

Pathogens

Mode of transmission

Example: Collagenases - degrade collagen [Connective tissue]

Part of Lipopolysaccharide - Release when cell dies

Therapy: anti-endotoxin or anti-TNF antibodies

E.g.: Diphtheria Toxin from Corynebacterium diphtheria

* Enterotoxins: Affect epithelia cell lining @ GI tract

* Neurotoxins: interfere with nerve impulse - Lockjaw [Muscle Contraction]

* Cytotoxins: inhibits protein synthesis

E.g.: Tetanus Toxin from Clostridium tetani

E.g.: Vibrio enterotoxin from Vibrio cholera

Systemic infection (Toxins are spread throughout the body via the circulatory system)

→ Activate cytokines production

→ Causing Septic shock

Description

Direct damage to the tissue Specific tissue-degrading enzymes

Reservoir

Portal of exit

Susceptible host

Portal of entry

Extent of the Infection

Damage Mechanism

Mechanism

Endotoxins

Exotoxins - Cytotoxins

Exotoxins - Neurotoxins

Exotoxins - Enterotoxins

· Local infection (Relative Small Area of Body)

Factors → more susceptible to disease.

M6 - Pathogenesis & Host Defense

3 more properties

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Learning Objectives For Pathogenesis

- 1. Study the spread of the infection: reservoirs, transmission of disease, and exit
- 2. Microbial pathogenesis: entry into the host, penetrate host defense and damage the host
- 3. Understand nonspecific host defense, specific host defense, and antibody-mediated immunity vs. cell-mediated immunity

The Infection Chain

| Pathogen | * Rapid identification of organisms | |
|----------------------|---------------------------------------|--|
| Reservoir | ★ Health surveillance | |
| | * Environmental sanitation | |
| | ★ Disinfection/sterilization | |
| Portal of exit | ★ Hand hygiene | |
| | ★ Control of excretions and secretion | |
| | ★ Trash and waste disposal | |
| Mode of transmission | ★ Hand hygiene | |
| | ★ Sterilization | |
| | ★ Standard precautions | |
| | * Airflow control | |
| | ★ Food handling | |
| | ★ Isolation | |
| | ★ Pest control | |
| Portal of entry | * Aseptic technique | |
| | ★ Catheter care | |
| | ★ Wound care | |
| Susceptible host | ★ Treatment of underlying diseases | |
| | * Recognition of high-risk patients | |

The Iceberg Concept of Infectious disease

Severity and Duration of a Disease

| Disease Type | Develop Time | Duration | Example |
|--------------|--------------|--------------------|--------------------------------|
| Acute | Rapidly | Short | Influenza, Hepatitis A |
| Chronic | Slowly | Long | Tuberculosis, Hepatitis B, HIV |
| Latent | Inactive | Can be reactivated | Herpes Simplex Virus |

Development of Disease

| Stage of Disease | Description |
|----------------------|--|
| Period of incubation | Between: Infection ~ First sign of symptoms |
| Illness | Exhibits symptoms |
| Decline | Symptoms subside |
| Convalescence | Regains strength → a state of health |

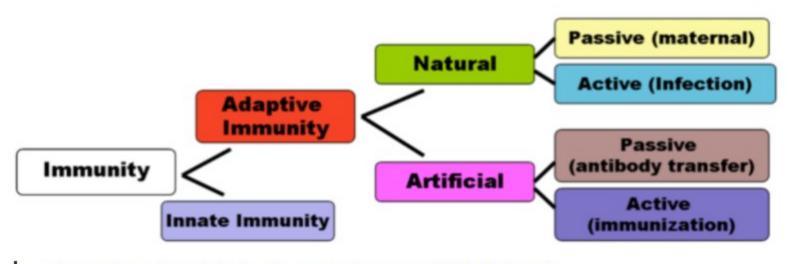
Predisposing Factors

| Gender / | Climate | Age | Fatigue | Stress | Nutrition |
|----------|---------|-----|---------|--------|-----------|

Learning Objectives For Host Defense Mechanism

- Understand nonspecific vs. specific host defense.
- Understand antibody-mediated immunity vs. cell-mediated immunity.

Introduction to Immunity



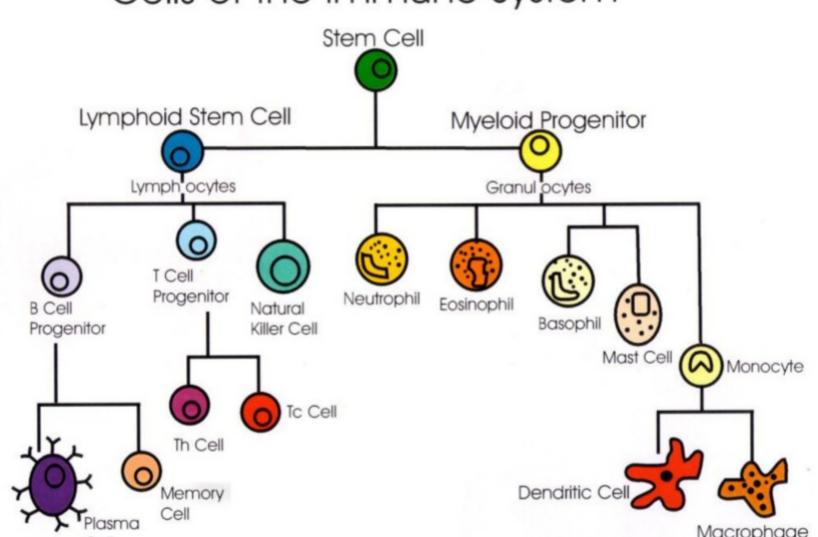
Resistance of organism to infection, disease, or other unwanted biological invasion

