CHS 2413
Pathology and Physiopathology

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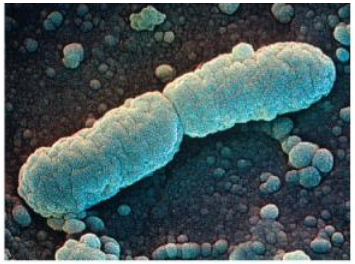
Immunopathology

Defense Against Disease



Nonspecific External Barriers
skin, mucous membranes

If these barriers are penetrated,
the body responds with

Innate Immune Response
phagocytic and natural killer cells,
inflammation, fever

If the innate immune response is insufficient,
the body responds with

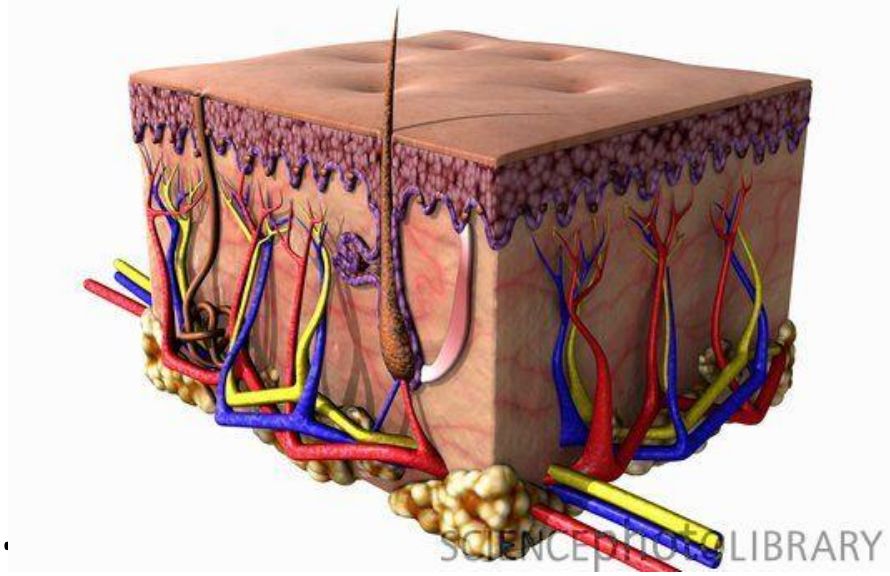
Adaptive Immune Response
cell-mediated immunity, humoral immunity

First line of defense

- Non-specific defenses are designed to prevent infections by viruses and bacteria. These include:
  - Intact skin
  - Mucus and Cilia
  - Phagocytes

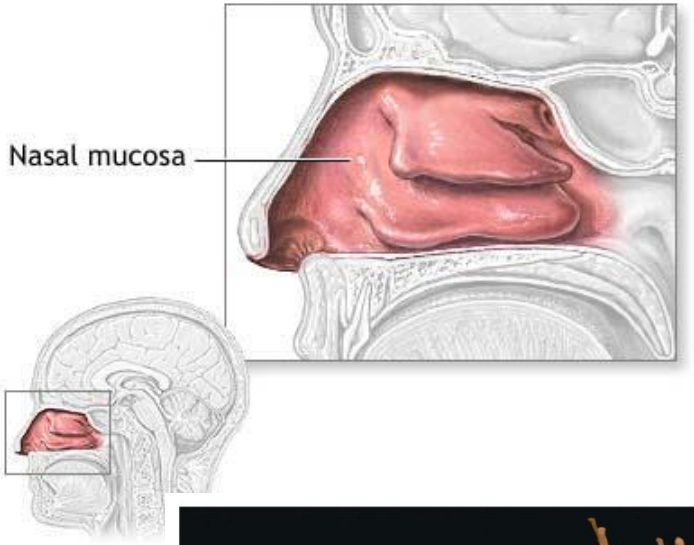
Role of skin

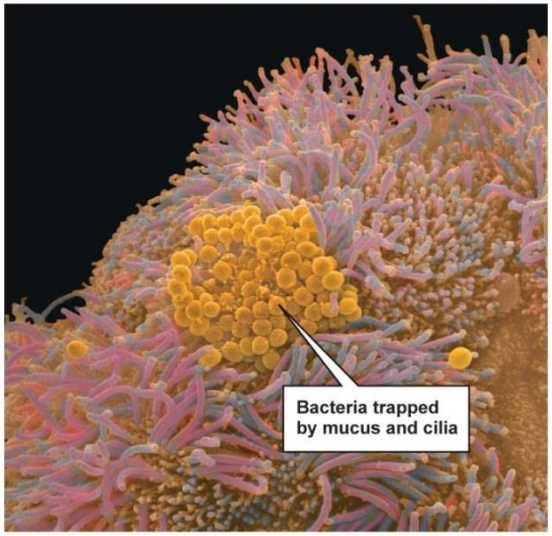
- Dead skin cells are constantly sloughed off, making it hard for invading bacteria to colonize.
- Sweat and oils contain anti-microbial chemicals, including some antibiotics.



Role of mucus and cilia

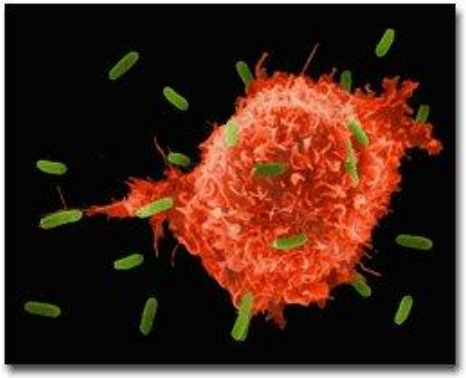
- Mucus contains lysozymes, enzymes that destroy bacterial cell walls.
- The normal flow of mucus washes bacteria and viruses off of mucus membranes.
- Cilia in the respiratory tract move mucus out of the lungs to keep bacteria and viruses out.

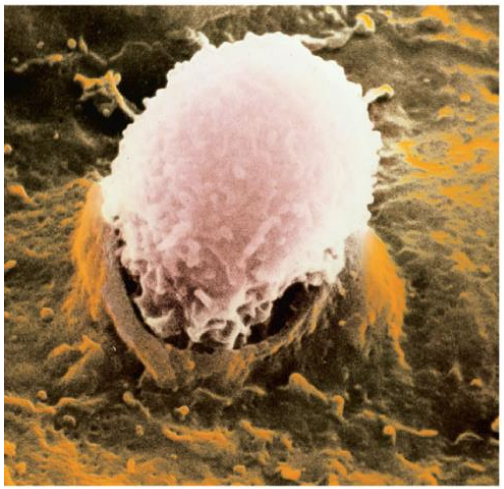




Role of phagocytes

- Phagocytes are several types of white blood cells (including macrophages and neutrophils) that seek and destroy invaders. Some also destroy damaged body cells.
- Phagocytes are attracted by an inflammatory response of damaged cells.





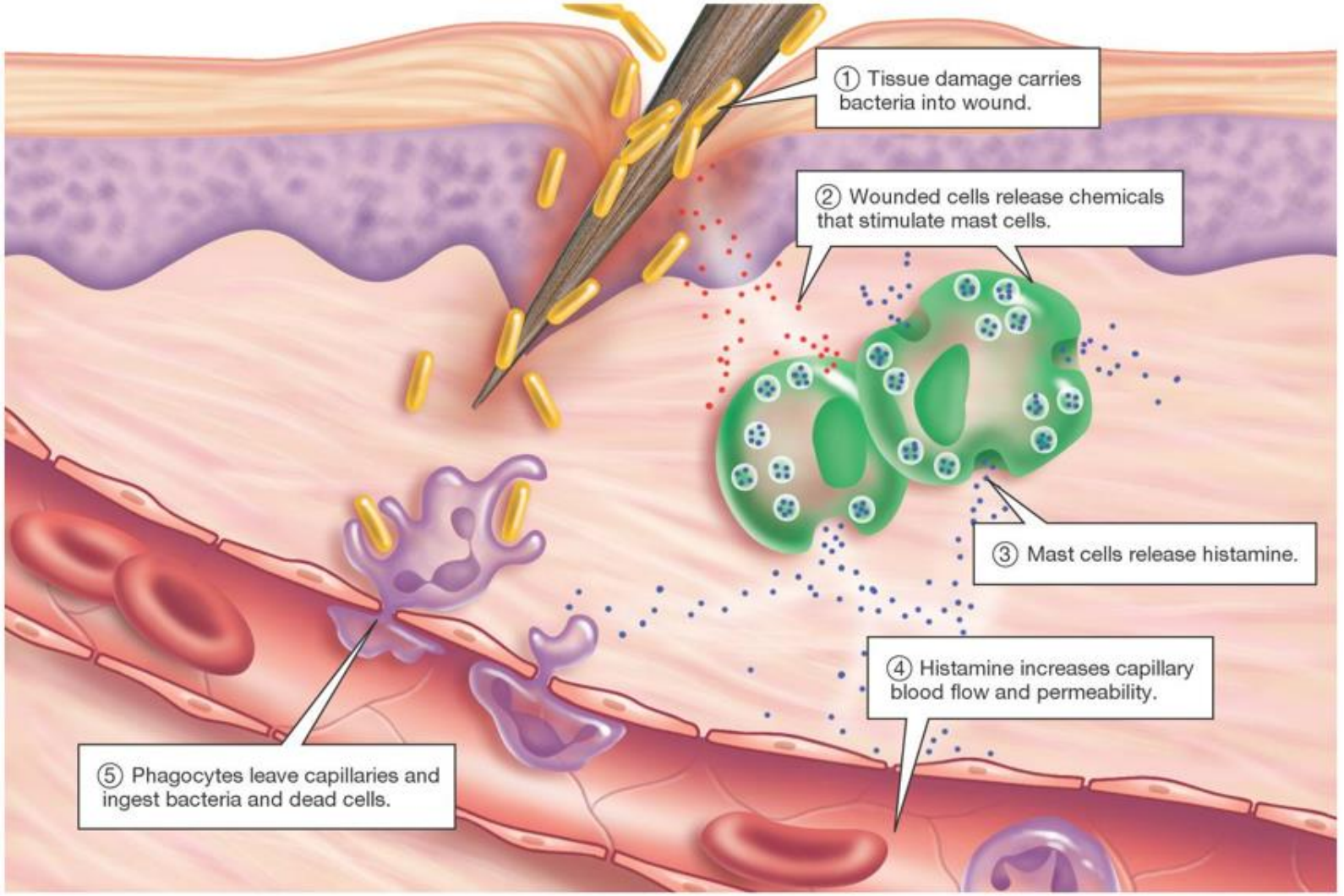
Role of inflammation

- Inflammation is signaled by mast cells, which release histamine.
- Histamine causes fluids to collect around an injury to dilute toxins. This causes swelling.
- The temperature of the tissues may rise, which can kill temperature-sensitive microbes.

