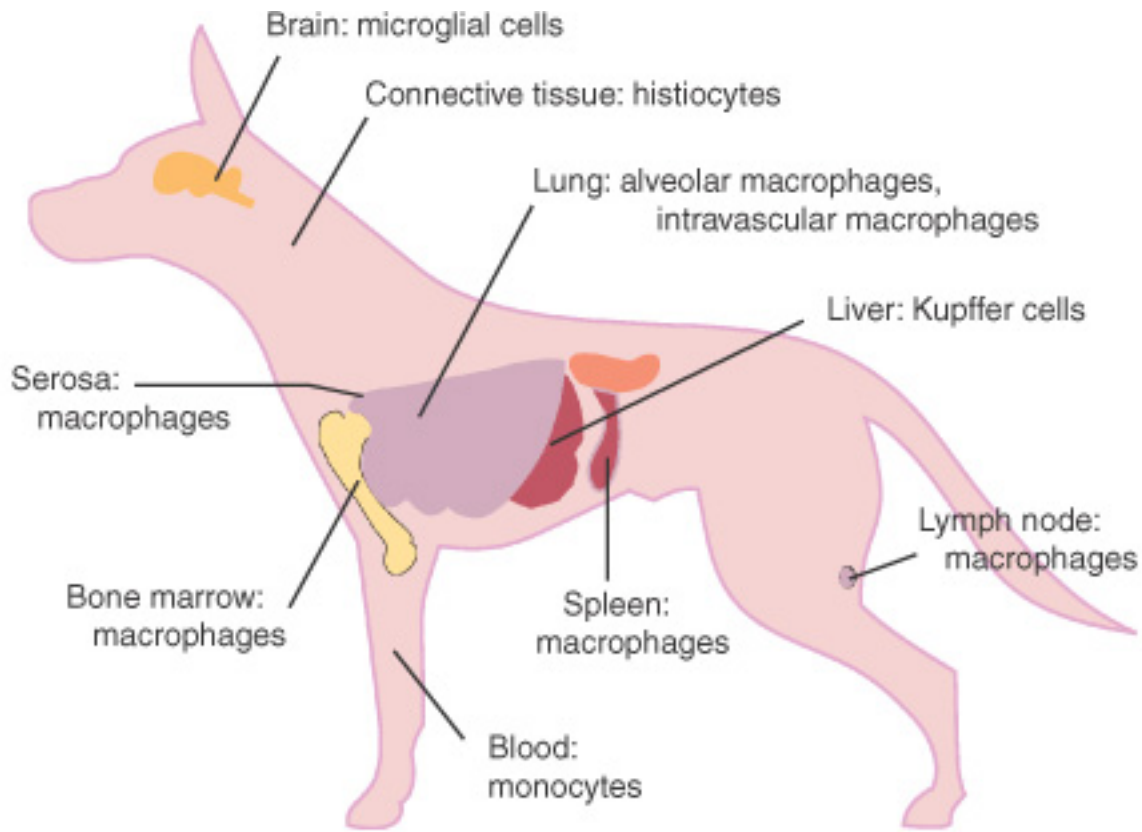



**FIGURE 2-5** The location of the cells of the mononuclear phagocyte system.

O que fazem os neutrófilos?

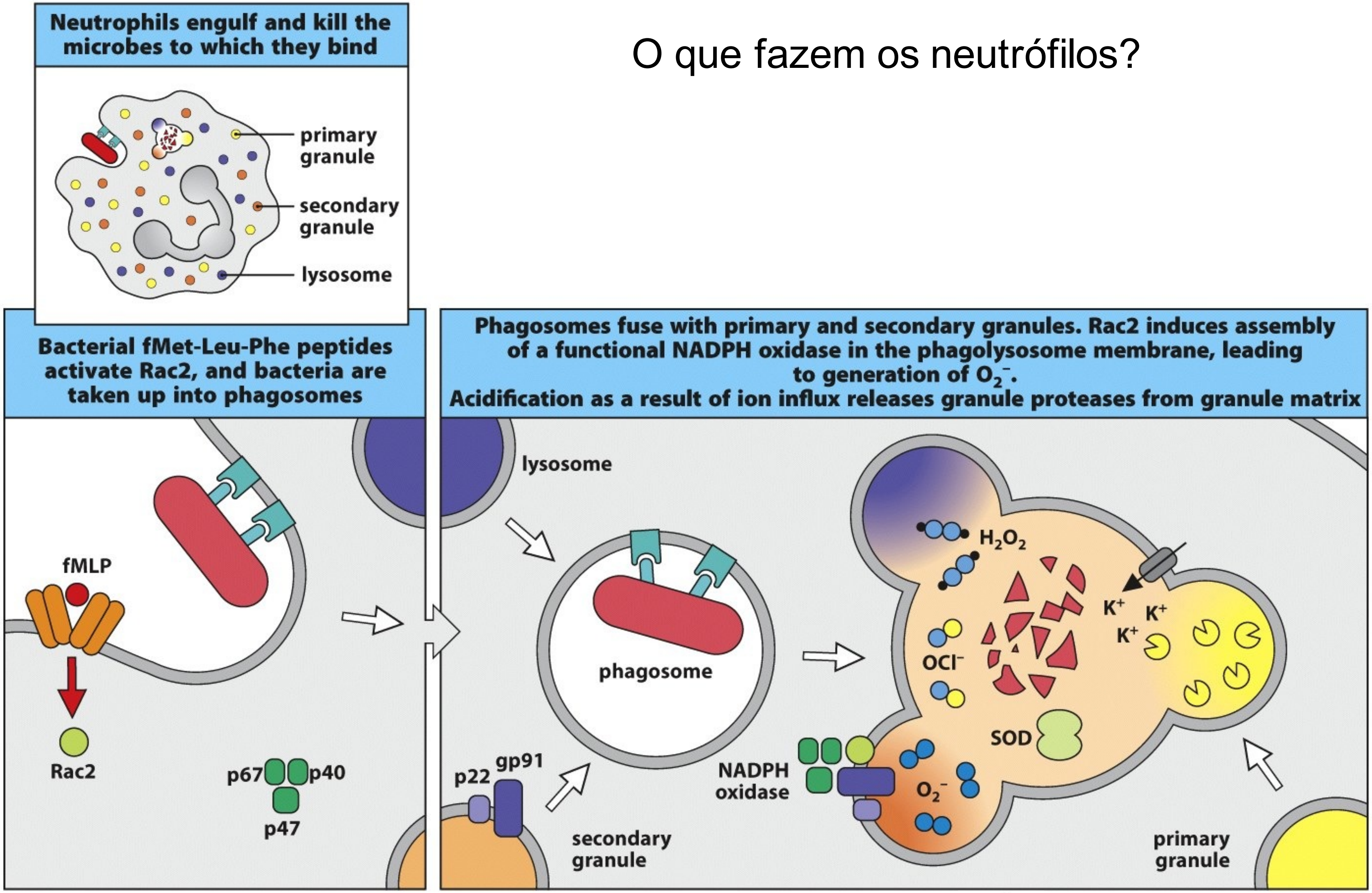
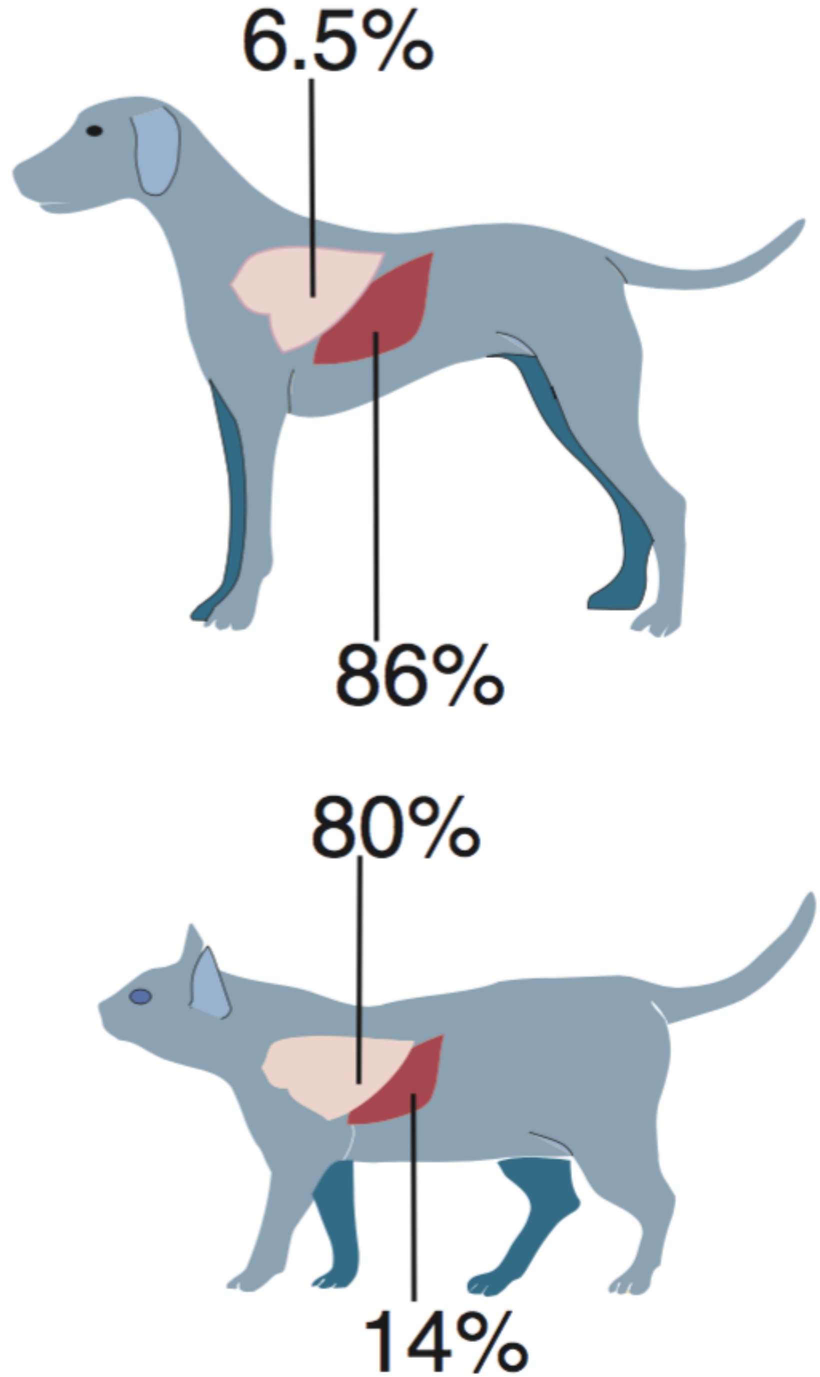



Figure 3.5 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

conceito importante!

**TABLE 6.1 Sites of Clearance of Particles From the Blood in Domestic Mammals**

| | LOCALIZATION (%) | |
|---|---|---|
| **Species** | **Lung** | **Liver/Spleen** |
| Calf | 93 | 6 |
| Sheep | 94 | 6 |
| Dog | 6.5 | 80 |
| Cat | 86 | 14 |
| Rabbit | 0.6 | 83 |
| Guinea pig | 1.5 | 82 |
| Rat | 0.5 | 97 |
| Mouse | 1.0 | 94 |



