Medical bacteriology

Host Defenses Against Pathogenic Bacterial

The Property of Host Defenses

The host and microbes are in a dynamic interaction. This relationship outcome depends on:

1. The virulence of the pathogen (invade, damage the host, resist the defenses of the host).
2. The degree of host resistance or defense (susceptibility of the host or effectiveness of the host defense mechanisms)

Host defense: A healthy human can defend itself against pathogens at different stages against infectious disease process. The host defenses may be:

- of such a degree that infection can be prevented entirely.
- Or, if infection does occur, the defenses may stop the process before disease is apparent.

Host defense mechanism

Host defense mechanisms are divided into two groups:

1. **Innate defenses,** provide protection against invasion by normal flora and pathogens.

   It is referred to as natural or constitutive resistance since they are host inherent .

- They are continually ready to respond
- Don't require a period of time to be induced
- Non specific
- Partially depending on genetics

innate immunity includes;

- anatomical and structural barrier
- inflammation
- complement
- phagocytosis
- the presence sof normal flora

Host defense mechanism

Host defense mechanisms are divided into two groups:

2. **Adaptive defenses,** this mechanism must be induced by host exposure to a pathogen during an infection and involve the immunological responses to a pathogen causing an infection. Unlike the innate defenses, they are not immediately ready to come into action until after the host is appropriately exposed to the pathogen

Also known as acquired or inducible immunity

- **Adaptive immunity is reaction is directed specifically against the invading pathogen**
- **Some pathogens overcome the nonspecific innate defense and are usually more suitable to specific inducible defenses**

Host defense mechanism

Adaptive immunity is divided into two types:

1. Active immunity:

- the host undergoes an immunological responses and produces cells and factors responsible for the immunity, i.e., the host produces its own antibodies and/or lymphocytes.
- Active immunity can persist a long time in the host.

