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Bovine Spongiform Encephalopathy: What Is Our Risk in America?

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Bovine Spongiform Encephalopathy (BSE) or what the news media insist on calling *"mad cow disease"* has been recognized for almost 15 years as a devastating disease of cattle. There are still many questions regarding this disease. However, there has been some progress and more is known about the mysterious condition. A more important question is, *What are the risks of introducing BSE in the United States?*

What is Bovine Spongiform Encephalopathy (BSE)?

BSE is a chronic degenerative disease that affects the central nervous system (brain & spinal cord) of cattle, first diagnosed in cattle in Great Britain in 1986. BSE belongs to a group of diseases known as Transmissible Spongiform Encephalopathies (TSEs) These TSEs include scrapie (sheep & goats), transmissible mink encephalopathy, feline spongiform encephalopathy (cats), chronic wasting disease of elk and mule deer, and BSE in cattle. Humans have a number of TSEs also and these include kuru, Creutzfeldt Jakob Disease (CJD), new variant Creutzfeldt Jakob Disease (nvCJD), Fatal Familial Insomnia, Gerstmann-Straussler syndrome (in humans). The clinical signs or symptoms in cattle appear as nervousness or aggression, abnormal posture, incoordination, weight loss, difficulty rising, progressing to death. There is no treatment for any of these conditions and currently there are no vaccines available for prevention.

How is BSE recognized or diagnosed?

BSE cannot be confirmed in the live animal. It has signs similar to rabies, polioencephalomalacia, *Hemophilus somnus* infection, and a number of other diseases. The microscopic examination of brain tissue is the only way BSE can currently be diagnosed. There is no "live animal test" for BSE; however, one is needed badly. A live animal test that could identify an "infected" animal well before it becomes ill would be particularly valuable.

What causes BSE?

The cause of BSE and the other TSEs in other species is not fully understood. Most of the scientific community feels the cause is a prion or abnormal protein. BSE (like all TSEs) has (1) a long incubation period (most often several years), (2) is smaller than a typical virus, (3) the agent is resistant to sunlight, radiation, and common disinfectants, and (4) the agent causes no detectable immune response in the host.

Why did BSE occur in Great Britain and subsequently spread to Europe?

The bottom line to this question is: *No one knows for sure*. The epidemiologic data suggests that BSE is associated with the feeding of meat and bone meal as a protein source to dairy cattle and other cattle. The causative agent is suspected to have come from either scrapie-infected sheep or from cattle with a previously unknown TSE. Changes in the rendering practices in the U.K., such as lowering the temperature of processing and/or removing the solvent step in processing, may have allowed the survival of the agent in the meat and bone meal. BSE had never been identified before 1986, when it was first recognized in Britain. BSE has been confirmed in native cattle in Ireland, Northern Ireland, France, Portugal, Germany, and Switzerland. It is now thought that meat and bone

meal exported from the U. K. was responsible for the infection of native cattle in these other countries. BSE has been identified in cattle exported from Britain to other countries. Prior to 1986, a small amount (14 tons) of ruminant protein was imported from the U.K. to the U.S.A. Our current regulations prohibit the import of ruminant proteins from any country affected with BSE. Overall, 99% of the BSE cases have occurred in the U. K. Currently, the BSE epidemic in Britain is winding down; however, cases of BSE have been identified at an alarming rate on mainland Europe.

Do we have BSE in the United States?

No. There have been no cases of BSE in the U.S.A. There was one case in Canada (which was in a cow imported from Britain). Before the ban on British cattle imports into the U.S.A. went into affect in 1989, there were 499 cattle brought to the U.S.A. from Britain. All of those cattle were carefully accounted for and none showed evidence of BSE.

Veterinarians and others in the U.S.A. have very aggressive surveillance programs for BSE. This includes the National Veterinary Services Laboratory in Ames, Iowa, the Centers for Disease Control, the USDA, and all state veterinary diagnostic laboratories. Surveillance of high-risk populations such as disabled dairy cattle has continued at a high rate, with more than 1,000 cattle from California alone examined for evidence of BSE to date. So far, there has been no evidence of BSE in the U.S.A.

What else has been done to prevent BSE from occurring in the U.S.A.?

