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Layout

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Logo Design

Ruth Cronje, and Jan Swanson;

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BSE *will* affect the swine industry

William D. Hueston DVM, PhD, DACVPM

Director, Center for Animal Health and Food Safety, College of Veterinary Medicine, University of Minnesota

Introduction

The Bovine Spongiform Encephalopathy (BSE) epidemic has spread throughout continental Europe and its human equivalent, variant Creutzfeldt-Jakob Disease (vCJD), has claimed more than 100 lives. "Mad Cow Disease" has achieved almost universal name recognition among American consumers despite the fact that no BSE or vCJD have been identified in the US. Furthermore, some lobby for additional prevention measures, fostering headlines warning "Is the US doing enough?" Ironically, BSE has affected the swine industry as well, even though no naturally occurring BSE has been identified in swine.

This paper discusses the impact that BSE is having on the swine industry. Both the real and perceived issues will be dissected in order to understand the present and future consequences of BSE for swine practitioners and pork producers.

Evidence to date

Bovine spongiform encephalopathy was first diagnosed in 1986 in British cattle and a kudu, an exotic antelope in the London Zoo. The disease manifests as a progressive neurologic disorder affecting sensation, mentation, and locomotion. The affected animal demonstrates no immunologic response. No effective treatments have been identified and the disease is universally fatal. The BSE epidemic evolved rapidly, with 1,000 new cases confirmed per week by the beginning of 1993. Over 180,000 cases of BSE have been confirmed to date, with more than 98% of the cases occurring in the United Kingdom (UK).

BSE belongs to a family of diseases called the Transmissible Spongiform Encephalopathies (TSEs). The TSE agent is remarkably resistant to common disinfectants and environmental stress, surviving temperatures greater than 500°C and gamma irradiation. All of the TSEs are associated with a unique misshapen and protease-resistant protein, called a prion. Scientific debate continues as to whether the prion alone is the TSE agent or whether additional co-factors might be involved. Regardless, the abnormal prion protein, known as PrP^{res}, is diagnostic of all of the TSEs. The TSEs include scrapie, transmissible mink encephalopathy (TME), and chronic wasting dis-

ease of deer and elk (CWD), as well as BSE. No naturally occurring transmissible spongiform encephalopathies have been identified in pigs.

The epidemiology of BSE was worked out by a thorough investigation of the first affected cattle farms. The only common and unique factor among the BSE-affected farms was the use of proprietary compounded feeds; the common feed ingredient found on virtually all affected farms was rendered animal protein, specifically meat and bone meal. A ban on the feeding of ruminant-derived meat and bone meal (MBM) from cattle diets has proved a very effective disease control strategy, reinforcing the conclusion that BSE represents a massive common source animal feed-borne epidemic. BSE is not contagious in that the disease is not spread from animal to animal through direct contact, with the possible—if infrequent—exception of maternal transmission.

Since the original case was identified in the UK, BSE has spread throughout continental Europe via trade in live cattle and MBM from the UK. The disease has been confirmed in native-born cattle in Belgium, Czech Republic, Denmark, France, Germany, Greece, Italy, Lichtenstein, Luxembourg, Netherlands, Portugal, Spain, and Switzerland, most likely owing to the contamination of their feedstuffs. In addition, cattle exported from the UK have succumbed to BSE in countries (Canada, Falklands, Oman) where no native cattle appear to have been affected. No cases of BSE have been identified in the USA in either imported or native cattle despite aggressive surveillance (USDA, 2001). International recommendations for the control of BSE concentrate on trade restrictions concerning meat and bone meal and live animals from BSE-affected countries, and bans on the feeding of ruminant-derived MBM to ruminants to prevent propagation of the epidemic if the agent is introduced (OIE, 2001).

Cases of BSE have occurred in species other than cattle owing to exposure to infective meat and bone meal. Exotic ruminants and cats, as well as domestic cats, have all demonstrated spongiform encephalopathies subsequent to the BSE epidemic in cattle. Examination of brain samples from these cases shows that the agents are indistinguishable from BSE. No cases of transmissible spongiform encephalopathies or BSE have been identified in UK pigs or anywhere else around the world.

