

**The People's Democratic Republic of Algeria**  
**Ministry of Higher Education and Scientific Research**  
**University Center of Mila**  
**Faculty of Science and Technology**  
**Department of Natural and Life Sciences**

**TD 07 – Immunology correction**

**Exercise 1:**

ACROSS:

1. **COMPLEMENT** — The protein complex designated by the symbol C that plays roles in opsonisation, inflammation, and direct lysis via MAC formation.
2. **PHAGOCYTOSIS** — The cellular process by which specialized cells (macrophages, neutrophils) ingest and destroy solid foreign particles on a micrometric scale.
3. **PARACRINE** — A cytokine secretion mode where the cytokine acts on nearby cells in the immediate vicinity of the secreting cell.
4. **CD4** — The co-receptor present on T helper cells that interacts with MHC Class II molecules on antigen-presenting cells.
5. **DIGESTION** — The stage of phagocytosis where the phagosome fuses with lysosomes to form a phagolysosome for microbial degradation.
6. **DENDRITIC CELL** — The cell type that bridges innate and adaptive immunity by capturing antigens in tissues and migrating to lymph nodes to present them to T cells.

DOWN:

7. **MAC (Membrane Attack Complex)** — The pore-forming complex composed of complement components C5b through C9 that creates transmembrane channels in target cells.
8. **IL-2 (Interleukin-2)** — The pro-inflammatory cytokine that serves as the major factor in lymphocyte growth and proliferation, acting as an autocrine growth factor for activated T cells.
9. **ANTIBODIES (Immunoglobulins)** — Soluble glycoproteins produced by plasma cells in response to a specific antigen; they bind specifically to that antigen via variable regions.
10. **DIAPYCNOSIS (Extravasation)** — The process by which leukocytes cross the endothelial wall of blood vessels to migrate from the bloodstream into infected tissues.

**Exercise 02:**

**Case 1: Bacterial Skin Infection**

**a) Innate barriers bypassed**

The deep laceration bypassed two primary physical barriers of the innate immune system: (1) the skin (the largest and most important physical barrier, providing a mechanical shield against pathogen entry), and (2) the mucous membranes (if the wound involved mucosal surfaces). Additionally, the breach disrupted the chemical barrier of the skin surface, including the acidic pH, antimicrobial peptides (defensins), and normal microbiota that compete with pathogens for resources and produce inhibitory substances.

**b) Inflammatory mechanisms**

The four cardinal signs of inflammation are explained by the following mechanisms involving specific cells and mediators:

Redness (erythema) is caused by vasodilation of arterioles at the injury site, mediated primarily by histamine released from mast cells and prostaglandins (PGE<sub>2</sub>) produced by damaged cells and macrophages. The increased blood flow brings more immune cells and oxygen to the area.

Swelling (edema/tumor) results from increased vascular permeability. Histamine, bradykinin, and leukotrienes cause endothelial cells of post-capillary venules to contract, creating intercellular gaps through which plasma proteins (including complement and acute-phase proteins) and fluid extravasate into the interstitial tissue space.

Heat (calor) is a direct consequence of the increased blood flow (vasodilation) at the site, bringing warm blood from the body core to the skin surface.

Pain (dolor) is caused by multiple mechanisms: (1) pressure on nerve endings from the accumulating fluid (edema), (2) direct stimulation of pain receptors by bradykinin and prostaglandins (which sensitize nerve endings to other stimuli), and (3) the action of C3a and C5a anaphylatoxins, which can directly activate nociceptors. The elevated C3a and C5a levels in this patient confirm active complement activation and anaphylatoxin release.

## Case 2: Recurrent Viral Infections

### a) Affected immune branch

The cellular immunity branch of adaptive immunity is primarily affected. The reduced CD8<sup>+</sup> T cell count directly impairs the cytotoxic T lymphocyte (CTL) response, which is essential for eliminating virus-infected cells. CD8<sup>+</sup> CTLs recognize viral peptides presented by MHC Class I on infected cells and destroy them through apoptosis. The reduced NK cell activity further compounds the deficiency in recognizing and killing virus-infected cells through the innate surveillance mechanism.

### b) Normal antibody responses despite infections

The humoral immunity branch (B cells and antibodies) remains functional because B-cell activation and antibody production can still proceed normally. Thymo-dependent B-cell activation requires LTh2 (CD4<sup>+</sup>) helper cells, not CD8<sup>+</sup> cells, and the LTh2 population appears intact in this patient. The cell cooperation between B cells (antigen presentation via MHC II), LTh2 cells (CD40L-CD40 co-stimulation and cytokine secretion), and the subsequent plasma cell differentiation pathway is unaffected, allowing normal antibody production against extracellular pathogens.

## Exercise 03:

### Sequence A: Classical Complement Activation

1. C1q binds to the Fc fragment of IgM or IgG antibodies (pathogen surface)
2. C1s cleaves C4 and C2 → C4a, C4b, C2a, C2b
3. C3 convertase (C4b2a) cleaves C3 → C3a and C3b
4. C5 convertase (C4b2a3b) cleaves C5 → C5a and C5b
5. C5b recruits C6, C7, C8, and (C9)<sub>n</sub> to form the MAC

### Sequence B: Phagocytosis

1. Opsonisation: C3b or antibodies coat the pathogen surface
2. Adhesion: phagocyte recognizes the opsonized pathogen via surface receptors (FcR, CR1)
3. Ingestion: pathogen is internalized into a membrane-bound phagosome
4. Digestion: phagosome fuses with lysosomes to form a phagolysosome; enzymes degrade the pathogen
5. Exocytosis: waste materials and undigested debris are expelled from the cell

## **Sequence C: Inflammatory Response**

- 1. Skin injury allows pathogens to bypass natural physical barriers**
- 2. Recognition: sentinel cells (macrophages, mast cells) identify the pathogen and produce chemical mediators (histamine, cytokines, prostaglandins)**
- 3. Vasodilation: blood capillaries dilate, causing erythema (redness) and edema (swelling)**
- 4. Diapedesis: granulocytes and monocytes cross vessel walls (extravasation) following chemotactic gradients**
- 5. Phagocytosis: macrophages ingest and digest the microorganisms at the infection site**