Role of fever

- Fever is a defense mechanism that can destroy many types of microbes.
- Fever also helps fight viral infections by increasing interferon production.
- While high fevers can be dangerous, some doctors recommend letting low fevers run their course without taking aspirin or ibuprofen.
- Fever is caused by your body's pyrogens signaling the hypothalamus
- a person with AIDS have a fever if they catch the flu because fever has nonspecific response.

Ouch!


① Tissue damage carries bacteria into wound.
② Wounded cells release chemicals that stimulate mast cells.
③ Mast cells release histamine.
④ Histamine increases capillary blood flow and permeability.
⑤ Phagocytes leave capillaries and ingest bacteria and dead cells.

Specific defenses

- Specific defenses are those that give us immunity to certain diseases.
- In specific defenses, the immune system forms a chemical "memory" of the invading microbe. If the microbe is encountered again, the body reacts so quickly that few or no symptoms are felt.

Innate and Adaptive Immunity

- Innate Immunity (Inherit, natural , native)

  First line of defense

  Always ready to prevent and eradicate infection

  Non-specific

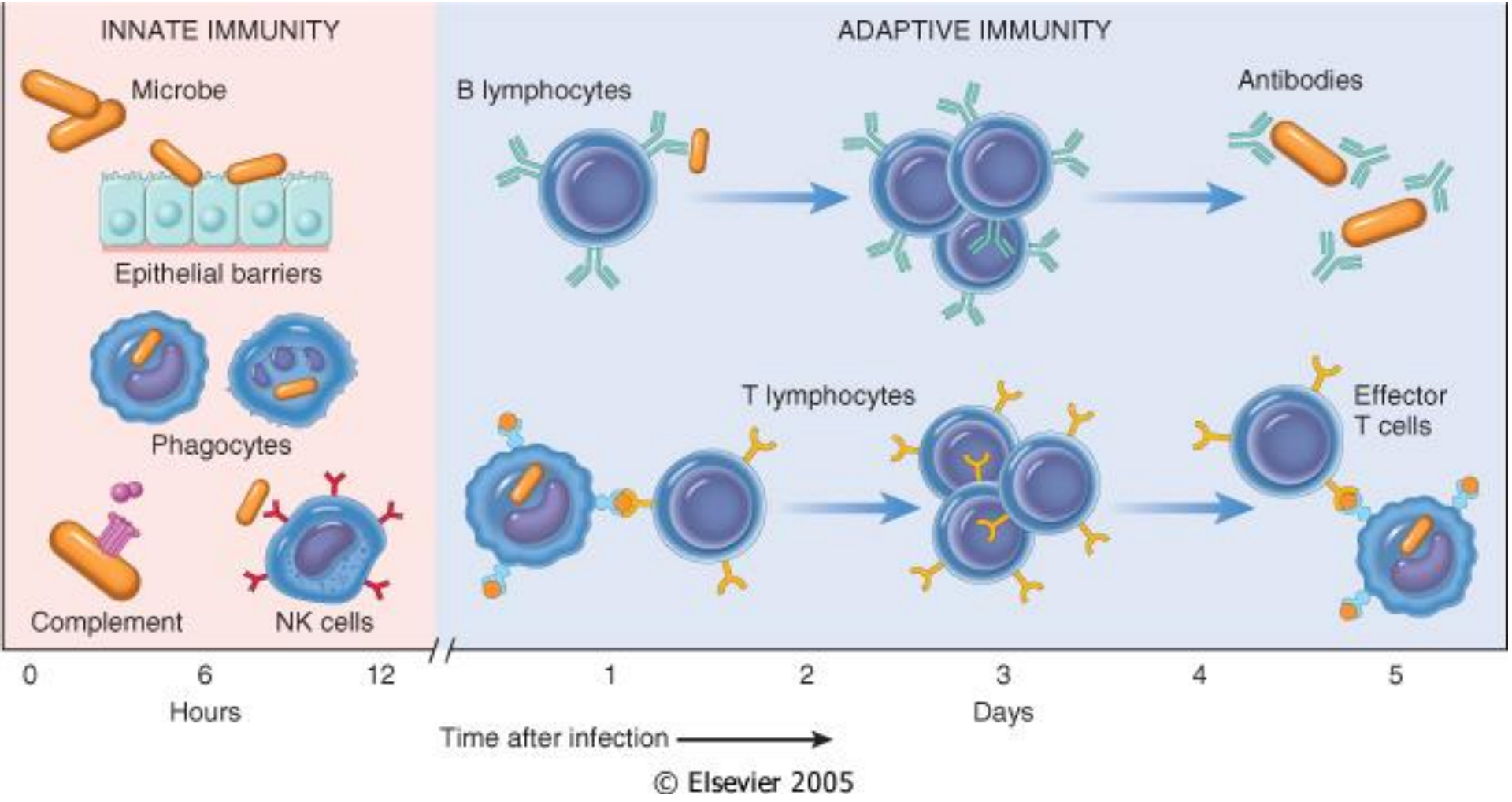
- Adaptive Immunity (acquired- , specific-)

  Develops later after exposure to microbes

  More powerful in combating infections

  Specific

Innate and Adaptive Immunity



Innate and Adaptive Immunity

Innate Immunity	Adaptive Immunity
• Phagocytes: Neutrophil, Macrophage • Microbes are internalized and destroyed by reactive oxygen, hydrolytic enzyme, resulting in inflammation. • Complement proteins: most important plasma proteins of the innate immunity • Lung surfactant, epithelial, mucus, cilia, cough reflex, HCl	• Mainly consists of lymphocytes and their products, including antibodies Humoral immunity • Defense against extracellular microbes and their Toxins Cell-mediated (cellular) immunity • Defense against intracellular microbes

Major players

- The major players in the immune system include:
  - Macrophage
  - T cells (helper, cytotoxic, memory)
  - B cells (plasma, memory)
  - Natural Killer Cells
  - Dendritic Cells (Langerhans cell)
  - Antibodies

Immunopathology

- Immunopathology: Diseases of the natural defense system that usually resists infections
- Immunity: Resistance to infection via the immune response to any substance perceived as foreign
  Therefore, not always protective

Immune Response

Inherited

- Independent of previous exposures to foreign substances

Include

- Mechanical barriers
- Phagocytic cells
- Natural killer cells
- Protective proteins

Acquired

- Based upon specific responses elicited by substances that act as antigens
- Antigens
- Chemical substance that can induce a specific immune response
- Immune system activated to destroy the antigen
- Anti bodies are formed

The specific immune response is triggered when A macrophage delivers an antigen to a T-helper cell

Cells of the Immune System

Lymphocytes

- Derived from bone marrow pre-lymphoid stem cells
- Primary lymphoid organs
  - Thymus: T lymphocytes mature here
  - Bone marrow: B lymphocytes remain here
- Secondary lymphoid organs: Colonized by T and B lymphocytes once they enter blood stream
  - Lymph nodes
  - Spleen
  - Gastrointestinal and Bronchial mucosa

T Lymphocytes

Lymphocytes that have matured in the thymus

- T helper /inducer: Actively participate in the immune response to antigens, helping B cells produce antibodies
- T suppressor/cytotoxic cells: Suppress unwanted antibody production. Mediate killing cells that are recognized by the body as foreign
- NK cells: Natural killer cells10-15% of peripheral blood lymphocytes. Not involved in T and B cell mediated immune reactions. React against virus-infected cells and to kill tumor and foreign cells without previous infection

Variant polypeptide chains of T cells

- CD3 molecular complex (Cluster of Differentiation-3)
- Other expressed molecules: CD4, CD8 serve as co-receptors for T-cell stimulation
- Normally, CD4/CD8 ratio = 2 : 1

T-Cell

- Constitute 60-70% of the lymphocytes in circulating blood, also in lymphoid organs
- Memory T-cells are formed, which can quickly divide and produce cytotoxic T-cells to quickly fight off the invader if it is encountered again in the future.
- While B-cells divide and differentiate, so do T-cells.
- Some T-cells become cytotoxic, or “killer” T-cells. These T-cells seek out and destroy any antigens in the system, and destroy microbes “tagged” by antibodies.
- Some cytotoxic T-cells can recognize and destroy cancer cells.
- Helper T-cells have receptors for recognizing antigens. If they are presented with an antigen, they release cytokines to stimulate B-cell division.
- The helper T-cell is the key cell to signal an immune response. If helper T-cells are disabled, as they are in people with AIDS, the immune system will not respond.

