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

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
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.

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

Fever

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