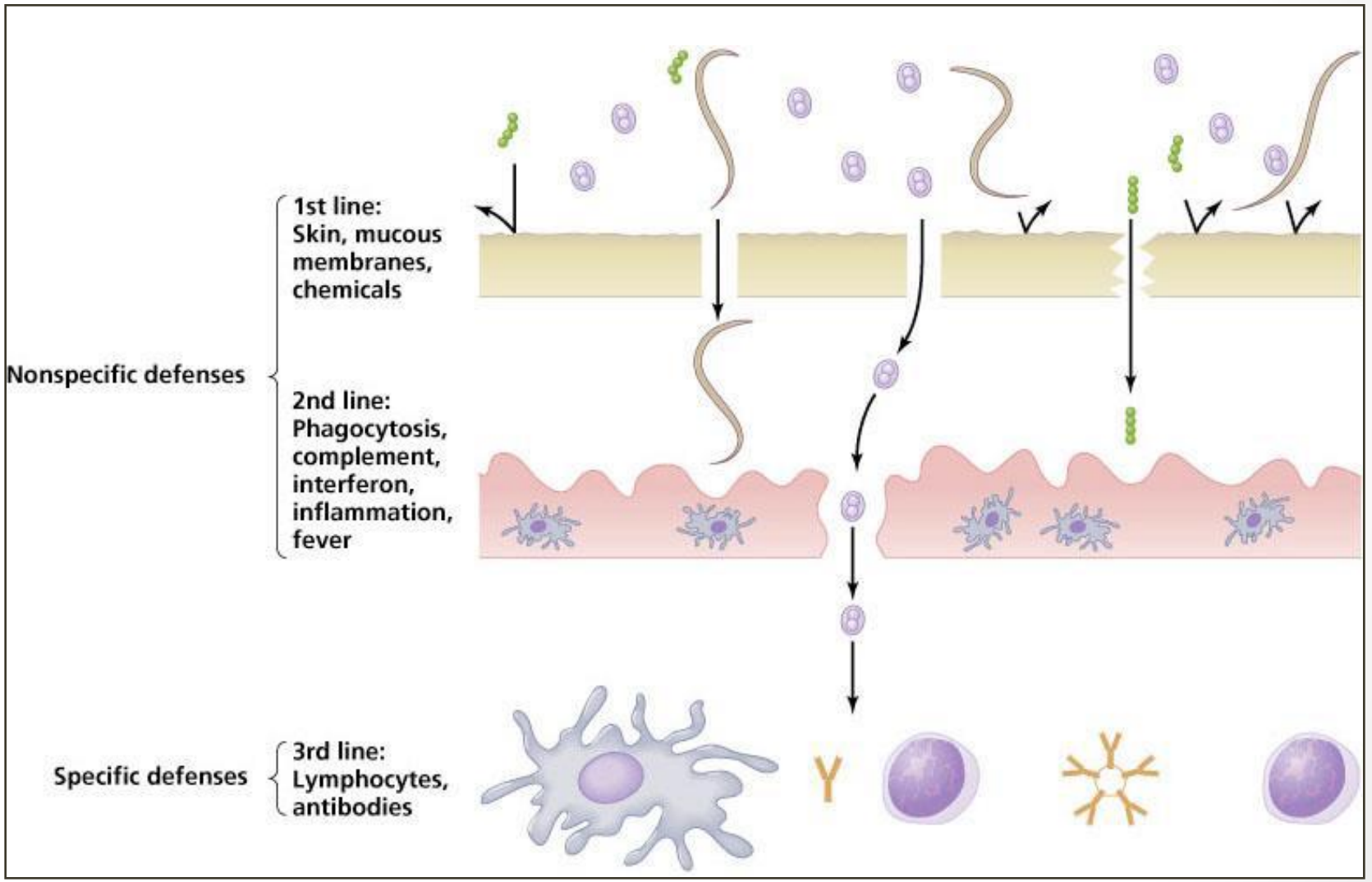
2. Passive immunity

- is acquisition by a host of immune factors which were produced externally in another animal, meaning that the host receives antibodies and/or immuno-reactive lymphocytes originally produced in another animal. Passive immunity is typically short-term.

Host defense mechanism

Innate immunity	Adaptive immunity
First line in host defense Operates when pathogens succeed in penetrating the skin or mucous membranes	Second line in host defense
Generally lacks specificity	Highly specific for a particular pathogen i.e. antigen-specific
Generally lacks memory	Possesses memory i.e. the onset of the response is fast successive exposures to the pathogen
Dendritic cells, mast cells, monocytes/ macrophages, natural killer (NK) cells, polymorphonuclear leukocytes (PMNL) cells are primarily involved	Antigen-presenting cells, T lymphocytes and B lymphocytes are the major cellular components Composed of cells, antimicrobial chemicals, but no physical barriers

