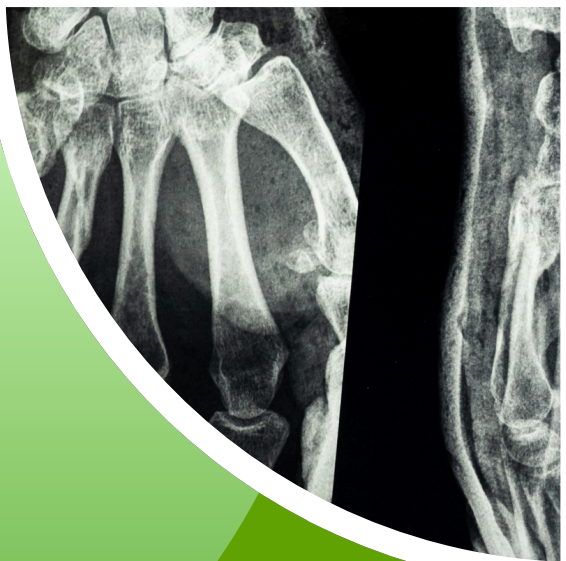
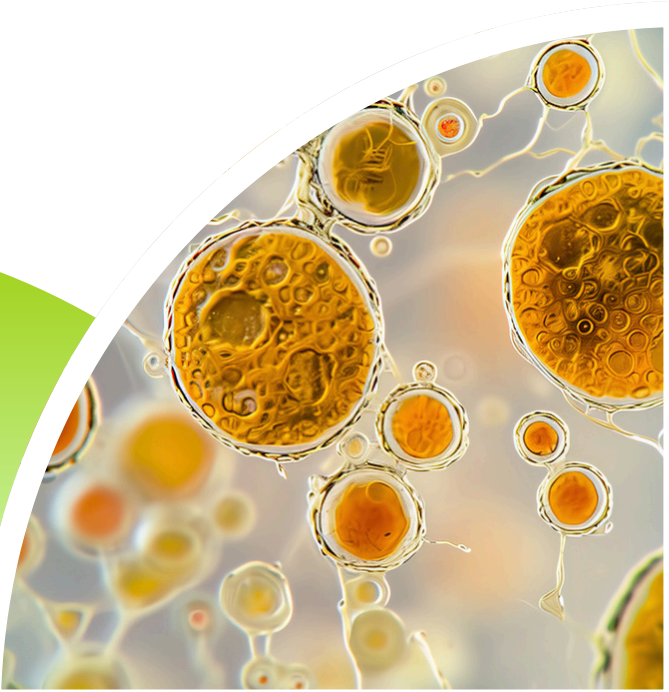


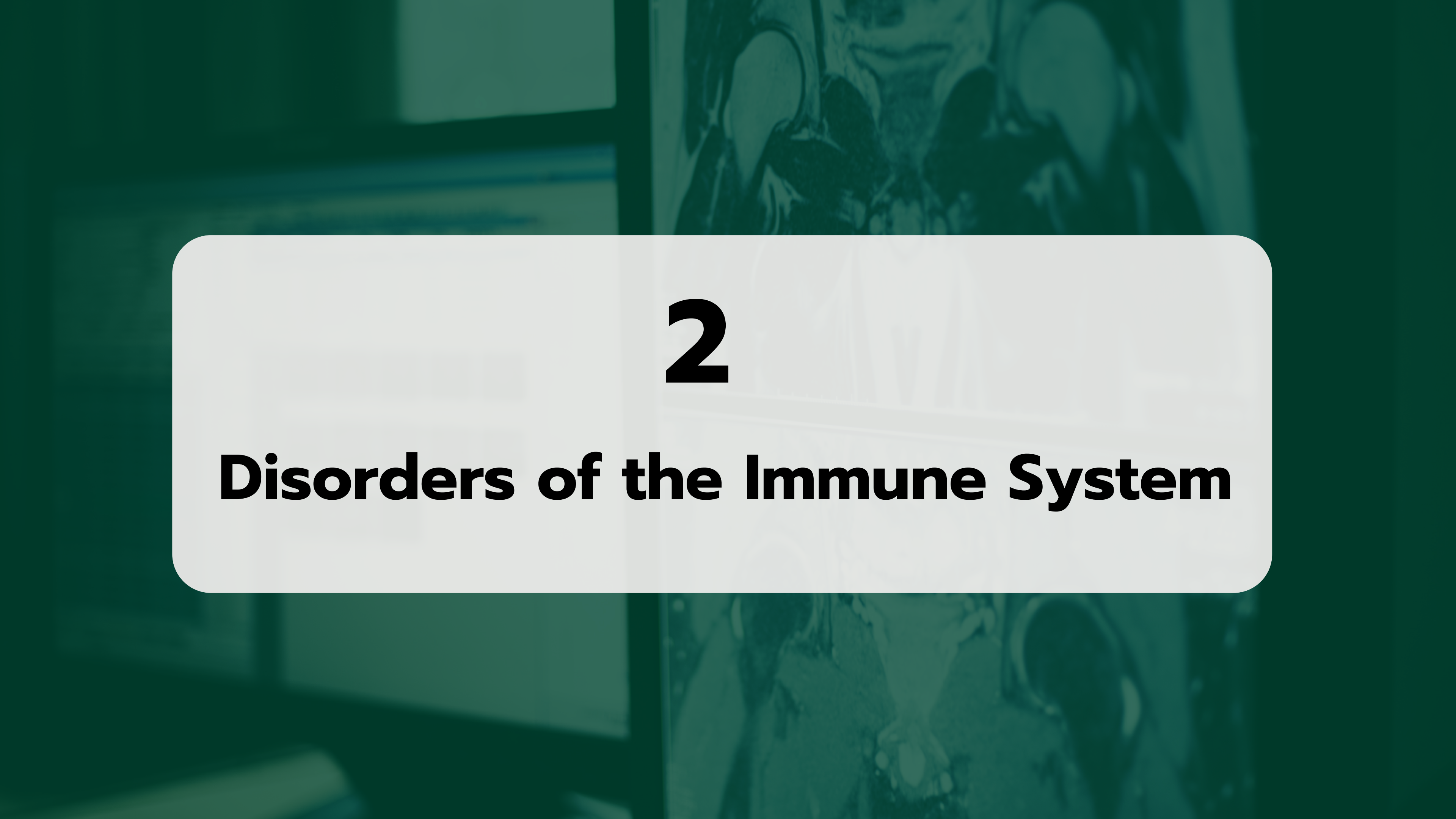


มหาวิทยาลัยราชภัฏนครปฐม

Mechanism and Illness of Human Body 2

*Lecturer Unchisa Rattanakunuprakarn
Asst.Prof. Jutatip Tepsuwan
Lecturer Napat Rattanahongsa*