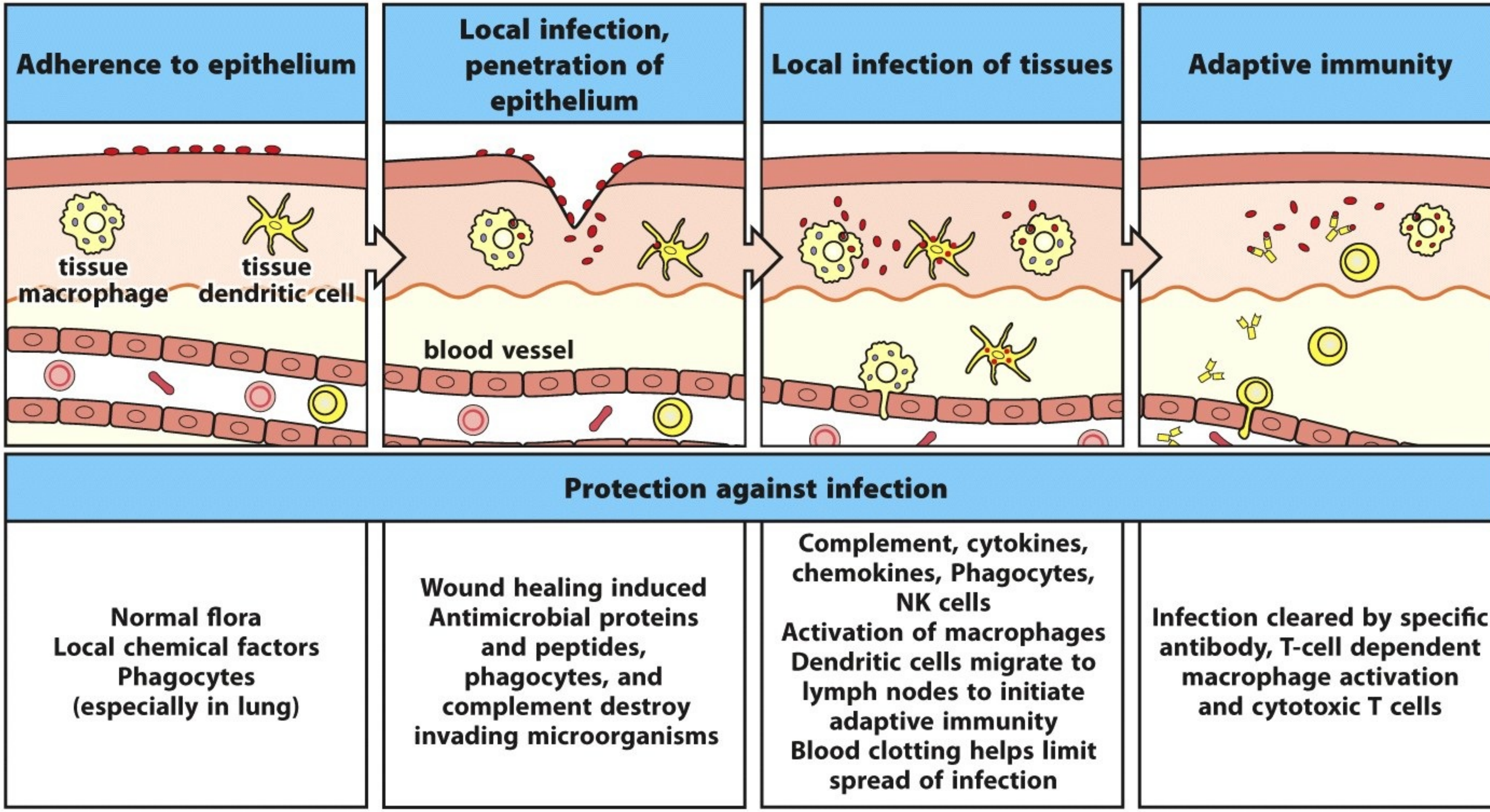



Figure 2.5 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

conceito importante!

Como os leucócitos chegam na lesão?

QUIMIOTAXIA

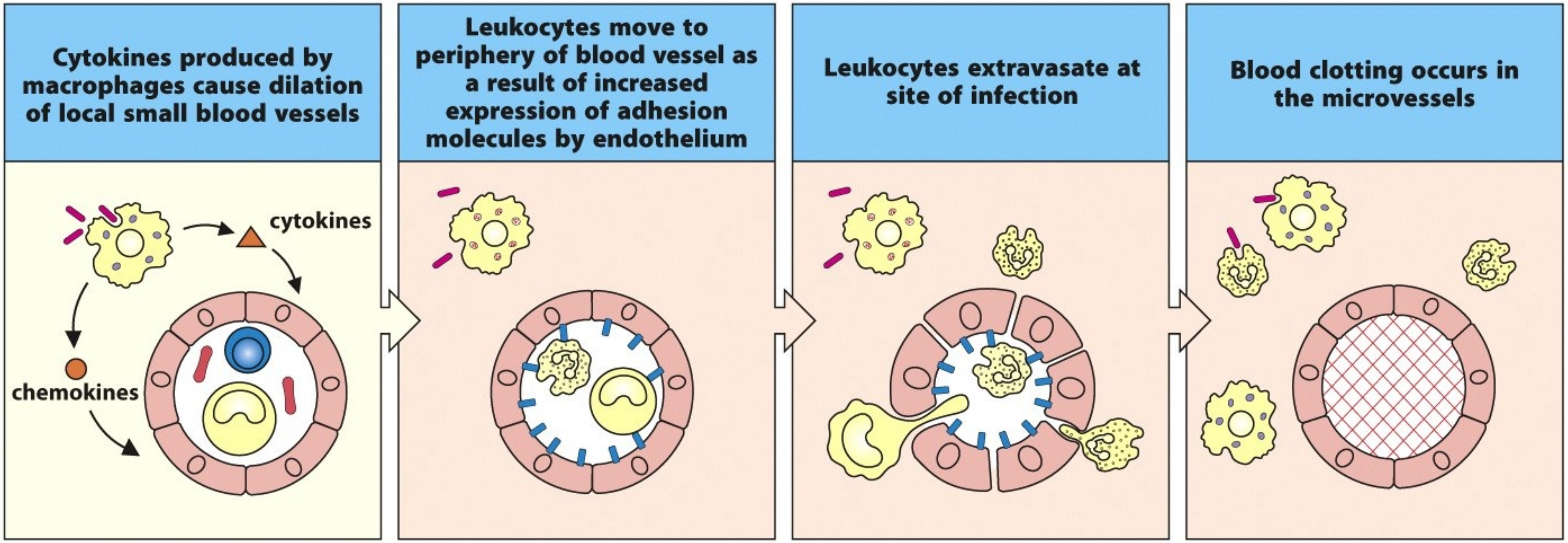



Figure 3.6 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

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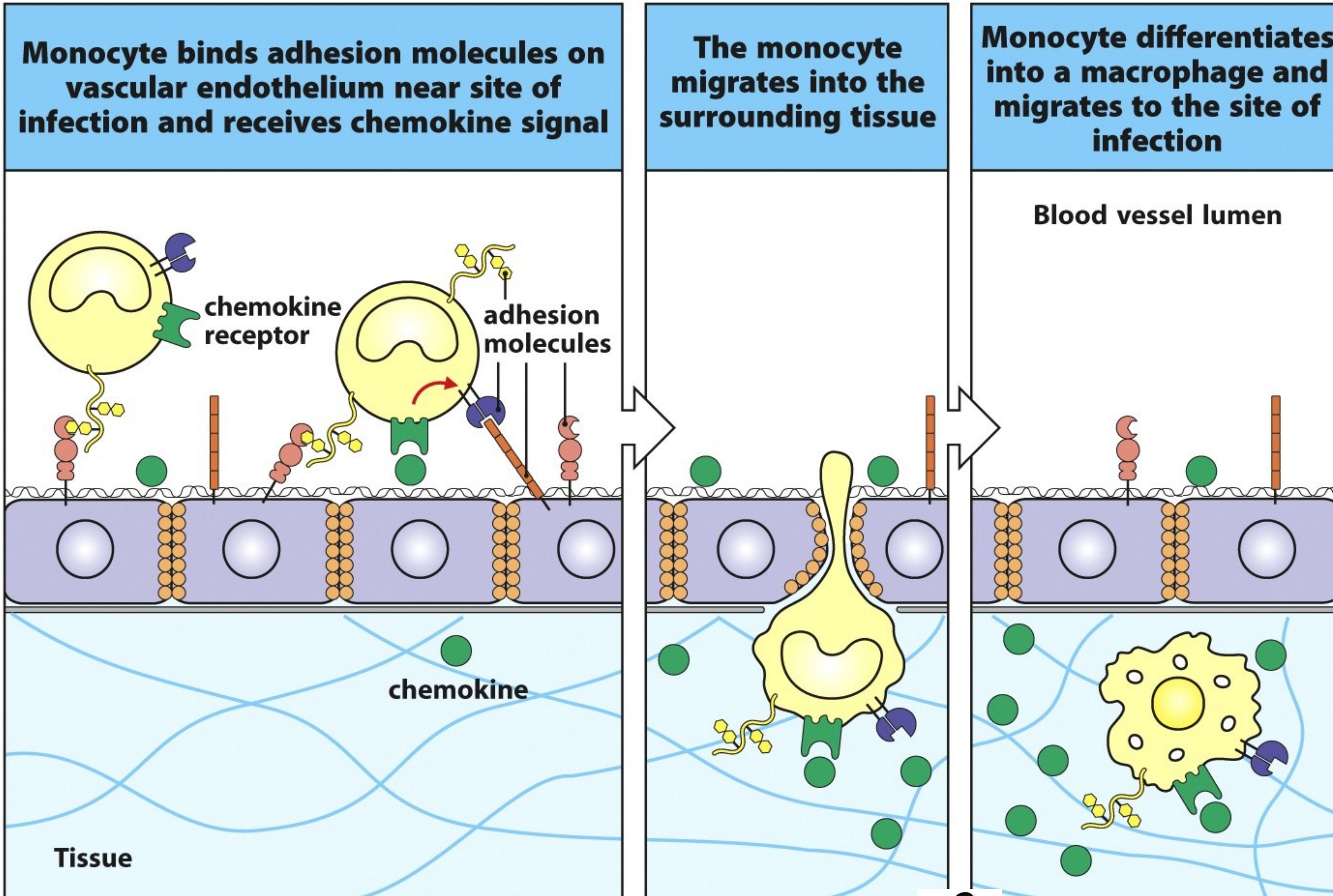
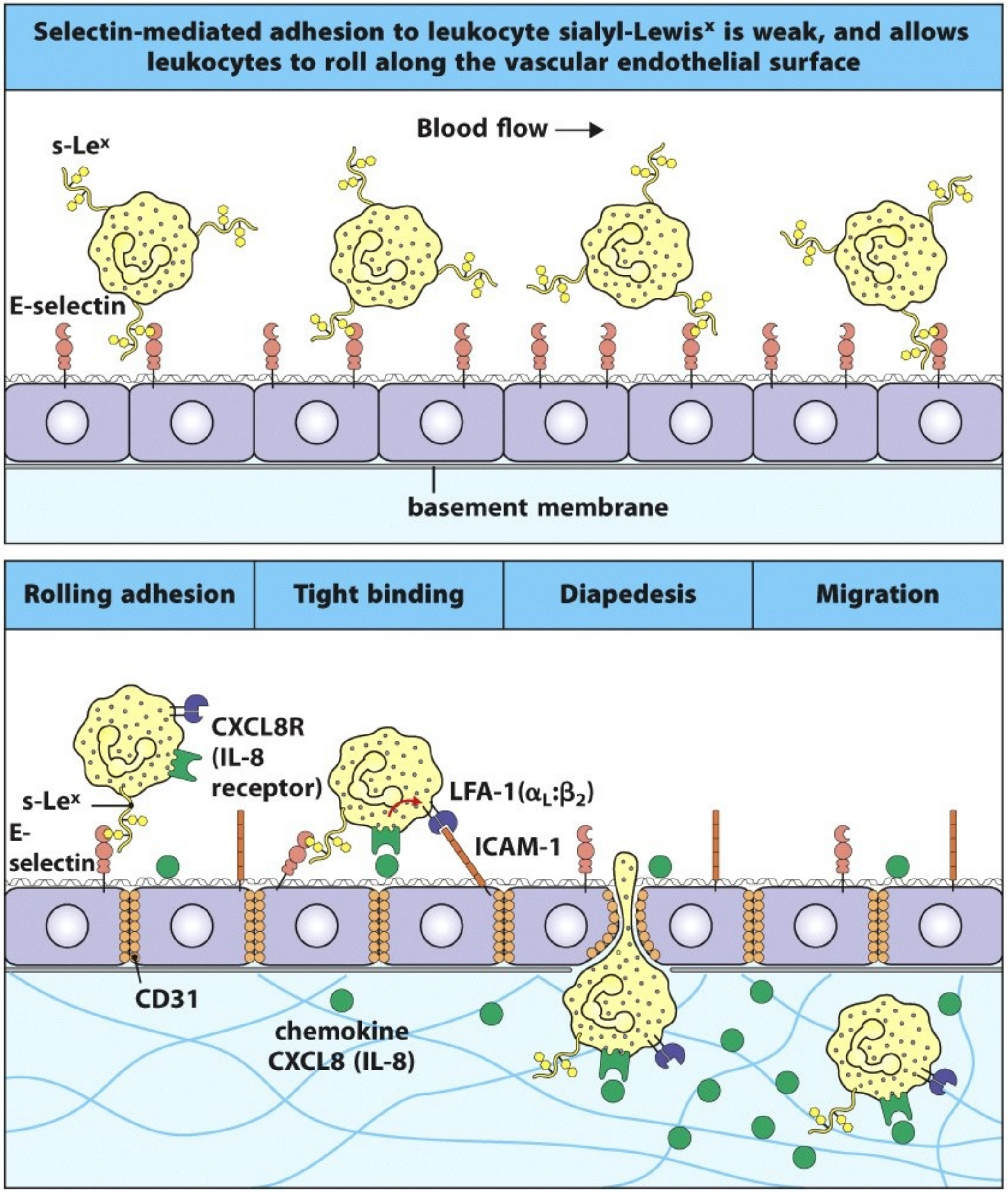



Figure 3.7 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

conceito importante!



