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**Bovine Spongiform Encephalopathy (BSE):
Risks and Implications for the United States**

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and

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Bovine Spongiform Encephalopathy (BSE): Risks and Implications for the United States

Practitioner's Abstract:

Mad cow disease has caused two major disruptions in European beef markets—first in the U.K. in 1996 following the announcement of a link to new variant Creutzfeldt-Jacob Disease in humans, and the second in late 2000 following the discovery of “homegrown” cases of the disease in Germany and Spain. In September 2001 the disease was discovered in Japan where it also resulted in an immediate and substantial reduction in beef demand.

The disease has not been found in the U.S. but the current scope of detection efforts provides little assurance that it does not exist at a very low level. The U.S. has taken a number of precautionary measures to reduce both the risk of importing the disease and the risk of the disease spreading if it were to appear. Those measures include a ban on the feeding of ruminant protein to ruminants—a measure that the General Accounting Office concluded was not being adequately enforced and which failed to halt the disease in the U.K. We present an overview of BSE in the U.K., the EU, and Japan and present an argument for implementing additional precautionary measures in the U.S.

Keywords: mad cow disease, BSE, beef demand, risk

Introduction¹

Bovine spongiform encephalopathy (BSE), or mad cow disease, was first recognized in the U.K. in the mid 1980's. The disease is one of several transmissible spongiform encephalopathies (TSE's), variants of which affect sheep (scrapie), elk and deer (chronic wasting disease), mink, and other species. In 1996, U.K. government scientists reported that new variant Creutzfeldt-Jacob Disease (vCJD), a fatal disease in humans, was linked to consumption of beef from animals infected with BSE. Unlike sporadic CJD, which affects elderly people and occurs worldwide at a rate of about 1 case per million individuals, vCJD affects younger patients - average age 29 years, as opposed to 65 for CJD.

As of June 2002, vCJD has been confirmed or suspected in the deaths of 113 individuals in the U.K., including at least 4 dairy farmers. A further 10 individuals were suspected to have the disease including one victim in the U.S. whose diagnosis was announced in April 2002 but who likely contracted the disease while living in the U.K. Deaths from vCJD have also occurred in France (6), Italy (1), and Ireland (1). Given uncertainty about the incubation period of the disease, estimates about the likely number of total victims vary from fewer than a thousand to over fifty thousand.

The 1996 announcement of the link to vCJD resulted in an immediate ban on U.K. exports of live cattle and all beef products, a 30 to 40 percent reduction in domestic beef consumption,² and significant losses to beef producers, agribusinesses,³ and the treasury. A report by the Food Standards Agency estimated the total annual direct costs of BSE controls to U.K. taxpayers at £425 million (approximately \$650 million).⁴ In late 2000, a similar set of events played out in the EU following the announcement of the first native-born cases of the disease in Germany, Spain, Denmark, and later in Italy. Importing countries, notably Egypt and Russia, banned beef from the EU, even from countries such as Ireland which were already known to have the disease and which had controls in place. The direct cost of BSE control measures to the EU farm budget was estimated at €1 billion (approximately \$1 billion) in 2001. The scenario was repeated for a third time when Japan announced its first case of the disease in September 2001.

In this paper we present an overview of the science of BSE, and of events associated with the disease in the U.K., Europe and Japan. We then review the situation in the U.S. where the disease has not yet been discovered. We describe the counter measures already in place, and argue, based on the experience with the disease in other countries, that counter measures should be strengthened.

1 Abbreviations used in the manuscript are listed in Table 1.

2 Average per capita consumption fell from 15.4 kg in 1995 to 12.6 kg in 1996. Consumption had already been in decline, down from 19.3 kg in 1986. During the same period, per capita consumption in the EU had fallen from 23 to 20 kg, and in the U.S. from 49 to 44 kg. In 1997, U.K. consumption recovered to about 87 percent of the 1995 level, and continued to increase in subsequent years. (All figures are carcass weight equivalents.)