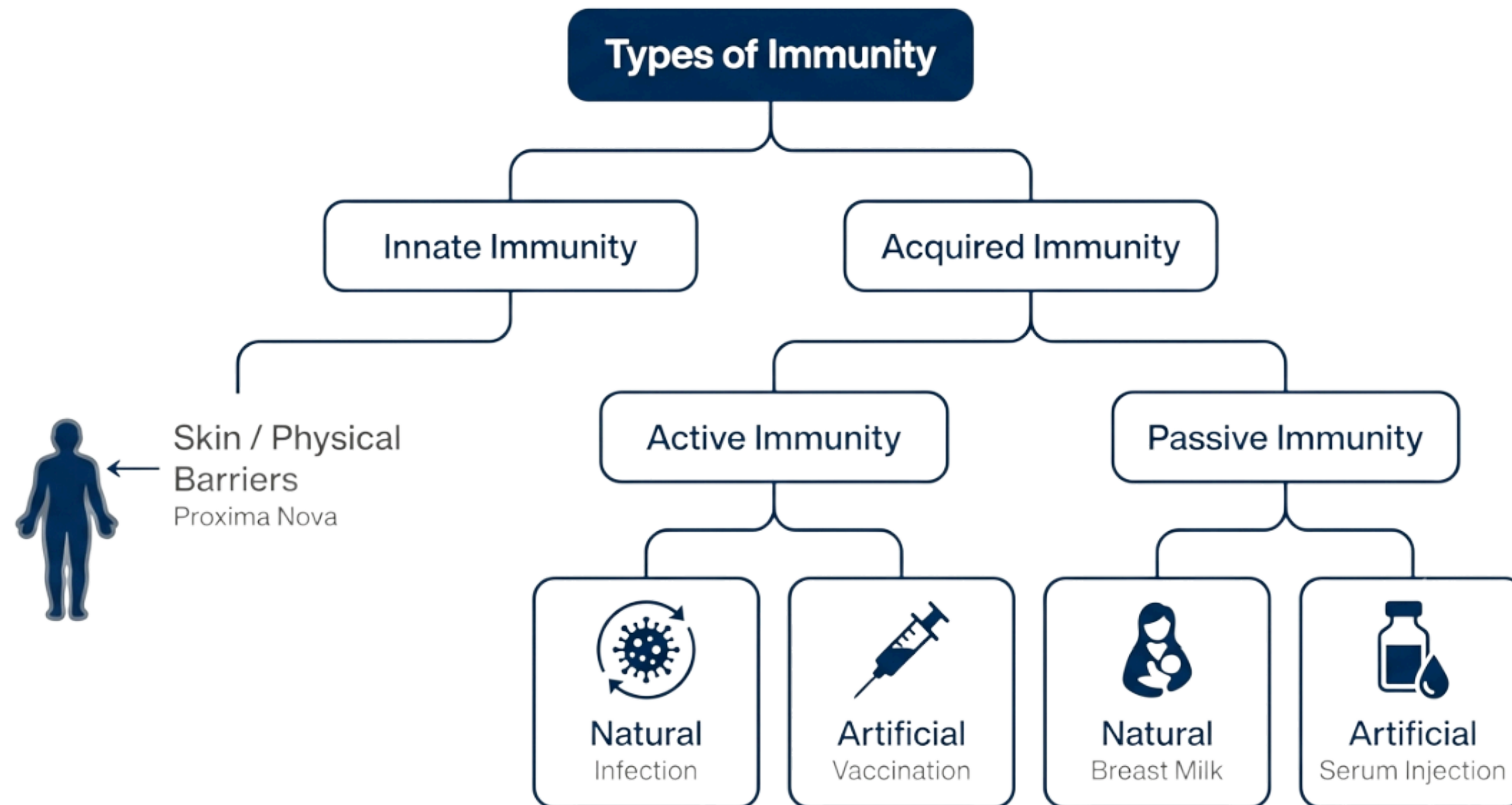




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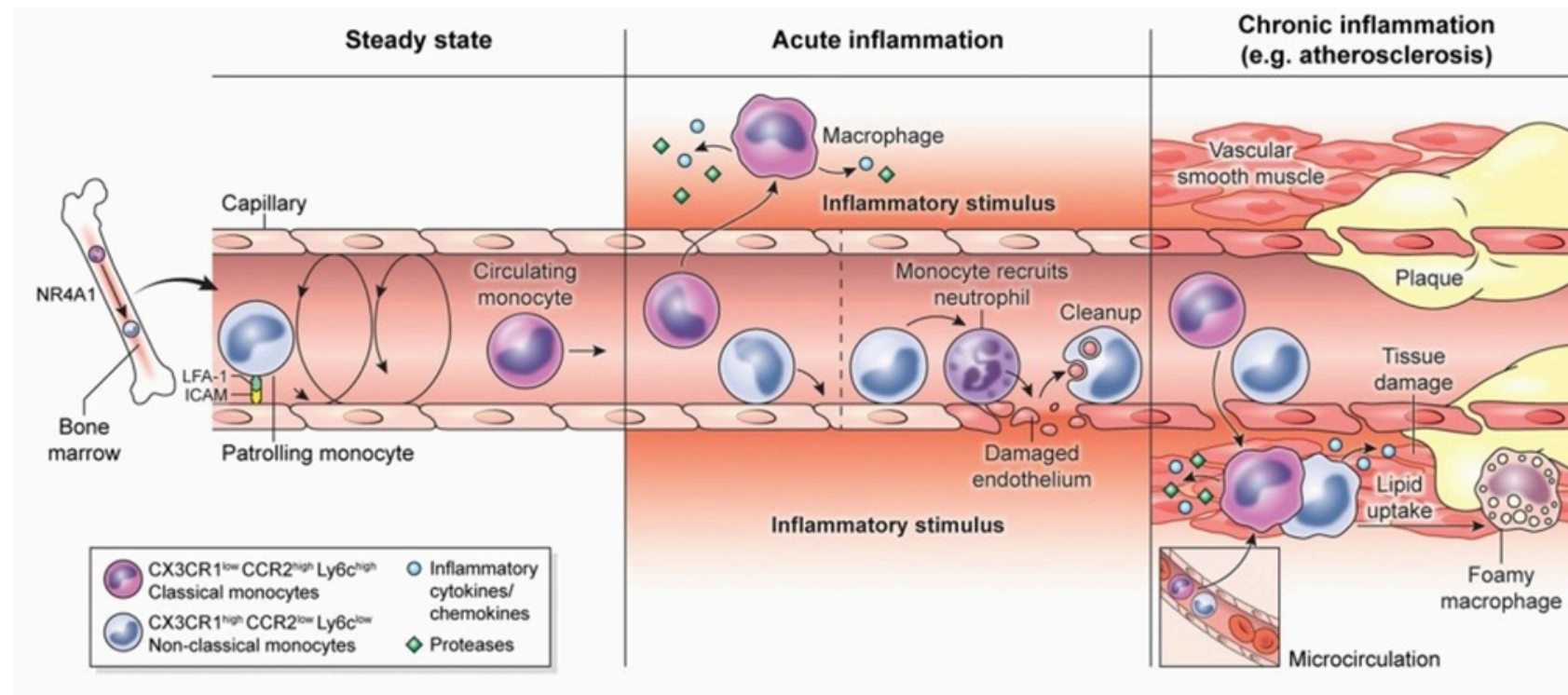
Disorders of the Immune System

Type of Immunity

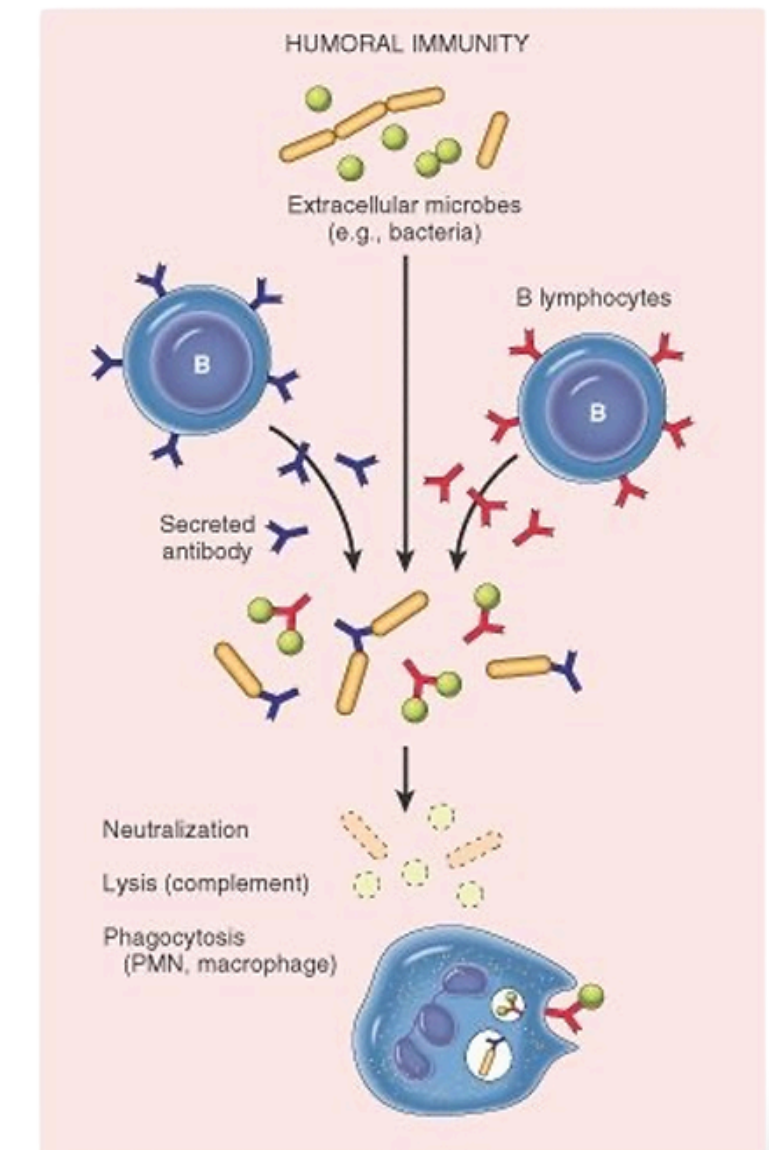
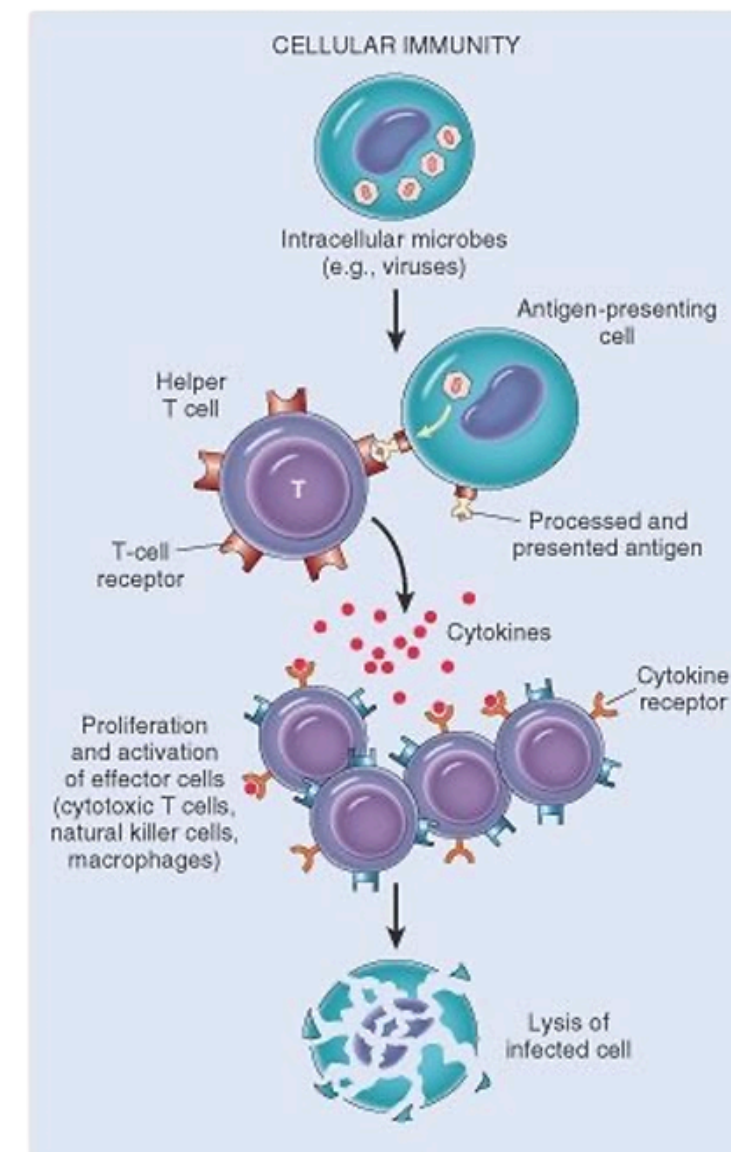


Response

Nonspecific response



Specific response



Classification of Immune Disorders



1. Immunodeficiency

Primary (Genetic) &
Secondary (Acquired)



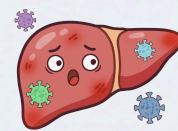
2. Hypersensitivity

Over-reaction
(Types I, II, III, IV)



3. Autoimmune Disease

Loss of self-tolerance
(Systemic & Organ-specific)



4. Immunoproliferative Disease

Malignant growth
(Leukemia, Lymphoma)





Immunodeficiency Overview



Definition: Failure of the immune system to protect the body against infection or malignancy.

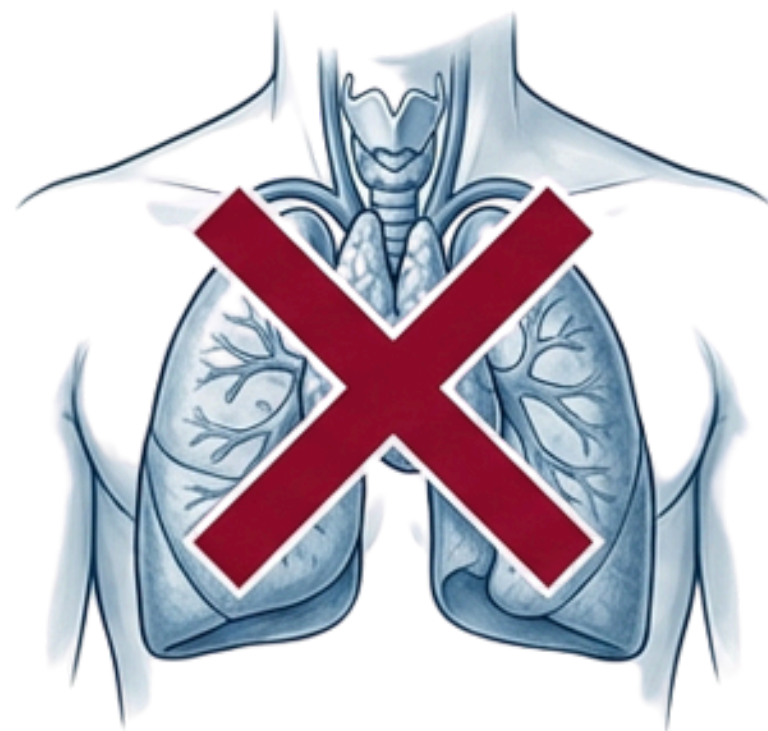
Classification:

Primary (Congenital): Genetic defects, present from birth.

Secondary (Acquired): Caused by external factors (e.g., infection, drugs).

Primary Immunodeficiency

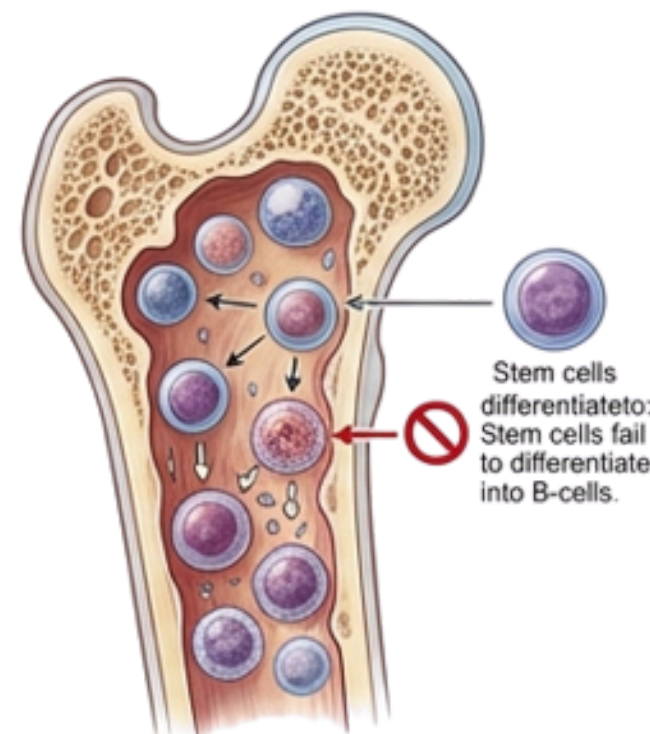
T-Cell Defects



DiGeorge Syndrome: Absent thymus & parathyroid.

Nezelof Syndrome: Absent thymus, normal parathyroid.