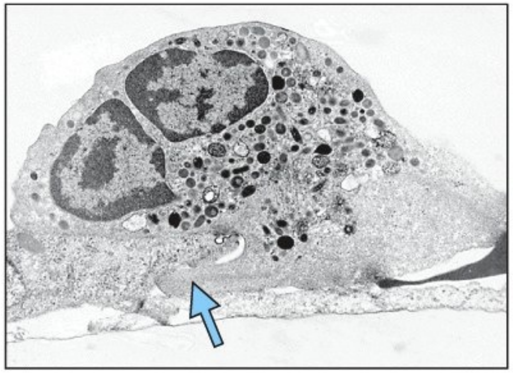



Figure 3.25 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

| Class | Chemokine | Produced by | Receptors | Cells attracted | Major effects |
|---|---|---|---|---|---|
| CXC | CXCL8 (IL-8) | Monocytes<br>Macrophages<br>Fibroblasts<br>Epithelial cells<br>Endothelial cells | CXCR1<br>CXCR2 | Neutrophils<br>Naive T cells | Mobilizes, activates and degranulates neutrophils<br>Angiogenesis |
| | CXCL7 (PBP, β-TG, NAP-2) | Platelets | CXCR2 | Neutrophils | Activates neutrophils<br>Clot resorption<br>Angiogenesis |
| | CXCL1 (GROα)<br>CXCL2 (GROβ)<br>CXCL3 (GROγ) | Monocytes<br>Fibroblasts<br>Endothelium | CXCR2 | Neutrophils<br>Naive T cells<br>Fibroblasts | Activates neutrophils<br>Fibroplasia<br>Angiogenesis |
| CC | CCL3 (MIP-1α) | Monocytes<br>T cells<br>Mast cells<br>Fibroblasts | CCR1, 3, 5 | Monocytes<br>NK and T cells<br>Basophils<br>Dendritic cells | Competes with HIV-1<br>Antiviral defense<br>Promotes $T_H1$ immunity |
| | CCL4 (MIP-1β) | Monocytes<br>Macrophages<br>Neutrophils<br>Endothelium | CCR1, 3, 5 | Monocytes<br>NK and T cells<br>Dendritic cells | Competes with HIV-1 |
| | CCL2 (MCP-1) | Monocytes<br>Macrophages<br>Fibroblasts<br>Keratinocytes | CCR2B | Monocytes<br>NK and T cells<br>Basophils<br>Dendritic cells | Activates macrophages<br>Basophil histamine release<br>Promotes $T_H2$ immunity |
| | CCL5 (RANTES) | T cells<br>Endothelium<br>Platelets | CCR1, 3, 5 | Monocytes<br>NK and T cells<br>Basophils<br>Eosinophils<br>Dendritic cells | Degranulates basophils<br>Activates T cells<br>Chronic inflammation |
| CXXXC ($CX_3C$) | CX3CL1 (Fractalkine) | Monocytes<br>Endothelium<br>Microglial cells | $CX_3CR1$ | Monocytes<br>T cells | Leukocyte–endothelial adhesion<br>Brain inflammation |

Figure 3.22 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

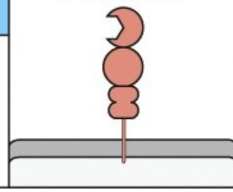
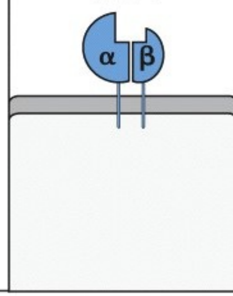
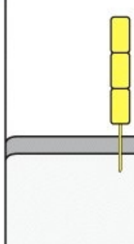
| Family | Function | Example | Name | Tissue distribution | Ligand |
|---|---|---|---|---|---|
| **Selectins** | Bind carbohydrates. Initiate leukocyte–endothelial interaction | P-selectin  | P-selectin (PADGEM, CD62P) | Activated endothelium and platelets | PSGL-1, sialyl-Lewis$^{x}$ |
| | | | E-selectin (ELAM-1, CD62E) | Activated endothelium | Sialyl-Lewis$^{x}$ |
| **Integrins** | Bind to cell-adhesion molecules and extracellular matrix. Strong adhesion | LFA-1  | $\alpha_L$:$\beta_2$ (LFA-1, CD11a:CD18) | Monocytes, T cells, macrophages, neutrophils, dendritic cells, NK cells | ICAMs |
| | | | $\alpha_M$:$\beta_2$ (CR3, Mac-1, CD11b:CD18) | Neutrophils, monocytes, macrophages, NK cells | ICAM-1, iC3b, fibrinogen |
| | | | $\alpha_X$:$\beta_2$ (CR4, p150.95, CD11c:CD18) | Dendritic cells, macrophages, neutrophils, NK cells | iC3b |
| | | | $\alpha_5$:$\beta_1$ (VLA-5, CD49d:CD29) | Monocytes, macrophages | Fibronectin |
| **Immunoglobulin superfamily** | Various roles in cell adhesion. Ligand for integrins |  ICAM-1 | ICAM-1 (CD54) | Activated endothelium, activated leukocytes | LFA-1, Mac1 |
| | | | ICAM-2 (CD102) | Resting endothelium, dendritic cells | LFA-1 |
| | | | VCAM-1 (CD106) | Activated endothelium | VLA-4 |
| | | | PECAM (CD31) | Activated leukocytes, endothelial cell–cell junctions | CD31 |

Figure 3.23 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

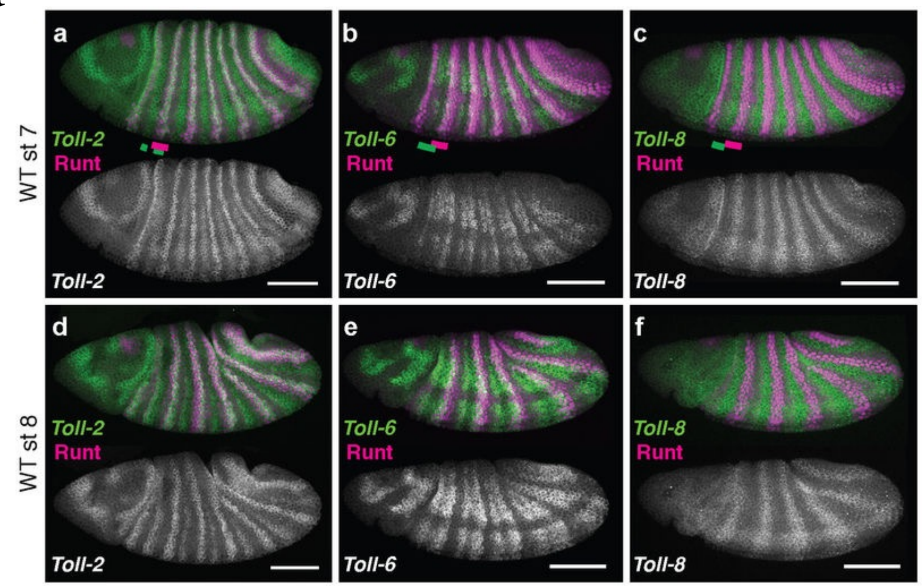
Charles Janeway Jr.
1943 - 2003

Predição teórica da existência de receptores que reconhecessem padrões moleculares para ativação do sistema inato


Christiane Nüsslein-Volhard

Envolvimento dos receptores Toll no **desenvolvimento** de invertebrados

The Nobel Prize in Physiology or Medicine 1995



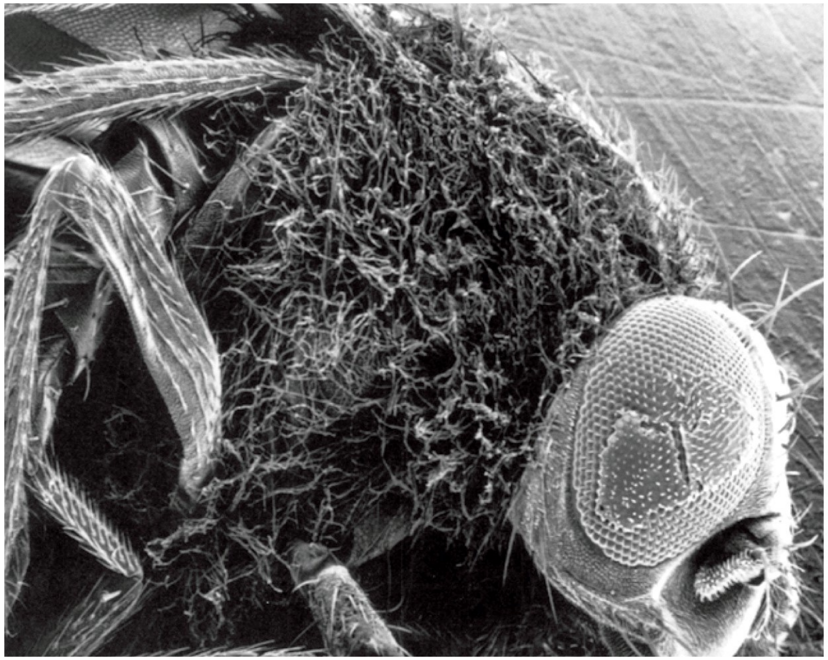


Figure 3.8 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

Envolvimento dos receptores Toll na **imunidade** de invertebrados

Jules Hoffmann

Physiology or

nature
International journal of science

Correspondence | Published: 09 November 2011

# Nobels: Toll pioneers deserve recognition

James P. Allison, Christophe Benoist & Alexander V. Chervonsky

*Nature* **479**, 178 (10 November 2011) | Download Citation

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3

# Nobel Prize for Immunologists Provokes Yet Another Debate

By Martin Enserink, John Travis | Dec. 16, 2011 , 2:36 PM

Lab Times online
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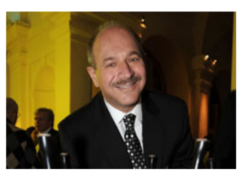
## Nobel Prize Critiques: Bruce Beutler

*(February 14th, 2012)* The 2011 Nobel Prize for Medicine has been dogged by controversy. Both Jules Hoffmann and Bruce Beutler have been accused of unfairly promoting their own contributions to the discoveries. Here, Jeremy Garwood investigates the "case" of Bruce Beutler and rival claims.

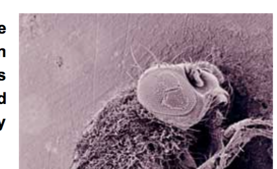


Lab Times online
News Magazine for the European Life Sciences



## A Nobel Prize not Immune from Error?

*(December 12th, 2011)* From 1993-1997, Bruno Lemaitre made ground-breaking discoveries on innate immunity in insects. Yet, although he clearly did this research, the lab's administrative manager, Jules Hoffmann, has now received the Nobel Prize. A case of mistaken identity? Jeremy Garwood looks at the evidence.



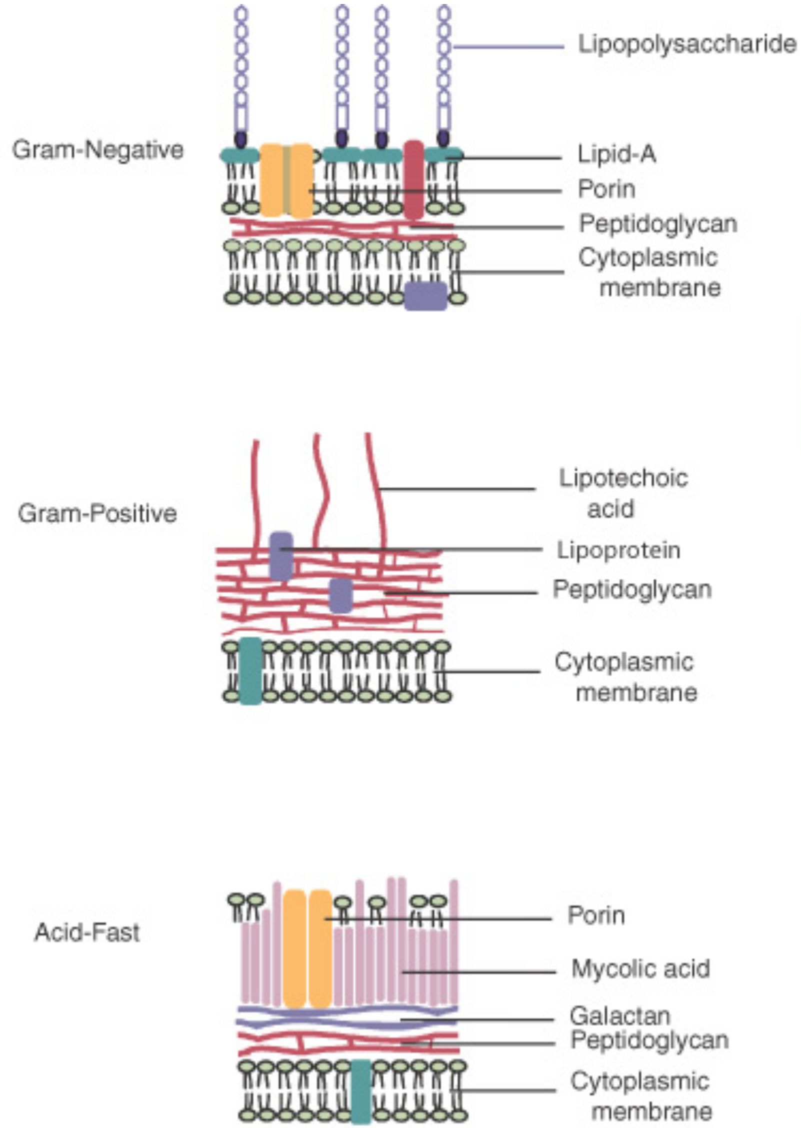



**FIGURE 2-2** The major structural features of the cell walls of Gram-negative, Gram-positive, and acid-fast bacteria. These conserved structural molecules serve as pathogen-associated molecular patterns and can bind to pattern-recognition receptors such as the toll-like receptors.