3 Henson and Mazzocchi analyze the significant negative returns in the U.K. beef sector businesses associated with this event.

4 The total cattle inventory in the U.K. is around 8 million, compared to about 96 million in the U.S.

The Science—what is known and what is not

Several questions remain unanswered about the cause, original source, incubation periods and routes of infection of BSE. As to the cause, most but not all scientists believe the infective agent to be a modified prion⁵ (or more correctly a prion protein). From a biological perspective prions are simple structures - they are not entire cells and do not have DNA, and are thus more difficult to destroy than viruses or bacteria. In fact, no feasible food processing intervention including irradiation destroys prions.

Experts still disagree as to the origin of the modified prion responsible for BSE. The report of the BSE inquiry (Phillips *et al.*) concluded that BSE “probably originated from a novel source early in the 1970s, possibly a cow or other animal that developed disease as a consequence of a gene mutation,” and that the cases identified beginning in 1986 resulted from the recycling of cattle infected with BSE and were not linked to sheep scrapie. However in a later report, Horn *et al.* lean toward the conclusion that BSE was caused by an unmodified version of sheep scrapie but agreed that the possibility of a prion mutation, in either cattle or sheep, could not be excluded.

Scrapie has long been known as a disease in sheep and is found world-wide with the exception of Australia and New Zealand. The first recorded case in the U.K. dates from 1732 and there are an estimated five to ten thousand cases there every year. Until the 1980’s, scrapie had not been known to cross the species barrier to cattle—or to humans. Why the disease may have spread from sheep to cattle in the U.K. at that time is not known with certainty, but Horn *et al.* point to: a) changes in the rendering⁶ system in the 1970’s - in particular the abandonment of solvent extraction of tallow which may have led to a ten-fold increase in the amount of potentially infective prions surviving the process, and b) the introduction of meat and bone meal (MBM) as a protein supplement in rations for young dairy calves—a practice that became prevalent in both the U.K. and Australia in the 1970’s.⁷

Regardless of the original source, once it became established in cattle, BSE spread within cattle and then to humans and other species as tissue from BSE-infected animals entered the animal and human feed chains. It is possible but not yet proven that maternal transmission can occur in cattle, and scientists have not ruled out the possibility of lateral transfer, via contaminated feces,

5 The word prion is derived from ‘proteinaceous infectious particle.’ Infectious prions associated with TSE diseases are modified forms of naturally occurring prion protein. Contact between normal prion protein and the infectious form causes the normal form to convert to the infectious form – this conversion process is not understood and has not been achieved experimentally (Horn *et al.*). Thus far only one strain of the infectious prion agent has been identified in cattle with BSE – and the same strain has been identified in other species suffering from BSE type diseases (cats and captive exotic ruminants such as eland), and in humans suffering from vCJD.

6 Rendering is the processing of offal (organs, intestines) and other parts of the carcass to produce meat and bone meal, and tallow. It involves drying and cooking, and separating the solids (meal) from the melted liquid fraction (tallow).

7 In favoring the “unmodified scrapie” hypothesis for the origin of BSE, Horn *et al.* also point to the fact that the U.K. has had one of the highest ratios of sheep to cattle in the world. It is not yet known whether sheep in the U.K. were/are infected with BSE, but the disease can be induced in sheep by feeding infected material.

between cohort animals.⁸

The majority of BSE cases occur in animals over four years old, although rare cases have been found in animals as young as 20 months. The pattern of disease in the U.K. suggests that most animals became infected as calves. During the incubation period infective prions accumulate in nervous tissue—spinal cord, eyes and brain⁹—before symptoms, which involve loss of motor and neurological control, appear. Once symptoms appear, progression of the disease is relatively quick with death occurring within a year. To date there is no known treatment in either humans or cattle and the disease can only be definitively diagnosed post-mortem.¹⁰

Infective doses for humans are not known with certainty but experiments with cattle and sheep have shown that as little as a half of a gram of infective material is sufficient to transmit BSE when administered orally. Genetic factors are known to play a role in determining both human susceptibility to vCJD and sheep susceptibility to scrapie. Research is ongoing to find corresponding genetic markers in cattle. Other co-factors such as throat infections or tonsillitis¹¹ have been hypothesized to play a role in transmission to humans. Thus far transmission to humans has only been associated with consumption of prion infected nervous tissue from bovines. It is believed unlikely that consumption of muscle tissue or milk from infected animals can cause the disease. However, Bosque *et al.* recently demonstrated the presence of infectious prions in muscular tissue in mice.