B-Cell Defects



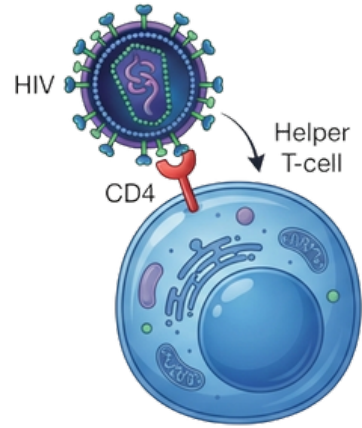
Bruton's Agammaglobulinemia: Stem cells fail to differentiate into B-cells.

Combined Defects



SCID (Severe Combined Immunodeficiency): Defect in stem cells or lymphoid tissue.

(Gennery & Cant, 2001)



Secondary Immunodeficiency (AIDS)

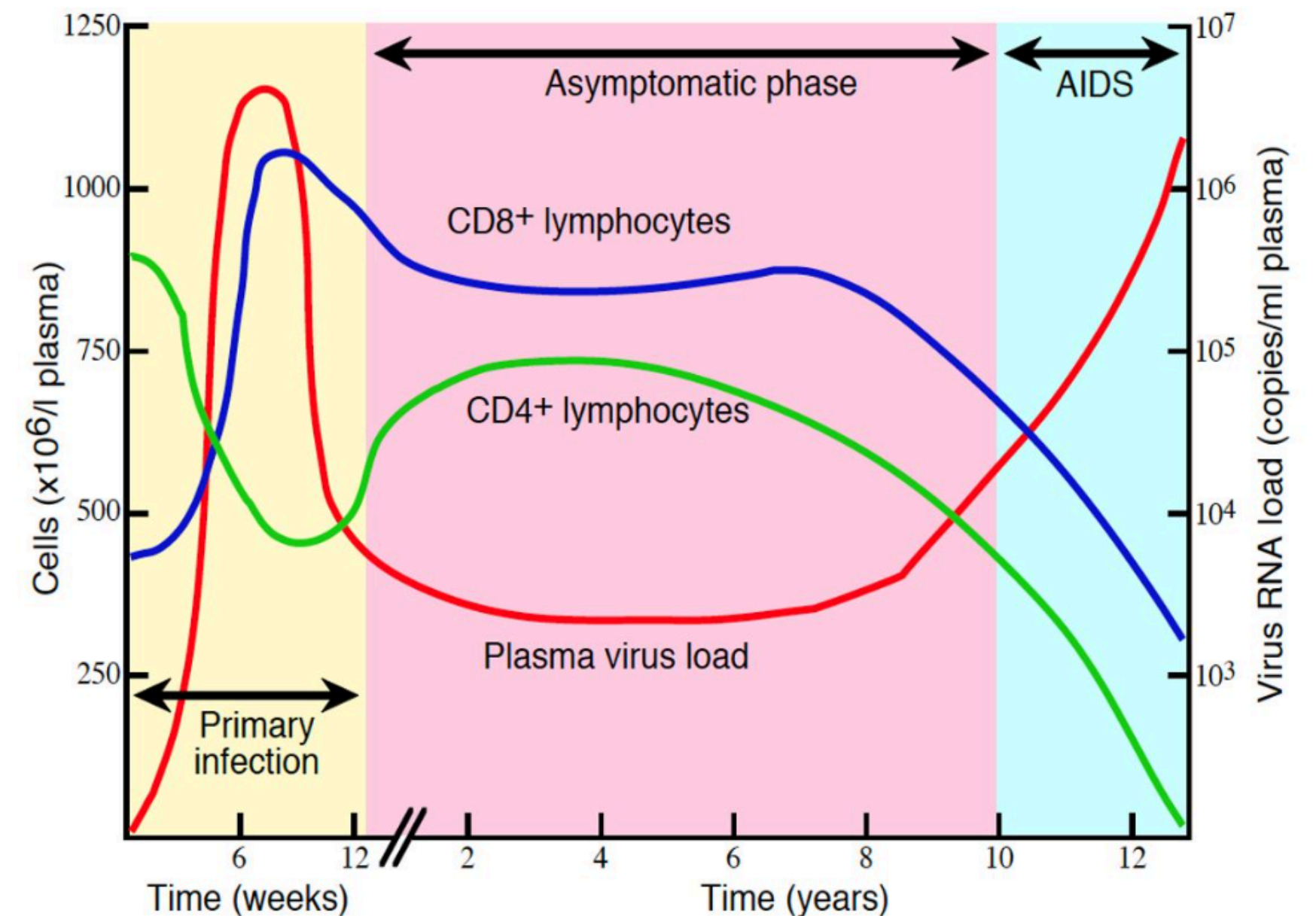


Etiology: HIV (Human Immunodeficiency Virus), LAV, HTLV-III.

Pathophysiology: Virus targets and destroys Helper T-lymphocytes (CD4+).

Significant decrease in Helper : Suppressor T-cell ratio.

Consequence: Susceptibility to opportunistic infections.

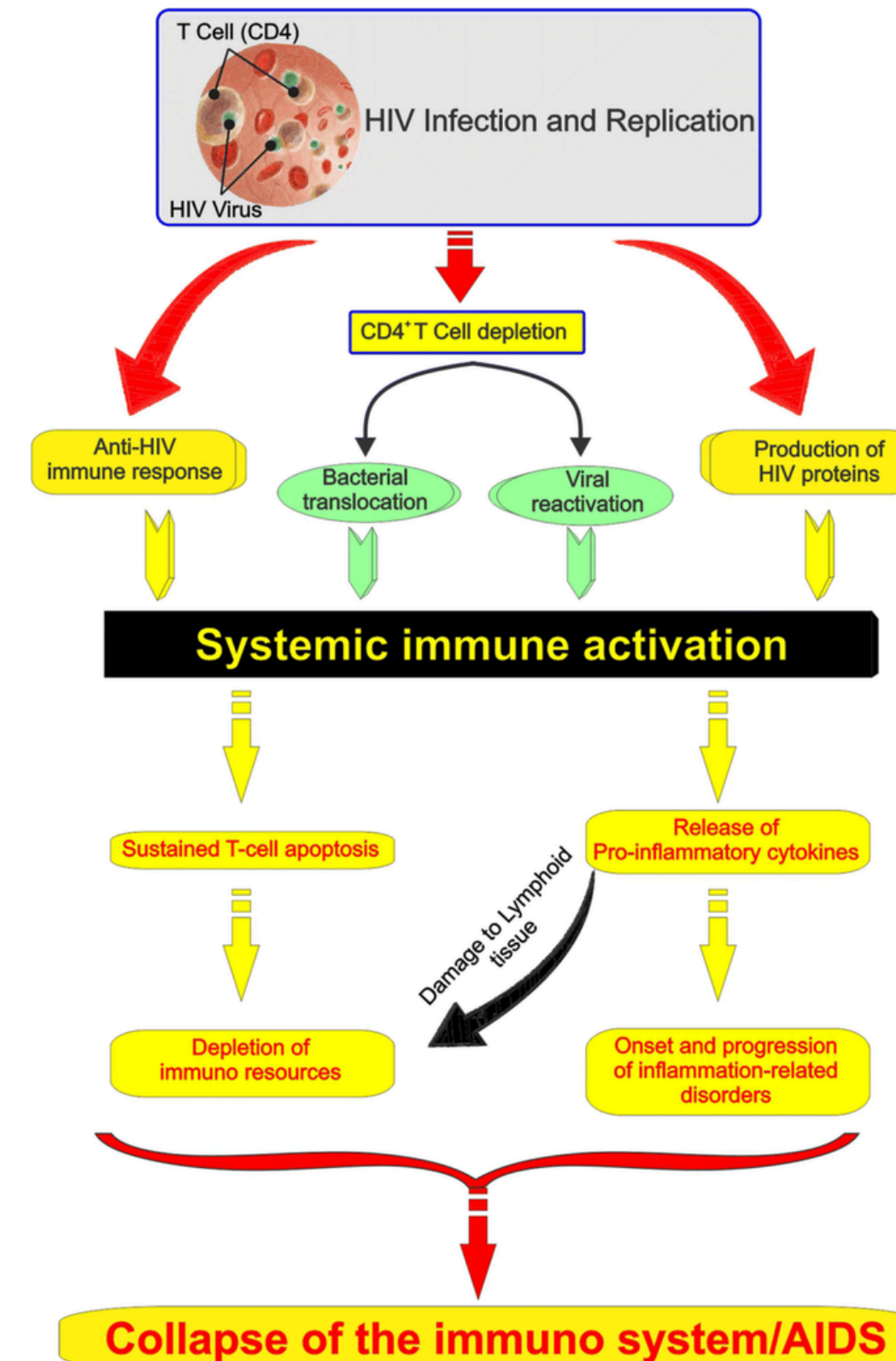
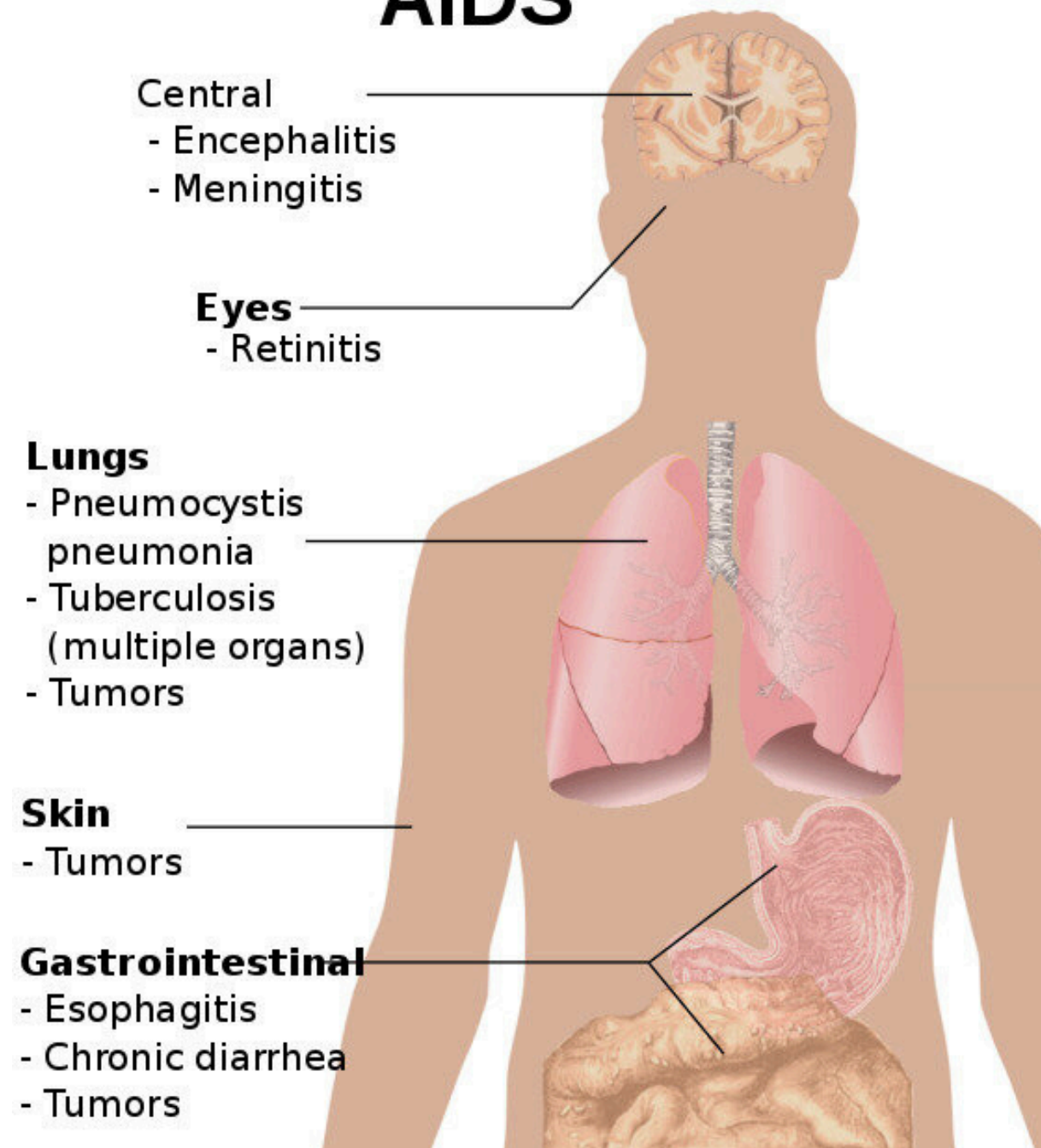


