# Non-Specific Defenses

1. Skin and Mucous Membranes
    - A. Mechanical Factors
        1. Skin
        2. Mucous membranes
        3. Ciliary escalator
        4. Lacrimal apparatus
        5. Salivary glands
    - B. Chemical Factors
        1. Mucus
        2. Sebum
        3. Perspiration
        4. Lysozyme
        5. Gastric juice

2. Normal Microbiota
    - Microbial antagonism

3. Phagocytic Leukocytes
    - Types of Leukocytes
        - A. Granulocytes
            1. Neutrophils (PMNs) -phagocytic
            2. Basophils and Mast Cells
            3. Eosinophils -phagocytic
        - B. Agranulocytes
            1. Monocytes and Macrophages -phagocytic
                - A. Fixed Macrophages
                - B. Free Macrophages
            2. Lymphocytes
                - A. T cell = cell-mediated immunity
                - B. B cell = antibody mediated immunity

    - Phagocytosis
        1. Chemotaxis and Adherence
        2. Ingestion
        3. Phagosome formation
        4. Phagolysosome formation
        5. Digestion
        6. Residual Body formation
        7. Exocytosis

4. Inflammation
    - A. Vasodilation and Increased Permeability
        1. Chemical release
        2. Blood clot formation
        3. Abscess formation
    - B. Phagocyte Migration and Phagocytosis
        4. Margination
        5. Emigration
        6. Phagocytosis
    - C. Tissue repair
        7. Repair

5. Fever

6. Antimicrobial Substances
    - A. Nitric Oxide
    - B. Complement Fixation
        1. MAC
        2. Opsonization
        3. Inflammation
    - C. Interferons
        - IFN Beta
        - IFN Alpha
        - IFN Gamma

# Body Defense

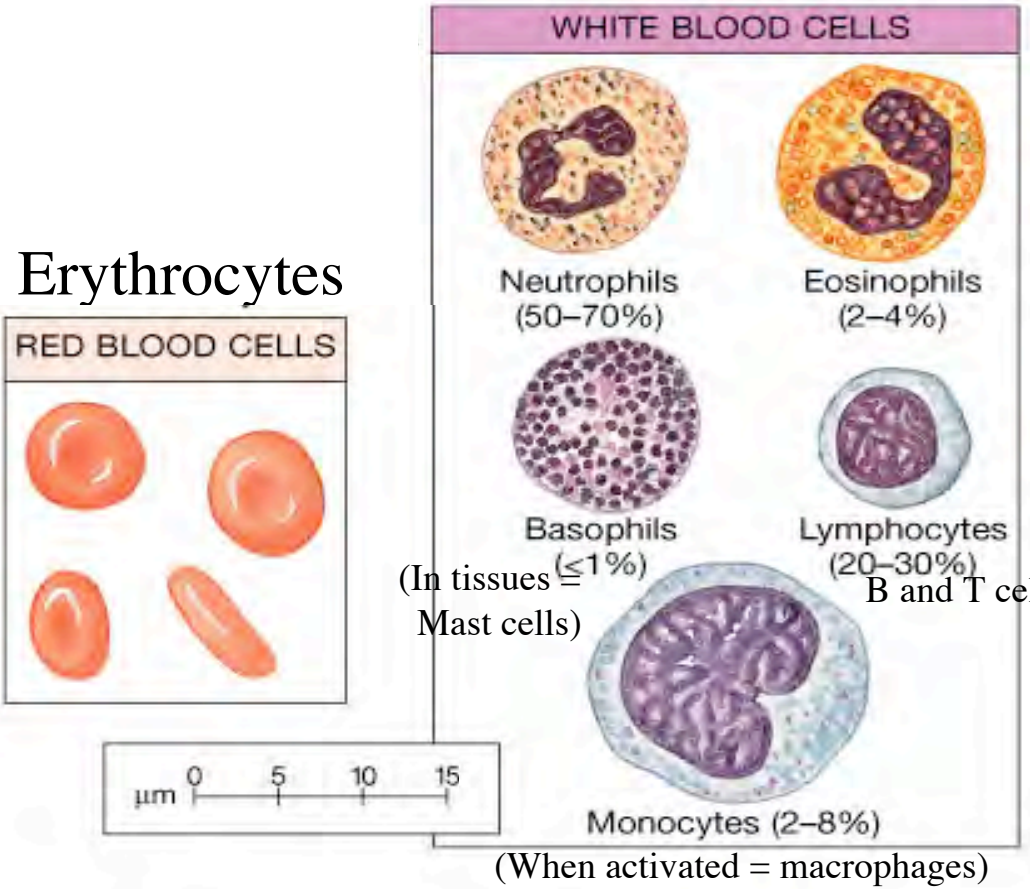
| Nonspecific Resistance | | Specific Resistance (Responses of the Immune System, Chapter 17) |
|---|---|---|
| **First line of defense** | **Second line of defense** | **Third line of defense** |
| • Intact skin<br>• Mucous membranes and their secretions<br>• Normal microbiota | • Phagocytic white blood cells<br>• Inflammation<br>• Fever<br>• Antimicrobial substances | • Specialized lymphocytes: B cells and T cells<br>• Antibodies |