BSE in the U.K.

The earliest recorded case of what was later recognized to be Mad Cow Disease was reported in 1984 on a farm in Sussex in the south of England. Within a year, six more cows from the same farm had succumbed to the illness, but it was not until late in 1986 that the new disease was confirmed as a bovine TSE (see Table 2). As of June 14, 2002 a total of 179,280¹² cases had been confirmed on over 35,000 farms (Figure 1). It is likely that for each confirmed case several others were slaughtered before developing clinical symptoms. Approximately eighty percent of cases have been in dairy cows, with sixty percent of U.K. dairy farms having at least one case.

Transmission of the new disease was soon linked to MBM and in July 1988 the U.K. banned the feeding of “animal protein” to ruminants.¹³ Animal protein was defined as any protein material derived from the carcass of a ruminant although an earlier draft of the legislation had apparently defined it as protein derived from the carcass of a mammal. The ban did not cover tallow, which

8 If these routes of transmission operated at significant levels, one would expect to see more than the 18 cases detected to date in animals born after the implementation of an enhanced feed ban in the U.K. in 1996.

9 Scientists believe that the infective agents reach the brain by spreading along nerves rather than via the bloodstream.

10 Recently a test has been developed to detect chronic wasting disease using tissue from the third eyelid in deer. Progress has also been reported on developing tests for TSE's using urine and blood samples.

11 This hypothesis is usually associated with Dr. Stephen DeArmond of University of California School of Medicine San Francisco.

12 Of the total, 641 were detected via active surveillance – primarily of fallen or casualty animals.

13 The ban was initially temporary – effective until the end of the year or until such time as rendering could be investigated with a view to recommending standards that would inactivate the BSE agent.

is essentially fat. Because most MBM was used for pig and poultry feed, with only around 10-12 percent used for cattle, it was believed that the ban would not have significant economic consequences.

Having lost a portion of their domestic market for MBM, U.K. renderers had an incentive to increase exports. Most MBM exported from the U.K. went to EU countries,¹⁴ where it was incorporated into animal rations that were often exported outside of EU.¹⁵ The U.K. was not particularly proactive in warning importers about the dangers of feeding MBM—considering it sufficient to communicate the risk via articles published in the U.K. *Veterinary Record* and a statement made at the annual meeting of the OIE¹⁶ in May 1989. A number of EU countries imposed restrictions on MBM in 1989¹⁷ but some U.K. officials were concerned that other importers would not realize the magnitude of the risk. It was not until February 1990, at the request of the agriculture minister, that the Chief Veterinary Officer communicated directly with counterparts in countries, which had imported MBM from the U.K.¹⁸ The U.K. government was subsequently criticized by the EU for failing to control exports of MBM.

The issue of potential human health implications of the new disease was examined by an expert group under the chairmanship of Sir Richard Southwood. Following its first meeting, the group recommended that carcasses of infected animals be condemned and destroyed by incineration. Legislation enforcing those recommendations, and providing compensation to producers at the rate of 50 percent of market value,¹⁹ was enacted in July 1988. The group subsequently recommended that the ruminant feed ban be extended indefinitely, and that milk from animals suspected of having BSE be destroyed. They also considered the use of bovine brain and other potentially infective tissue in foods for human consumption, and the use of those tissues in vaccines and other medicinal products. However, other than recommending that manufacturers of baby food avoid use of bovine offal, the group did not consider that other measures related to human consumption of those tissues, including labeling, were warranted—a conclusion for which they were later criticized. In short, the Southwood group concluded²⁰ that the risk to human health was remote.

