Chapter 16/17: Immune system

**Lecture**

Chapter 16: Nonspecific defenses
- First line of defense
- Formed elements
- Second line of defense
- Complement system

Chapter 17: Specific defenses
- Antibodies
- Humoral response
- Cellular response

**Lab**

Check results from Tues and inoculate glucose and lactose broths for Enteric A and B

Lab EXAM
Susceptibility - Lack of resistance to a disease

Resistance/ immunity - Ability to ward off disease

Innate (nonspecific) immunity - Resistance to all microbes; present from birth (can be species specific)

Adaptive (specific) resistance - Resistance to a specific pathogen
# Host defense systems

<table>
<thead>
<tr>
<th>Innate (Nonspecific) Immunity</th>
<th>Adaptive (Acquired) Immunity (Chapter 17)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First line of defense</strong></td>
<td><strong>Second line of defense</strong></td>
</tr>
<tr>
<td>Intact skin</td>
<td>Natural killer cells and phagocytic white blood cells</td>
</tr>
<tr>
<td>Mucous membranes and their secretions</td>
<td>Inflammation</td>
</tr>
<tr>
<td>Normal microbiota</td>
<td>Fever</td>
</tr>
<tr>
<td></td>
<td>Antimicrobial substances</td>
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<tr>
<td></td>
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<td></td>
<td><strong>Third line of defense</strong></td>
</tr>
<tr>
<td></td>
<td>Specialized lymphocytes: T cells and B cells</td>
</tr>
<tr>
<td></td>
<td>Antibodies</td>
</tr>
</tbody>
</table>

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First lines of defense

(Table 16.3)
First line of defense - skin

- Physical factors
  * Dermis and epidermis
  * Lots of keratin
  * Dry conditions, low temperature
First line of defense- skin

-Physical factors
  * Dermis and epidermis
  * Lots of keratin
  * Dry conditions, low temperature

-Chemical factors
  * Sebum (includes fungistatic and bacteriostatic fatty acids)
  * Low pH
  * High salt
  * Lysozymes (sweat)
  * IgA (sweat)
First line of defense - skin

- Physical factors
  - Dermis and epidermis
  - Lots of keratin
  - Dry conditions, low temperature

- Chemical factors
  - Sebum (includes fungistatic and bacteriostatic fatty acids)
  - Low pH
  - High salt
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- Normal microbiota
First line of defense - mucosal surfaces

- Physical factors
  * Mucous production
  * Cilia
  * Hairs

- Mucous production
- Cilia
- Hairs
First line of defense - mucosal surfaces

- Physical factors
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  * Cilia
  * Hairs

- Chemical factors
  * Lysozymes
  * Lactoferrin
  * α and β Defensin
  * IgA
First line of defense - mucosal surfaces

- Physical factors
  * Mucous production
  * Cilia
  * Hairs

- Chemical factors
  * Lysozymes
  * Lactoferrin
  * α and β Defensin
  * IgA

- Normal microbiota
First line of defense - lacrimal apparatus

- Physical factors
  - Tears

- Chemical factors
  - Lysozyme
  - β Defensin
  - IgA
Second line of defense: Formed Elements in Blood

**TABLE 16.1 Formed Elements in Blood**

<table>
<thead>
<tr>
<th><strong>I. Erythrocytes (Red Blood Cells)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>4.8–5.4 million per μl or mm³</td>
</tr>
<tr>
<td>Function: Transport of O₂ and CO₂</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>II. Leukocytes (White Blood Cells)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>5000–10,000 per μl or mm³</td>
</tr>
</tbody>
</table>

A. Granulocytes (stained)

1. Neutrophils (PMNs) (60–70% of leukocytes)
   - Function: Phagocytosis

2. Basophils (0.5–1%)
   - Function: Production of histamine

3. Eosinophils (2–4%)
   - Functions: Production of toxic proteins against certain parasites; some phagocytosis

B. Agranulocytes (stained)

1. Monocytes (3–8%)
   - Function: Phagocytosis (when they mature into macrophages)

2. Dendritic cells
   - Functions: Derived from monocytes; phagocytosis and initiation of adaptive immune responses

3. Lymphocytes (20–25%)
   - Natural killer (NK) cells
     - Function: Destroy target cells by cytolysis and apoptosis

   - T cells
     - Function: Cell-mediated immunity (discussed in Chapter 17)