Host defense mechanism



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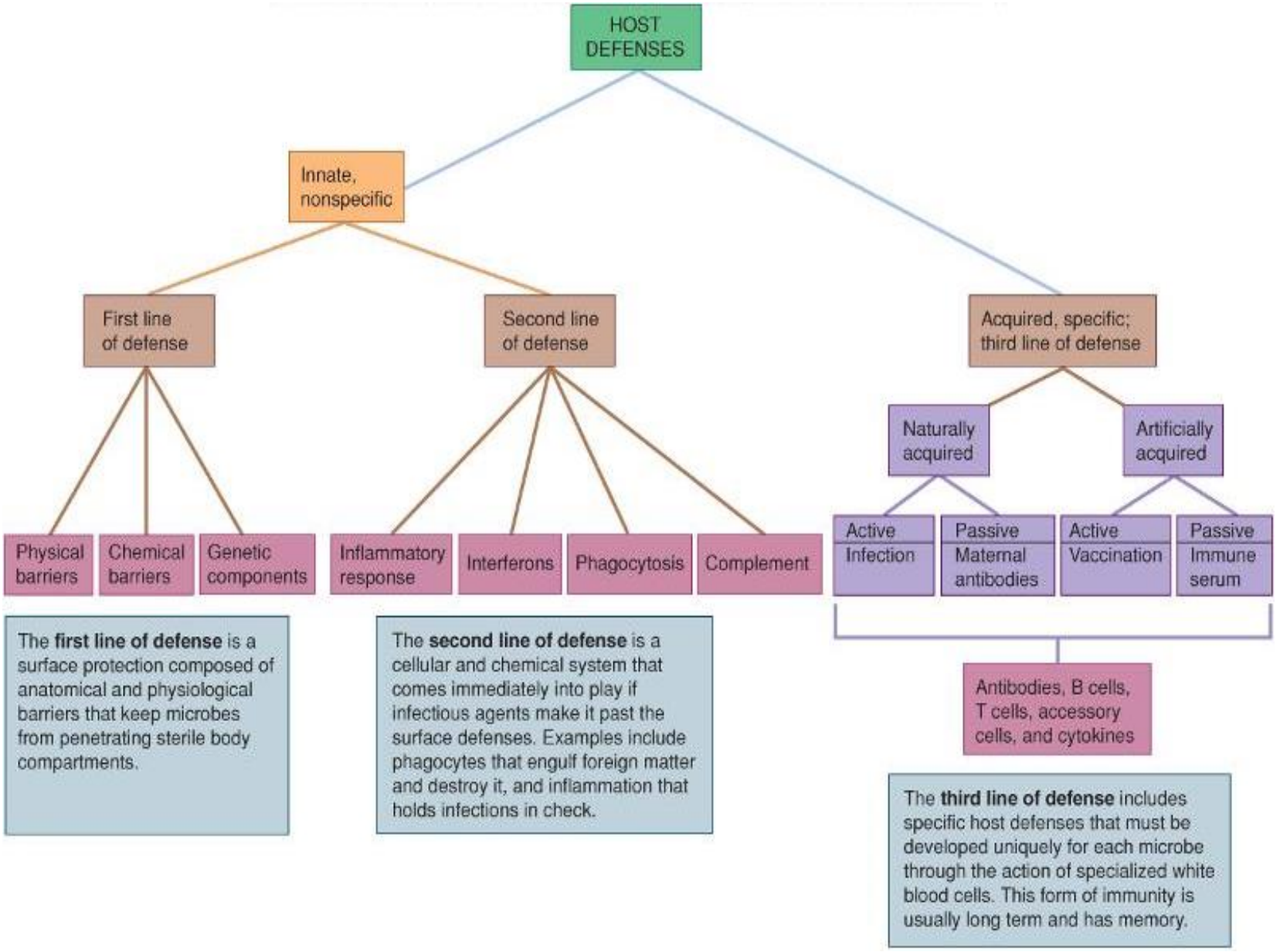
HOST DEFENSES
Innate, nonspecific
Acquired, specific; third line of defense
First line of defense
Second line of defense
Naturally acquired
Artificially acquired
Physical barriers
Chemical barriers
Genetic components
Inflammatory response
Interferons
Phagocytosis
Complement
Active Infection
Passive Maternal antibodies
Active Vaccination
Passive Immune serum
Antibodies, B cells, T cells, accessory cells, and cytokines
The first line of defense is a surface protection composed of anatomical and physiological barriers that keep microbes from penetrating sterile body compartments.
The second line of defense is a cellular and chemical system that comes immediately into play if infectious agents make it past the surface defenses. Examples include phagocytes that engulf foreign matter and destroy it, and inflammation that holds infections in check.
The third line of defense includes specific host defenses that must be developed uniquely for each microbe through the action of specialized white blood cells. This form of immunity is usually long term and has memory.