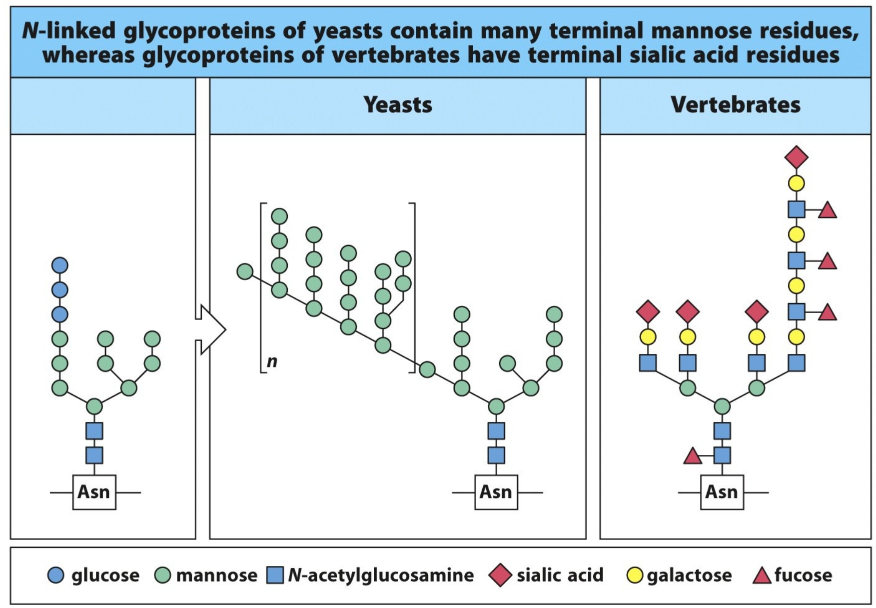
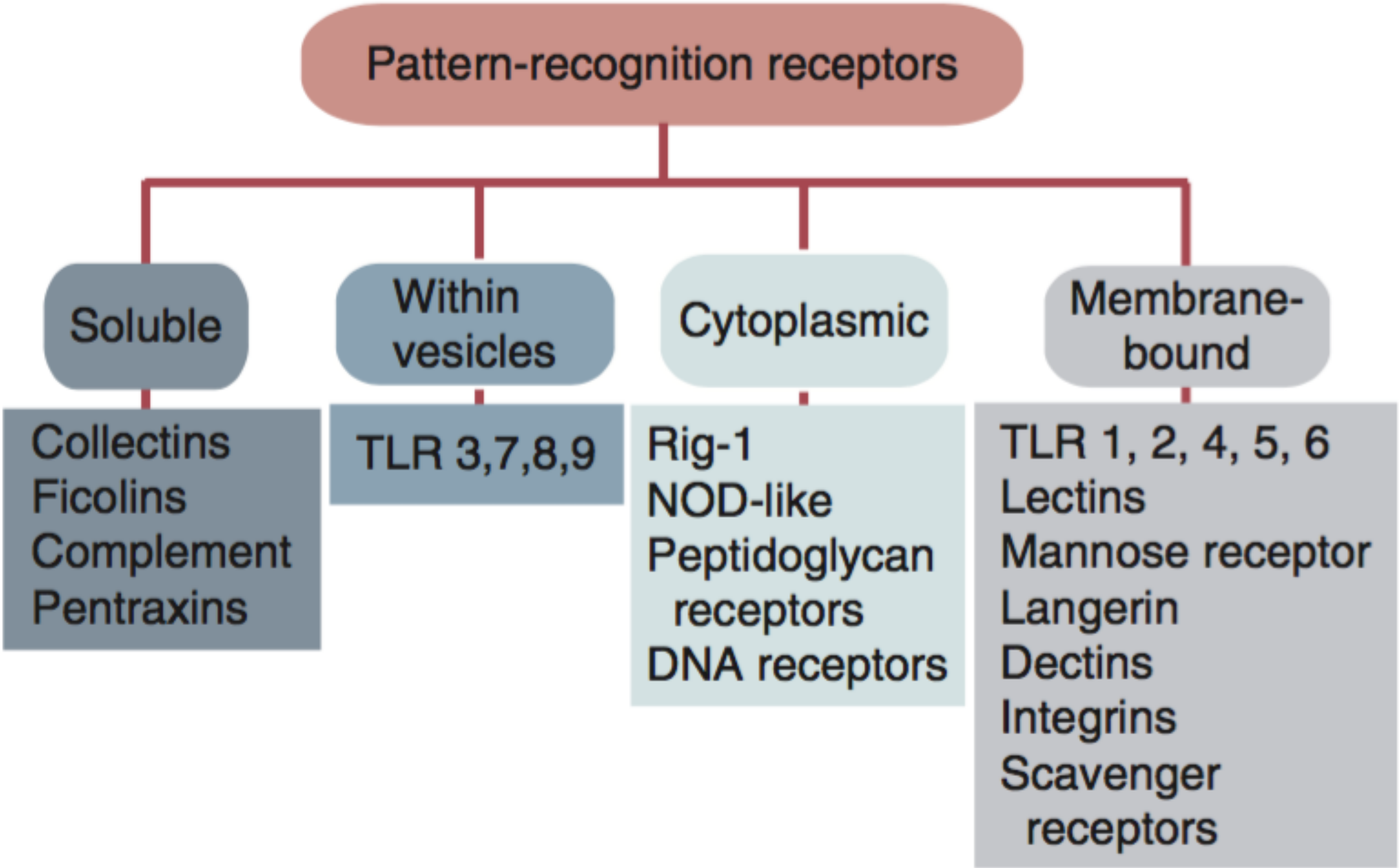



