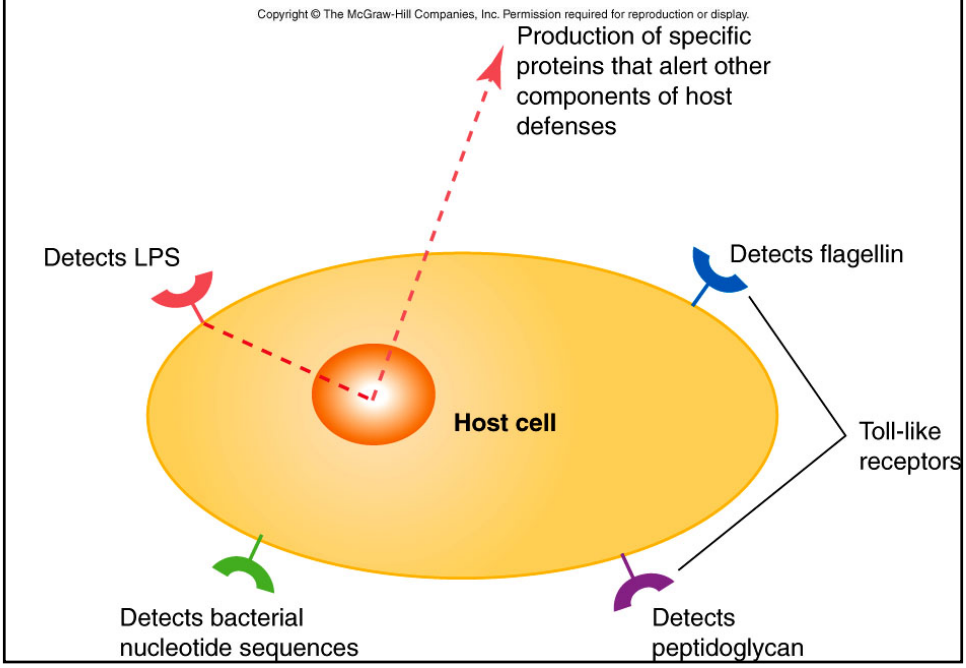
Topics

Sensor systems
Phagocytosis
Inflammation
Interferons
Fever

Sensor systems

- **Toll – like receptors**
- **Complement system**
  - **Classical pathway**
  - **Alternate pathway**
  - **Lectin pathway**

Figure 15.6 - Toll – like receptors (TLRs)