Host defense mechanism

1. EXTERNAL (PRIMARY): Physical barrier of gross surface area; e.g., skin, respiratory tract, gastrointestinal tract, genitourinary tract

- **Mechanical and Physical Factors:** sweat, fatty acids, pH, indigenous competitive flora (microbial antagonism), peristalsis, hair, cilia, urinary flushing, mucus, [tears, nasal secretions, saliva (lysozyme)], semen (spermine), mucosal secretory antibody (IgA predominant)

Host defense mechanism

2. INTERNAL (SECONDARY): When an infecting parasite succeeds in penetrating the skin or mucuos membranes, cellular defense mechanisms include local macrophages and blood-borne phagocytic cells. Mononuclear phagocytes (**monocytes** and **macrophages**) and **polymorphonuclear leukocytes** (**PMNs**) are the most important phagocytic cells targeting bacterial infections.

- **MONONUCLEAR PHAGOCYTE SYSTEM:** total pool of monocytes and cells derived from monocytes; predominantly **macrophages** (phagocytic cells)

Host defense mechanism

Other types of host defence mechanism:

NON-SPECIFIC: oxygen metabolites (superoxide anion radical, hydrogen peroxide, hydroxyl radicals, halide radicals), kinin forming system related to **clotting**

HOST-GENERATED PROTEINS: complex array of **humoral and cellular mediators**; e.g., lysosomal enzymes, lipid mediators, prostaglandins, histamine, heat-shock proteins (stress proteins)

Host defense mechanism

- **CELLULAR IMMUNE RESPONSE:** any immune response directed at the cellular level; includes **INFLAMMATION** and **PHAGOCYTOSIS** processes

INFLAMMATORY RESPONSE: a protective response of tissues affected by disease or injury characterized by **redness**, localized **heat**, **swelling**, **pain**, and possibly **impaired function** of the infected part

PHAGOCYTOSIS: the process by which certain phagocytes can **ingest extracellular particles** by engulfing them; particles **OPSONIZED** with antibody are more rapidly and efficiently ingested

T-LYMPHOCYTES and **CYTOKINES**

Host defense mechanism

HUMORAL IMMUNE RESPONSE: the sum total of components of the immune response circulating in the blood or body fluids ; includes **ANTIBODY** and **COMPLEMENT** systems

B-LYMPHOCYTES and **ANTIBODY PRODUCTION:**
a class of proteins produced as a result of the introduction of an antigen that has the ability to combine with the antigen that caused its production