No beef or beef products, including items such as fetal bovine serum for use in research laboratories, have been imported from counties known to have BSE, such as Britain since 1989. Also, no beef products were imported from Britain to the U.S.A. prior to 1989 because no FSIS-approved establishments for export to the U.S.A. existed in the U.K. Current regulations prohibit the importation of ruminant protein from all countries affected with BSE. As mentioned above, surveillance is continuing at a very high rate.

Does BSE cause new variant Creutzfeldt Jakob Disease (nvCJD) in humans?

CJD is a slow progressive disease of humans that affects the central nervous system, causing dysfunction, progressive dementia, and death. CJD occurs throughout the world at a rate of about 1-2 cases per million people per year. There has been a major change in CJD in Britain since the outbreak of BSE in cattle. This change has been the recognition of a new variant CJD or nvCJD in humans. To date, there have been more than 70 cases of nvCJD recognized in humans in Britain and one case in France. The exact cause of this new form of the disease is not entirely known; however, circumstantial evidence suggests that BSE may be involved. It is this possibility that nvCJD could jump the "species boundary" from cattle to humans that has everyone so nervous. The abnormal protein in the brain of nvCJD patients and the abnormal protein in the brain of BSE cattle appear closely related. While the exact relationship between BSE and nvCJD is not fully understood, the prudent course has been to assume a possible link. This has prompted the effort to eliminate BSE in Britain (and now Europe) and to work even harder to prevent BSE from occurring in the U.S.A.

What is currently being done to prevent BSE in the U.S.A.?

In addition to importation bans on cattle and ruminant protein sources from countries with BSE, surveillance in the U.S.A. continues at a very high rate. Also, in 1997 the FDA enacted a ruminant feed ban here in the U.S.A. This prohibits feeding protein derived from mammals (such as meat and bone meal) to be fed to ruminants. There are some exceptions to this rule, but in general it is very strict and would certainly help limit any outbreak should one occur. We have some TSEs that occur in the U.S.A. These include such diseases as scrapie in sheep, chronic wasting disease in elk and deer, and transmissible encephalopathy in mink. Monitoring of all of these TSEs is occurring and active research is also ongoing on these conditions. There has been a large increase in the efforts to eliminate scrapie in sheep. New, more accurate diagnostic tests in sheep are being developed and when use of these tools becomes widespread, it may be possible to completely eradicate scrapie in sheep. Currently, there is no known risk to the cattle population of the U.S.A. with regard to BSE

and there is no risk to people consuming beef products in this country. Obviously, this problem has decimated the cattle industries of the U.K. and other countries, and we must all continue to work hard to prevent this problem in the U.S.A. This disease illustrates how important research of cattle diseases is to the health of the cattle industry and to public health and confidence.

Why all the BSE reports in the press lately?

The first reason is the alarming number of new cases of BSE in European cattle. The public was lead to believe that the threat of BSE spreading to Europe was very low and the spread of new cases has occurred despite the efforts in Europe. Secondly, the ban on ruminant-to-ruminant by-product feeding in the U.S.A. has been in effect since 1997 and the FDA (the agency responsible for enforcing the ban) has not been very active with regard to on site inspections or education of feed mills relative to these regulations, according to some sources. Thus, recently, the FDA "discovered" that some meat and bone meal had inadvertently been fed to about 1,000 head of cattle in a Texas feedlot. Of course, none of that bone meal was from outside the U.S.A. and the cattle have been identified and isolated. Presently, no one has indicated that this constitutes a risk for American cattle or consumers. It may simply be the FDA responding to criticism for not doing inspections on a routine or thorough basis. It does point out the fact that industry and the regulatory agencies charged with protecting us must all work together in a coordinated manner to avoid the type of disaster that occurred in Britain and Europe.

What must we do to prevent BSE from occurring in America?

If a BSE case were to occur in the U.S.A. it would be economically and politically devastating to the cattle industry. This problem needs to be avoided, period! Stringent bans on cattle, cattle by-products, and beef products from at-risk nations need to be put into place and maintained. The regulatory agencies need to aggressively monitor these bans. Producers need to support efforts by their state and national associations to ensure that science-based policies on cattle health and food safety are implemented. This support should include volunteer efforts and membership support. The cattle industry has been very proactive on this issue. Just last week, both ConAgra Beef and IBP announced they will be asking cattle producers to sign affidavits that cattle have not been fed mammalian derived protein products (such as meat and bone meal). Also, audits and inspection of feeding operations are expected as part of these new industry programs. By treating these issues seriously U.S. producers will avoid the problems now occurring in Europe.