Figure 2.14 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

# Receptores de reconhecimento de padrão (PRR)



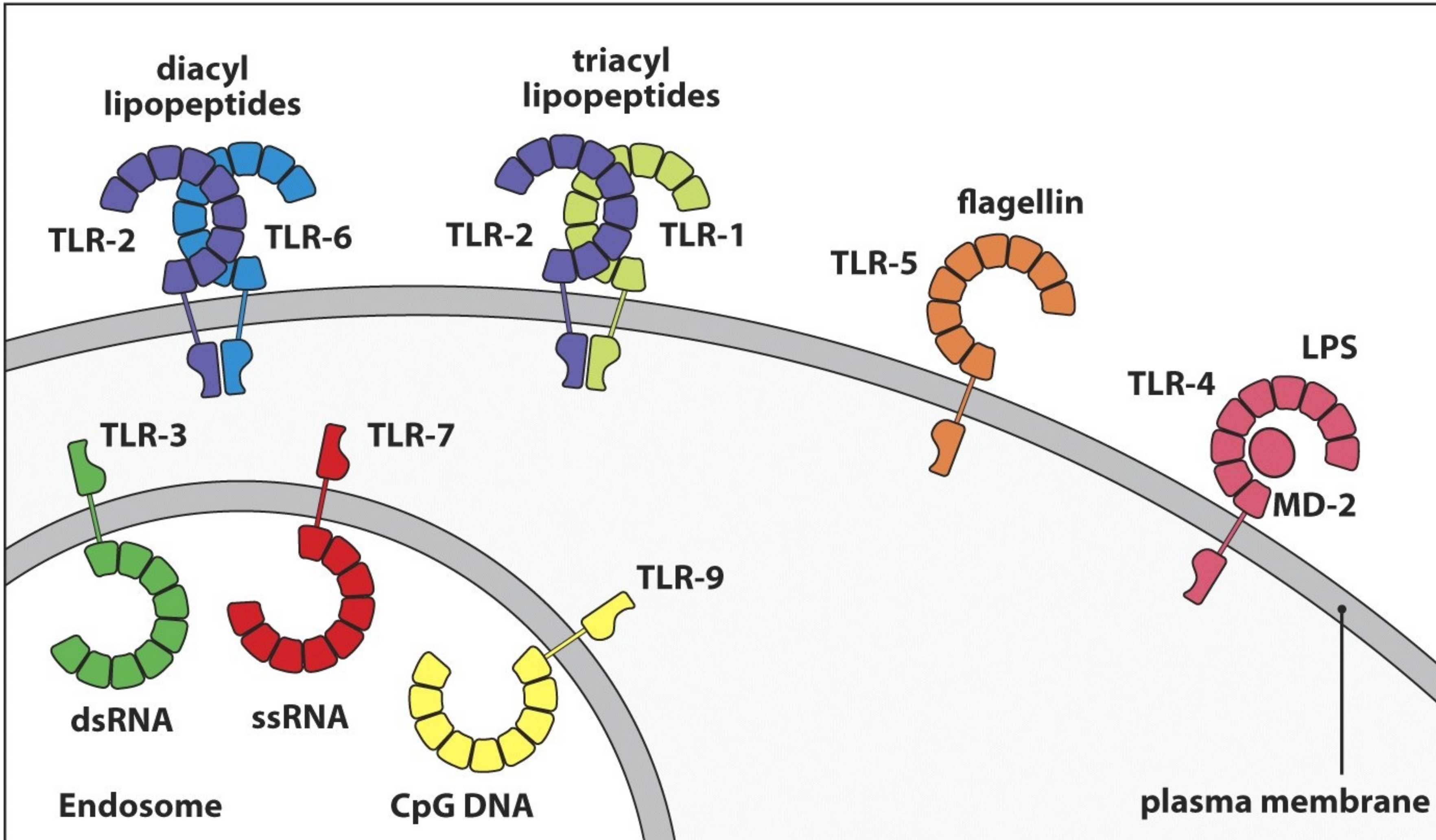



Figure 3.10 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

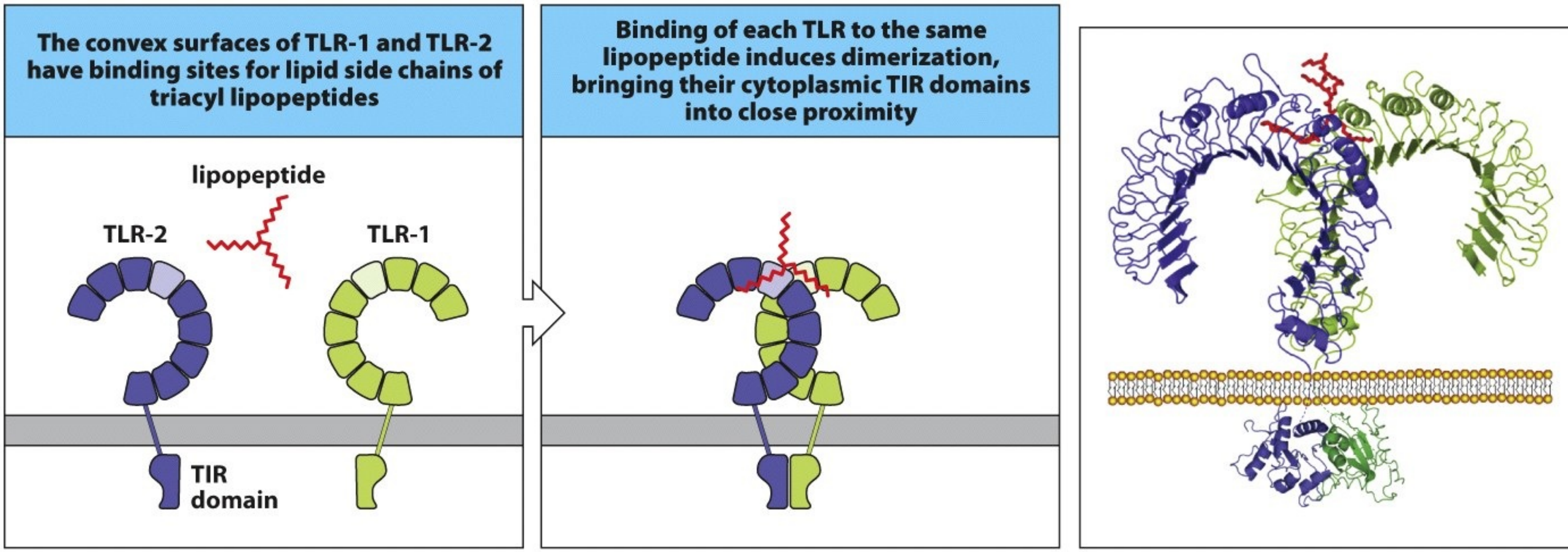



Figure 3.11 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

| Innate immune recognition by mammalian Toll-like receptors | | |
|---|---|---|
| **Toll-like receptor** | **Ligand** | **Cellular distribution** |
| TLR-1:TLR-2 heterodimer<br>TLR-2:TLR-6 heterodimer | Lipomannans (mycobacteria)<br>Lipoproteins (diacyl lipopeptides; triacyl lipopeptides)<br>Lipoteichoic acids (Gram-positive bacteria)<br>Cell-wall β-glucans (bacteria and fungi)<br>Zymosan (fungi) | Monocytes, dendritic cells, mast cells, eosinophils, basophils |
| TLR-3 | Double-stranded RNA (viruses) | NK cells |
| TLR-4 (plus MD-2 and CD14) | LPS (Gram-negative bacteria)<br>Lipoteichoic acids (Gram-positive bacteria) | Macrophages, dendritic cells, mast cells, eosinophils |
| TLR-5 | Flagellin (bacteria) | Intestinal epithelium |
| TLR-7 | Single-stranded RNA (viruses) | Plasmacytoid dendritic cells, NK cells, eosinophils, B cells |
| TLR-8 | Single-stranded RNA (viruses) | NK cells |
| TLR-9 | DNA with unmethylated CpG (bacteria and herpesviruses) | Plasmacytoid dendritic cells, eosinophils, B cells, basophils |
| TLR-10 | Unknown | Plasmacytoid dendritic cells, eosinophils, B cells, basophils |
| TLR-11 (mouse only) | Profilin and profilin-like proteins (*Toxoplasma gondii*, uropathogenic bacteria) | Macrophages, dendritic cells, liver, kidney, and bladder epithelial cells |

**Figure 3.9 Janeway's Immunobiology, 8ed.** 

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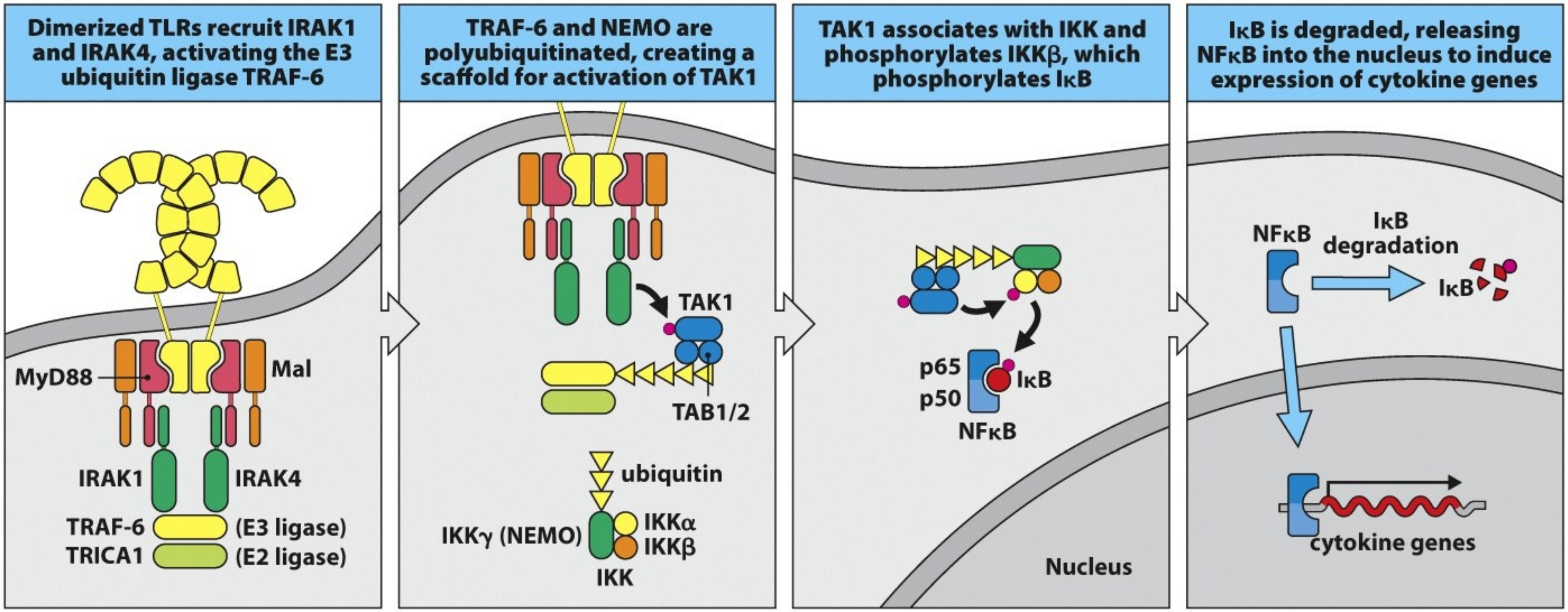



Figure 3.13 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

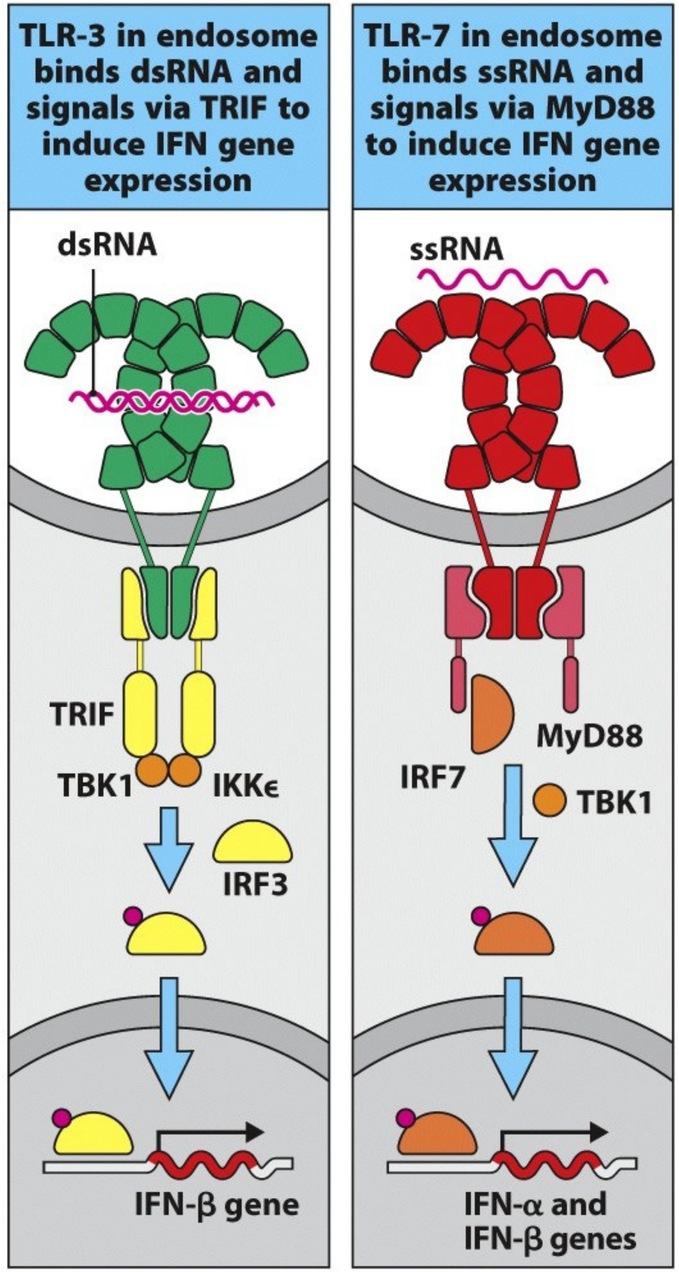



Figure 3.14 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

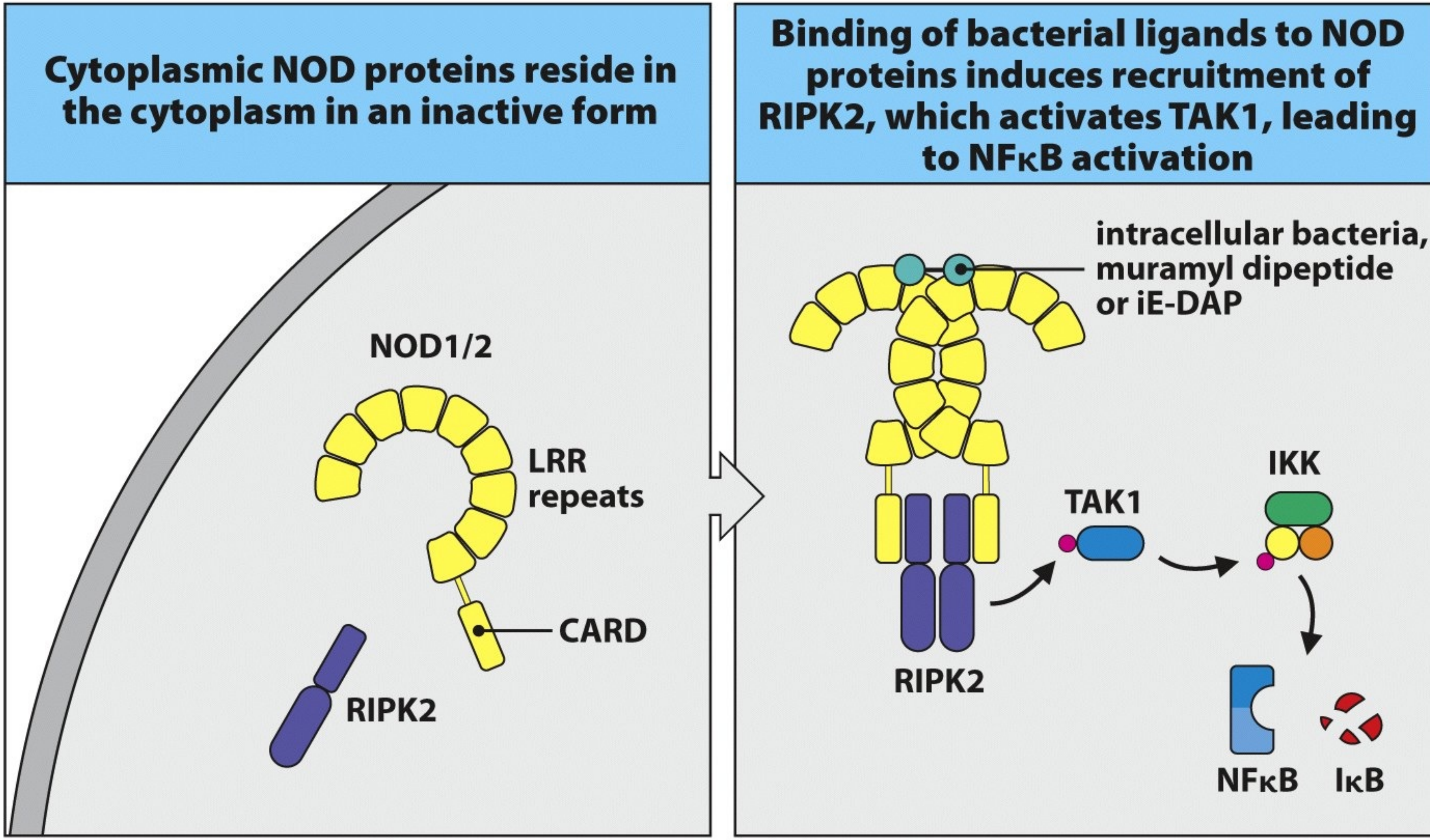
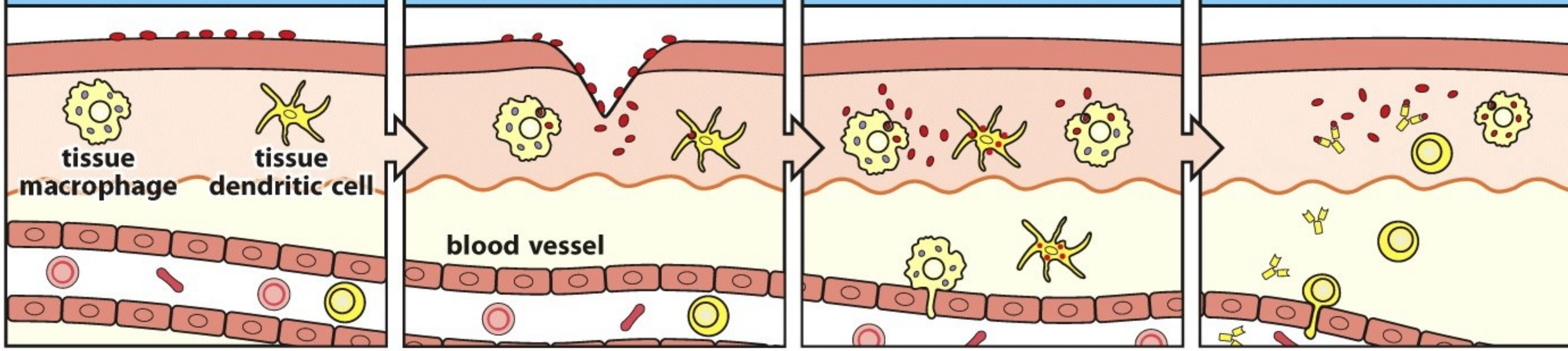