14 U.K. exports of MBM to the EU totaled 12,553 tonnes in 1988, rose to 25,005 tonnes in 1989 – the year following the U.K. feed ban, then fell to 10,005 tonnes in 1990 and to between 1 and 3,000 tonnes in 1991-94 as EU countries imposed restrictions.

15 For example, the emergence of BSE in Switzerland was attributed to the feeding of contaminated MBM in rations imported from Belgium.

16 Office International des Epizooties (OIE), based in Paris is the animal health equivalent of the World Health Organisation (WHO).

17 France, Germany, and Italy banned imports of U.K. MBM while the Netherlands imposed a feed ban similar to that in place in the U.K.

18 The letter was sent to 25 countries outside of the EU which had imported ruminant-based meat and bone meal from the U.K. The countries were Norway, Sweden, Switzerland, Czechoslovakia, Hungary, Nigeria, Thailand, South Africa, Malaysia, Taiwan, Hong Kong, South Korea, Japan, Canada, U.S.A, Turkey, Kenya, Malta, Liberia, Lebanon, Saudi Arabia, Sri Lanka, Puerto Rico, Curaçao and Finland.

19 In February 1990, compensation was increased to 100 percent of market value in order to eliminate any incentive on the part of herd-owners to send suspected BSE cases into the food chain. The compensation rate was adjusted downward marginally in 1994 to save money and to reflect the fact that most cases were in older animals.

20 While the report did include certain caveats as to the risk to humans if certain assumptions were to be proven

In June 1989, the U.K. government went beyond the recommendations of the Southwood report by banning the use of Specified Bovine Offal²¹ for human consumption, thereby removing from the food chain potentially infective tissue from animals not showing symptoms of the disease. While many considered this measure unnecessary, the decision was taken partly in the light of increased media interest in measures to protect the public, and partly because of difficulties implementing a ban that would apply only to baby food (as per the Southwood recommendation). In 1990, it was discovered that BSE could be transmitted to mice via feeding. In the same year, the first case of spongiform encephalopathy was diagnosed in a domestic cat,²² and scientists demonstrated that BSE could be transmitted, by inoculation, to pigs. Following these discoveries, and on advice from the newly commissioned Spongiform Encephalopathy Advisory Committee, the ban on specified bovine offal (SBO) was extended to all animal feed²³ in September 1990. With both the ruminant feed ban and SBO ban²⁴ in operation, government officials felt confident that the disease would be contained. The official line was that BSE was not a risk to humans and that it was safe to eat beef. As quoted in the Phillips report, a scientist affiliated with the meat industry claimed that “*a human would have to eat an impossible amount of pure cow brain at the height of infection to reach an equivalent dose to that needed to infect a cow.*”

In March 1991 came the first case of BSE in an animal born after the 1988 feed ban (referred to as a ‘BAB’—born after ban). BABs were initially attributed to delays in the clearing of MBM stocks and while there was some evidence of deliberate breaches of the regulation, officials remained confident until late 1993 that the ban would be effective in eradicating the disease. However, in 1994 there were almost 10,000 BABs, suggesting serious problems with cross-contamination²⁵ in mills producing feed for both ruminants and non-ruminants, and, by extension, a very high level of infectivity in tissues derived from sub-clinical animals (since clinical cases were being destroyed).²⁶ In July 1994 came the first reports of BAB’s born in

incorrect, this, at least, is how the report was perceived.

21 Initially SBO was defined to include brain, spinal cord, tonsils, spleen, thymus and intestines from cattle more than 6 months old - tissues in which the BSE agent was most likely to be present. The SBO list was expanded several times in subsequent years as more was learned about the disease. The BSE inquiry faulted officials for not including mechanically recovered meat (MRM) from the spinal column in the initial ban. Following a period of consultation, the ban was implemented in November 1989.

