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)

D deteets LPS

D deteets flagellin

D deteets bacterial nucleotide sequences

D deteets peptidoglycan

Host cell

Production of specific proteins that alert other components of host defenses

Mast cells also stimulated

C5a is a chemoattractant

Not effective against Gram-positive bacteria

Figure 15.7

Complement system

Binds C3b receptors on phagocytes; C3a and C5a cause phagocytes to produce more receptors

Mast cells also stimulated

C5a is a chemoattractant

Not effective against Gram-positive bacteria
**Figure 15.8**
Membrane Attack Complex of Complement (MAC)

**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
- **Have Toll-like receptors and are stimulated by microbial substances**
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)
  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**

(a) Normal blood flow in the tissues as injury occurs.

(b) Substances released cause dilation of small blood vessels and increased blood flow in the surrounding tissue.

(c) Phagocytes attach to the endothelial cells and then squeeze between the cells into surrounding tissue.
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
- Damage to surrounding tissue
- Eliminate invading pathogen
Interferons

- Glycoproteins
- Control viral infections

Figure 15.11 - Interferons
Fever

- Hypothalamus controls temperature
- Pyrogens (endogenous or exogenous)
  
  *cytokines that induce fever via hypothalmus*

- High temperature inhibits pathogen growth