(Samuel & Carsten, 2012)

Secondary Immunodeficiency (AIDS)

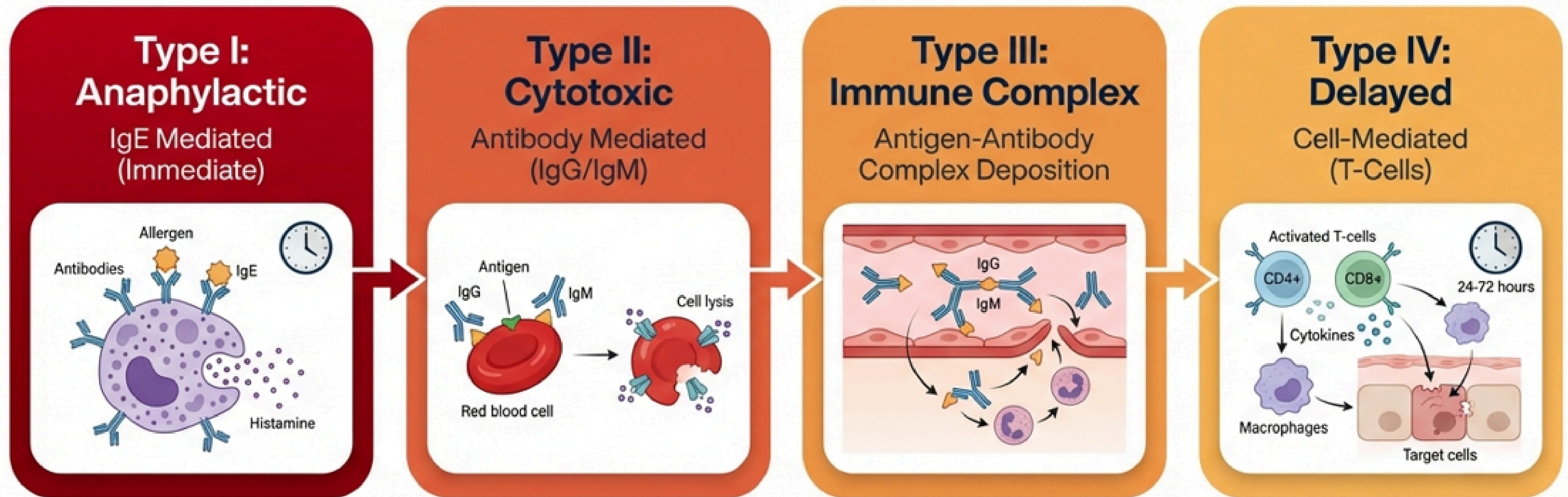


Main symptoms of AIDS





Hypersensitivity Reactions



Type I Hypersensitivity (Anaphylactic)

Mechanism: IgE-mediated.

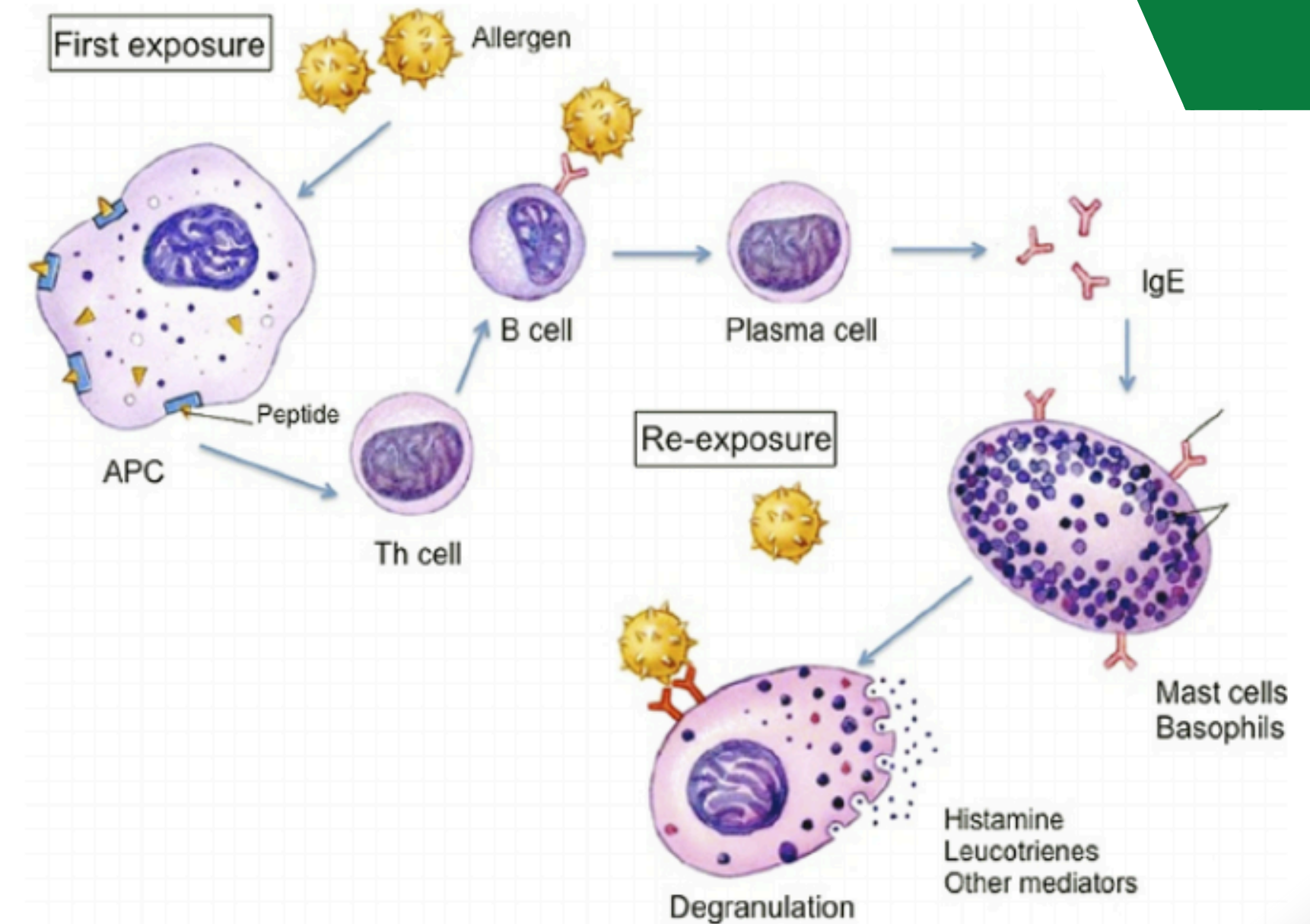
Key Cells: Mast cells, Basophils.

Process: Antigen binds to IgE on mast cells
Degranulation



Release of Histamine/Heparin.

Examples: Asthma, Allergic Rhinitis,
Anaphylactic shock.



Type II Hypersensitivity (Cytotoxic)

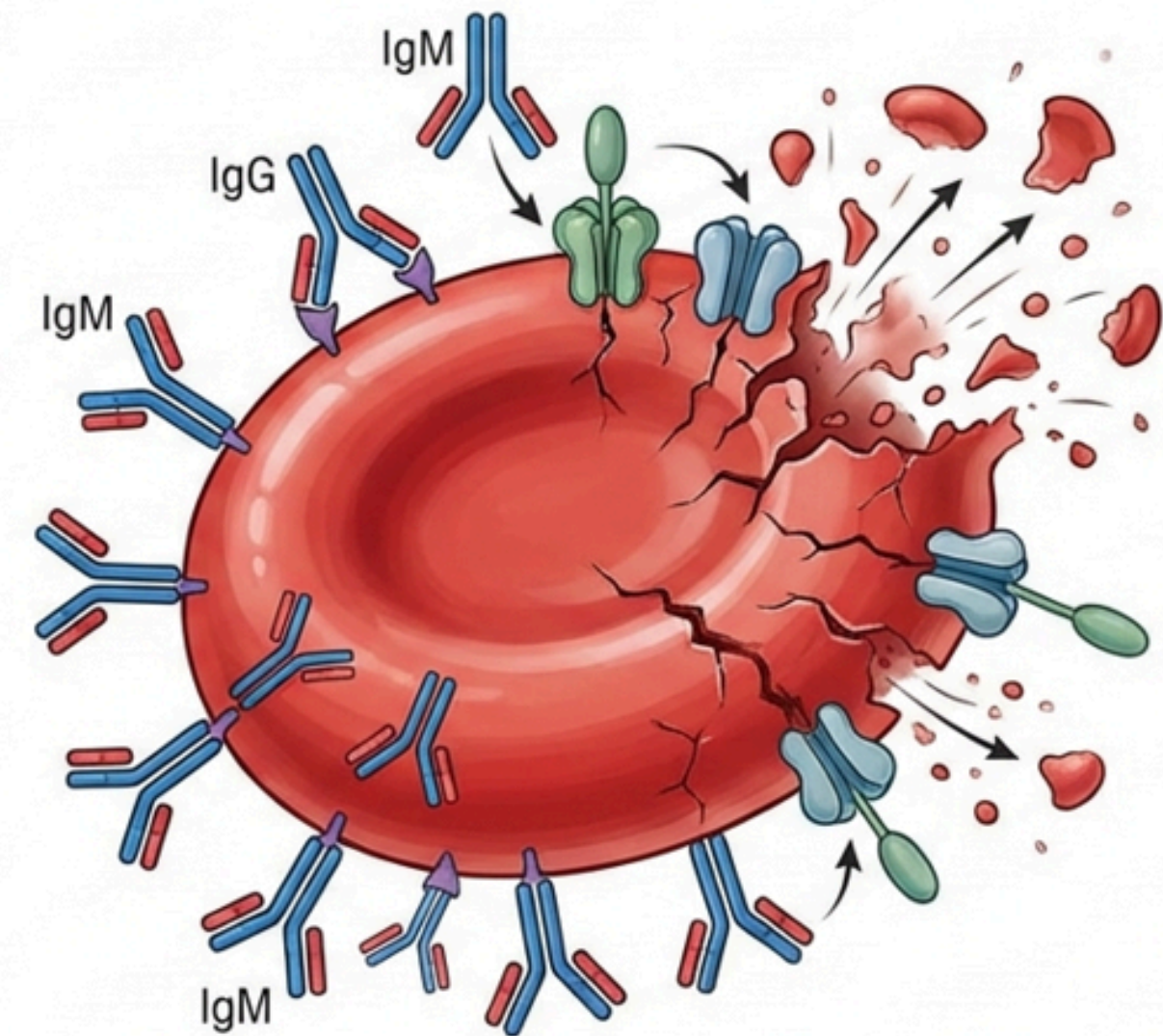
Mechanism: Antibody (IgG/IgM) binds to antigens on cell surfaces.

Pathogenesis:

Opsonization: Enhanced phagocytosis.

Complement Activation: Cell lysis.

Examples: Blood transfusion reactions,
Autoimmune Hemolytic Anemia.