22 Scientists had previously transmitted CJD to a cat by inoculation, but attempts to transmit scrapie had not succeeded.

23 In fact, the pet food industry and many animal feed manufacturers were already observing a voluntary ban on the use of SBO – by refusing to purchase MBM manufactured with SBO. The agriculture ministry was concerned that the feed industry would cease using MBM entirely – a move which would hurt animal producers and lead to disposal problems.

24 In 1992 the SBO ban was amended to prohibit the removal of meat from skulls that had been split to remove the brain. An amendment in 1995 required that brains and eyes not be removed and the entire skull be disposed of as SBO. That amendment also required that SBO be separated from all other material, dyed with blue stain, and processed in dedicated facilities. In 1995 the government also introduced a ban on MRM from the spinal column—an issue of concern to several scientists from the outset due to incomplete removal of spinal cord and dorsal root ganglia.

25 A problem here was the lack of a reliable test for ruminant protein in compound feeds.

26 It had been discovered in 1990 that an orally administered dose of a half of a gram of BSE infected brain was sufficient to cause the disease in sheep. In 1994, it was shown that 1 gram (the lowest dose used in the experiment) was sufficient to cause BSE in cattle.

1991. In total, over 12,000 cases have been detected in BAB's born after the September 1990 animal SBO ban, suggesting that, in addition to cross-contamination in mills, SBO was not being properly removed.

Figure 2 describes the infection pathways and the objectives of the measures taken to halt the disease. All three of the numbered pathways must operate for the disease to persist. Destroying clinically infected animals was not sufficient to halt the disease, because sub-clinically infected animals are also infective. The ruminant feed ban sought to block pathway number 3, but failed because of cross-contamination. The SBO ban, which would block pathway 2, failed because of non-compliance. Part of the problem, as identified by the inquiry, was that the animal SBO ban was poorly designed and essentially unenforceable with no way of knowing whether MBM contained SBO.

Despite continued reassurance from the government, public concern about the risk to humans had been growing since BSE was first reported. When it was learned in 1990 that the disease had spread to cats, the media began to report that some scientists were concerned about the risk to humans. One scientist, Professor Richard Lacey, was quoted as saying that due to BSE, "*in the years to come our hospitals will be filled with thousands of people going slowly and painfully mad before dying.*" In 1990, local school districts started to ban beef from school menus. In 1993 it was reported that two dairy farmers had died from CJD but the official response remained unchanged—i.e., there was no risk to humans. In fact, as late as December 1995 a health minister was quoted as saying "there is no conceivable risk of BSE being transmitted from cows to people."

Early in 1996, officials at the ministry of agriculture were informed that ten people had died of a new strain of CJD. Unlike sporadic CJD which strikes elderly people, all the victims were in their teens or twenties. On March 20, 1996, the government announced that there was probably a link between BSE and this new type of CJD. New control measures announced included a ban on the use of all MBM of mammalian origin in farm animal feed, and the removal of beef from animals over thirty months from the human food chain. This later measure was implemented via the over-thirty-months-scheme, which remains in operation and has accounted for over 5 million animals. As of June 6, 2002 there have been 18 as yet unexplained cases of BSE in animals born after the enhanced feed ban was enforced on August 1, 1996.

Spreading it around

BSE spread from the U.K. through exports of live animals and contaminated feed. Infected animals exported from the U.K. were detected in Canada, Oman and the Falkland Islands, in addition to six European countries. The EU banned imports of live animals from the U.K. in 1989, but the ban applied only to animals born prior to the July 1988 feed ban—on the premise that the feed ban would prevent new infections. The import ban was extended the following year to only allow imports of animals aged less than six months on condition that they are slaughtered in the destination country before reaching the age of six months. These restrictions were not significant for the U.K. since the majority of its live exports were young calves destined for veal production. Other countries reacted more vigorously than the EU in terms of restricting imports.