B Lymphocytes

- 10-20% in circulating blood, bone marrow, lymph nodes, spleen, tonsils, GI tract
- After stimulation, B cells form "plasma cells" that secrete immunoglobulins (antibody)
- 5 basic Immnuglobulins isotypes: IgG, IgM, IgA, IgE, IgD
- Lymphocytes that primed to differentiate into immunoglobulin-producing plasma cells

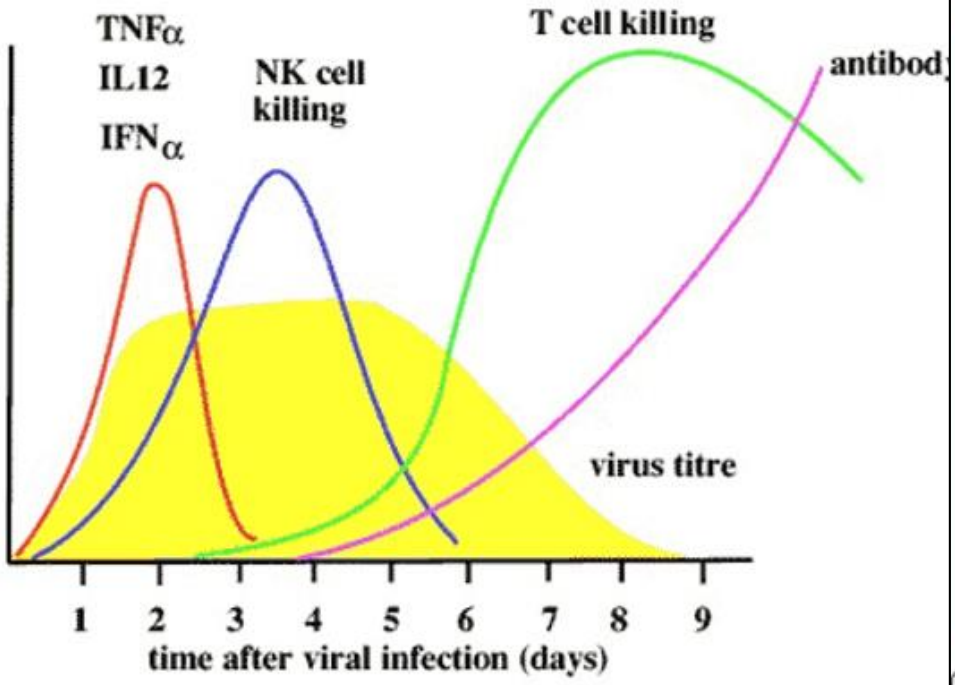
Plasma cells

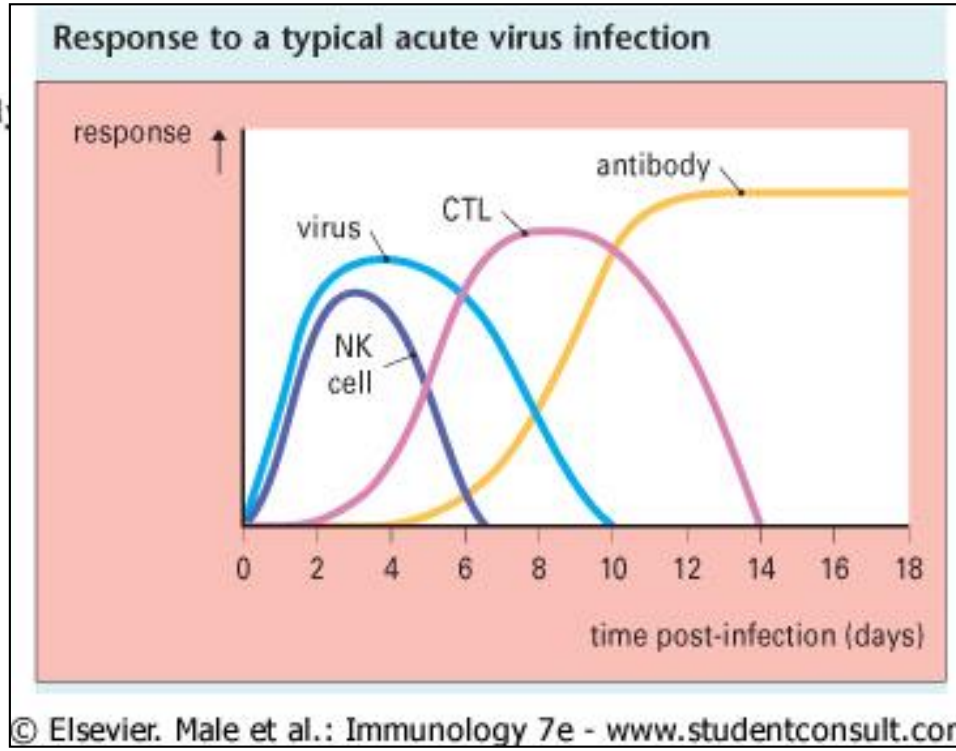
- Mature descendants
- Contain an abundance of ribosomes
- Secrete immunoglobulins

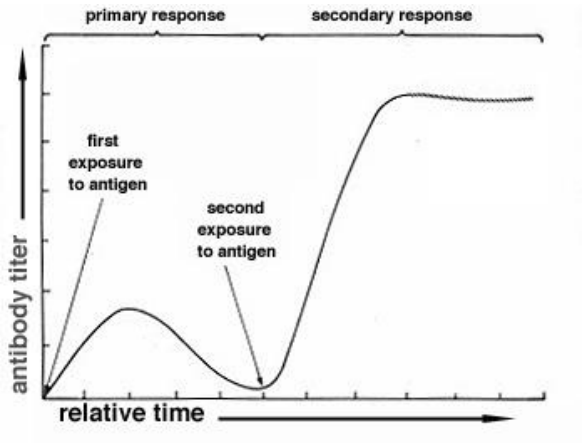
Antibodies

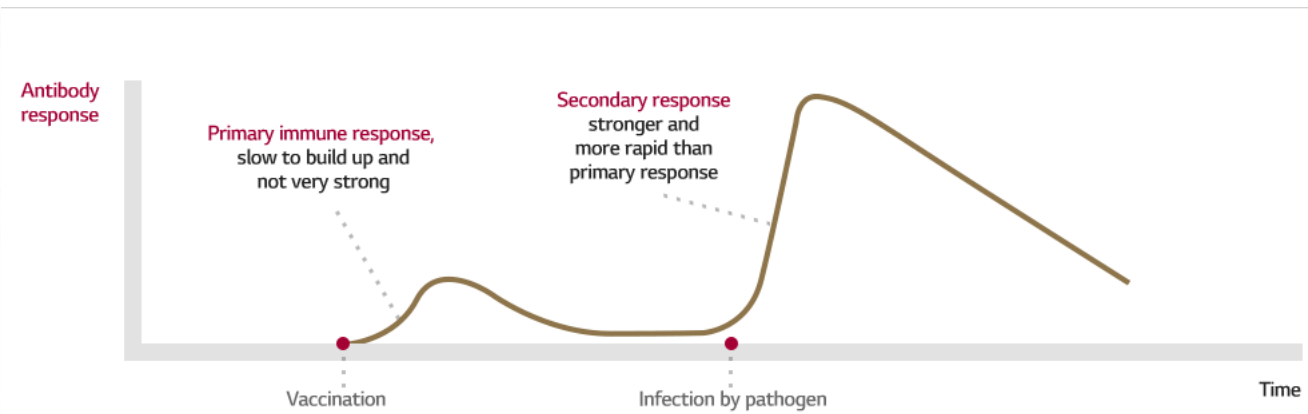
- Antibodies are assembled out of protein chains.
- Antibodies: Serum proteins of the immunoglobulins secreted by the plasma cells
- Antibody Production: Begins with contact between antigen and the cells of the immune system
- There are many different chains that the immune system assembles in different ways to make different antibodies.
- Antibodies can attach to B cells, and serve to recognize foreign antigens
- Antibodies released into the blood stream will bind to the antigens that they are specific for.
- Antibodies may disable some microbes, or cause them to stick together (agglutinate). They "tag" microbes so that the microbes are quickly recognized by various white blood cells.
- B-cells in general produce antibodies. Those with antibodies that bind with the invader's antigen are stimulated to reproduce rapidly.
- B-cells differentiate into either plasma cells or memory B-cells. Plasma cells rapidly produce antibodies. Memory cells retain the "memory" of the invader and remain ready to divide rapidly if an invasion occurs again.