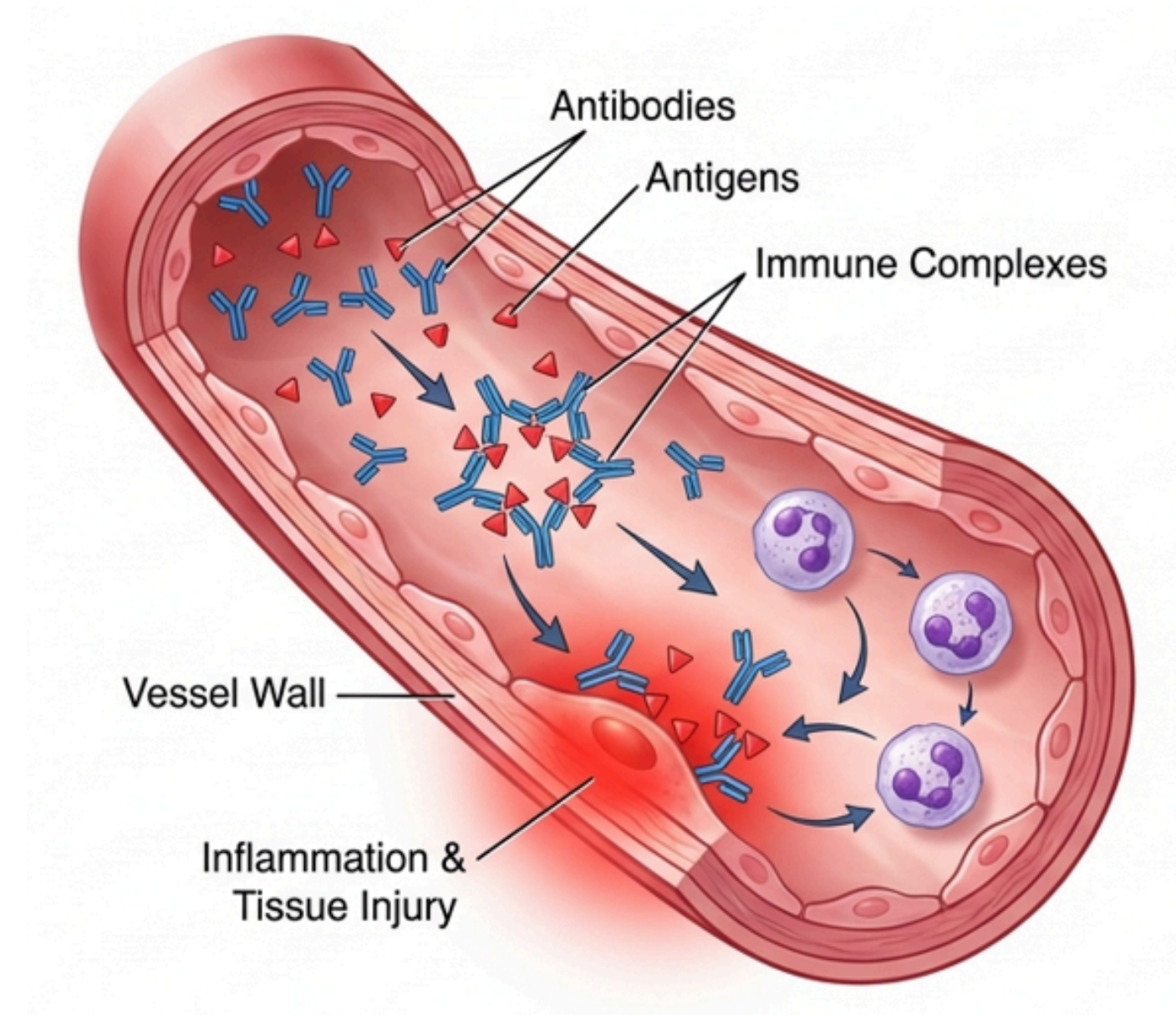
Type III Hypersensitivity (Immune Complex)

Mechanism: Formation of Antigen-Antibody (Ag-Ab) complexes.

Pathogenesis:

- Complexes deposit in tissues/blood vessel walls.
- Activation of Complement (Classical Pathway).
- Inflammation and tissue damage.

Examples: Systemic Lupus Erythematosus (SLE), Serum Sickness.



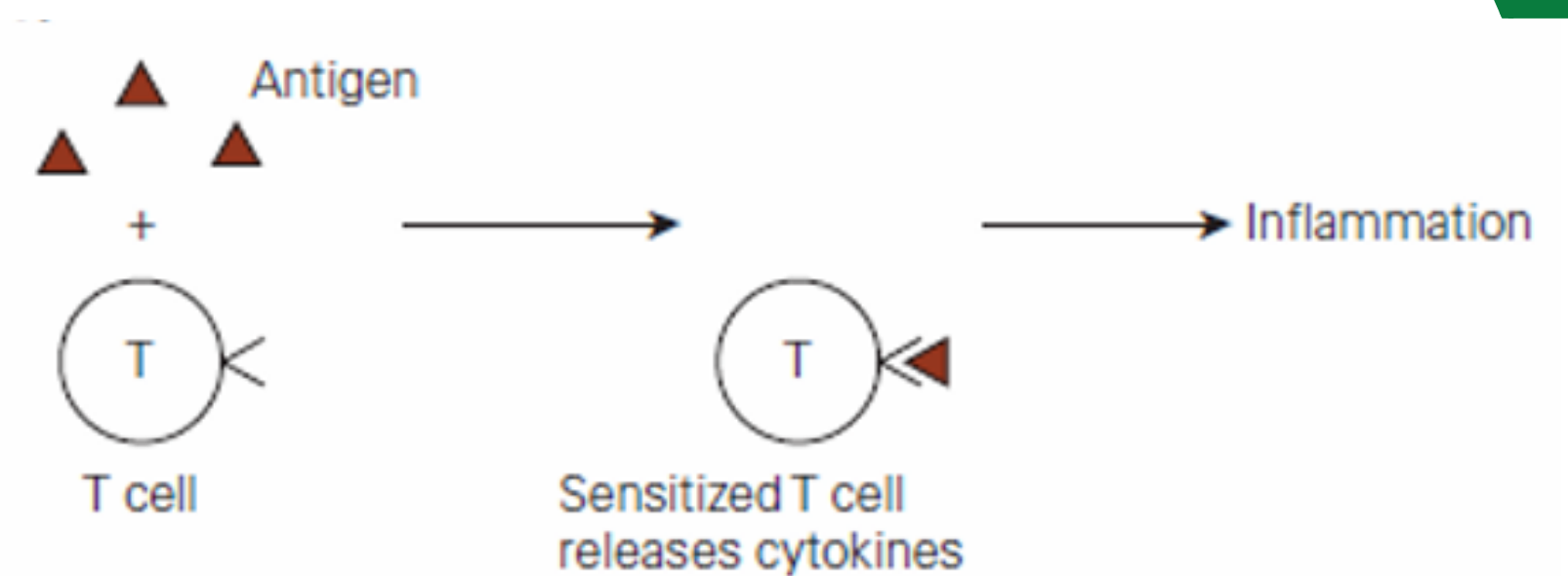
Type IV Hypersensitivity (Cell-Mediated)

Mechanism: T-lymphocyte mediated (Not antibody-mediated).

Pathogenesis:

- Sensitized T-cells release Lymphokines.
- Activation of Macrophages causing tissue damage.

Examples: Tuberculin skin test (TB), Contact dermatitis, Graft rejection.



Positive tuberculin skin test

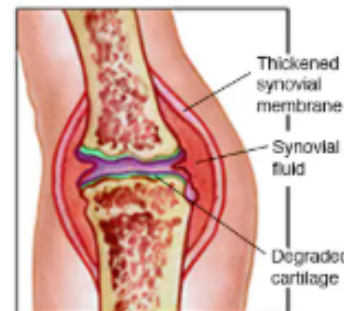


Autoimmune Diseases



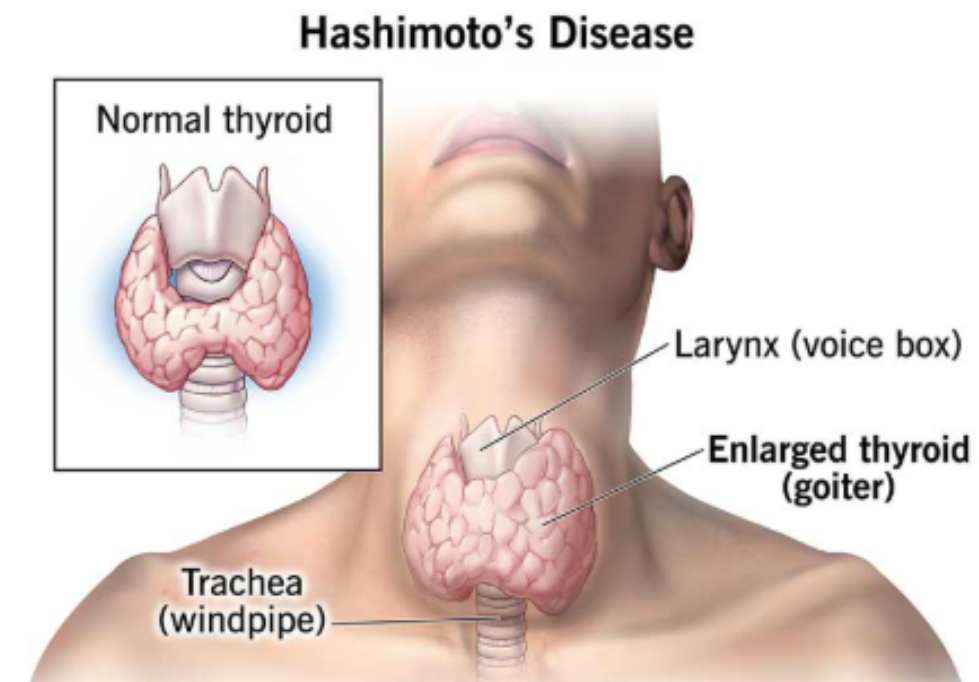
Definition: Breakdown of self-tolerance; immune system attacks self-antigens

Systemic Autoimmune Disease:
Affects multiple organs
(e.g., SLE, Rheumatoid Arthritis)



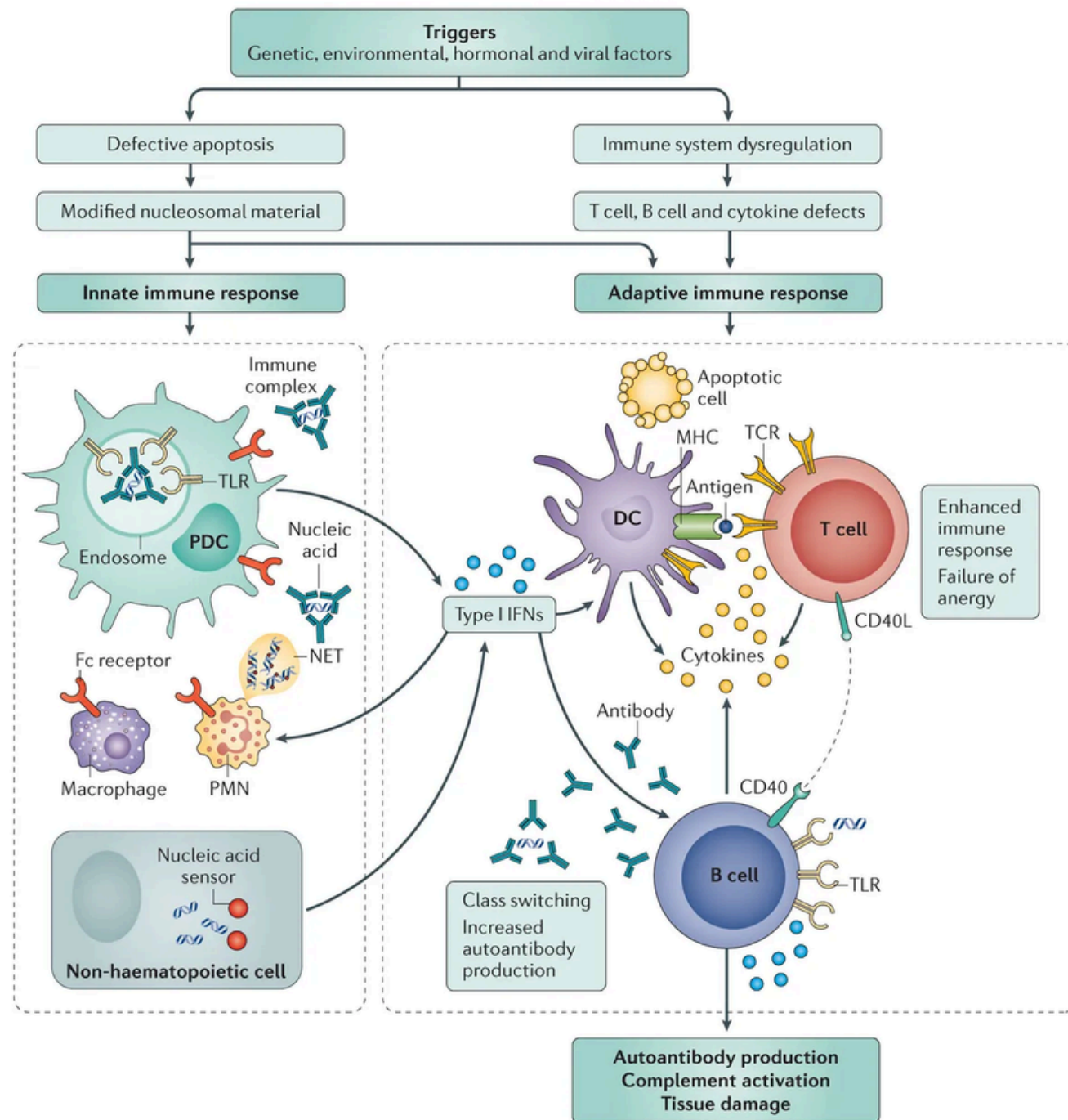
(Cleveland Clinic, 2023)

