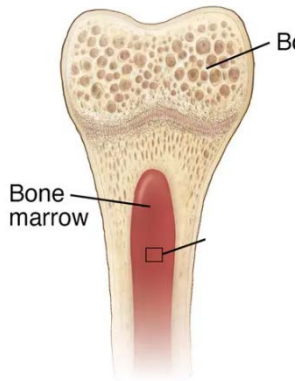
Nutrition in Health course

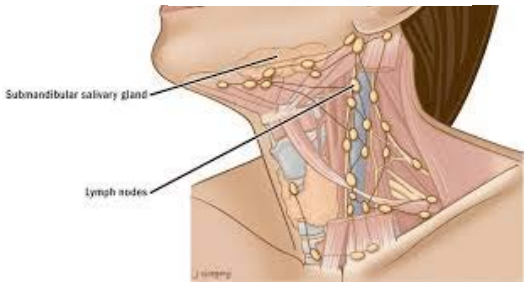
Module 4: Nutrition and Immune Fundamentals

I. The Immune System: Core Functions and Organization

The principal function of the immune system is host protection from infectious diseases caused by pathogens (viruses, bacteria, fungi, protozoa, or parasites).

1. **Defense Roles:**
    - **Pathogen Clearance:** Fighting professional pathogens (like the measles virus or *Vibrio cholerae*).
    - **Control:** Managing commensal organisms (usually benign) that colonize the skin and gut.
    - **Injury Response:** Reacting to sterile injury (tissue damage without microorganisms).
2. **Organization (Where Cells Develop and Meet):**
    - **Primary Organs:** Bone marrow (origin of all white blood cells/leukocytes) and thymus (T cell maturation).
    - **Secondary Organs:** Lymph nodes and spleen. These are "meeting places" where information from the innate system is transferred to the adaptive system.

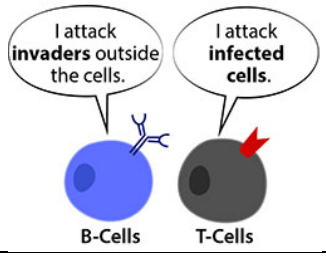




II. The Two Immune Teams: Innate vs. Adaptive

The immune system functions as an integrated whole, comprising two main components: innate and adaptive immunity.

Feature	Innate Immunity (The General/Fast Response)	Adaptive Immunity (The Specific/Memory Response)
Speed	Fully functional at birth; rapid response.	Slower to develop upon initial infection.
Recognition	Recognizes **Pathogen-Associated Molecular Patterns (PAMPs)**—signature molecules common to groups of microorganisms (e.g., bacterial flagella).	Responds to a **specific pathogen** (e.g., measles virus specifically).
Memory	No memory response; responses are the same for all individuals.	Develops **immunologic memory** for faster, more efficient second encounters.

Mechanisms	**Barrier defenses** (skin, gut epithelia), **Phagocytosis** (killing by macrophages and neutrophils)	Relies on specialized cells: **T cells** (helper, cytotoxic) and **B cells** (produce antibodies). 
Systemic Effect	High activity triggers the **Acute Phase Response** (fever, malaise). The host decreases serum iron and zinc levels to inhibit bacterial acquisition of these minerals.	Antibody production (Immunoglobulins like IgG, IgA). **T-Helper (Th) cells** promote B cell or Cytotoxic T Lymphocyte (CTL) responses.

III. Key Nutritional Impacts on Immunity

Nutrients play crucial roles in regulating the function, proliferation, and protective mechanisms of immune cells.

A. Vitamins

Nutrient	Core Immune Role	Key Deficiency Impact
Vitamin A	**Maintains epithelial barrier defenses**. Promotes development of iTreg cells (tolerance to gut flora).	Impairs barrier defense (squamous metaplasia) and reduces antibody responses (especially secretory IgA). Supplementation reduces infant mortality in high-burden areas.
Vitamin D	**Innate Immunity:** Macrophages produce calcitriol, which increases antimicrobial peptides (like cathelicidin).	Deficiency is postulated to increase the risk of autoimmune disease. Vitamin D may have an overall immunosuppressive activity in adaptive immunity (inhibiting Th1/Th17 cells and increasing regulatory T cells).
Vitamin C	Involved **in maintaining Th1 function**. Protects phagocytic cells (neutrophils) from the oxidative stress associated with bacterial killing.	Deficiency reduces delayed-type hypersensitivity (DTH) skin responses.
B Vitamins (B6, B12, Folate)	**Essential for nucleic acid and protein synthesis**.	Deficiency impairs both T-cell and B-cell function, leading to reduced proliferation and decreased antibody synthesis.
Vitamin E	Promotes Th1 responses. Important for improving declining immune responses in the aged.	Enhanced the formation of immune synapses between TCRs and APCs in purified T cells.

B. Minerals and Trace Elements

Nutrient	Core Immune Role	Key Deficiency Impact
Zinc	Essential for B and T cell activities.	Deficiency causes thymic atrophy and reduced Th1 function (decreased IL-2 and IFN-gamma production). Supplementation reduces the risk of infectious diseases (like diarrhea) in children.
Iron	Required for Th1 cell function (IFN-gamma production).	Th1 cells are sensitive to deficiency. The host actively reduces serum iron levels during the acute phase response to restrict iron availability to opportunistic pathogens.
Selenium	Component of **antioxidant enzymes** (glutathione peroxidase).	Deficiency impairs T-cell proliferation and B-cell Ig synthesis. Associated with increased virulence and mutation rates of viruses in mouse models.
Copper	Component of antioxidant enzymes (superoxide dismutase).	T-cell proliferation is reduced in copper-deficient animals and humans.

C. Fatty Acids (Eicosanoid Precursors)

Fatty acids (PUFAs) modify immune function primarily through the production of eicosanoids and effects on cell signaling.

1. **Omega-6 (Arachidonic Acid - AA):**
    - AA (C20:4, n-6) is the precursor for pro-inflammatory mediators.
    - Produces 2-series prostaglandins (PGE2) and 4-series leukotrienes (LTB4). LTB4 enhances leukocyte chemotaxis and killing.
2. **Omega-3 (EPA and DHA):**
    - **EPA** is a precursor for less inflammatory mediators (e.g., LTB5 has lower activity than LTB4). Increased EPA intake has anti-inflammatory effects.
    - **DHA** has independent anti-inflammatory effects related to the production of specialized mediators called resolvins and protectins. DHA can also block signaling initiated by bacterial LPS (Toll-like receptor signaling).

End of module 4…