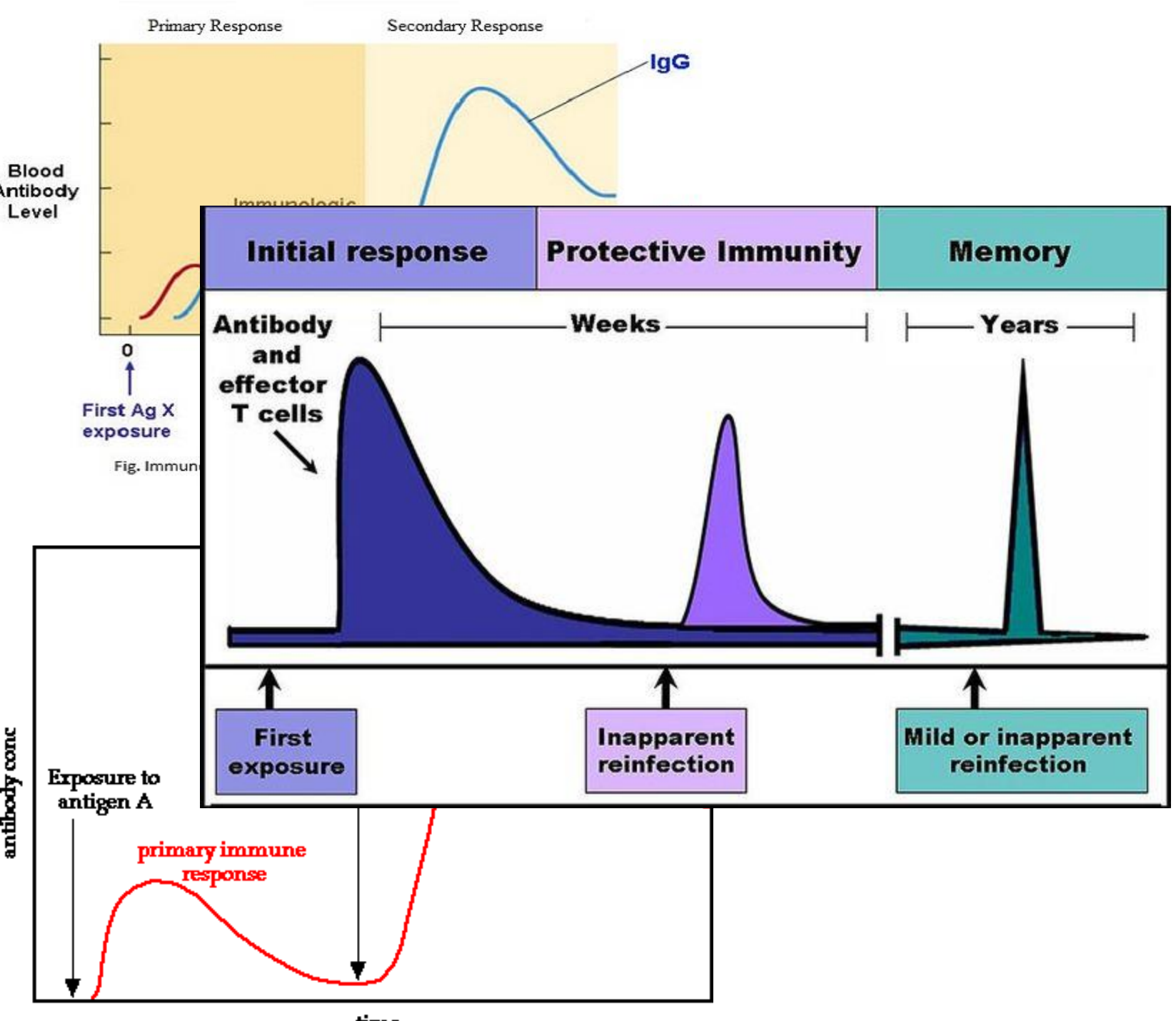
Immune response to Infectious Agents









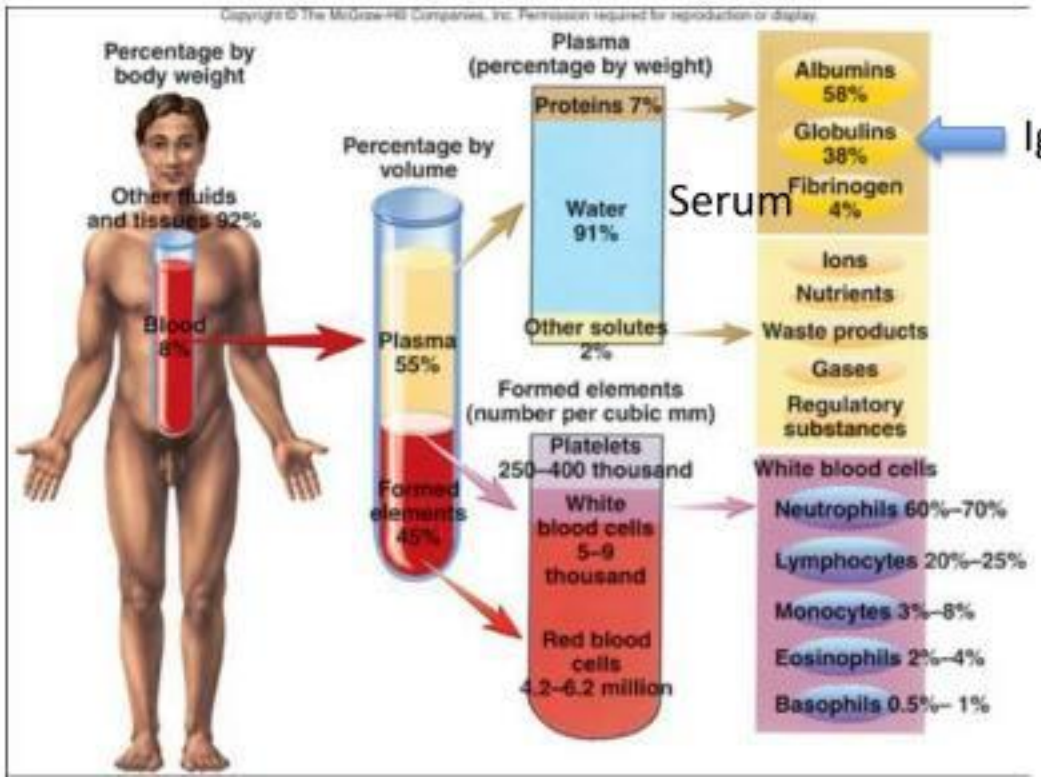

Primary Response
Secondary Response
IgG
Blood Antibody Level
0
First Ag X exposure
Initial response
Protective Immunity
Memory
Antibody and effector T cells
Weeks
Years
First exposure
Inapparent reinfection
Mild or inapparent reinfection
antibody conc
Exposure to antigen A
primary immune response

Antibody (Ab)

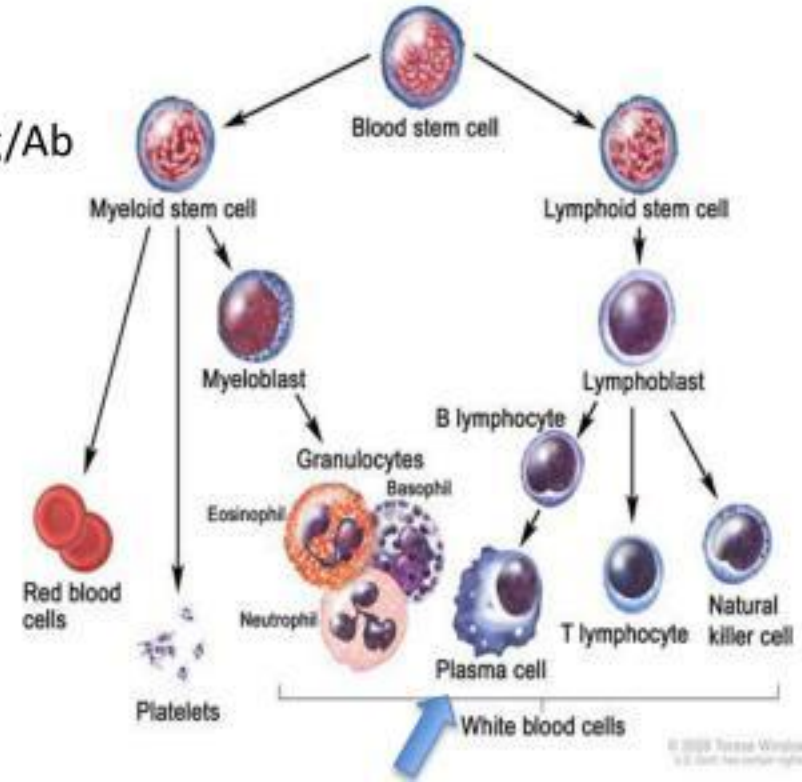
คือสารไกลโคโปรตีน (Glycoprotein) ที่อยู่ในเลือด (blood serum) ซึ่งสร้างมาจาก Plasma cells (WBC) เกิดจากการตอบสนองของระบบภูมิคุ้มกันต่อ antigenic determinant ที่แปลกปลอม

Ab ส่วนใหญ่อยู่ใน serum ส่วน γ-globulin และเนื่องจาก Ab เป็น globulin ที่ทำหน้าที่เกี่ยวกับภูมิคุ้มกันของร่างกายจึงเรียกว่า Immunoglobulin (Ig)

Ab ทำหน้าที่ต่อสู้หรือป้องกันการติดเชื้อโรคจากแบคทีเรีย ไวรัส หรือสิ่งแปลกปลอมอื่นๆที่เข้ามาในร่างกาย โดยจะทำปฏิกริยาที่มีความจำเพาะต่อแอนติเจน (Ag)

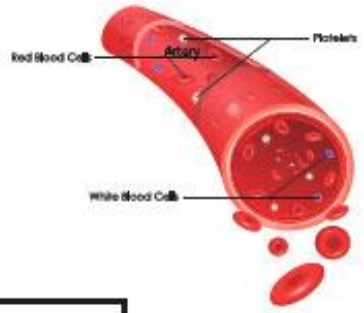


Ig/Ab



The Immune System
Our Ultimate Line of Defence

Immune System



External
(Skin, mucous membrane, nasal hair)
Barriers

Internal
(Immunological cells)
Barriers

1st Line Defense

- **Phagocytes**
  - Inflammation
  - Identify & Exterminate
- **Natural Killer Cells**
  - Attack mutated/cancer cells

Innate Immunity

2nd Line Defense

- **T Cells**
  - Receive message from Phagocytes, eliminate enemies, inform B cells
- **B Cells**
  - Produce antibodies, stores memory

Adaptive Immunity

Adaptive Force

Cy

Cytotoxic T cell

อัศวินหนุ่มผู้เป็นโรคขาดความมั่นใจ...

One

T helper 1

สาวเชียร์ผู้คอยให้กำลังใจ Cy อยู่เสมอ

B

B cell

จอมเวทสุดชิลที่วันๆ เอาแต่นอน

Two

T helper 2

ฝาแฝดของ One ที่คอยเคี่ยวเข็ญ B ให้ทำงาน

Reg

Regulatory T cell

พี่สาวใจดีที่คอยดูแลทุกคนไม่ให้ทำเกินกว่าเหตุ

Innate Force

Mas

Mast cell

นักเคมีมือระเบิดสุดขวัญอ่อน

Den

Dentritic cell

สายสืบแห่งสนามรบผู้เก็บหลักฐานมือฉมัง

Kil

Natural Killer cell

นายทหารผ่านศึกผู้มีสัมผัสอันเฉียบคม

Mac

Macrophage

นักรบแนวหน้าผู้มีรสนิยมในการกินอันพิสดาร...