Figure 3.15 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

| Adherence to epithelium | Local infection, penetration of epithelium | Local infection of tissues | Adaptive immunity |
|---|---|---|---|




**Protection against infection**

| Adherence to epithelium | Local infection, penetration of epithelium | Local infection of tissues | Adaptive immunity |
|---|---|---|---|
| Normal flora<br>Local chemical factors<br>Phagocytes<br>(especially in lung) | Wound healing induced<br>Antimicrobial proteins and peptides, phagocytes, and complement destroy invading microorganisms | Complement, cytokines, chemokines, Phagocytes, NK cells<br>Activation of macrophages<br>Dendritic cells migrate to lymph nodes to initiate adaptive immunity<br>Blood clotting helps limit spread of infection | Infection cleared by specific antibody, T-cell dependent macrophage activation and cytotoxic T cells |

Figure 2.5 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

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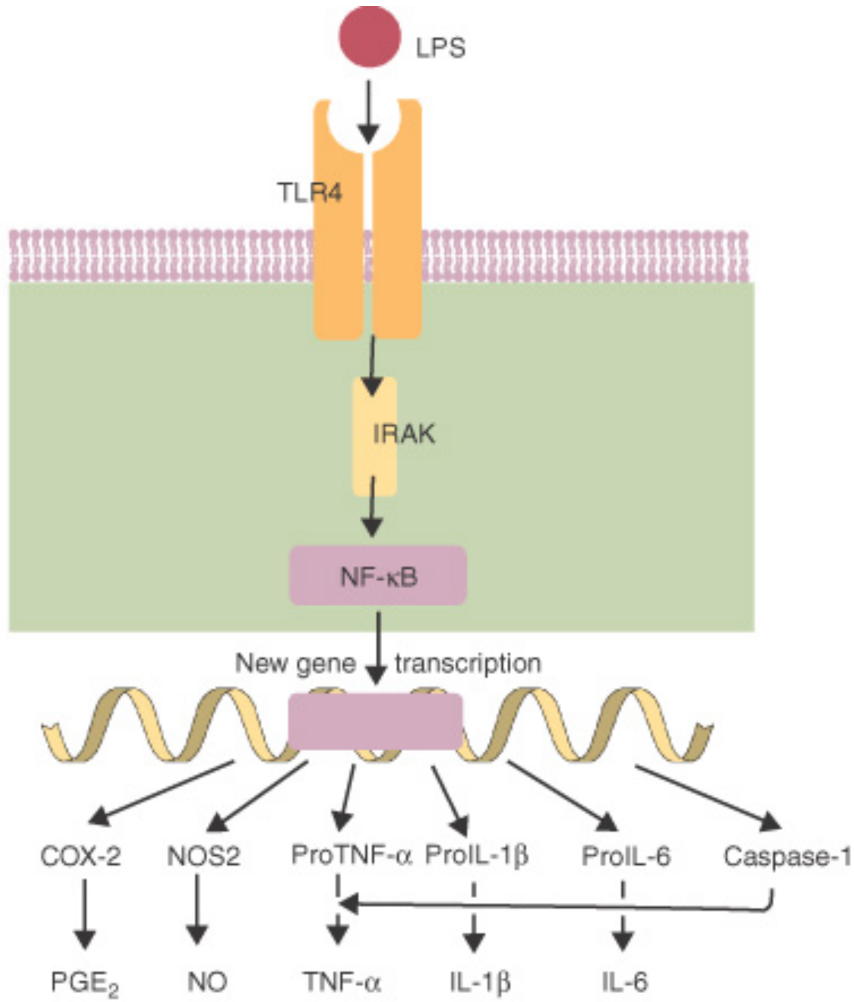


**FIGURE 2-3** Binding of a pathogen-associated molecular pattern such as lipopolysaccharide to a toll-like receptor *(TLR)* leads to generation of a transcription factor called nuclear factor kappa-B *(NF-κB)*. NF-κB turns on the genes for three major cytokines, interleukin-1 *(IL-1)*, IL-6, and tumor necrosis factor-α *(TNF-α)*. It also turns on the genes for nitric oxide synthase 2 *(NOS2)* and cyclooxygenase-2 *(COX-2)*. These two enzymes generate nitric oxide and prostaglandins and leukotrienes, respectively.



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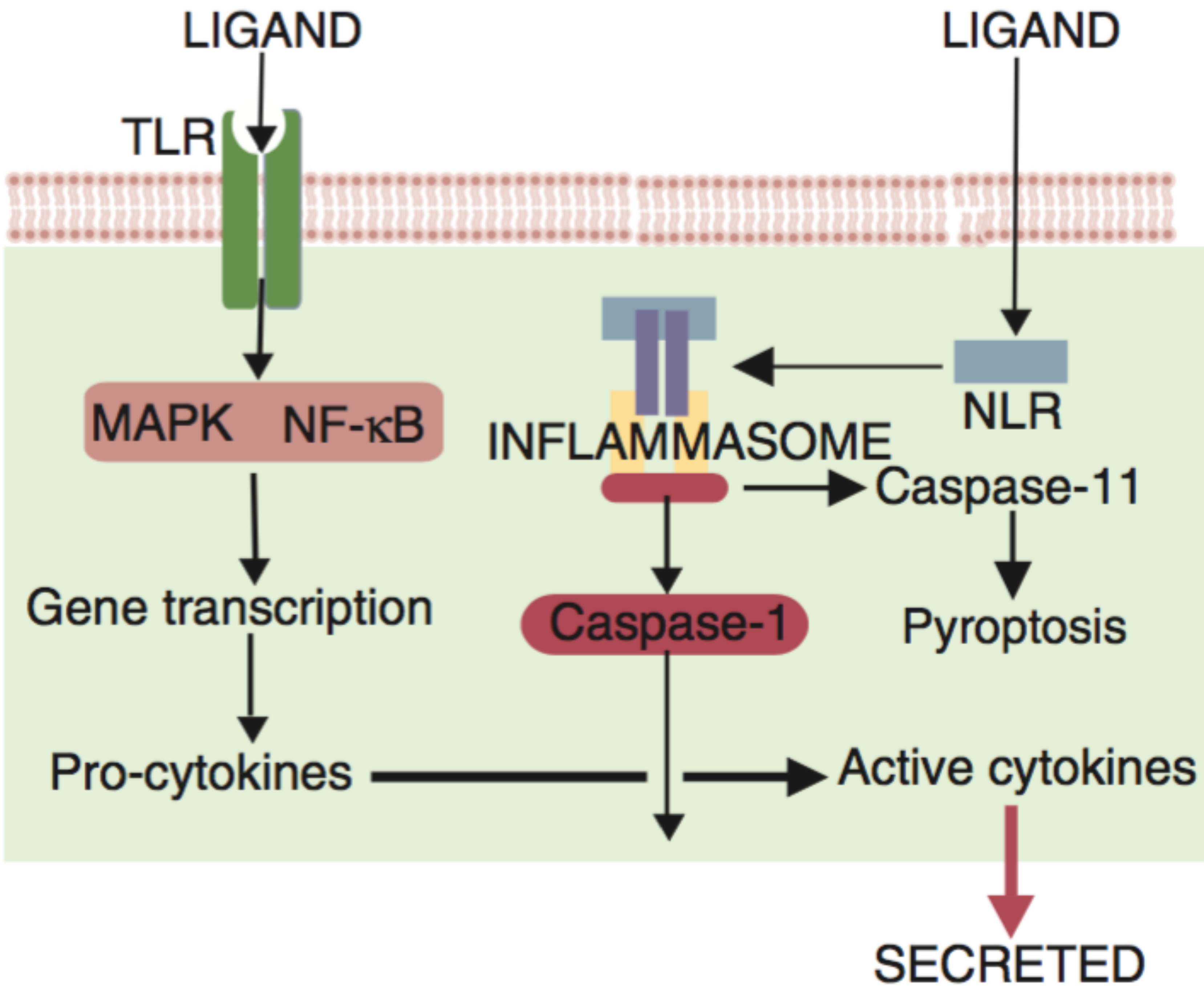


LIGAND
LIGAND
TLR
MAPK
NF-κB
INFLAMMASOME
NLR
Caspase-11
Gene transcription
Caspase-1
Pyroptosis
Pro-cytokines
Active cytokines
SECRETED

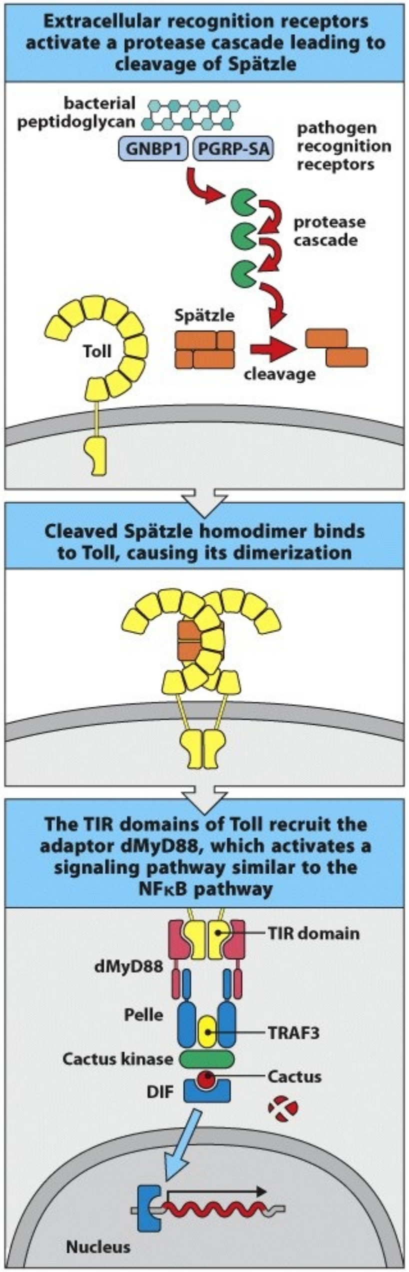
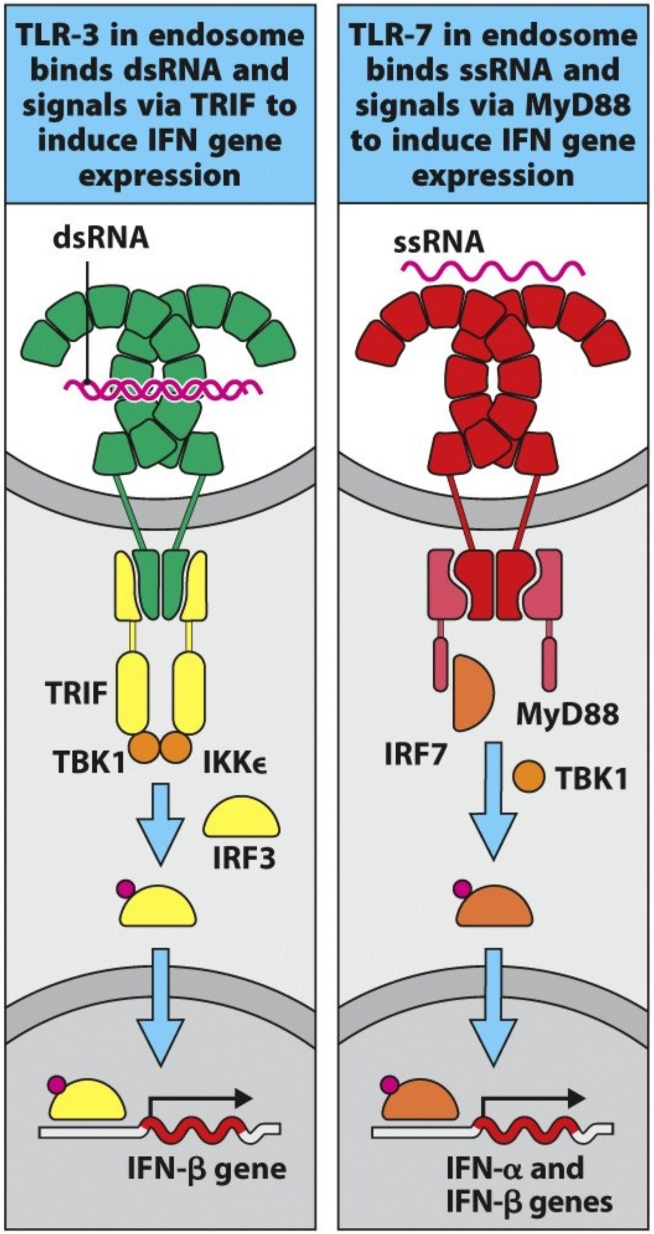