---

# Human Blood Cells

## Leukocytes



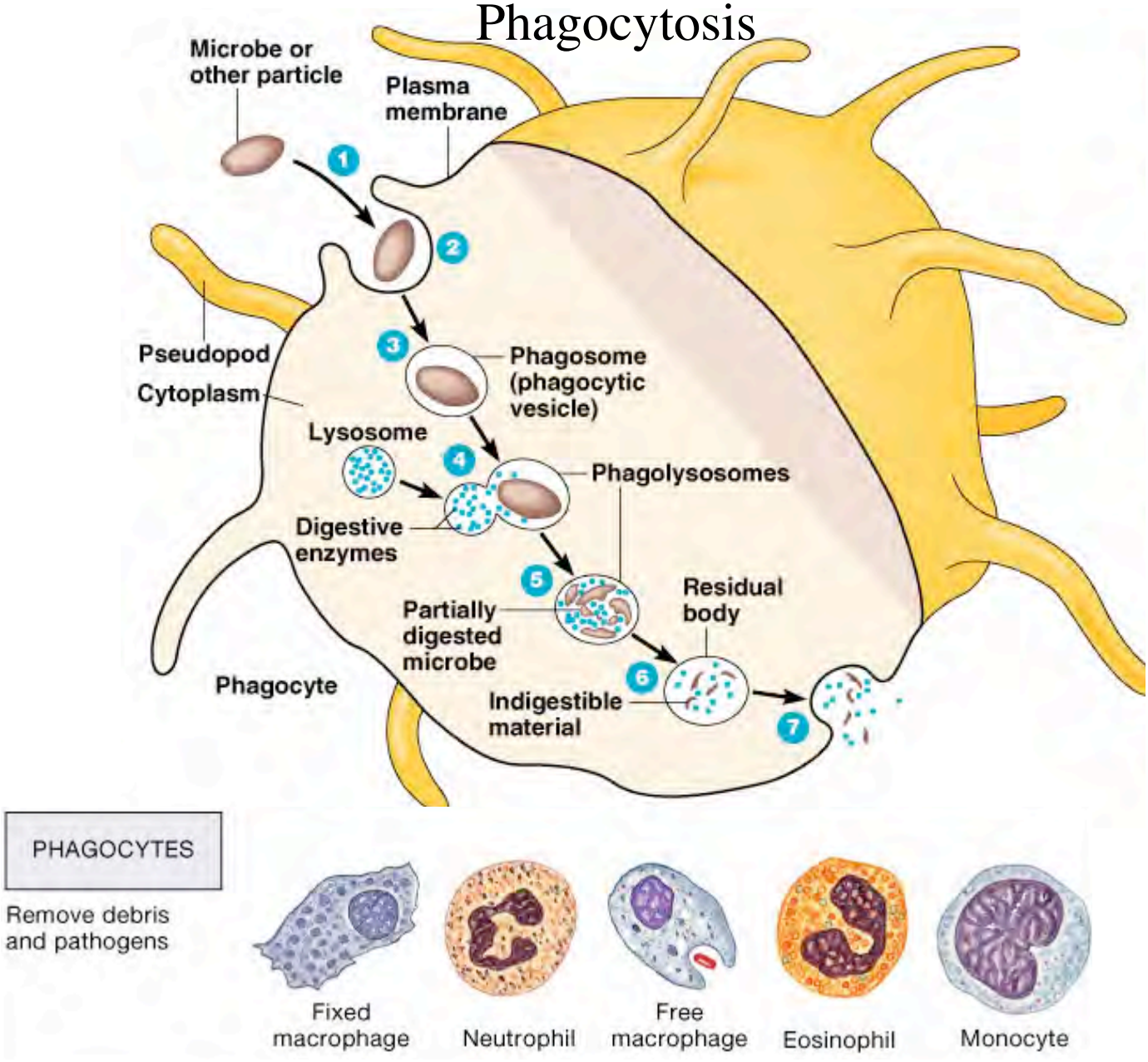

(In tissues = Mast cells)

B and T cells

(When activated = macrophages)

## Erythrocytes

# Phagocytosis




1. **Chemotaxis and Adherence**
   Chemotaxis = chemical attraction of phagocyte to microbe
   Adherence = attachment of plasma membrane of phagocyte to microbe; made easier by opsonization
      Opsonization = coating of the microbe with antibodies or complement proteins
2. **Ingestion**: phagocyte extends pseudopods around microbe
3. **Phagosome formation**: fusion of pseudopods around the microbe encloses it in a phagocytic vesicle called a phagosome which now floats in the cytoplasm