"N"

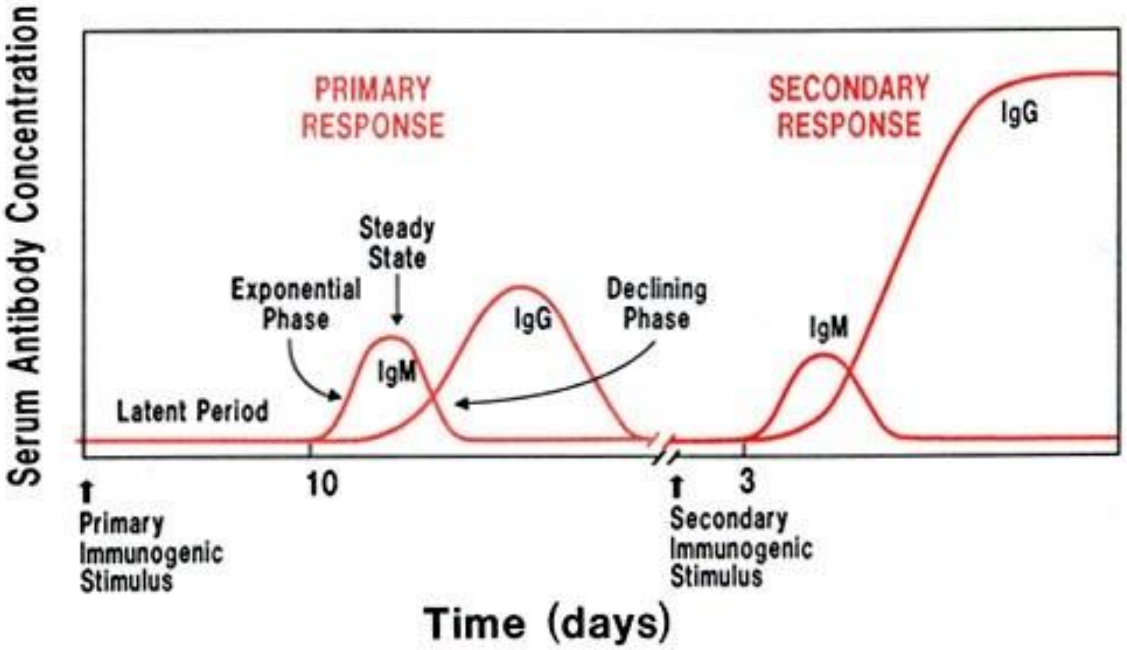
Neutrophi

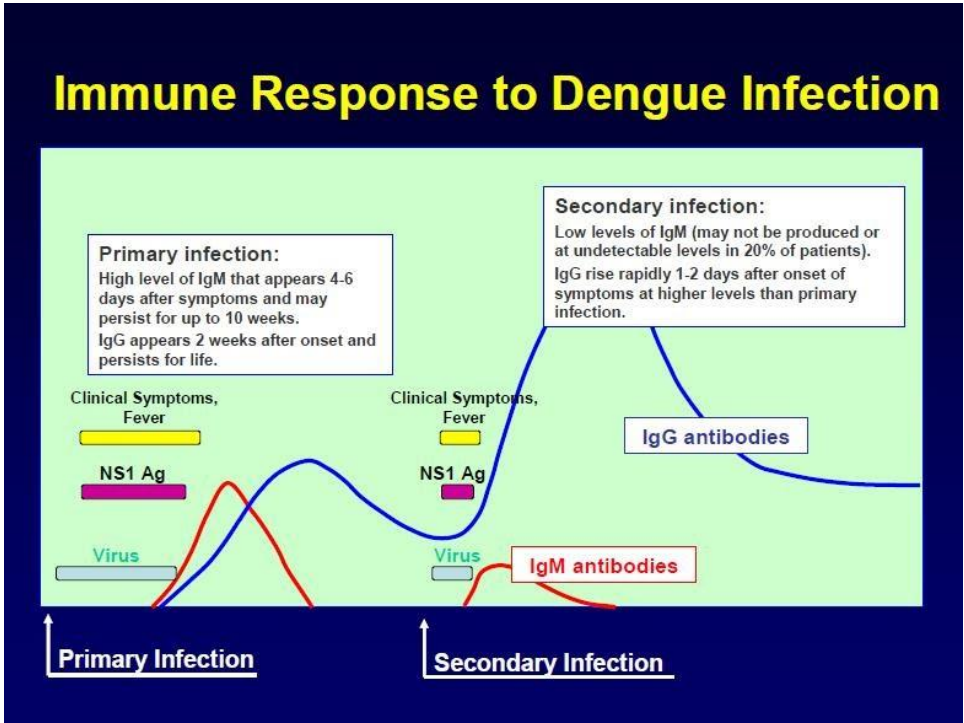
ทหารตัวประกอบที่พร้อมจะมาตายเป็นเบือ

"C"

Complement

ผู้ช่วยสำคัญของเหล่าImmune Force

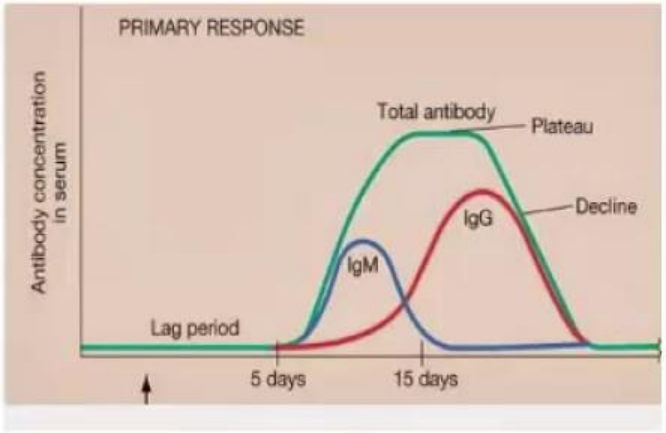

Serum Antibody Concentration
PRIMARY RESPONSE
SECONDARY RESPONSE
IgG
Steady State
Exponential Phase
Declining Phase
IgG
IgM
IgM
Latent Period
10
3
Primary Immunogenic Stimulus
Secondary Immunogenic Stimulus
Time (days)

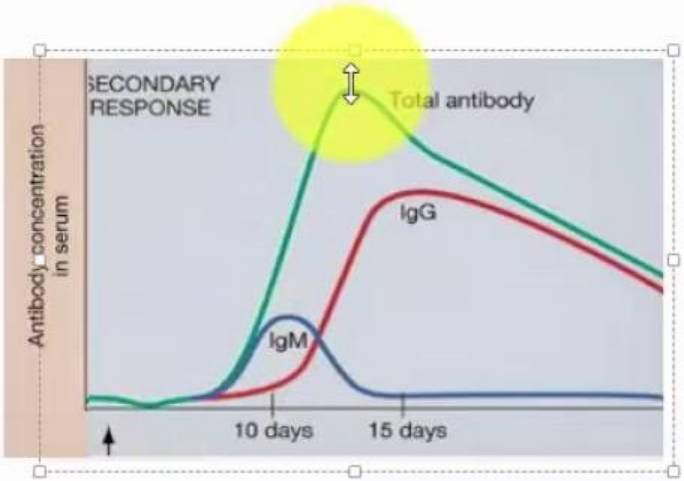

Immune Response to Dengue Infection
Primary infection:
High level of IgM that appears 4-6 days after symptoms and may persist for up to 10 weeks.
IgG appears 2 weeks after onset and persists for life.
Secondary infection:
Low levels of IgM (may not be produced or at undetectable levels in 20% of patients).
IgG rise rapidly 1-2 days after onset of symptoms at higher levels than primary infection.
Clinical Symptoms, Fever
NS1 Ag
Virus
Clinical Symptoms, Fever
NS1 Ag
Virus
IgG antibodies
IgM antibodies
Primary Infection
Secondary Infection

PRIMARY AND SECONDARY IMMUNE RESPONSE

08 March 2017 12:42

Primary	Secondary	Characteristics
1.First exposure to a specific Antigen .	1.After second exposure to the same antigen	Exposure to antigen
2.1 Week delay	2.Within hours	Time of onset
3.Low affinity antibodies are Made .	3.High affinity antibodies are made.	Affinity of antibodies
4. Response is generally smaller.	4.Usually larger	Strength
5.Activated by all Immunogens.	5.Mostly by peptide antigens.	Activation agent
6.Short life ,for only few weeks. 7.IgM	6.Forms antibodies for many months. 7.IgG	Duration





HYBEING

COLOSTRUM

เสริมสร้างภูมิคุ้มกันอย่างมีประสิทธิภาพ

IgG
เสริมสร้างภูมิคุ้มกัน
อย่างมีประสิทธิภาพ

IgE
ป้องกันต่อต้าน
เชื้อปรสิตในลำไส้
และอยู่ในปฏิกิริยา
ความไวต่อนม

IgM
ต่อสู้เชื้อโรคเพิ่มความสามารถ
ในการกลืนกินและกำจัดการ
รุกรานของเชื้อโรค จึงทำลาย
เชื้อโรคที่เข้ามาบุกได้

IgA
ด่านแรกของการป้องกัน
เชื้อโรคเข้าสู่ร่างกาย พบใน
เยื่อบุผิวช่องทางเดินอาหาร
ทางเดินหายใจ และป้องกัน
การจู่โจมของเชื้อโรค

IgD
กระตุ้นเซลล์
เม็ดเลือดขาวให้ผลิต
สารภูมิต้านทานต่างๆ

LINE

Hypersensitivity Reactions

- An abnormal immune response to exogenous antigens
- A reaction to endogenous auto-antigens
- Basis of hypersensitivity diseases

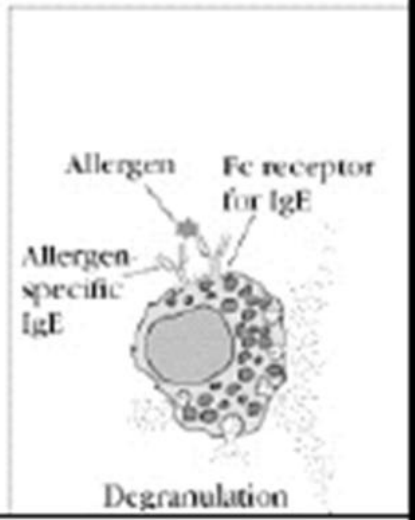
Hypersensitivity Reactions (4 types)

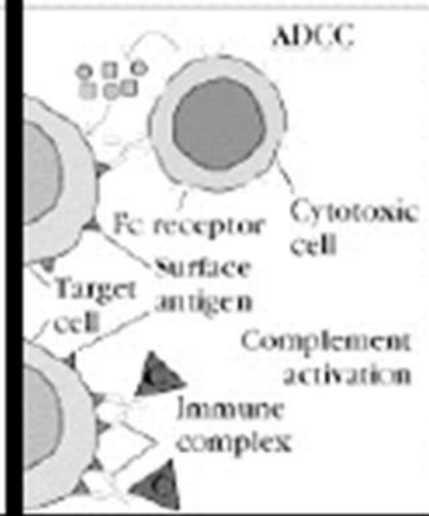
Type I : Allergy and Anaphylaxis
Type II : Antibody Dependent
Type III : Immune Complex – Mediated
Type IV : Cell – Mediated

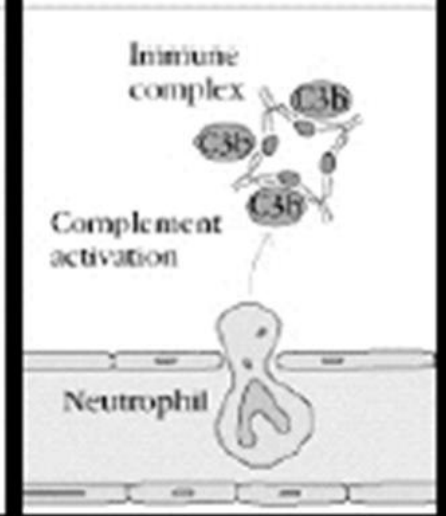
Transplant Rejection

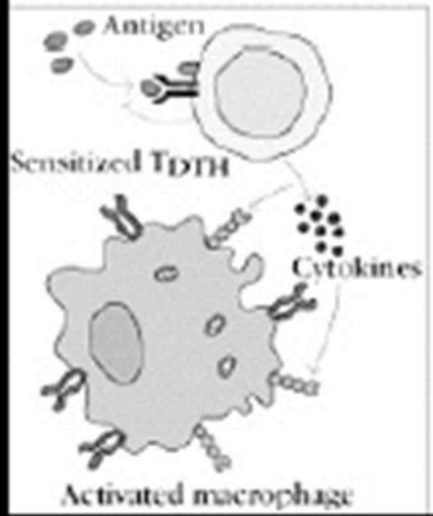
Hyperacute rejection (within minutes – few hrs)
Acute rejection (within days – weeks)
Chronic rejection (within months – years)

Gel and Coombs classification of hypersensitivities.