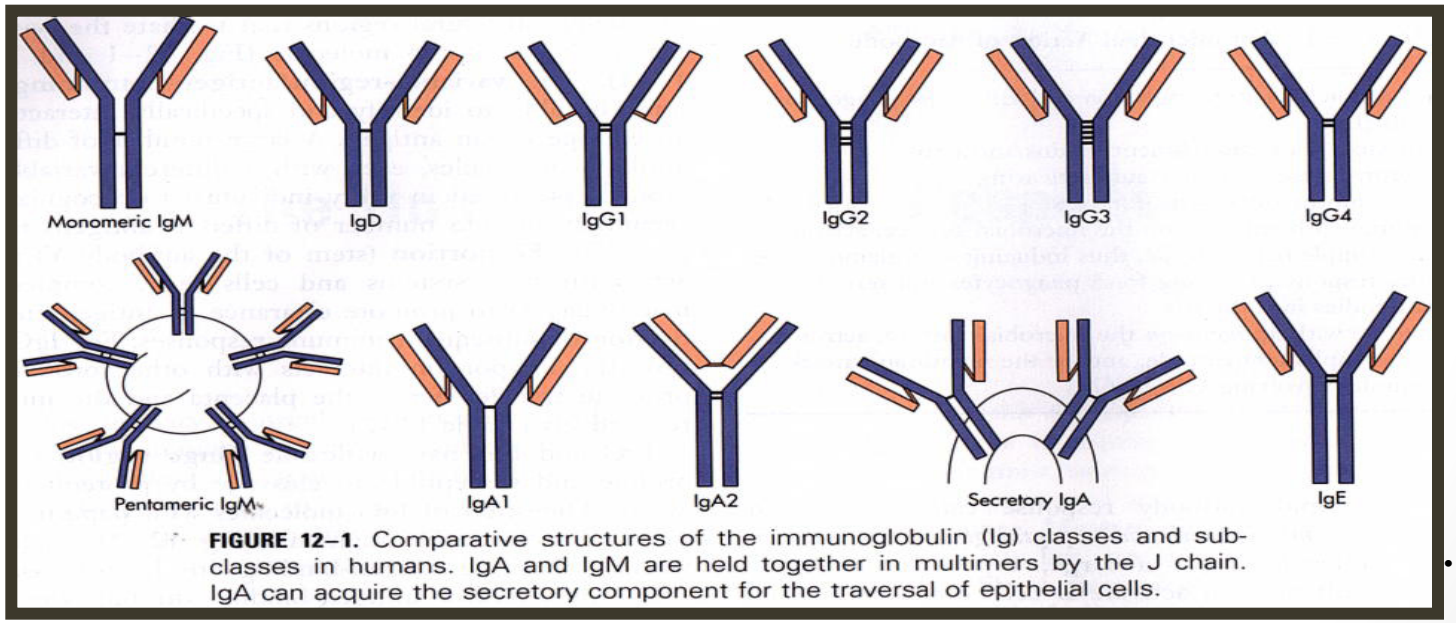

FIGURE 12–1. Comparative structures of the immunoglobulin (Ig) classes and subclasses in humans. IgA and IgM are held together in multimers by the J chain. IgA can acquire the secretory component for the traversal of epithelial cells.

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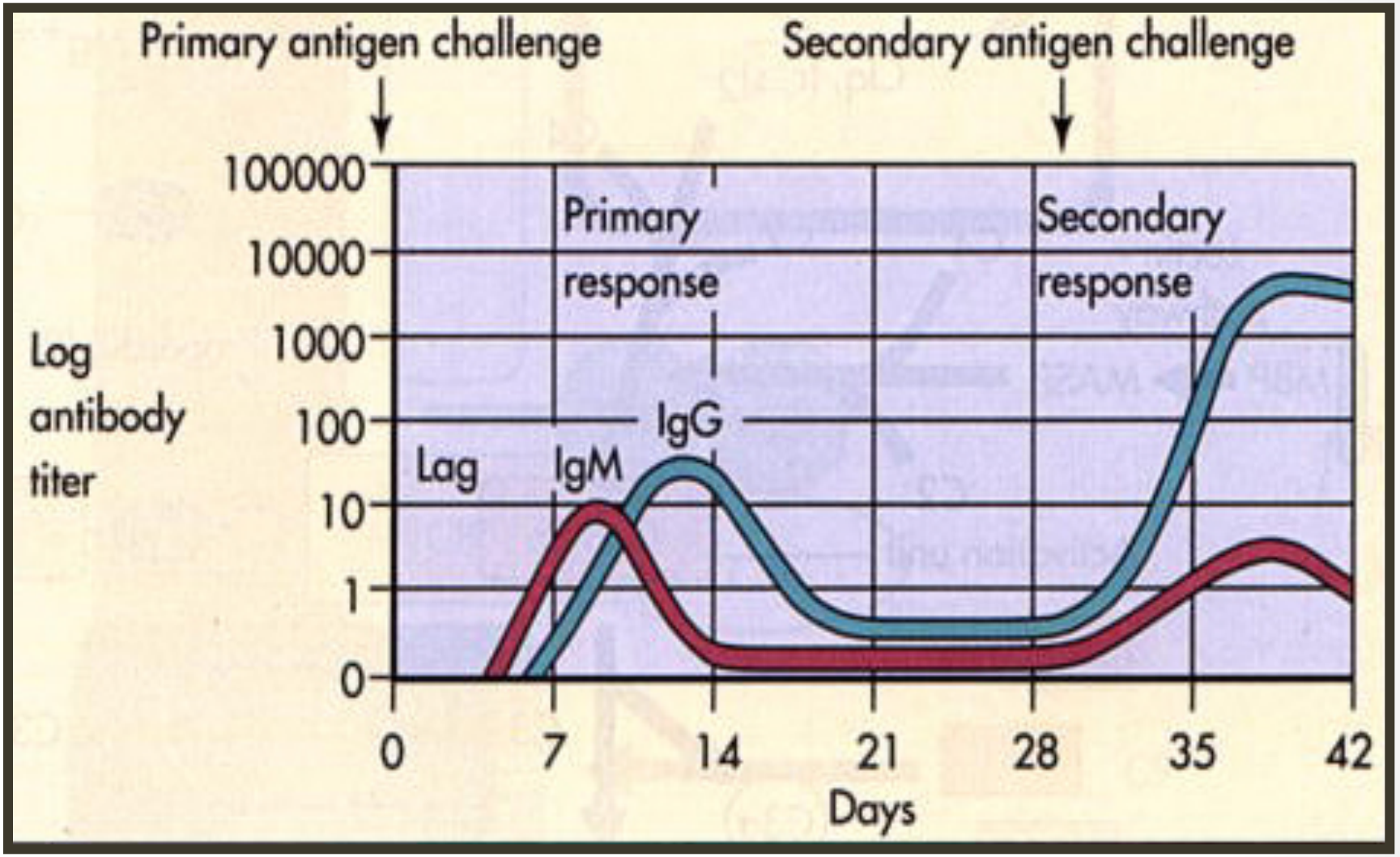
Primary antigen challenge
Secondary antigen challenge
Log antibody titer
100000
10000
1000
100
10
1
0
Primary response
Secondary response
IgG
IgM
Lag
0
7
14
21
28
35
42
Days