Scientists have experimentally exposed a number of species both to BSE and to other TSEs. Experimental inoculation of pigs yielded mixed results. No transmission was documented from experimental parenteral challenge of pigs with kuru (a human TSE) and scrapie. However, parenteral challenge with BSE resulted in clinical disease and histopathology in seven of ten pigs. The other three pigs died or were removed from the study early in the incubation period, so they too may have developed the disease if they had survived for the full study period. Oral challenge of pigs with BSE infective material failed to achieve transmission. Further assay of a range of tissues from these orally challenged pigs did not detect any BSE infectivity.

Discussion

Ruminant-derived meat and bone meal (MBM) has been a significant component of swine rations in many parts of the world, including the UK. In fact, swine rations had higher inclusion rates of MBM than did cattle rations. The lack of any BSE cases in pigs during the BSE epidemic and the failure of oral challenge experiments to transmit the disease to pigs suggests that the disease is not transmitted to swine under natural conditions.

Why have the parenteral challenges been successful while the oral challenges failed? The successful parenteral challenge experiments cloud the issue of swine susceptibility to BSE. Several factors may explain the discrepancy. First, the parenteral route is recognized to be more efficient than the oral route. Consequently, the size of the oral inoculum may simply have been too small to achieve transmission. Another logical explanation stems from the recognition that the latency period of TSEs (time from exposure to clinical disease) is inversely related to the size of the exposure dose. Therefore, the orally exposed pigs simply may not have lived long enough to develop the disease. Alternatively, the pathogenesis of the disease in pigs may be different than that in ruminants. Regardless of the scientific explanation of the lack of oral transmission, the successful parenteral challenge has raised concerns that pigs may harbor the BSE agent and/or develop disease under certain conditions.

If ruminant-derived MBM continue to be used as part of swine diets, then pigs will be exposed to the BSE agent in countries where BSE exists or where contaminated bovine products are used from animal feeds. No evidence exists to suggest that the pig digestive tract can denature the BSE agent. Therefore, if the BSE agent exists in the feed, then either the agent is taken up by the gut or the agent is excreted in the feces, or both. Several research studies have examined the potential for silent carriers, that is, exposed animals in which the agent does not replicate but where infectivity remains detectable in specific tissues. Marsh et al (1969) and Race and Cheseboro (1998)

reported recovery of TSE agents (TME and hamster scrapie strain 263K, respectively) from non-susceptible species after parenteral challenge. Race and Cheseboro suggested that certain food animal species might harbor persistent BSE infectivity with replication after oral exposure.

Conclusions

No naturally occurring TSE has been identified in swine and experiments to date do not demonstrate transmission of BSE to swine through feeding of infective materials. However, concerns remain about the potential for swine to serve as silent carriers of BSE and for swine manure to harbor infectivity subsequent to the ingestion of contaminated feedstuffs. The potential for unaffected species to harbor infectivity subsequent to exposure has been shown in experimental settings but has not been observed in nature. Therefore, these concerns about the silent carrier remain hypothetical. Based on current scientific knowledge, the risk that pigs could serve as silent carriers depends on

- the susceptibility of swine (and the individual pig) to BSE;
- the efficiency of the oral route of exposure;
- the size of the exposure, including both the concentration of the agent and the number of exposure events over time; and
- the length of time between exposure and clinical disease.

Should BSE-exposed swine prove to be silent carriers, then bans on the feeding of swine-derived animal protein products would appear to be necessary for BSE prevention and control. Similarly, if infectivity remains in swine manure after feeding of contaminated feedstuffs, then limits on the use of manure would also be justified.

Regardless of the final resolution of the silent carrier issue, BSE has and will have an impact on the swine production and trade. The BSE epidemic has focused global attention on the potential for trade in animals and animal products to spread disease around the world. Concern over BSE and other animal diseases has led to tightening of health requirements, additional requirements for individual animal identification, and further guarantees concerning husbandry practices.

In the absence of scientifically sound and statistically reliable information, perception becomes reality. Public perceptions about the dangers associated with animal diseases and global trade have been affected by the extensive media coverage of BSE, foot and mouth disease, and west Nile virus, among others. The swine industry can expect further public scrutiny of production practices and

animal health, as well as the increased regulatory requirements. Addressing these needs with sound and reliable scientific information is the best protection against public mistrust and excessive government regulation.

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