Type I	Type II	Type III	Type IV
IgE Mediated	IgG/IgM Mediated	IgG Mediated	T cell
Classic Allergy	rbc lysis	Immune complex Disease	Delayed Type Hypersensitivity

Type I: Anaphylactic reaction

- Classic allergy
- Sensitization to foreign antigens
- Triggers release of vasoactive substances histamine
- Occurs 4-6 hours after exposure to allergens but late phase response can also occur
- Hay fever, allergic rhinitis, bronchial asthma, atopic dermatitis
- Repeat stimulation memory cells cause a stronger reaction
- Anaphylactic Shock (type I):Life threatening. Massive release of histamine and other vasoactive substances. Choking (laryngeal edema),Wheezing (bronchial spasm), Pulmonary edema, Systemic circulatory collapse & fainting Hypotension due to vasodilation and increased leakage of fluid from hyperpermeable blood vessels.

Type II-Hypersensitivity: Cytotoxic Antibody Reaction

- RBC lysis
- Mediated by cytotoxic antibodies that react with antigens in cells or tissue components, such as basement membranes.
- Antibody may be Extrinsic or foreign antigens ie.**Drugs, chemicals**
- Hypersensitivity occurs upon re-exposure to the pathogenic antigen Persistent antigens
- Intrinsic ie. **Autoimmune diseases**
- Hemolytic anemia, **Miss match transfusion**
- Grave's Disease **hyperthyroidism**
- Myasthenia Gravis: Severe muscle weakness, mediated by antibodies to the receptor for acetylcholine on the surface of striated muscle cells. No messages reach the muscle

Type III Immune Complex Hypersensitivity

- Mediated by immune complexes that are formed between antigens and appropriate antibodies
- Most reactions are short lived however
- Sustained production of immune complexes leads to chronic conditions e.g. Lupus, Rheumatoid arthritis

Type IV Delayed type hypersensitivity

Type IV Hypersensitivity: Cell mediated hypersensitivity

- Delayed type hypersensitivity
- Takes more than 12 hrs to develop after antigenic challenge
- Examples include: contact dermatitis and tuberculin reaction
- Antigens include large molecules or small molecules (haptens) linked to carrier molecules
- T lymphocytes become sensitive to presence of antigen and produce delayed response when exposed to this antigen
- Tuberculosis granulomas-provide the ability to test to see whether someone has been exposed to TB
- **Contact dermatitis**
- allergy to allergens; **rubber gloves**, poison ivy,

Antigens

- Free antibodies can bind to antigens, which "tags" the antigen for the immune system to attack and destroy
- Cells of the immune system are "trained" to recognize "self" proteins vs. "not self" proteins.
- If an antigen ("not self") protein is encountered by a macrophage, it will bring the protein to a helper T-cell for identification.
- If the helper T-cell recognizes the protein as "not self," it will launch an immune response

Human Antigens

Minor Blood Group Antigens

- ABO antigens have corresponding natural antibodies:
  "A" contain anti-B
  "B" contain anti-A
  "O" contain both anti-A and anti-B universal donor
  AB= contain none of anti-A and anti-B = universal recipients

Rh blood antigens

- Expressed on the surface of human RBCs
- There are no natural antibodies
- Positive or negative
- Hemolysis results when there is a difference

Histocompatibility Molecules = membrane protein

- Human Leukocyte Antigen (HLA) complex

Types of Immunity

Passive Immunity

- Using antibodies created by another person to prevent infectious disease
  - Natural
    - When antibodies from the mother pass through the placenta to the developing fetus
  - Acquired
    - When antibodies are acquired through an injection (Immunoglobulin)
    - Short lived but fast acting

Active Immunity

- Antibodies created by the person themselves
  - Natural
    - Protection conferred following survival from an infectious disease
  - Acquired
    - Injection or ingestion of either altered pathogenic microorganisms or products of those microorganisms – immunization with a vaccine

Diseases of Immunity

Immunodeficiency Diseases

Primary immunodeficiencies

- X-Linked hypogammaglobulinemia: Bruton Disease
- DiGeorge syndrome
- Severe Combined Immunodeficiency (SCID)
- Wiskott-Aldrich Syndrome

Secondary immunodeficiencies

- Acquired Immunodeficiency Syndrome (AIDS)

Autoimmune Diseases

- Systemic Lupus Erythematosus (SLE)
- Sjögren Syndrome
- Scleroderma
- Polymyositis / Dermatomyositis

Acquired Immunodeficiency Syndrome (AIDS)

- Develops as a consequence of a severe acquired immunosuppression caused by: Human immunodeficiency viruses (Small RNA viruses)
- “1% of all college-aged people (18-25 years of age) have serologic evidence of HIV infection.)
- The HIV virus fools helper T-cells into thinking its proteins are “self,” and so is able to infect the cells that trigger specific immunity
- The virus forces T-cells to make more viruses, killing the T-cells when the new viruses burst out.
- HIV is a fragile virus that cannot live outside the human body for more than a few minutes.
- Preventing HIV spread comes down to preventing exposure to body fluids of an infected person
- Because it attacks the immune system directly, finding a vaccine has been difficult.
- Some drugs can slow down HIV reproduction, but no cure exists yet. Prevention is still the best “cure.”

Systemic lupus Erythematosus

- Multisystemic involvement
- 10 times more common in women
- May occur at any age, but most often young adults
- Etiology: Poorly understood
- Malfunctioning of suppressor T cells
- Clinical Symptoms (highly variable): Inflammation of the joints, organ involvement, CNS problems, Vision problems “Butterfly” rash
- Prognosis: More than 30% of the patients are alive 10 years post dx

Rheumatoid Arthritis (RA)

- RA is thought to be ***T-Cell*** mediated
- Most widely accepted hypothesis:
  - Professional APC encounters some “unknown” antigen
  - It presents this “unknown” antigen to a CD4 T-helper Cell
  - In a genetically predisposed individual, this starts an immune chain reaction

Allergies

- Allergies are an immune system reaction to harmless antigens.
- Some, such as pollen, may get in through the respiratory system. Fragments of food proteins may get through the digestive system.
- The next time these proteins are encountered, the immune system attacks them.

Allergens

- Allergens are nonparasite antigens that can stimulate a type I hypersensitivity response

- Allergens bind to IgE and trigger degranulation of chemical mediators.

Allergens

Proteins

- Foreign serum
- Vaccines

Plant pollens

Drugs

- Penicillin
- Sulfonamides
- Local anesthetics

Foods

- Nuts
- Eggs

Insect products

- Bee venom

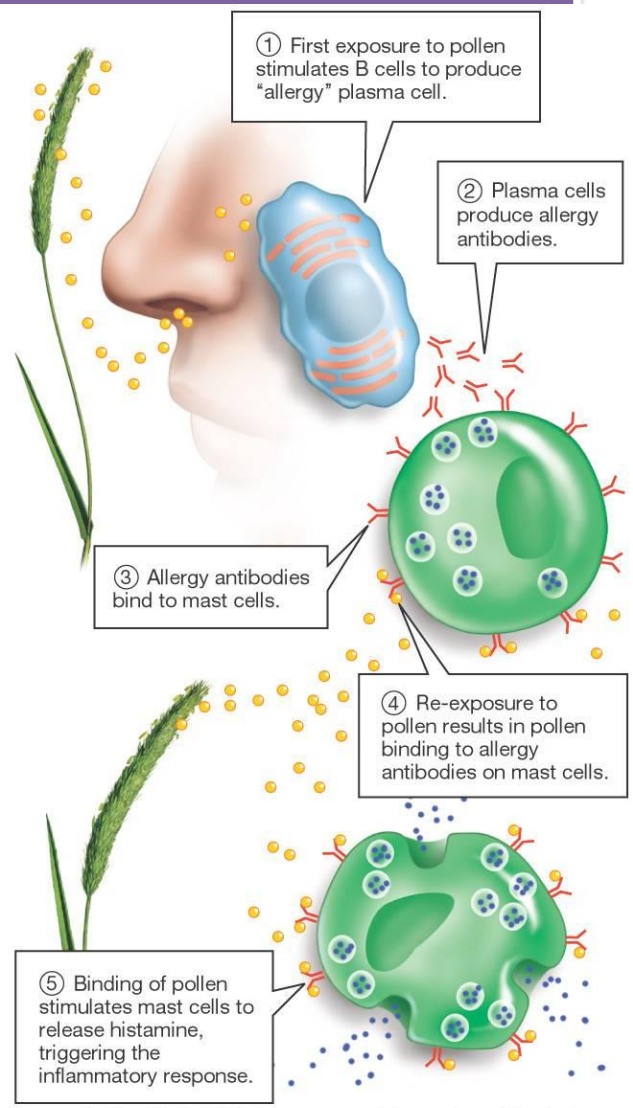
- Dust mites

Mold Spores

Animal hair and dander

Achoo!

