

Introduction

Proper nutrition has long been recognized as a prerequisite for a healthy immune function. Ancient writings from the Egyptians, Indians, and Greeks all include admonitions about foods and health; and further stress that to fully understand disease, the diet must be evaluated. However, the first truly scientific evidence linking nutrition and immunity came in 1810 when J.F. Menkel described thymic atrophy in malnourished patients. These early works eventually led to the discovery of the roles of lipids, proteins, energy, vitamins, and minerals in the body's defense mechanisms. Although all nutrients are important for immune function, this bulletin will focus solely on the roles of trace minerals and their interaction with immunity.

Deficiencies and excesses of the trace minerals have a direct effect on the immune system and can make an animal more susceptible to infection. Specific ways that nutrition affects the immune system include:

- anatomical development of lymphoid tissue
- mucus/lung surfactant production
- synthesis of immunologically active substances
- cellular proliferation
- cellular activation and movement
- intracellular killing
- detoxification of free radicals produced by phagocytic cells
- modulation and regulation of immune processes

The trace minerals most often associated with immune function, and possibly most studied, are Cu, Zn, and Se. All of

these minerals play an important role in antioxidant defense, Se through glutathione peroxidase, and Cu and Zn through Cu/Zn superoxide dismutase. The remainder of this bulletin will focus on the roles of these three minerals in immune function.

Copper

Copper is involved in all aspects of immune responsiveness. Through its role in Cu/Zn superoxide dismutase, Cu aids in the scavenging of free radicals, thus preventing oxidative damage. Copper deficient animals exhibit a decrease in the number of circulating neutrophils (Koller et al., 1987). Boyne and Arthur (1981) reported that respiratory burst and microbicidal activity of bovine peripheral blood neutrophils was decreased by Cu deficiency. Copper deficiency has also been shown to decrease activity of peritoneal macrophages (Babu and Failla, 1990) and reduce humoral and cell-mediated immune responsiveness (Bonham et al., 1990). Galyean et al. (1995) noted that adding organic Cu to the receiving diet tended to decrease the percentage of morbid steers compared with the control diet.

Selenium

The trace mineral Se is very important to immune processes. Selenium affects the development of nonspecific, humoral (antibody), and cell-mediated immune responses. A Se deficiency appears to result in immunosuppression, whereas supplementation results in augmentation

and/or restoration of immune function. Specifically, Se deficiency inhibits:

- resistance to microbial and viral infections,
- neutrophil function,
- antibody production,
- proliferation of B and T lymphocytes in response to mitogens, and
- cytodestruction by T lymphocytes and natural killer cells.

The exact mechanisms by which Se affects the immune system are largely speculative. The role of Se in glutathione peroxidase represents only one of many regulatory mechanisms.

Selenium research has shown that neutrophils from Se deficient calves had a decreased ability to kill *Candida albicans* (Boyne and Arthur, 1981). Reffett et al. (1988) found that Se-adequate calves had greater serum antibody titers to IBRV challenge than Se-deficient calves after a second challenge on d 35 of their study. Beck et al. (2005) noted that macrophage phagocytosis was increased in calves supplemented with Se yeast compared with control and sodium selenite supplemented calves. Furthermore, skin swelling responses after injection with PHA tended to be increased by Se supplementation.

Zinc

A nutritional deficiency of Zn is consistently associated with increased morbidity and mortality (Kincaid et al., 1997). Zinc deficiency can induce the following effects on immune function:

- Thymic atrophy and loss of T helper cell function
- Reduction in thymic hormones and Thy 1 positive lymphocytes

- Suppressed lymphocyte blastogenic response
- Decreased production of antibodies to T-dependant antigens
- Impaired natural killer cell activity, phagocytosis of macrophages and neutrophils, and chemotaxis and generation of oxidative burst

Johnson et al. (1988) reported that supplemental Zn increased gain, decreased medical treatments, and decreased morbidity. In a similar study, Galyean et al. (1995) found that morbidity from BRD was decreased by 52% in newly weaned steers fed 70 ppm supplemental Zn compared with steers receiving only 35 ppm Zn. Skin swelling response to PHA was greater for animals receiving supplemental Zn compared with control animals, suggesting a more responsive cell-mediated immune response (Engel et al., 1995 and Kegley et al., 2001). Salyer et al. (2004) reported no differences in morbidity and mortality between calves receiving Zn polysaccharide complex and Zn sulfate; however, calves receiving Zn polysaccharide complex had greater antibody titers in response to ovalbumin than calves fed Zn sulfate, an indication of increased humoral immunity.

References available on request. For more information on this topic, please visit our website at: <http://www.qualitechco.com> or call us at 800-328-5870.