Figure 3.19 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

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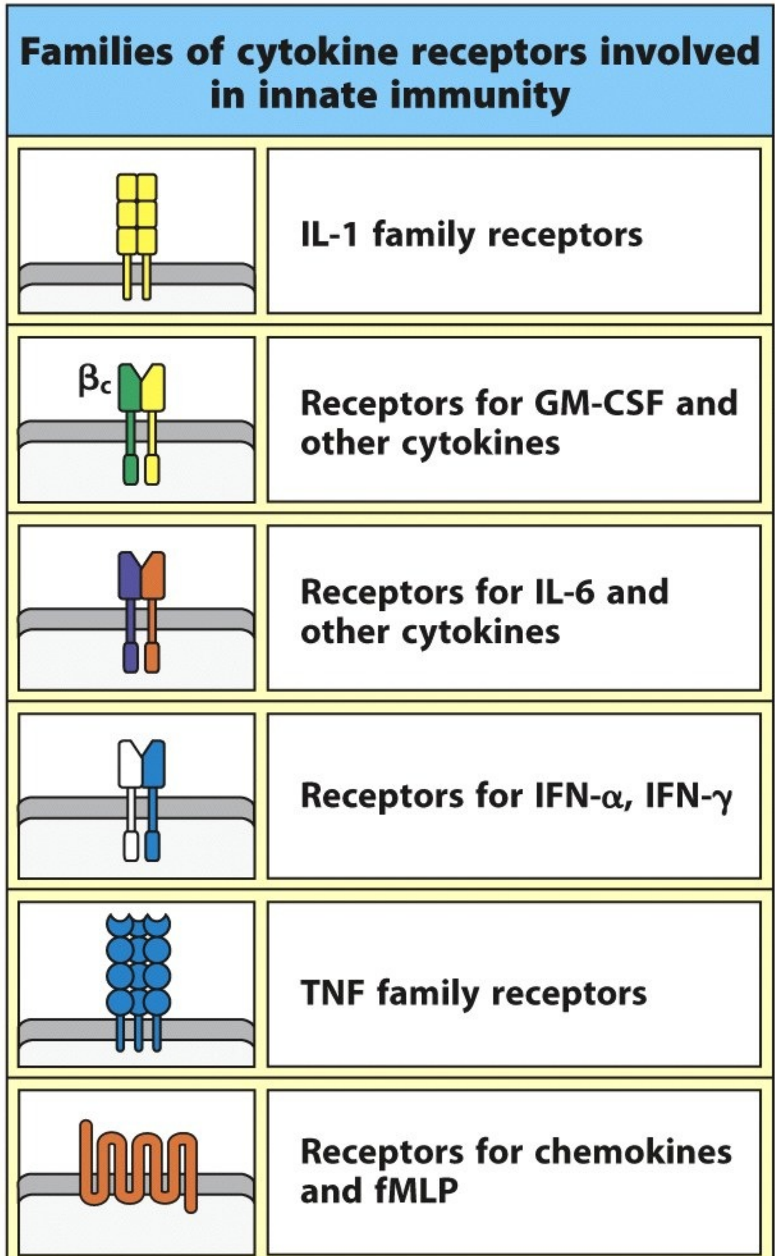


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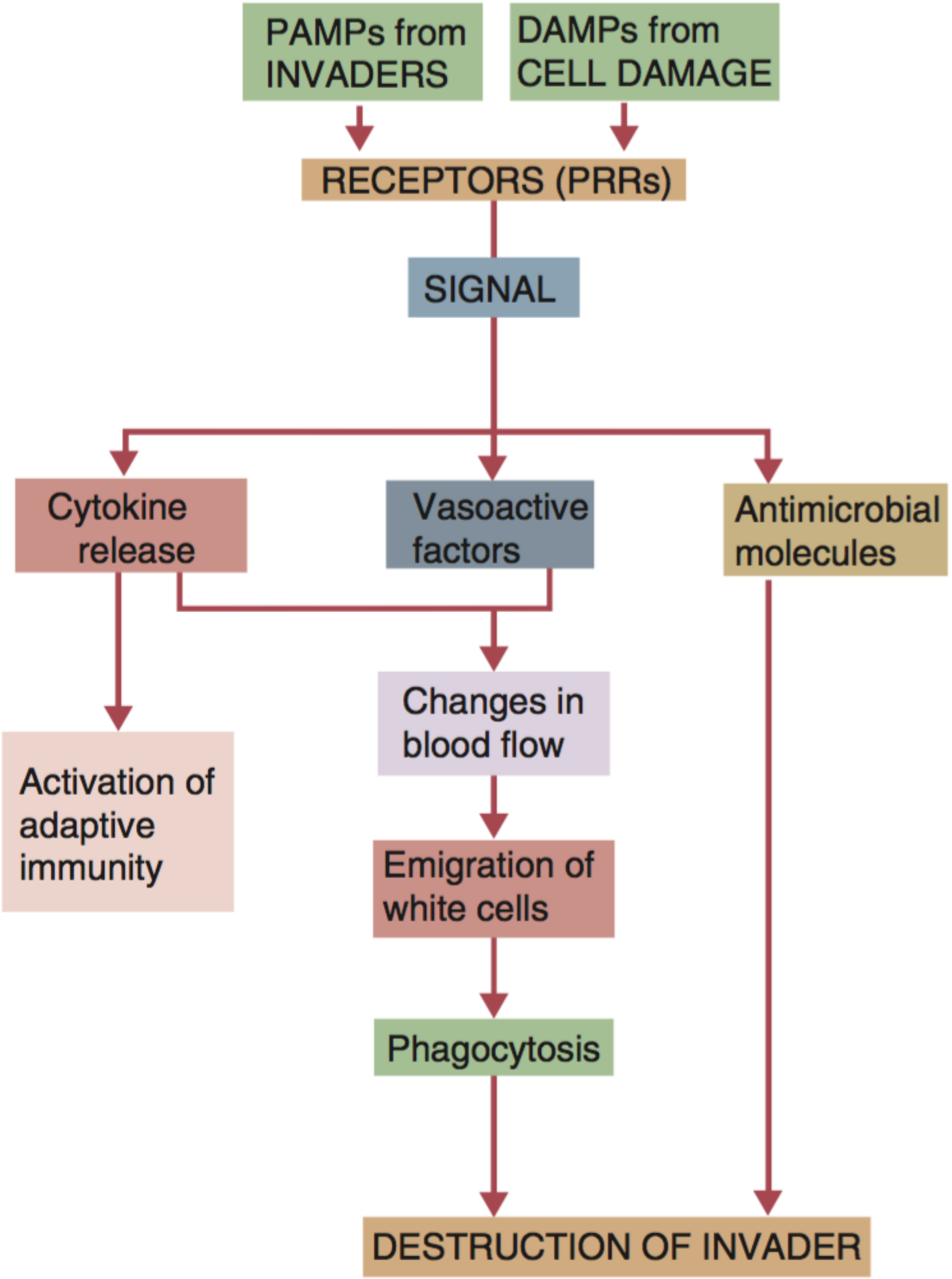
PAMPs from INVADERS
DAMPs from CELL DAMAGE
RECEPTORS (PRRs)
SIGNAL
Cytokine release
Vasoactive factors
Antimicrobial molecules
Activation of adaptive immunity
Changes in blood flow
Emigration of white cells
Phagocytosis
DESTRUCTION OF INVADER

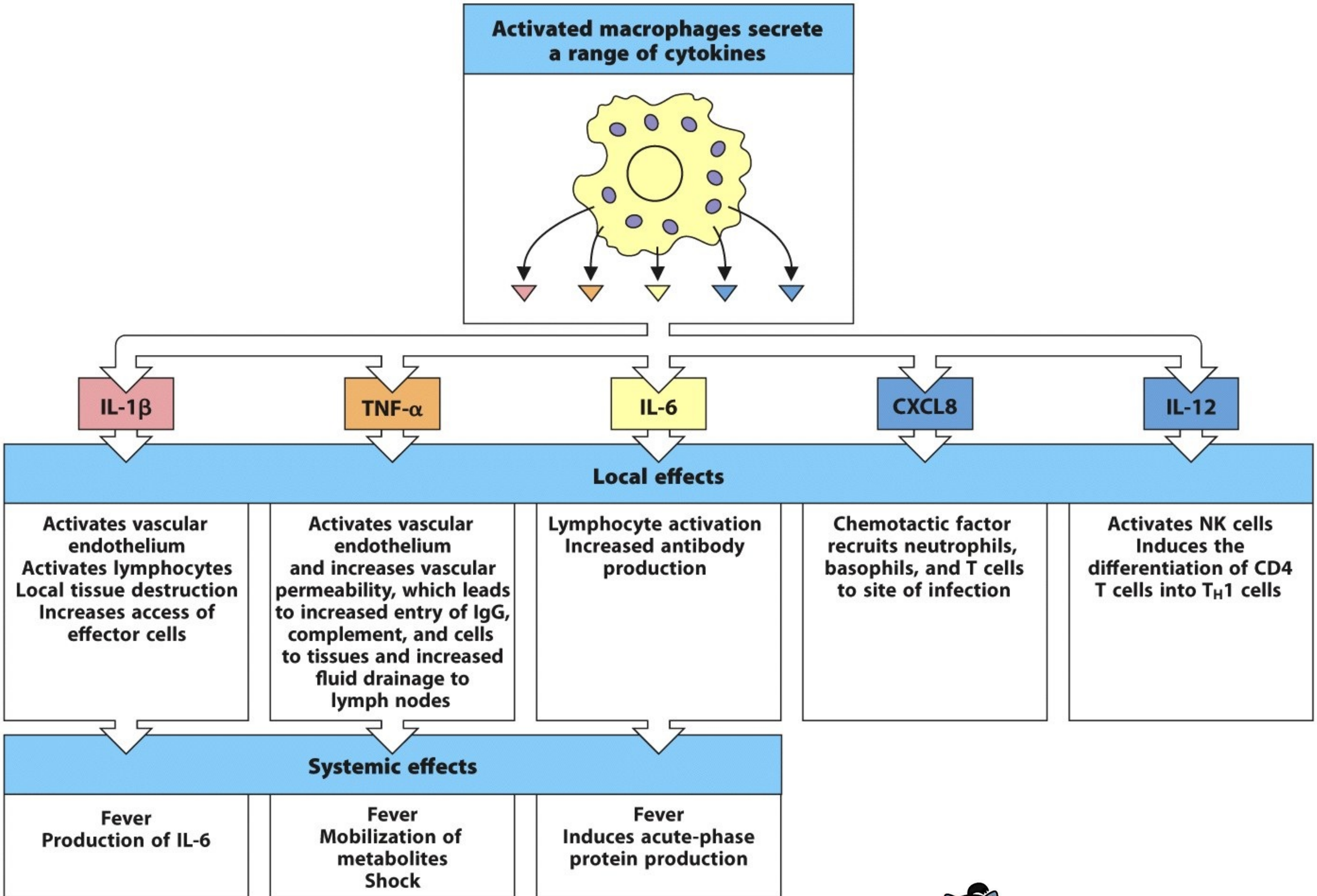


Figure 3.21 Janeway's Immunobiology, 8ed. (© Garland Science 2012)