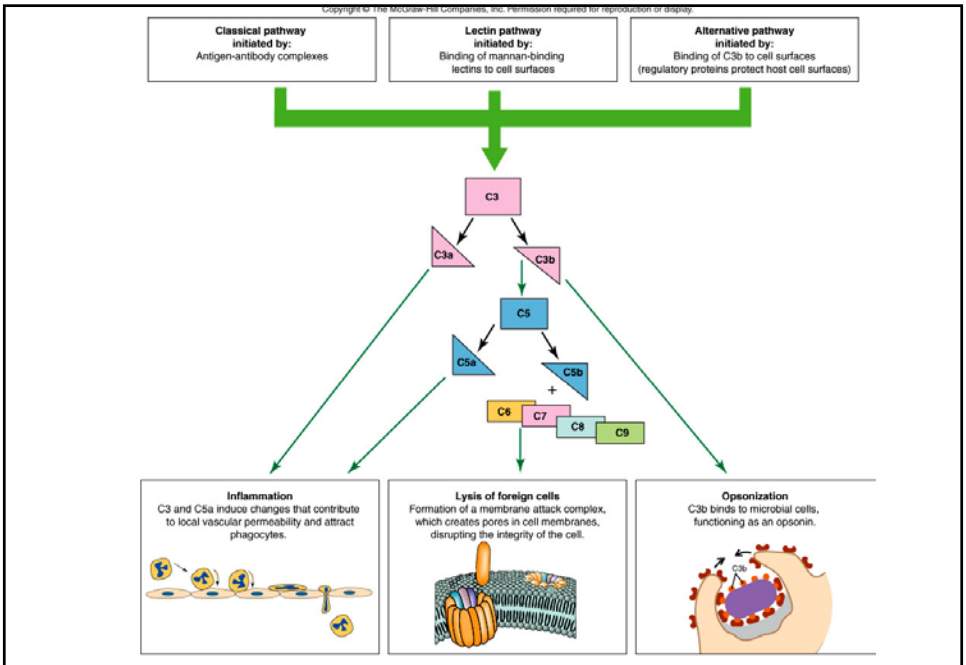


Figure 15.7 - Complement system

Phagocytosis

- **Process of phagocytosis**
- **Macrophages**
- **Neutrophils**

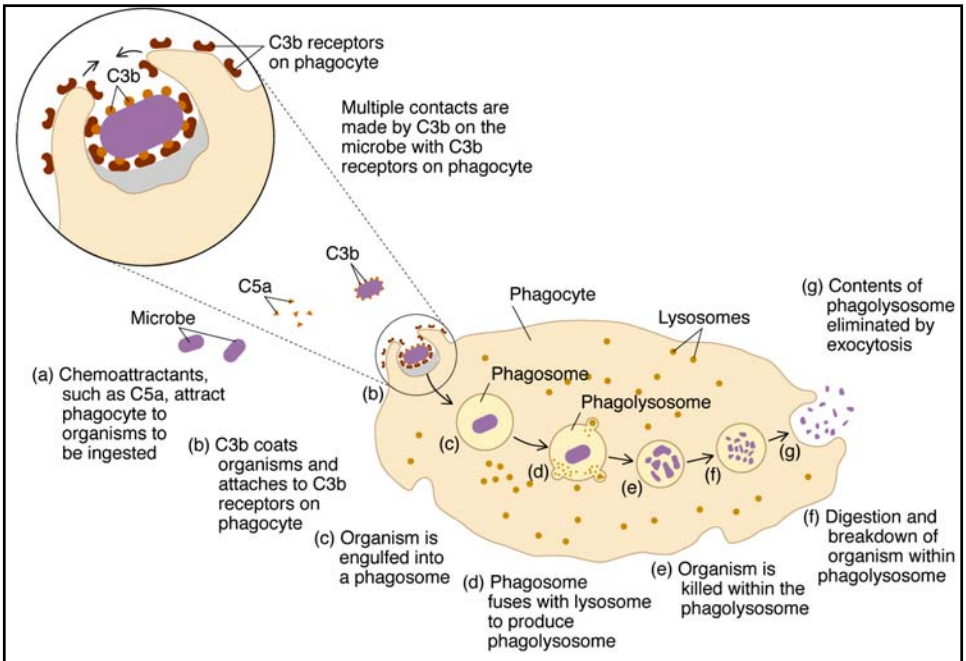
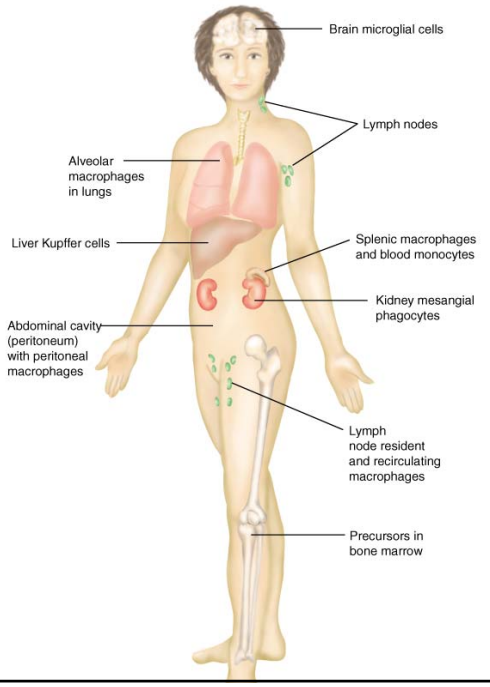