Organ-Specific Autoimmune Disease:
Affects a single organ
(e.g., Hashimoto's thyroiditis)



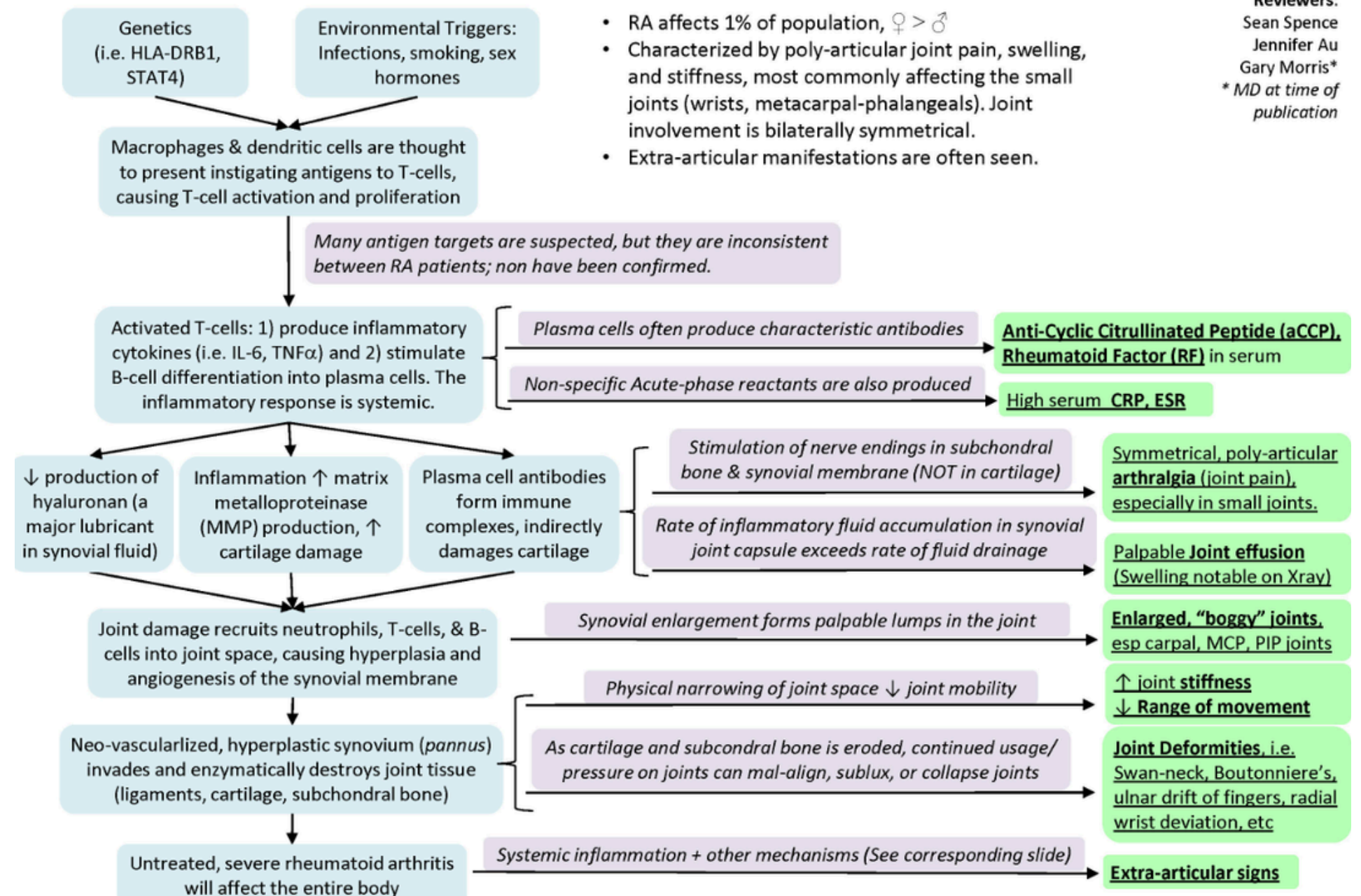
(Cleveland Clinic, 2023)

SLE



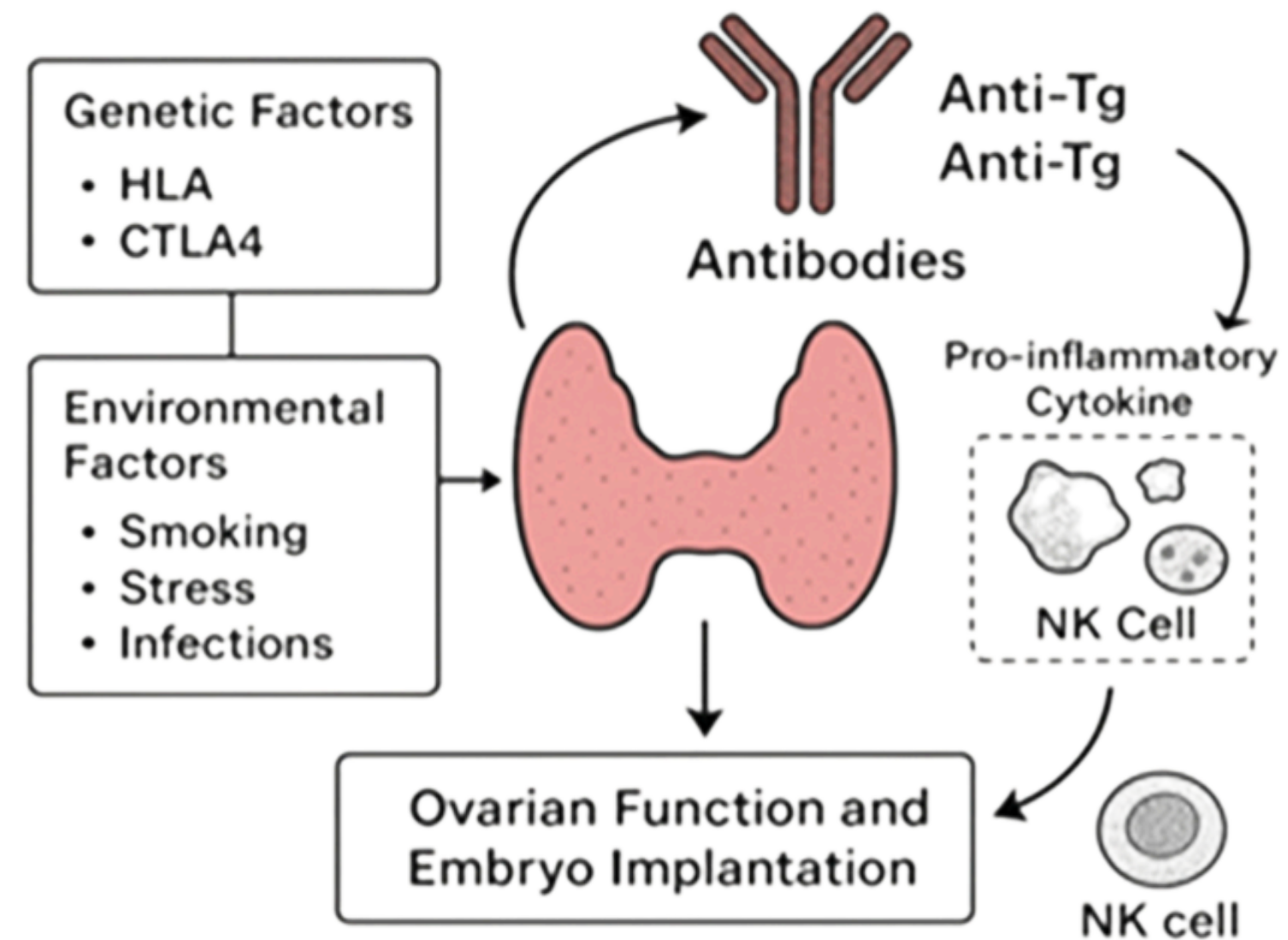
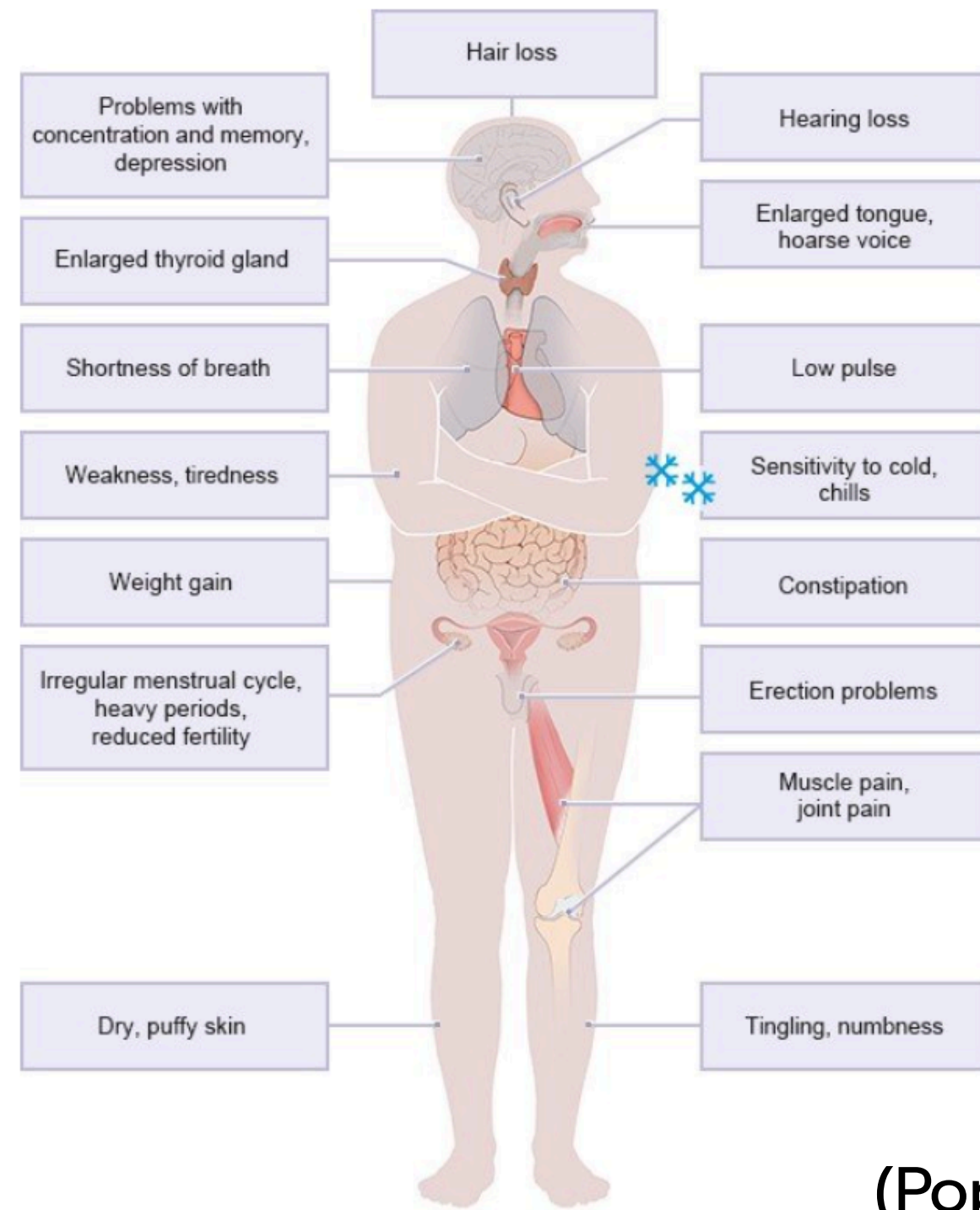
Rheumatoid Arthritis