   - B cells
     - Function: Descendants of B cells (plasma cells) produce antibodies
SEM of formed elements

Bruce Wetzel (photographer). Harry Schaefer (photographer) National Cancer Institute
### Complete blood count (CBC)

<table>
<thead>
<tr>
<th>Type of cell</th>
<th>Increase</th>
<th>Decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td>Erythrocytosis, Polycythemia</td>
<td>Anemia</td>
</tr>
<tr>
<td>WBCs</td>
<td>Leukocytosis</td>
<td>Leukopenia</td>
</tr>
<tr>
<td>- lymphocytes</td>
<td>Lymphocytosis</td>
<td>Lymphocytopenia</td>
</tr>
<tr>
<td>- granulocytes</td>
<td>Granylocytosis</td>
<td>Granulocytopenia</td>
</tr>
<tr>
<td>- neutrophils</td>
<td>Neutrophilia</td>
<td>Neutropenia</td>
</tr>
<tr>
<td>- eosinophils</td>
<td>Eosinophilia</td>
<td>Eosinopenia</td>
</tr>
<tr>
<td>Platelets</td>
<td>Thrombocytosis</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>ALL cell lines</td>
<td></td>
<td>Pancytopenia</td>
</tr>
</tbody>
</table>

![Image](https://via.placeholder.com/150)
Self-study for Thursday

- Preview the following processes:
  - Phagocytosis
  - Fever
  - Inflammation
  - Complement proteins
Second line of defense: Phagocytosis
### Microbial evasion of phagocytosis

<table>
<thead>
<tr>
<th>Evasion Mechanism</th>
<th>Bacteria/Pathogen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhibit adherence: M protein, capsules</td>
<td><em>Streptococcus pyogenes, S. pneumoniae</em></td>
</tr>
<tr>
<td>Kill phagocytes: Leukocidins</td>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>Lyse phagocytes: Membrane attack complex</td>
<td><em>Listeria monocytogenes</em></td>
</tr>
<tr>
<td>Escape phagosome</td>
<td><em>Shigella</em></td>
</tr>
<tr>
<td>Prevent phagosome-lysosome fusion</td>
<td>HIV</td>
</tr>
<tr>
<td>Survive in phagolysosome</td>
<td><em>Coxiella burnetti and Mycobacteria spp</em></td>
</tr>
</tbody>
</table>
Second line of defense: Fever

- Usually set at 37° C

- Some chemical signals set it higher
  - Cytokine interleukin-1
  - Cytokine alpha-tumor necrosis factor
  - Prostaglandins reset hypothalamic thermostat
Second line of defense: Inflammation

1. Chemicals released
   1. Histamine
   2. Kinins
   3. Prostaglandins
   4. Leukotrienes

2. Vasodilation

3. Increased permeability

4. Activation of acute phase proteins

(5. Clot formation, abscess, tissue repair)
### Inflammation - Chemical Signals

<table>
<thead>
<tr>
<th>Chemical Signal</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histamine</td>
<td>Vasodilation, increased permeability of blood vessels</td>
</tr>
<tr>
<td>Kinins</td>
<td>Vasodilation, increased permeability of blood vessels</td>
</tr>
<tr>
<td>Prostaglandins</td>
<td>Intensity histamine and kinin effect</td>
</tr>
<tr>
<td>Leukotrienes</td>
<td>Increased permeability of blood vessels, phagocytic attachment</td>
</tr>
</tbody>
</table>
Inflammation

MORE DETAIL:

Vasodilation/ increased Permeability
- Margination WBCs
- Emigration WBCs

Activation of acute-phase proteins
- Cytokines
- Kinins
- Complement proteins
- (Interferons)
Complement system

Opsonization:
Enhancement of phagocytosis by coating with C3b

C3
- C3b
- C3a

Histamine
Mast cell

Inflammation:
Increase of blood vessel permeability and chemotactic attraction of phagocytes
(see also Figure 16.12)

Cytolysis:
Loss of cellular contents through transmembrane channel formed by membrane attack complex C5–C9
(see also Figure 16.11)

Microbial plasma membrane
C5b, C6, C7, C8, C9
Independent Study

- Review the following processes:
  - Phagocytosis
  - Fever
  - Inflammation
  - Complement proteins