4. **Phagolysosome formation**: the phagosome fuses with a lysosome creating a phagolysosome putting he microbe in contact with digestive enzymes and bacteriocidal substances
5. **Digestion**: most microbes are killed and hydrolyzed in 10-30 min
   Oxidative burst kills the microbe: toxic oxygen radicals
   Enzymes and acids hydrolyze the microbe into component organic molecules
6. **Formation of the residual body**: useful small organic molecules are absorbed into the cytoplasm and the acids and enzymes are neutralized. Residual body = all remaining undigested material in a vesicle
7. **Exocytosis**: residual body contents are discharged outside the cell

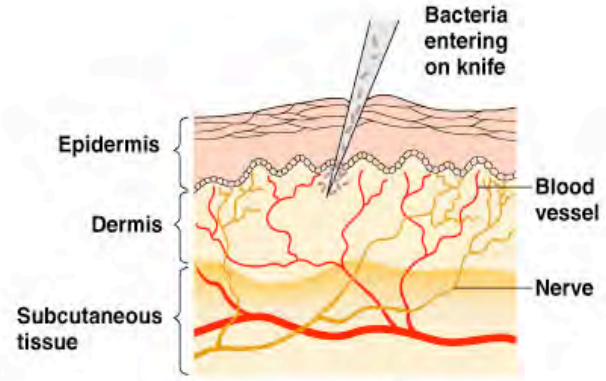
# Inflammation

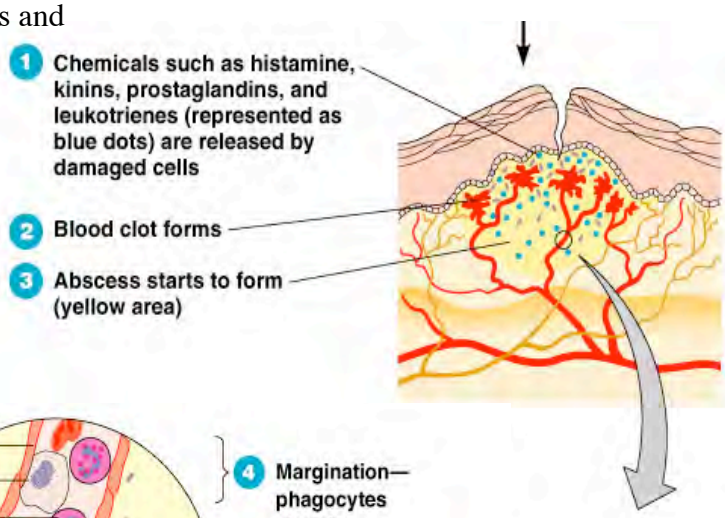
Begins with damage to the tissue:

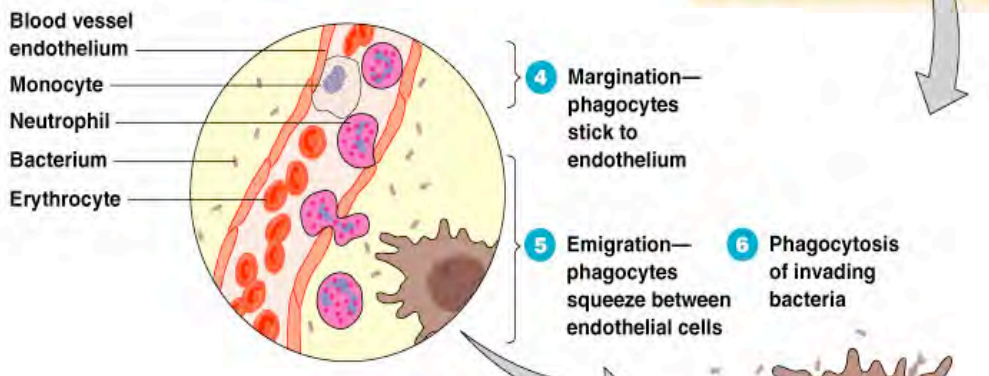
A. Vasodilation and Increased Blood Vessel Permeability
   1. Damaged cells release chemicals (histamine, prostaglandins, leukotrienes) which promote vasodilation, increased permeability of vessel walls, and attraction of phagocytes
   2. Blood clot forms: clotting factors solidify fibrin at the injury site to prevent spread
   3. Abscess forms: abscess = localized collection of dead cells, body fluids and microbes trapped under the clot

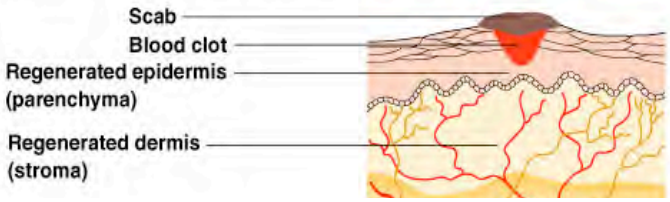
B. Phagocyte Migration and Phagocytosis
   4. Margination: phagocytes attracted to the injury adhere to the endothelium of the local blood vessels
   5. Emigration: marginated phagocytes move into the tissues via pseudopods (ameoboid motion) exiting the vessel between the loose endothelial cells, and chemotax toward the damaged cells and microbes
   6. Phagocytosis
      Neutrophils arrive first: fast acting but short lived, phagocytose pathogens
      Macrophages arrive second: engulf necrotic tissue, dead neutrophils, and remaining pathogens

C. Tissue repair
   7. Repair: begins only after all harmful substances and microbes have been neutralized
      Tissue = parenchyma (cells) + stroma (CT)
      parenchyma growth = reconstruction
      stroma growth = scar