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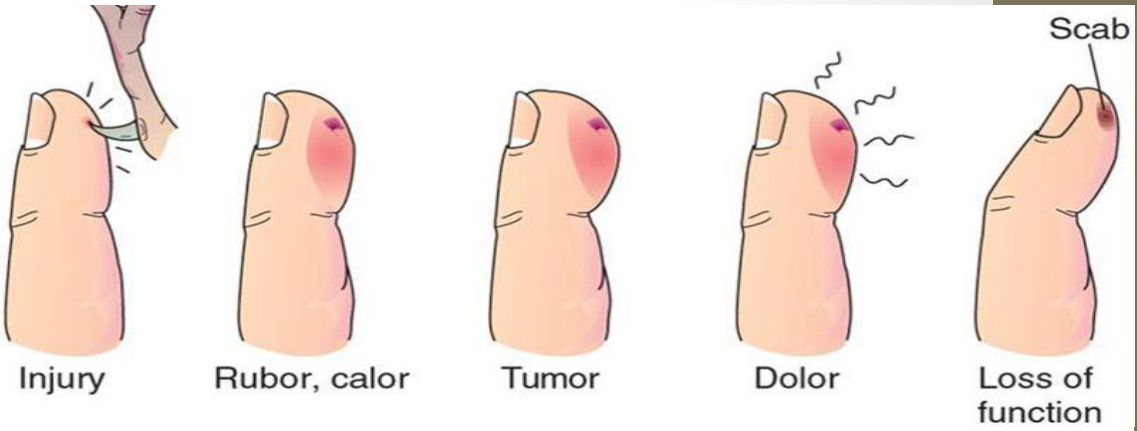
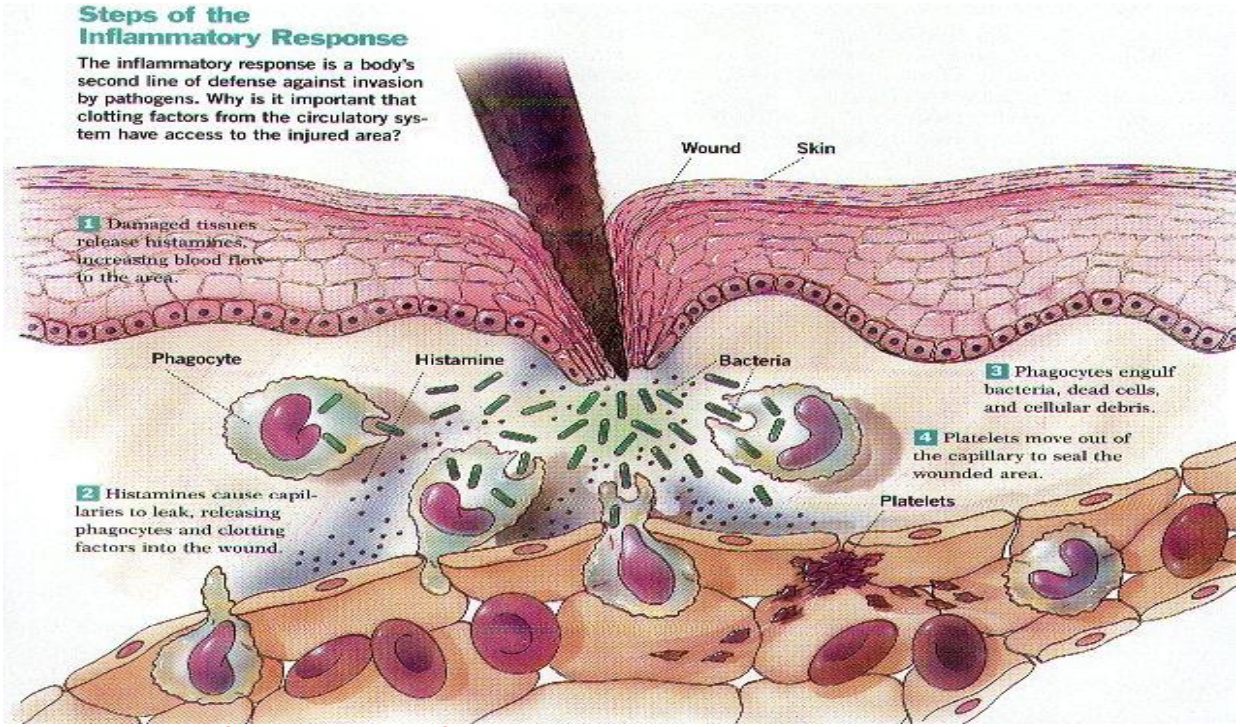
Signs of Inflammation