Rheumatoid Arthritis (RA): Pathogenesis and clinical findings



Authors:
Yan Yu
Reviewers:
Sean Spence
Jennifer Au
Gary Morris*
* MD at time of publication

Hashimoto's thyroiditis



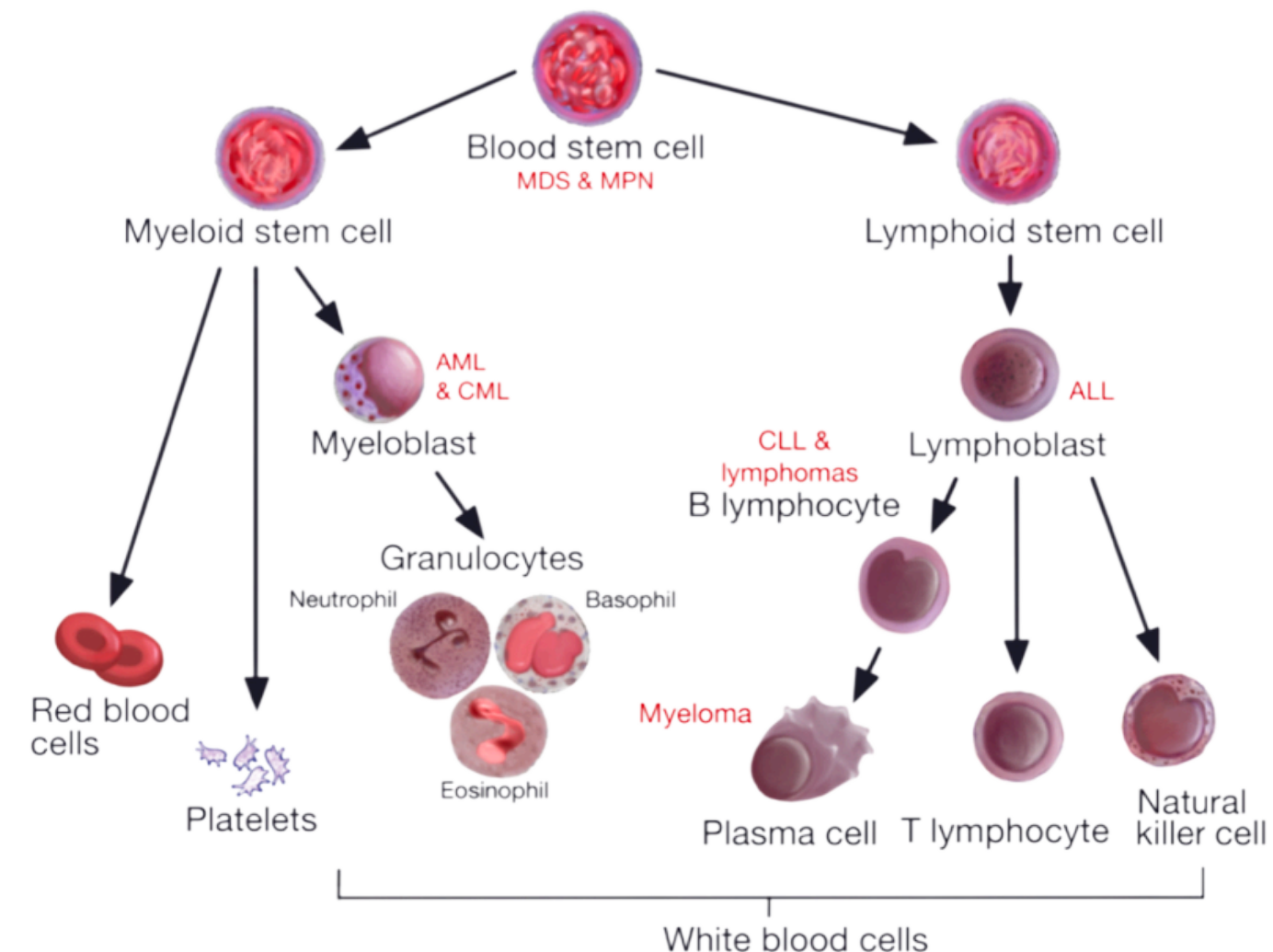
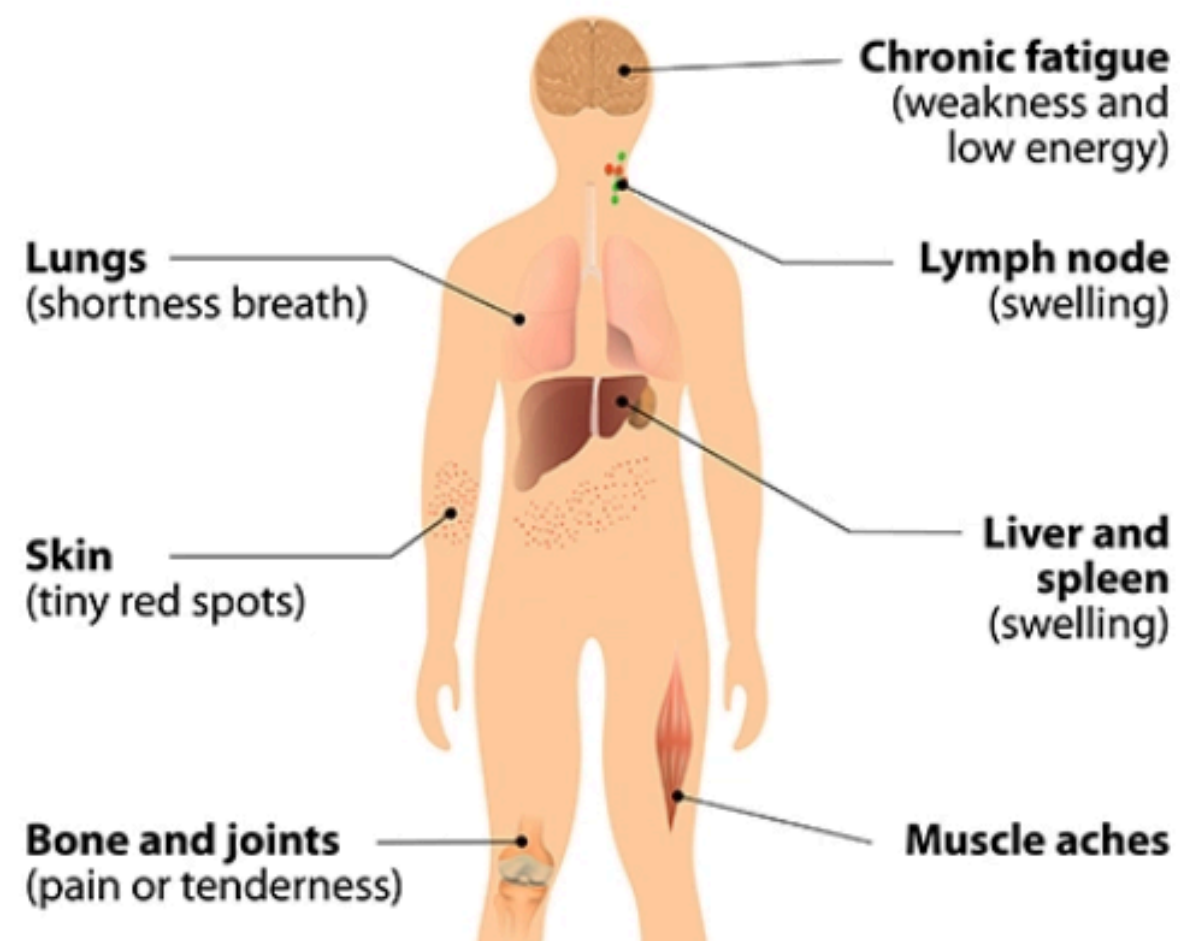
(Popa, et al., 2025)

