REPROGRAMMING OF THE IMMUNE SYSTEM DURING ZINC DEFICIENCY

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■ **Abstract** Thymic atrophy, lymphopenia, and compromised cell- and antibody-mediated responses that cause increased rates of infections of longer duration are the immunological hallmarks of zinc deficiency (ZD) in humans and higher animals. As the deficiency advances, a reprogramming of the immune system occurs, beginning with the activation of the stress axis and chronic production of glucocorticoids that accelerate apoptosis among pre-B and -T cells. This reduces lymphopoiesis and causes atrophy of the thymus. In contrast, myelopoiesis is preserved, thereby providing protection for the first line of immune defense or innate immunity. Changes in gene expression for cytokines, DNA repair enzymes, zinc transporters, signaling molecules, etc., suggest that cells of the immune system are attempting to adapt to the stress of suboptimal zinc. Better understanding of the molecular and cellular changes made in response to inadequate zinc should lead to the development of immunotherapeutic interventions.