Inflammation is characterized by **4 signs:** redness, heat, swelling & pain





Inflammatory Response involves several steps:

1) Skin/cells are damaged.

2) Mast cells release Histamine-histamine causes blood vessels to expand. As a side effect, histamine causes itchiness.

3) From the openings of blood vessels, phagocytes (eaters of pathogens), clotting factors, and platelets come out (healing)

Fever is triggered to kill pathogen.

BASIC EFFECTS of ENDOTOXIN

- **FEVER:** any elevation of body temperature above normal
- **LEUKOPENIA/LEUKOCYTOSIS:** abnormal reduction in number of leukocytes in blood, ($\leq$5000/mm^3) / abnormally large number of leukocytes in blood, as during hemorrhage, infection, inflammation, or fever ($\geq$12,000mm^3)
- **METABOLIC EFFECTS :** pathogenic organisms can affect any of the body systems with disruptions in metabolic processes, e.g., hypotension, hypoglycemia, etc.
- **RELEASE OF LYMPHOCYTE FACTORS:** a granular leukocyte concentrated in lymphoid tissue; active in immunological responses, including production of antibodies

BASIC EFFECTS of ENDOTOXIN

- **CELLULAR DEATH:**

  SEPTIC SHOCK: associated with overwhelming infection resulting in vascular system failure with sequestration of large volumes of blood in capillaries and veins; activation of the complement and kinin systems and the release of histamines, prostaglandins, and other mediators may be involved

  DISSEMINATED INTRAVASCULAR COAGULATION (DIC): disorder characterized by a reduction in the elements involved in blood coagulation due to their utilization in widespread blood clotting within the vessels; late stages marked by profuse hemorrhaging

  ORGAN NECROSIS: the sum of morphological changes indicative of cell death and caused by the progressive degradative action of enzymes