- Pollen is a harmless protein, yet we can become allergic to it.
- Most of the symptoms are caused by histamines released by mast cells. That is why antihistamines are used to treat allergies.



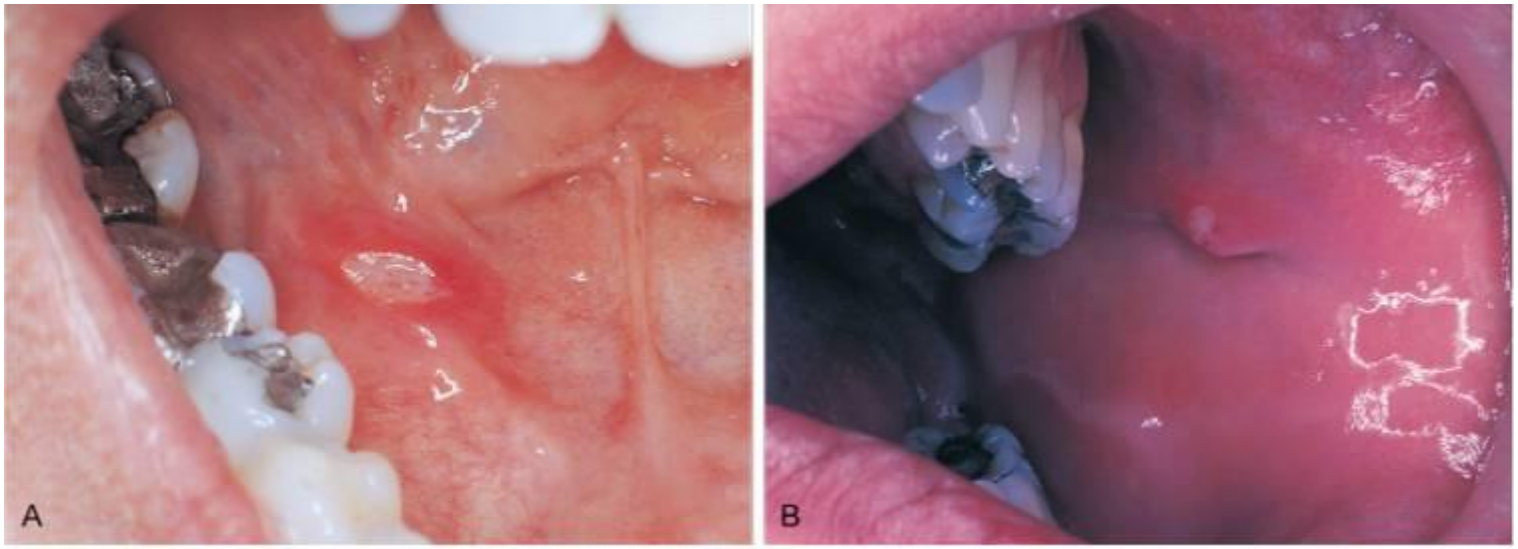
Diseases with Immunologic Pathogenesis

- Aphthous ulcers
- Urticaria and angioedema
- Contact mucositis and dermatitis
- Fixed drug eruptions
- Erythema multiforme
- Lichen planus
- Reactive arthritis (Reiter syndrome)
- Langerhans cell disease

Aphthous Ulcers

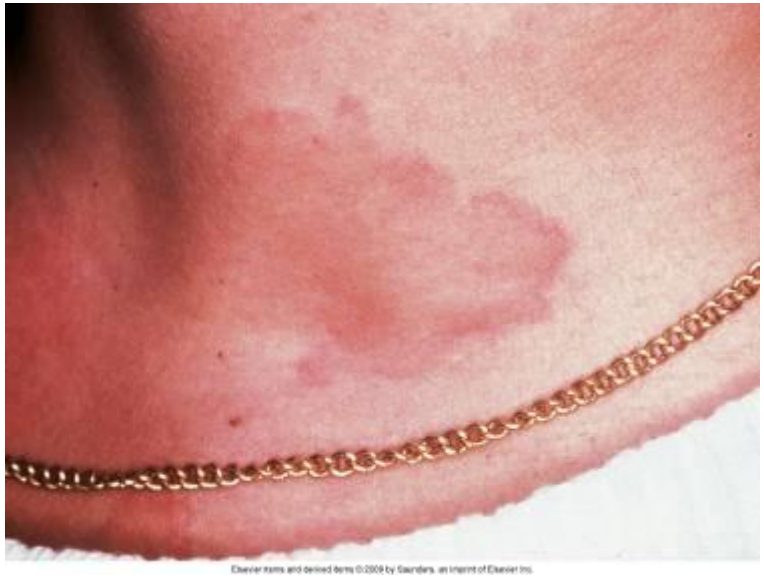
- Painful oral ulcers with an unclear cause
  - Occur in about 20% of the population
  - Trauma is the most common precipitating factor.
    - May be caused by emotional stress or certain food
  - May be associated with certain systemic diseases
  - Thought to have an immunologic pathogenesis
  - Occur in three forms: minor, major, and herpetiform

Minor Aphthous Ulcers (cont.)



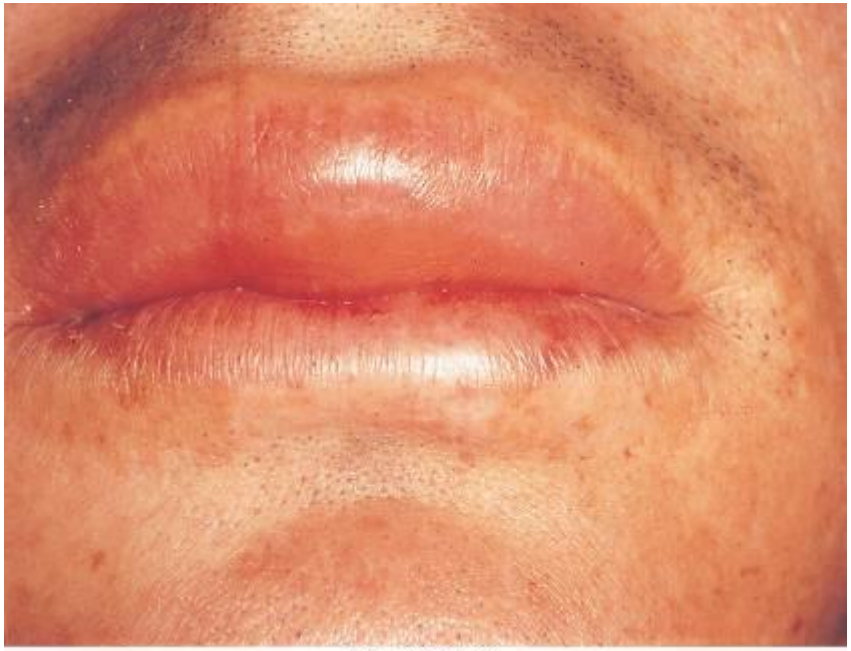
Urticaria

- Appears as multiple areas of well-demarcated swelling of skin
  - May have itching (pruritis)
- Lesions caused by localized areas of vascular permeability in superficial connective tissue



Angioedema

- Lesions caused by diffuse swelling due to increased permeability of deeper blood vessels
  - The skin covering the swelling appears normal
  - Usually do not have itching



Contact Mucositis and Dermatitis

- Lesions resulting from contact of an allergen with skin or mucosa
- Involves CMI (cell-mediated immunity)
  - The mucosa initially becomes erythematous and edematous.
    - Often there is burning and pruritus
  - Later, the area becomes white and scaly.
- Treatment
  - Topical and/or systemic corticosteroids

Fixed Drug Eruptions

- Lesions that appear in the same site each time a drug is introduced
  - Generally appear suddenly after a latent period and subside when the drug is discontinued
- May be single or multiple slightly raised, reddish patches or clusters of macules on the skin, or sometimes the mucous membranes
  - May have pain or pruritis

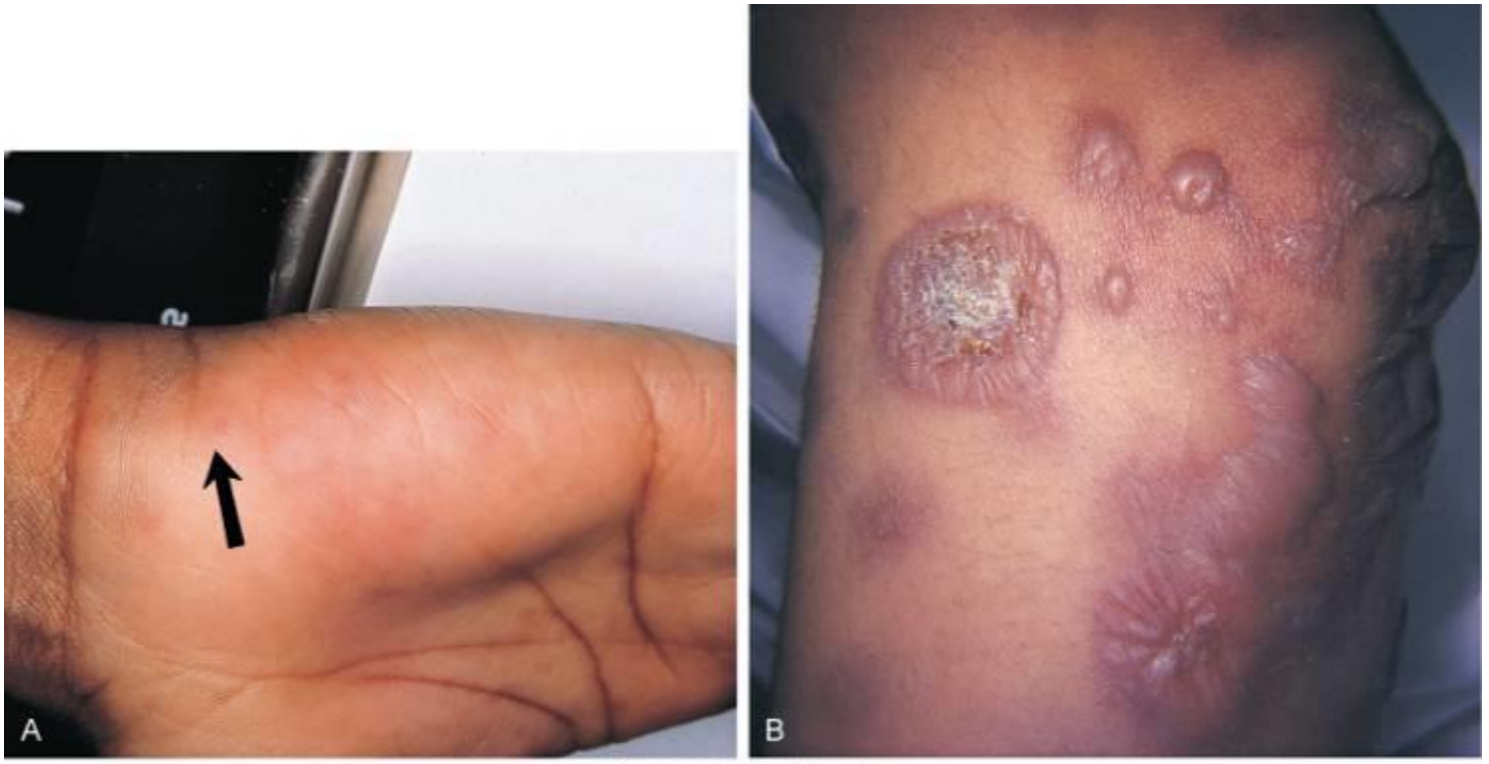
Fixed Drug Eruptions (cont.)

