Chapter 24
Nutrition-Mediated Immunity in Autoimmune Disorders at Molecular Pharmacological Approach:
A Conceptual Framework

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ABSTRACT

In autoimmune disorders (AD), innate defenses are inborn abilities of the immune system to detect, attack, and eliminate (or at least restrain) pathogenic invaders. In summary, micronutrients are essential for fine-tuning the development and function of immune cells. Altered homeostasis of micronutrients as seen in various autoimmune diseases could critically influence immunity and promote autoimmune dysregulations. The geo-epidemiological distribution of autoimmune diseases (ADs), their correlation with socioeconomic status, and their rapid increase in developed countries, together with observations in migrant populations, suggest that environmental factors, rather than genetic ones, are chiefly driving these evolutionary processes. This also paves the way for adaptive immune recognition that provides long-lasting immunity. The co-stimulation signals ensure nonreactivity when encountering self-antigens, in which case peripheral tolerance (through deletion or

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energy) would be induced. However, it is complicated how deviations would occur and specifically, how the breach of self-tolerance and autoimmunity would develop different antigen-based disorders.

1. INTRODUCTION TO NUTRITION MEDIATED IMMUNITY IN AUTOIMMUNE DISEASE

Autoimmune diseases (ADs) are a family of at least 80 diseases that result from an individual's immune system attacks the body's own tissues; the pathogenesis and etiology are not fully understood, but environmental factors (lifestyle, diet, drugs, infections) and certain genetic backgrounds have been proposed as risk factors. The incidence of ADs, approximately 3–5% worldwide, is increasing in westernized societies, as confirmed by epidemiological studies; these suggest that multiple sclerosis (MS), type 1 diabetes (T1D), inflammatory bowel diseases (mainly Crohn's disease), systemic lupus erythematosus (SLE), primary biliary cirrhosis, myasthenia gravis, autoimmune thyroiditis, hepatitis, and Tuberculosis mediated rheumatic diseases are steadily increasing. Recognition of these molecular patterns allows for rapid host defense while "mostly" maintaining nonreactivity to self (thorough recognizing only PAMPs and DAMPs).*(Huang S.,.2012)*

2. NUTRITION MEDIATED IMMUNITY IN AUTOIMMUNE DISORDERS AT MOLECULAR PHARMACOLOGICAL APPROACH

It is categorized by enhanced fluid accumulation, incendiary cell differentiation of the joints, and stimulates the release of immune cells (IL-1, IL-17 and TNF-α). TNF-α and IL-6 antagonists have been used in experiments to be beneficial against RA. Decreased development of immunoregulatory mediators such as IL-11, IL-13 and IL-10 leads to the immune response. Chronic inflammatory disorders such as RA are caused by an elevated percentage of pro-inflammatory to anti-inflammatory cytokines, which is induced by helper T cell type 1 (Th1) .TNF-α, IL-17A, IL-17F and IFN (-interferon) are produced by helper T17 (Th17) cells (which protect from foreign microorganisms) and contribute to the pathophysiology of RA. Several receptors that identify pathogen-virulence factors, such as TLRs, regulate RA by activating host-defense mechanisms and maintaining adaptive inflammatory reactions. TLRs control nuclear factor kappa-B ligand (NF-B), osteoclast formation and promote the elevated generation of TNF-α, IL-6, IL-12, IL-18 and a variety of other proinflammatory cytokines. TLRs are found in the cartilaginous fluid, where they