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

- Mast cells also stimulated
- C5a is a chemoattractant
- Binds C3b receptors on phagocytes;
- C3a and C5a cause phagocytes to produce more receptors
- 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 tissue as injury occurs.
(b) Bulbous vessels release vaso-dilator of small blood vessels and increased blood flow to the immediate area.
(c) Phagocytes attach to the endothelial cells and then squeeze between the cells into surrounding tissue.

Site of tissue damage and inflammation

Pus formation

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
- 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 hypothalamus

• High temperature inhibits pathogen growth