- A type of allergic reaction (Type III)
  - Immune complexes are deposited along the endothelial walls of blood vessels.
  - Inflammation causes vasculitis with damage to the vessel wall.
  - This creates erythema and edema in superficial layers of skin or mucosa.
- Treatment
  - The drug causing the reaction should be identified and discontinued.

Erythema Multiforme

- Cause is not clear; may be a hypersensitivity reaction
  - Most commonly occurs in young adults, affects men more commonly than women
- Target lesion
  - Characteristic skin lesion with concentric erythematous rings alternating with normal skin color

Erythema Multiforme (cont.)



(**A** courtesy Dr. Edward V. Zegarelli.)

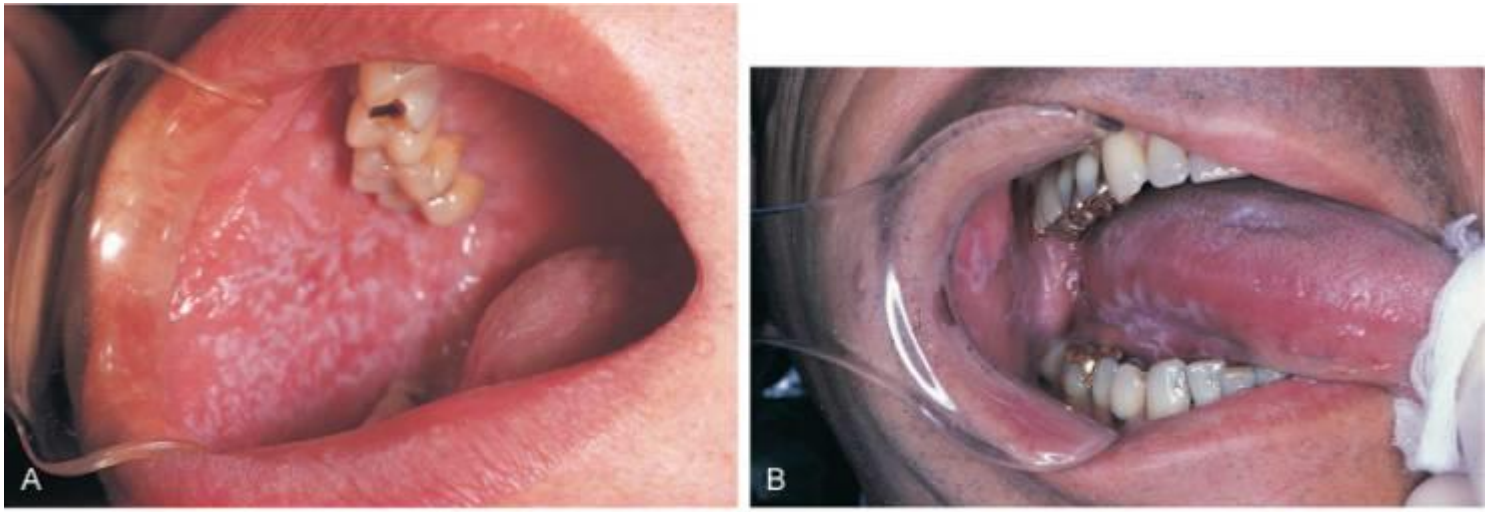
Erythema Multiforme (cont.)

- Diagnosis
  - Based on clinical features and by exclusion of other diseases
- Treatment and prognosis
  - Topical or systemic corticosteroids
  - Eye lesions may lead to blindness.

Lichen Planus

- A benign, chronic disease affecting skin and oral mucosa
  - Unknown cause
  - Lesions have characteristic Wickham striae
- Most commonly on buccal mucosa
  - Lesions may be on the tongue, lips, floor of mouth, and gingiva.
- Present in about 1% of the U.S. population
  - Most common in middle age
  - Slightly more common in women

Lichen Planus (cont.)

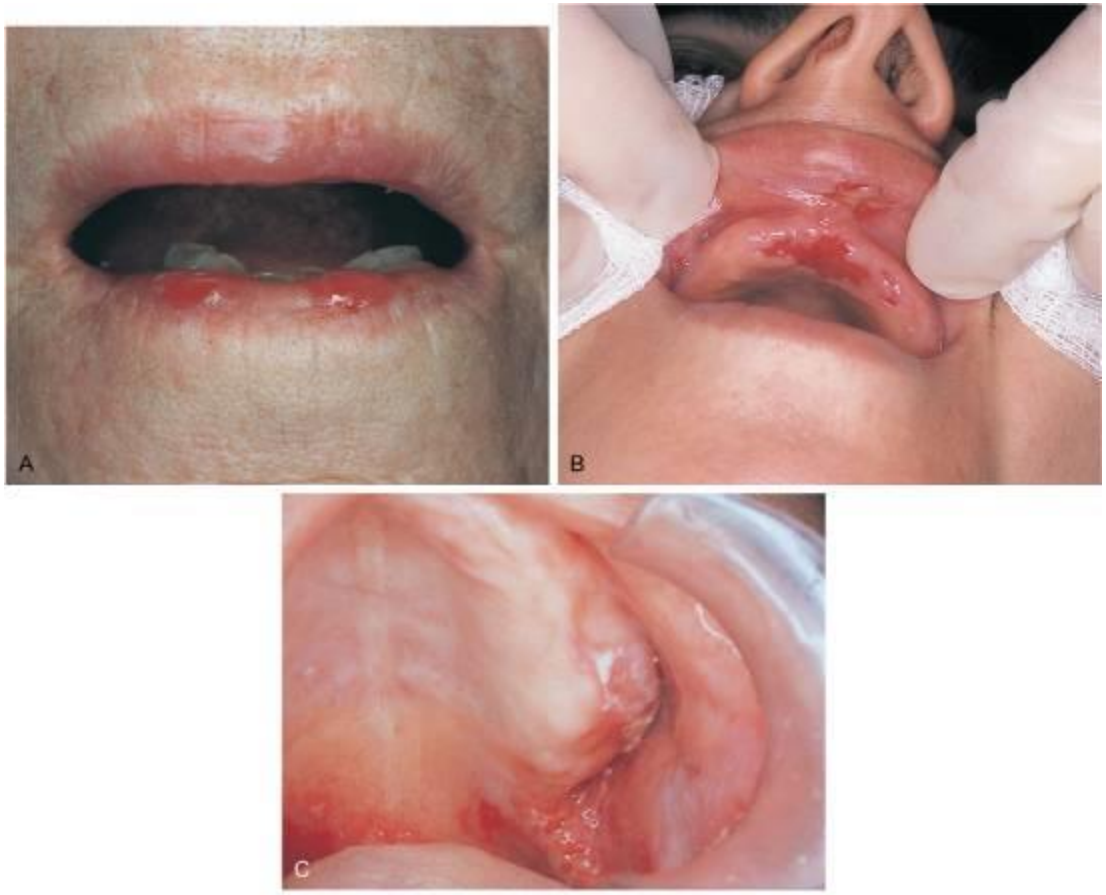


(**A** courtesy Dr. Edward V. Zegarelli.)

Pemphigus Vulgaris

- A severe, progressive autoimmune disease affecting skin and mucous membranes
  - Characterized by intraepithelial blister formation resulting from acantholysis, a breakdown of cellular adhesion between epithelial cells
- Genetic and ethnic factors have been reported.
  - Often seen in Ashkenazic Jews

Pemphigus Vulgaris (cont.)



(B courtesy Dr. Fariba Yousai, C courtesy Dr. Sidney Eisig.)

Bullous Pemphigoid

- Some investigators believe bullous and mucous membrane pemphigoid are variants of a single disease, but 80% of these patients are older than 60.
  - Oral lesions are less common than in cicatricial pemphigoid.
- Treatment
  - Systemic corticosteroids and nonsteroidal antiinflammatory drugs

Vaccine

- Expose body to harmless version of pathogen
- Body creates antibodies to the disease, but not the actual symptoms/pathology of the disease
- Vaccines stimulate the production of Memory cell
- Vaccination causes the body to learn to defend itself
- Vaccines are not less effective than a “natural” infection with the illness. The immunity is the same, and a mild response to a vaccine is much less risky than a full-blown infection
- Modern vaccines are created from killed bacteria or viruses, or fragments of proteins from these microbes.
- The proteins are recognized as antigens by our immune systems. This causes a mild immune response. Memory T-cells and B-cells remain ready to fight off the illness if it is encountered again

Antibiotic myths

- Antibiotics are not antibodies.
- Antibiotics do not weaken our immune system. They help it by weakening bacteria.
- Humans do not become “immune” to antibiotics. Bacteria that resist antibiotics and are not completely destroyed may multiply, producing more antibiotic-resistant bacteria.
- Antibiotics help destroy bacteria (but not viruses).
- Antibiotics work in one of several ways:
- Slowing bacteria reproduction.
- Interfering with bacterial cell wall formation