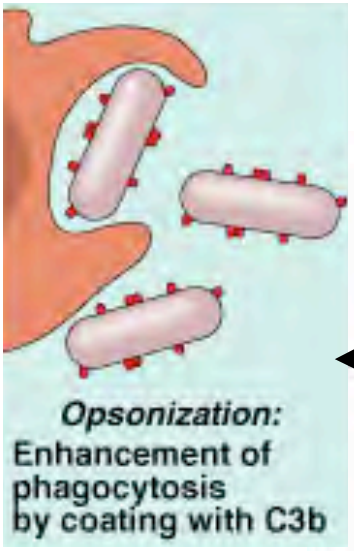



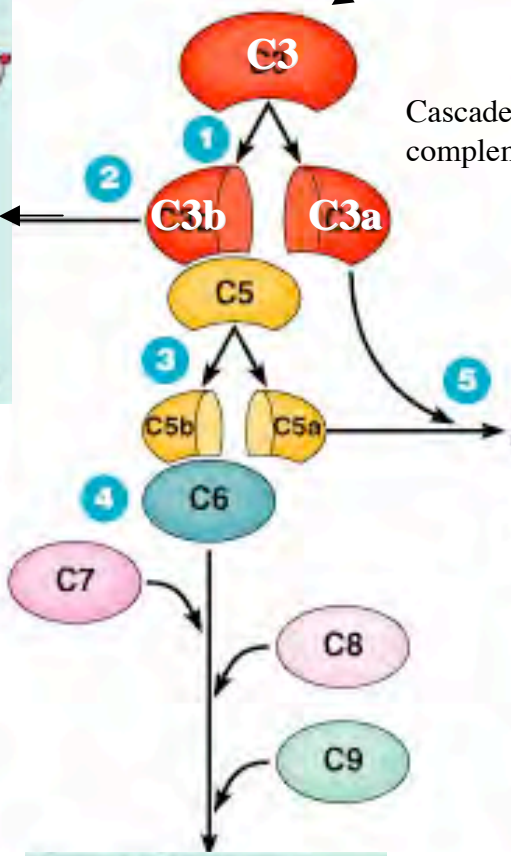



# Complement System

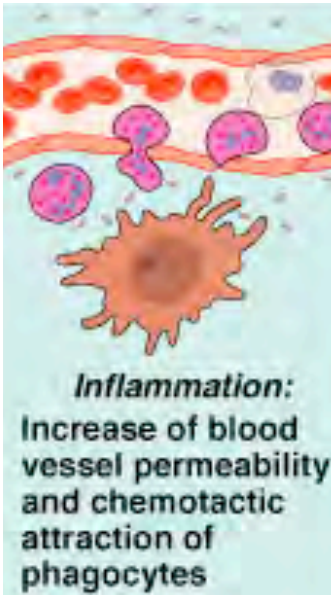
## Complement Fixation

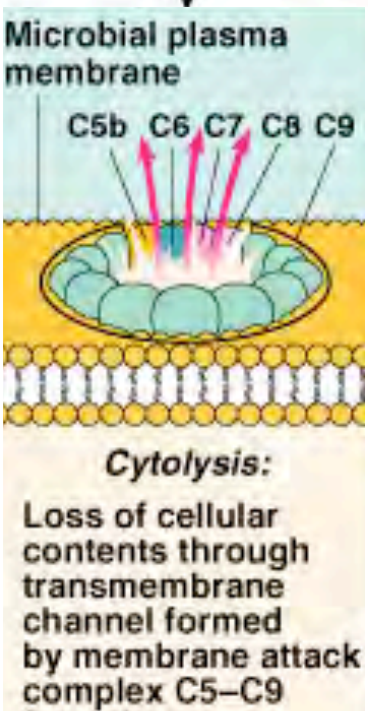



Activated by:
1. Antibody binding to microbe
2. Complement protein binding to microbe




Cascade is triggered in blood: one complement protein turns on the next




<u>Three Antimicrobial Results of Complement Fixation:</u>

1. MAC formation = Cytolysis
   - C5-C9 form the Membrane Attack Complex: the proteins assemble into a pore on the microbe membrane resulting in cell lysis
2. Opsonization = Phagocytosis
   - C3b opsonizes bacteria to enhance phagocytosis
3. Inflammation = Phagocytosis
   - C3a triggers histamine release from Mast cells thus triggering inflammation