Irrigated Pasture Fertility Management

By
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Tissue Testing

For tissue sampling, the simplest and most direct diagnostic methodology is to sample areas of good forage production and corresponding poor production areas in close proximity. By studying the differences between those two types of areas, quick analysis can be made of the most limiting nutrient deficiencies. Be sure to collect about 10-15 leaves from 10-15 spots and composite for each sample, one from the good producing area and one for the bad, for your analysis.

For clovers the top leaf portion should be the sample submitted for fertility evaluation on irrigated pastures. To evaluate grasses, the top or flag leaf portion on the upper part of the stock should be used. In late maturing grasses only the flag leaf should be used, not the stock. In earlier stages of growth the upper 4 to 6 inches, the leaf, not the stem, should be the only portions sampled.

The need for sulfur can be diagnosed best by the use of a tissue test and not a soil test. The following are tissue level numbers for acceptable ranges in grass:

- Nitrogen: younger growth 2.5 - 4 %, older growth 1.5 - 2.5 %
- Phosphorus: >0.25 % in younger grass and >0.2 % in older grass.
- Sulfur: >0.15 %, and zinc , >20 ppm.

Soil Sampling

Fertility analysis for soils may not be as reliable as leaf analysis, particularly for sulfur. A soil probe or shovel can be used, and a composite of 15-20 cores from the 6-8 inch depth, would categorize the zone that is required for managing fertility in irrigated pastures. As mentioned above, take separate soil samples from areas of good pasture growth as well as nearby areas that do not produce as well. Along with nutrients, you should also check the soil pH. Low soil pH can limit the nitrogen fixation of clovers. Soils with a pH below 5.8 should receive a lime application.

Phosphorus
The greatest increase in clover growth usually results from the application of phosphorus and sulfur but potassium may also be necessary. Soil test levels should be 8-10 ppm or higher (bicarbonate-P test). About 20 to 25 lbs. of P_20_5 per acre should be applied when phosphorus levels are low. The most concentrated form of phosphorus is 0-45-0 or 11-52- 0. Nitrogen is used in this formulation as it assists in formation of the granules in the manufacturing process. Plant growth response to phosphorus as well as potassium will usually not occur until 60-90 days after fertilizer application. Clover and other legumes respond much more to phosphorus and potassium than grasses do.

Potassium
Soil potassium levels should be 80 to 100 ppm or higher. As you get closer to the Sacramento Valley foothills on the Sierra side, soil potassium levels are often lower. In the Inter-Mountain valleys, soils originating from volcanic deposits may also have low potassium levels. About 30 to 50 lbs. or more of K_20 per acre should be applied when potassium levels are low. The most economical form of potassium fertilization is 0-0-60 or muriate of potash.

Nitrogen
Normally nitrogen fertilization is not necessary, particularly if legumes like clovers, trefoil, vetch or even alfalfa are present. If 25% of the irrigated pasture stand is clover in the form of canopy cover it should be sufficient to supply enough nitrogen for the grasses on a year around basis. If additional forage production is needed, a nitrogen application of 40 to 50 lbs. of N per acre followed by an irrigation can be applied 3-4 weeks prior to the requirement for more forage. Nitrogen applications made in July can improve forage production during the summer from cool season perennial grasses in pastures. High nitrogen fertilization can often mask other nutrient deficiencies.

Zinc
Irrigated pasture fields that have had recent land leveling requiring a high degree of cuts in them may require zinc fertilization. Identifying those areas and limiting your fertilization practices for zinc to only the known cut areas would decrease the cost and increase the efficiency of the application. Zinc deficiencies are more likely to be found in grasses and can be expected when soil test values are <0.5.

Water Test

The water you are applying may need to be tested if infiltration rates are poor. A proper calcium to magnesium ratio should be 1 to 1 or more calcium. If you have higher magnesium and/or high sodium, you may want to consider applying gypsum to the soil. Application of ½ to 1 ton of gypsum per year would be a way to evaluate an improvement in water infiltration. At the present price, that would put it at approximately $35 per acre.

If you have questions or would like additional information on the topics covered in the newsletter, please get in touch with me.

Sincerely,

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