Immunoproliferative Disorders

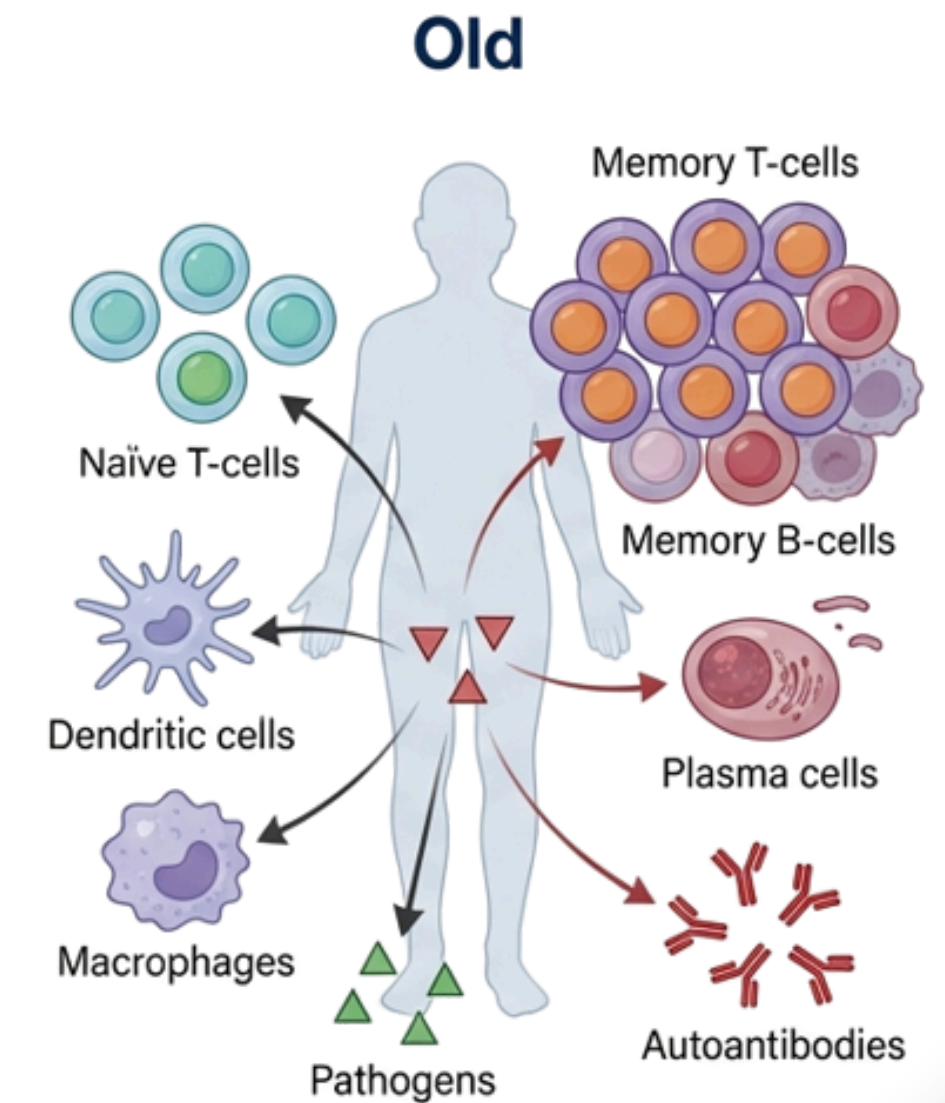
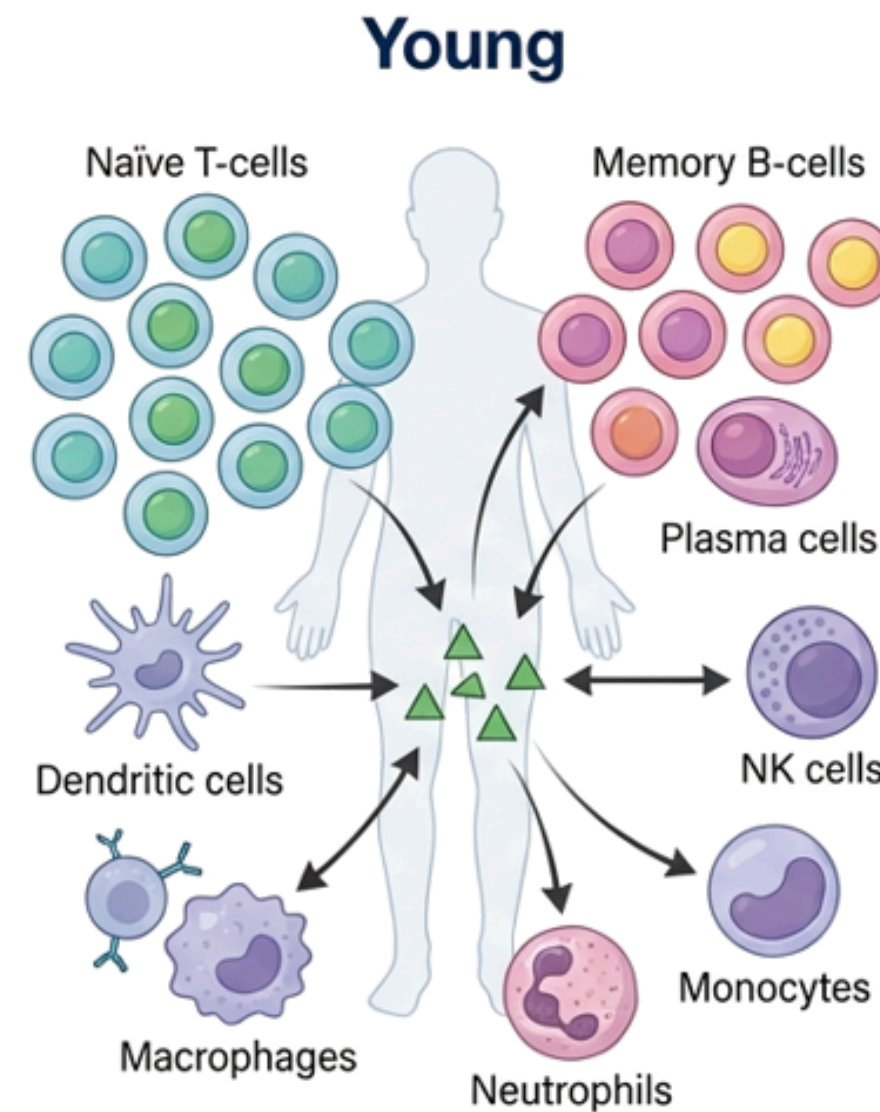
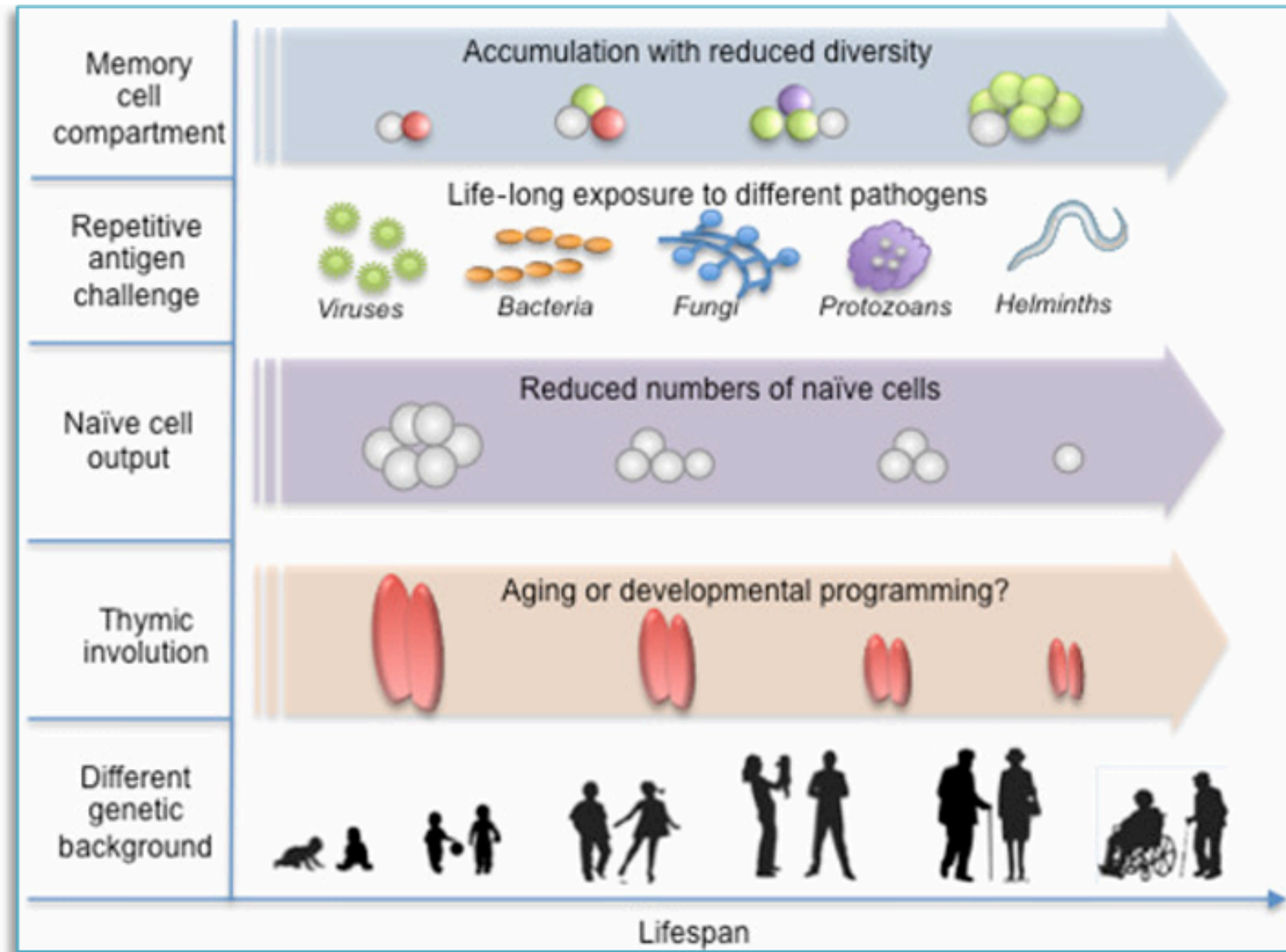
Definition: Abnormal proliferation of cells of the immune system.

Types: Reactive Lymphoproliferation:
Response to infection/inflammation

Malignant Lymphoproliferation: Leukemia, Lymphoma



The Aging Immune System



(Müller & Pawelec, 2016)

Case study

Patient Profile: Mrs. J, a 72-year-old female, comes to the hospital with a 2-month history of **severe fatigue, weight loss, and shortness of breath that worsens with deep breathing** (pleuritic chest pain).

History:

She complains of **morning stiffness in her wrists and knees**.

She denies having any skin rashes (No butterfly rash).

Investigations:

- CBC: **Pancytopenia (Low WBC, Low RBC, Low Platelets)**.
- Autoantibody Screen: Positive ANA (1:320), Positive Anti-dsDNA. Negative Anti-Histone **(ruling out drug-induced lupus)**.





Question

1. what is the specific pathophysiological mechanism causing tissue damage in this patient?
2. How does "Immunosenescence" (aging of the immune system) contribute to the development of autoimmunity in this patient?

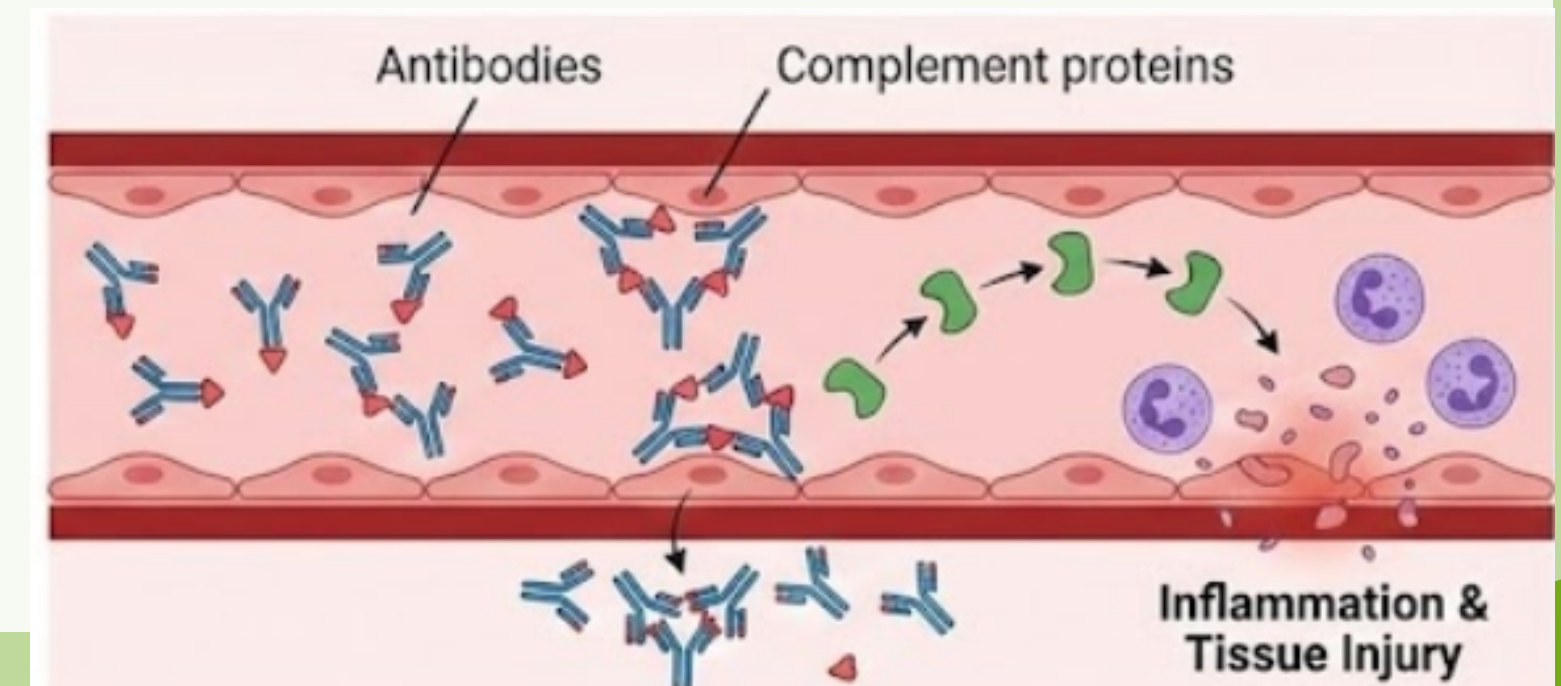
Answer

1

Type III Hypersensitivity (Immune Complex Mediated)

Mechanism:

1. Formation of **Antigen-Antibody complexes** in the circulation.
2. Deposition of these complexes in vessel walls (**Vasculitis**) or serosal membranes (Pleura).
3. Activation of the **Complement System**.
4. Recruitment of neutrophils -->
Release of lysosomal enzymes -->
Tissue injury (**Pleuritis/Cytopenia**)



Answer

2

Defective Self-Tolerance: The aging thymus produces fewer T-cells, and the mechanisms to delete self-reactive T-cells become less efficient.

Accumulation of Self-Reactive Cells:

Memory T-cells that are exhausted or senescent may secrete pro-inflammatory cytokines (Inflam-aging), triggering an autoimmune response against self-antigens.

Young vs. Elderly SLE

Classic SLE (Young)	Late-Onset SLE (Elderly)
 Prominent Malar Rash (Butterfly)	 Serositis (Pleural Effusion/Pericarditis)
 Photosensitivity	 Pulmonary Involvement
 Lupus Nephritis (Kidney)	 Hematologic Abnormalities (Cytopenias)
 Arthritis	 Less Common Rash/Kidney



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