Figure 15.9 - Process of phagocytosis

Macrophages

- **Located throughout the body (Kupffer cells, alveolar, etc.)**
- **Produce cytokines**
- **Interact with T helper cells – activated macrophages**
- **Help form granulomas**

Figure 15.5 – Mononuclear Phagocytes



Neutrophils

- First to arrive during an immune response
- Involved in inflammation
- Inherently have more killing power than macrophages

Inflammation

- Initiation
- Inflammatory process
- Outcomes of inflammation

Initiation

- **Microbial products (LPS, flagellin, DNA)**
  - **trigger toll-like receptors on macrophages**
  - **macrophages make cytokines (TNFα) and chemokines**
  - **TNFα causes liver to secrete acute phase proteins**
  - **acute phase proteins facilitate phagocytosis and complement activation**
- **Complement cascade**
  - **Triggered by microbial surfaces**
  - **Activates mast cells to secrete inflammatory cytokines**
- **Tissue damage**

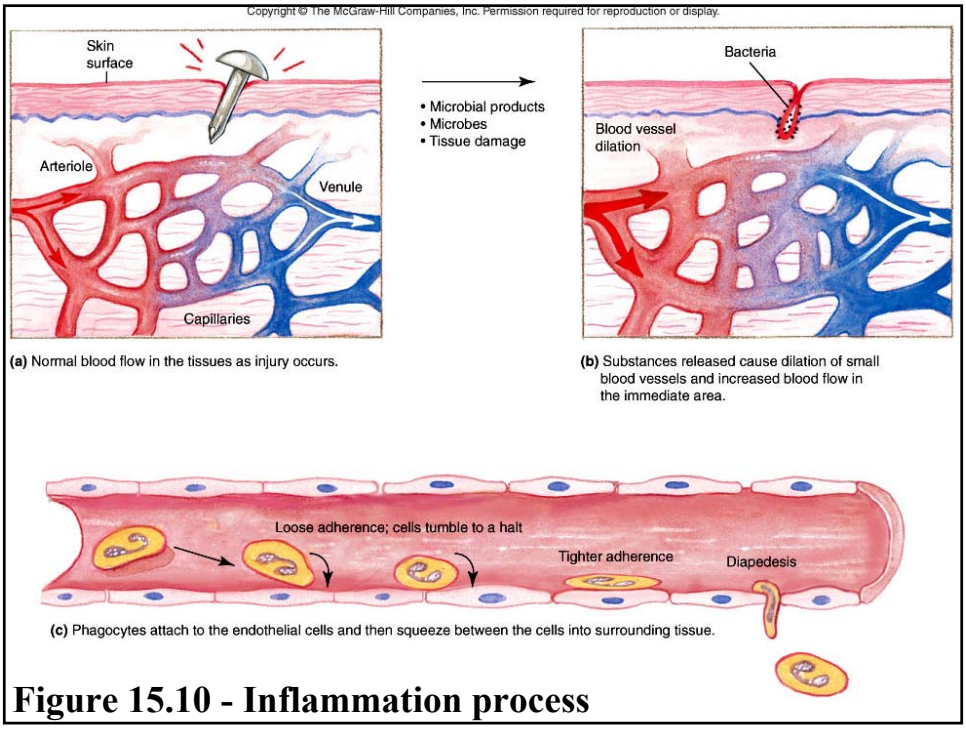
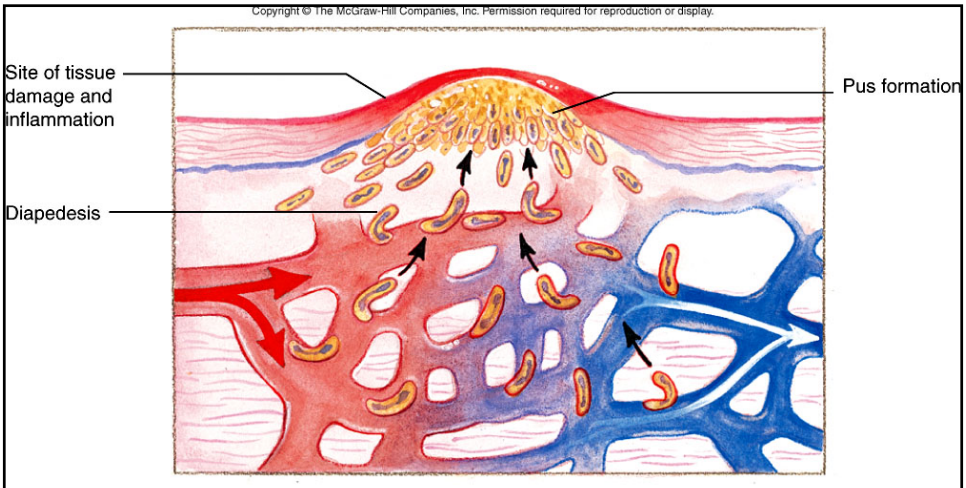