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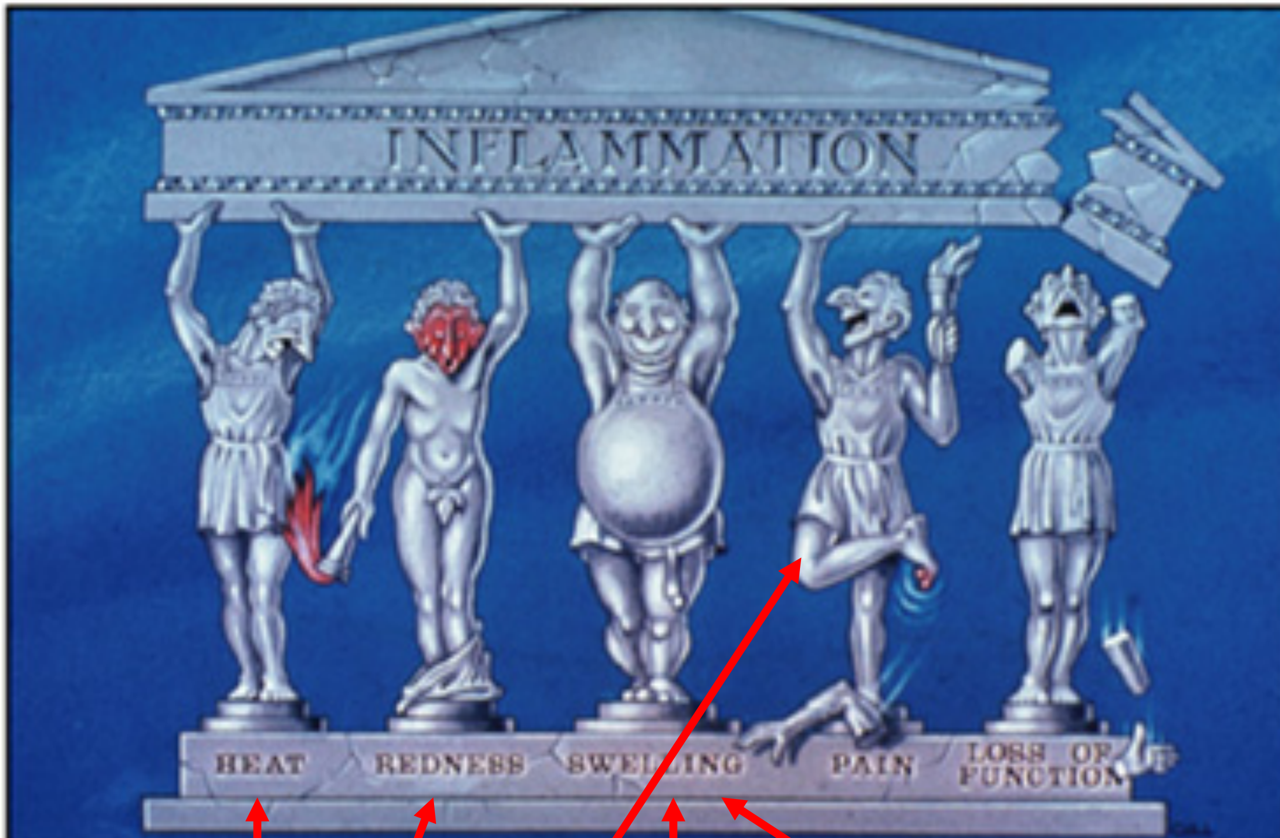


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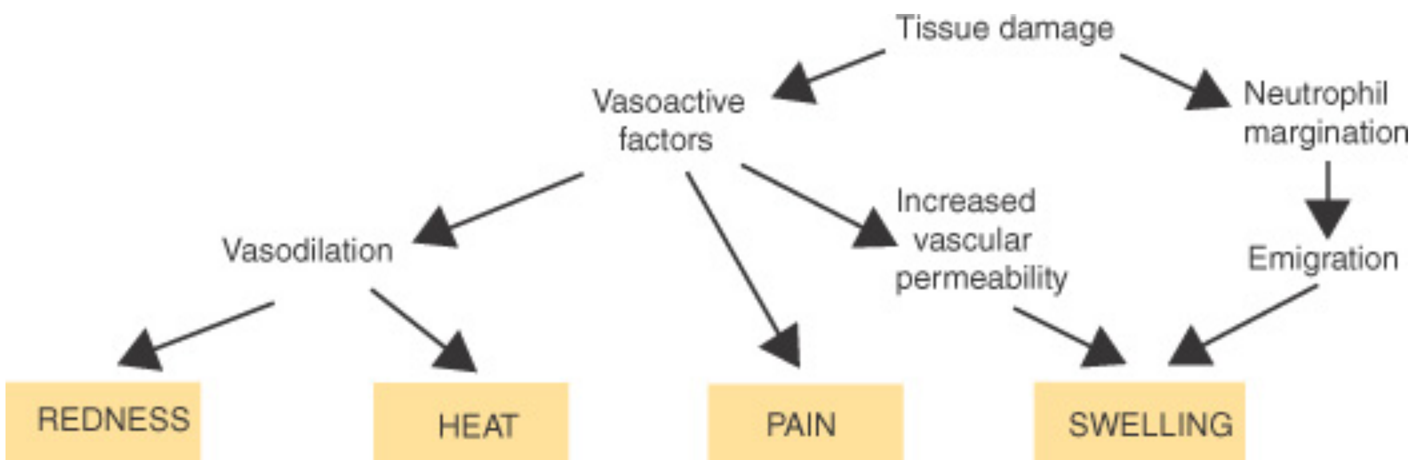


**FIGURE 2-17** The cardinal signs of acute inflammation and how they are generated.

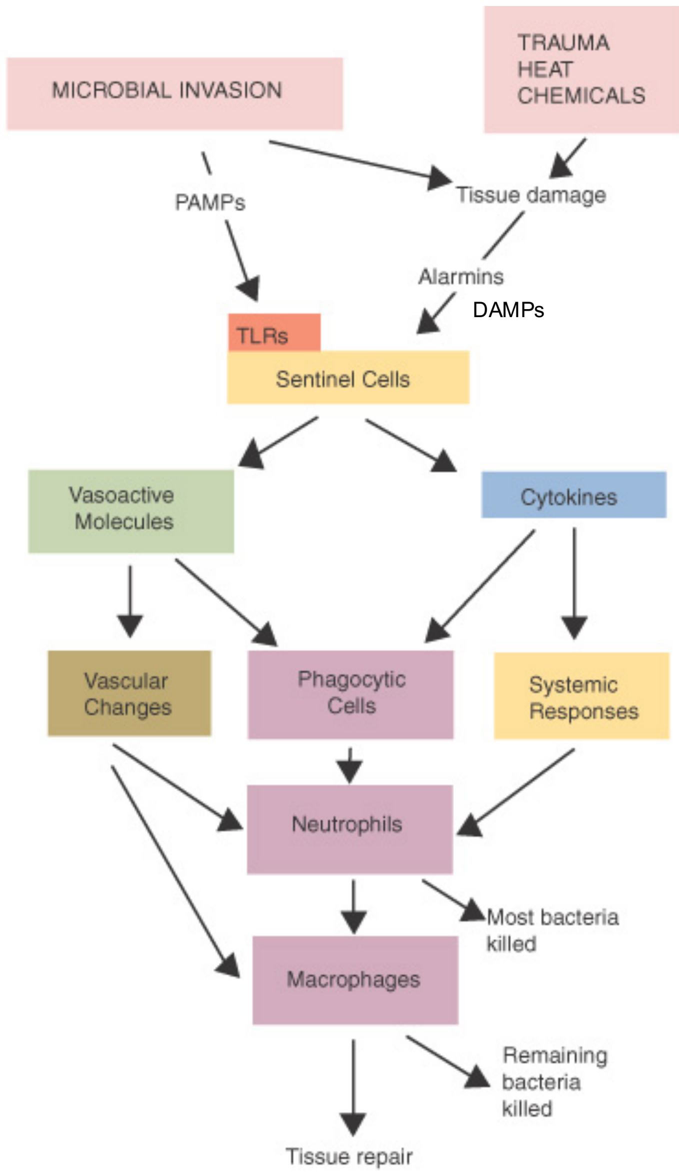


**FIGURE 2-1** An overview of the essential features of acute inflammation, an innate mechanism for focusing cells and other defensive mechanisms. It is triggered by microbial invasion and tissue damage.

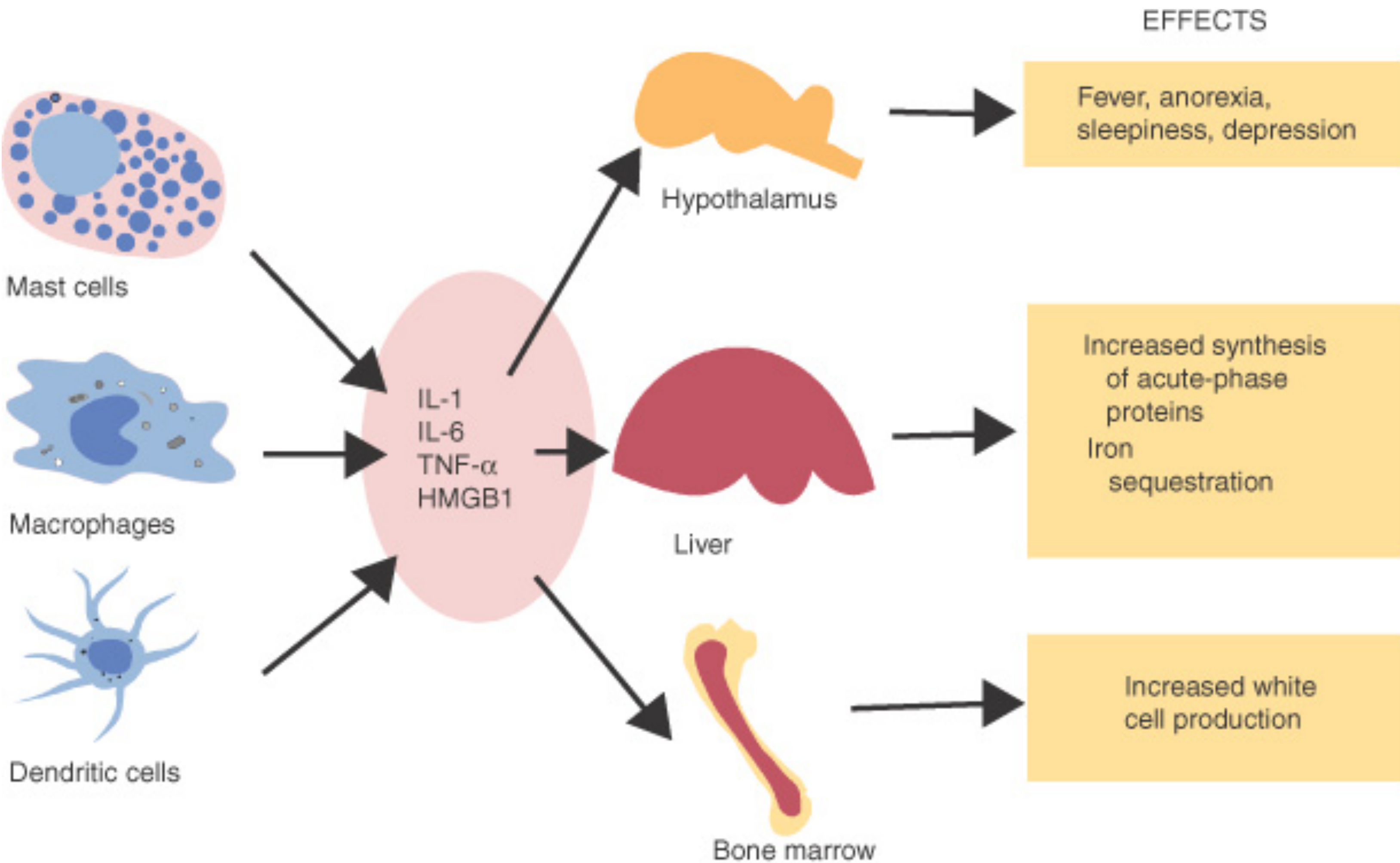


**FIGURE 4-13** Sickness behavior is part of the response of the body to inflammatory stimuli. Multiple systemic effects are due to the four major cytokines secreted by sentinel cells, mast cells, macrophages, and dendritic cells. The major sickness-inducing cytokines are interleukin-1 *(IL-1)*, IL-6, tumor necrosis factor-α *(TNF-α)*, and high mobility group box protein-1 *(HMGB1)*.



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