

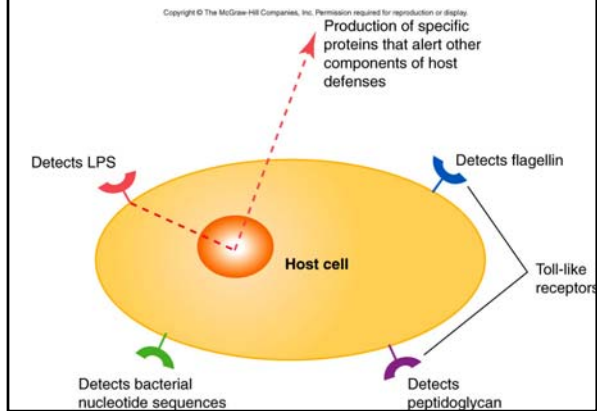
Topics

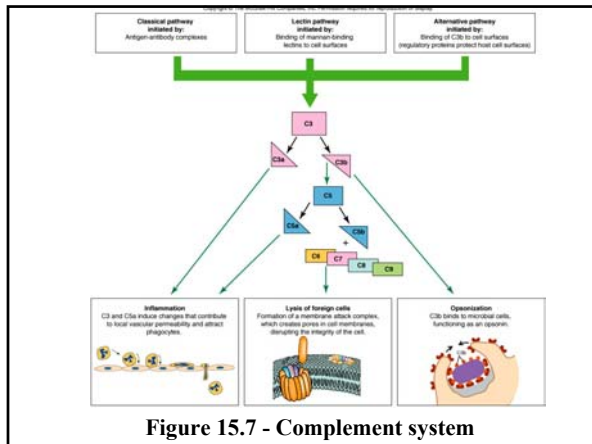
Sensor systems
Phagocytosis
Inflammation
Interferons
Fever

Sensor systems

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

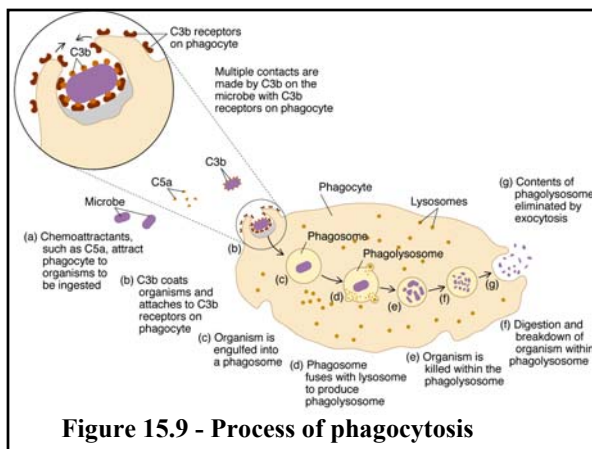
Figure 15.6 - Toll – like receptors (TLRs)





Phagocytosis

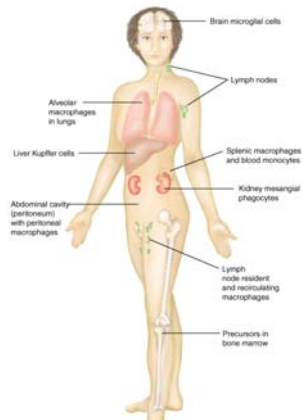
- Process of phagocytosis
- Macrophages
- Neutrophils



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 ($TNF\alpha$) and chemokines
 $TNF\alpha$ 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

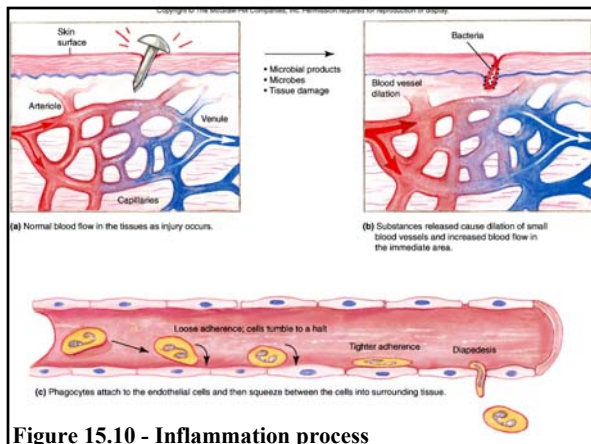
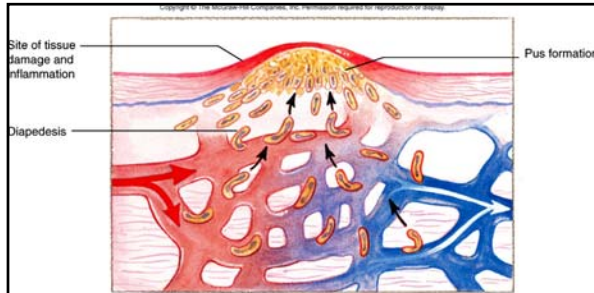


Figure 15.10 - Inflammation process

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 $\text{TNF-}\alpha$, 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

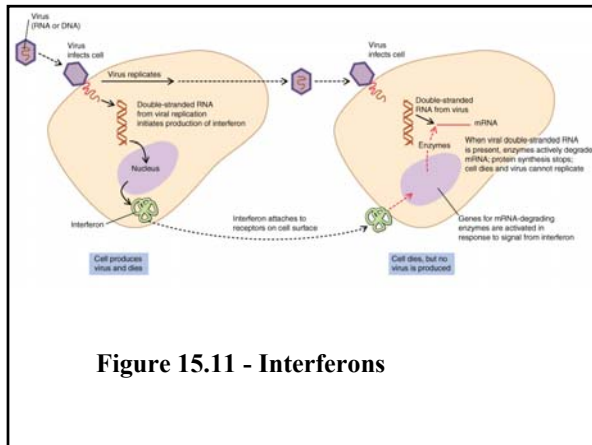


Figure 15.11 - Interferons

Fever

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