Remarks of the Immune Systems

- Phagocytic Cells: Macrophages, Neutrophils, Dendritic cells
- Mast cells: produce Histamine & Prostaglandin → Vasodilation & † permeability of blood vessels
- Interferons:

| Function | ★ Induce uninfected cells to produce antiviral proteins ★ Modulate immune response |
|--------------------------|---|
| Innate Immunity | IFN- α & IFN- β |
| Acquired Immune Response | IFN-γ |

Cells of the Immune System



Non-specific defense



Introduction to Barriers of Non-Specific Defense

| Name of Barriers | Description | Type of Barriers |
|---|--|---------------------|
| Intact skin | Against the penetration of microorganism | Physical Barriers |
| Saliva | Washes microorganisms from teeth and gums | Physical Barriers |
| Mucus | Traps microorganisms @ RR and GI tracts | Physical Barriers |
| Ciliate | Moves the mucus up | Physical Barriers |
| Sebum from sebaceous glands [Skin] | Production of fatty acid that helps protect the skin | Chemical Barriers |
| Lysozyme @ tears & saliva | Breaks down bacterial cell walls | Chemical Barriers |
| Hydrolytic enzymes in saliva Acid in stomach Degradative enzymes in small intestine | Break down and degrade microorganisms | Chemical Barriers |
| Normal flora | Prevent colonization & Infection | Biological Barriers |

Innate Immunity

| Type of Innate Immunity | Inflammation | Fever |
|-------------------------|---|---------------------------------|
| Cause | Cell damage | Interleukin-1 → hypothalamus |
| Symptoms | Redness, pain, heat, and swelling | Abnormal High Body Temperatures |
| Response | Vasodilation and ↑ permeability of blood vessels → Caused by histamine & prostaglandin from mast cells | / |
| Benefits | Phagocytic cells squeeze through endothelial linings → Phagocytosis → Facilitate tissue repairing | No consistent benefits |
| Related Systems | Complement system (>20 Different Proteins → Attacks surfaces of pathogens) | CNS |

Specific host dense

- Adaptive immune immunity ° Cell-mediated response - [T-Cell] T for <u>thymus</u>
- o Humoral response [B-Cell] B for bone marrow
- ▼ Humoral response Extracellular
 - · Production and Circulation of antibodies
 - Taget: Extracellular pathogens & Circulating toxins. · Primary Infection:
 - $B \text{ Cells} \rightarrow Plasma \text{ Cell} \rightarrow Release Antibodies$ Secondary Infection:
 - $\text{Long-living Memory Cell} \rightarrow \text{Multiply} \rightarrow \text{Plasma Cell}$ Antibodies from B cells → Bind to pathogens → Mark them for destruction by Immune Cells

▼ Cell-mediated response - Intracellular

- Activation of T cells Recognize and Attack infected cells
- Target: Intracellular pathogens, Cancer cells • Cytokines from T cells → Coordinate the immune response

| | Helper T cells (CD4+) | Cytotoxic T cells (CD8+) | Memory T |
|----------------|-----------------------|---|--------------------|
| Responsibility | modulate T and B cell | Release Lesion & Perforin → Destroy target cells | secondary immunity |

▼ Remarks:

Difference between Natural killer cells (Innate) & T-Cells (Adaptive)

| | T-Cells | Natural Killer (NK) Cells A minor type of T Cells |
|-------------|--|--|
| Origin | Mature in the thymus gland | Originate from the bone marrow |
| Function | Recognize specific antigens → directly attack and kill those cells Release cytokines to regulate the immune response | Missing-self Process. Part of the innate immune system. Killing abnormal cells Antibody-mediated cytotoxicity |
| Specificity | High degree of specificity. | Recognize based on lack of certain surface marker |
| Activation | Require antigen presentation become activated - By antigen-presenting cells (APCs) - Example: Dendritic & Macrophages | kill without the need for antigen presentation. |
| Memory | Memory T Cell | Do not exhibit memory response. |

- ▼ Working Mechanism of T-Cells
 - APCs present antigen and pass Interleukin-1 (prevent fever) → Th.
- Th. help to clone the cytokines to the B-cell & T-cell → Killing abnormal cell.