



Salt and Trace Minerals for Livestock, Poultry and Other Animals

BSE: POTENTIAL ROLE OF Trace Minerals

BSE is a short for Bovine Spongiform Encephalopathy, more commonly referred to as “Mad Cow Disease”. BSE was first observed in Great Britain in 1984 and was identified as a specific disease in 1986. The scope of the disease developed rapidly so that by June of 1990, there were over 14,000 confirmed cases out of a population of approximately 10 million cattle in Great Britain. The epidemic peaked in 1992-93 with approximately 1,000 confirmed cases per week. Since 1986 over 200,000 head of cattle have been destroyed after showing signs of BSE.

The exact cause of the disease is still under scientific investigation. Bacteria and viruses have been ruled out as the root cause. Most scientists believe the disease is caused by a self-replicating protein called a prion. But the question still remains as to why the disease exploded in the British cattle population. Recently a new theory that is getting a great deal of attention is that a trace mineral imbalance may be the root cause.

The term Bovine Spongiform Encephalopathy is the technical name for this disease because brain tissue sections of infected cattle appear spongy and infiltrated with a starch-like plaque when examined under a microscope. BSE causes a progressive degeneration of the central nervous system in cattle

The infective dose required for BSE transmission in cattle is quite small. An oral dose of 0.5 to 1.0 gram (0.02 to 0.04 oz.) of infected brain tissue is all that is required.

The contagious agent has only been found in brain, spinal cord, and retina of infected cattle. Consumption of meat or milk has not caused the disease. The mechanism by which the infectious agent is transferred from the digestive tract to the central nervous system has not been explained.

Prion protein is a glycoprotein produced by nerve tissues. This protein may protect the nerves in that it has superoxide dismutase activity (280). Deactivating harmful oxygen free radicals appears to be a key role. Most diseases involving prions such as CJD, scrapie in sheep, and chronic wasting disease in deer arise sporadically without a known cause. Other diseases are associated with point mutations in the prion protein. Prion diseases are characterized by the conversion of the normal cellular form of the prion protein to an altered isoform. It appears that when the isoform is introduced into the central nervous system, it can catalyze the conversion of the normal prion protein to the abnormal isoform. This isoform then is resistant to normal proteinase enzymes and accumulates within the brain causing the neurological degeneration and behavioral signs. This seeding theory helps to explain why the disease develops very slowly at first and then as more isoforms are developed they spout additional isoforms and the animals deteriorate rapidly.

Recently it has been shown that normal prion protein contains copper. This copper can be utilized at the nerve synapse or incorporated in copper/zinc superoxide dismutase. The prion will bind up to four atoms of copper and assumes a structure that is susceptible to proteinases. However, Brown et al., (281) showed that the prion was also capable of binding manganese and nickel. But more importantly, when manganese replaced copper the three-dimensional structure was changed such that the resulting prion was over 100 times more resistant to proteinase degradation. In addition, as the manganese bound prion aged it became increasingly resistant to the proteinase in vitro. Proteinase resistance and the resulting fibril formation is the most distinguishing characteristic of the isoform.

When the exact mechanism of BSE is finally understood there will still be the sporadic prion diseases that occur in nature without any known infective agent. Therefore some environmental factor that predisposes certain animals or humans to the disease is a distinct possibility. Recent research into finding a factor common among localized areas associated with scrapie in sheep (Iceland), CJD in humans (Slovakia), and chronic wasting disease in deer (Colorado), showed that the soil in these areas is low in copper and high in manganese (282).

Of course these finds do not explain why BSE has become such a problem in Great Britain. However a farmer, Mark Purdey in Britain has studied this problem extensively and made some interesting observations (283). Mark Purdey contends that the incidence of BSE is not associated with the feeding of meat and bone meal as much as it is with the use of an organophosphate called phosmet to control Warble fly maggots. One of the traits of Phosmet according to Purdey is that it binds copper making it unavailable to biological systems. Could it be possible that the manganese containing prion was present in the meat and bone meal and that small amounts were absorbed and because Phosmet bound the available copper this acted as the seed prion to initiate the development of BSE? The relationship of copper and manganese in the development of prion diseases is gaining credibility. Researchers at