Figure 15.10 - Inflammation process

Leukocyte motility is mediated by interaction of selectins expressed on the endothelial cell surface with ligands on the leukocyte cell surface, slowing leukocyte motility and inducing cell rolling.

Diapedesis is initiated via stronger interactions of integrins expressed on the leukocyte cells surface with adhesion molecules (ICAM-1 and ICAM-2) on endothelial cells.

Activation of the endothelium is driven by macrophage cytokines such as TNF-α, causing selectin expression and synthesis of ICAM-1 by the endothelial cells.



(d) The attraction of phagocytes causes them to move to the site of damage and inflammation. Collections of dead phagocytes and tissue debris make up the pus often found at sites of an active inflammatory response.

Figure 15.10 - Inflammation process

Outcomes of inflammation

- **Damage to surrounding tissue**
  caused by toxic products of phagocytes
- **Release of bacterial endotoxins**
  released as LPS from Gram negative bacteria
  stimulates inflammation, loss of blood pressure
  bloodstream infection = septic shock
- **Eliminate invading pathogen**

Interferons

- **Glycoproteins**
- **Control viral infections**

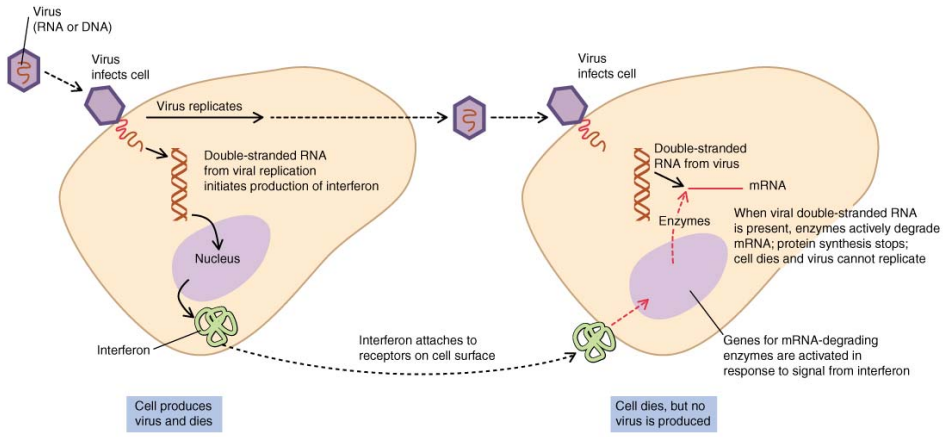


Figure 15.11 - Interferons

Fever

- **Hypothalamus controls temperature**
- **Pyrogens (endogenous or exogenous)**
- **High temperature inhibits pathogen growth**