Israel banned cattle imports from the U.K. in 1988, and the following year, Australia, New Zealand, Sweden, and the U.S. took a similar step. Of course, not all infected animals that were exported were detected—many ended up in the food chain, both animal and human, and likely contributed to the spread of both BSE and potentially vCJD.²⁷ However it is likely that exports of contaminated MBM were responsible for the greater part of the spread of BSE beyond the U.K.

Exports of MBM from the U.K. were relatively small during the 1980's, reaching approximately 15,000 tons (4 percent of production) in 1988, most of which went to EU countries, but also to third countries including Indonesia, Thailand and Sri Lanka, and indirectly via animal rations produced in the EU to the Middle East and North Africa. After the ruminant feed ban in 1988, exports to the EU and other countries increased markedly—essentially doubling between 1988 and 1989 (Figure 3). Because MBM was still allowed in pig and poultry feed, the U.K. did not ban MBM exports—essentially taking the position that importers, even though they did not have ruminant feed bans, were aware of the risk and could initiate restrictions. The preferred option for the U.K. was an EU-wide ruminant feed ban, similar to its own, but the EU did not adopt a feed ban until 1994.²⁸ Starting in 1989, individual EU countries imposed bans on imports of MBM from the U.K. or initiated their own ruminant feed bans. These measures resulted in a significant decrease in MBM exports to the EU in 1990, with the loss being offset by increased exports to non-EU countries.

It was not until September 1990, when the SBO ban was extended from human to animal feed, that the U.K. prohibited exports of MBM containing SBO to EU member states, and not until 1991 that the same prohibition was extended to other countries. It is also worth noting that the definition of what constituted SBO was expanded several times in the early 1990s to include eyes, thymus, and finally the entire skull in August 1995. Up until 1995 therefore, MBM continued to be manufactured in the U.K. containing potentially infective material, and exported to countries that did not have a ban on the feeding of ruminant protein to ruminants.

BSE in Europe

Following the initial EU ban on imports of U.K. cattle born before the feed ban, restrictions were gradually tightened. In 1990 live exports were limited to animals less than 6-months old, exports of SBO were prohibited, and U.K. exports of bone-in beef were permitted only from herds free of BSE. As noted above, individual European countries banned imports of MBM from the U.K. or adopted ruminant feed bans in 1989-90.

But, by the time these measures were taken, it was too late. The first country outside the U.K. to detect BSE was Ireland with 10 non-imported cases in 1989. Subsequently, the disease was discovered in Switzerland (2 cases in 1990), France (5 cases in 1991), and Portugal (12 cases in

27 From the Inquiry: "During 1985-1990 the U.K. exported 57,900 cattle. These animals would have resulted in 1,668 cases of BSE had they remained in Great Britain, but only a small fraction of these cases have been reported by the recipient countries. It has been suggested that only one in six probable cases of BSE within the EC has been reported." Because importing countries had no bans on human consumption of SBO, these exports also constituted a threat to human health.

28 The June 1994 decision prohibited the feeding of mammalian protein to ruminants throughout the EU.

1994). In 1994, the EU banned the use of mammalian protein in ruminant feed and in March 1996, following the announcement of the link to CJD, banned exports of all cattle and beef from the U.K. On January 1, 1998, new legislation required the destruction of Specified Risk Material (SRM) from cattle, sheep, and goats—introducing to other EU countries the restrictions on SBO in place in the U.K. As had occurred with SBO in the U.K., the definition of what constituted SRM continued to expand as more was learned about potentially infective tissues. In December 2000, for example, the definition was amended to include intestines of bovines of all ages.

From the mid to late 1990's additional BSE cases were discovered in France, Portugal and Switzerland at relatively low rates,²⁹ with cases also appearing in Belgium and the Netherlands in 1997. Then in the fall of 2000, the first “homegrown” cases were discovered in Germany, Spain, and Denmark, followed by Italy's first case in early 2001. The market reaction was instant and dramatic. Beef consumption fell by about 30 percent, and exports by the EU to third countries—notably Russia and Egypt - were halted. German consumers, having been continually reassured by their Government that their beef supply was free of BSE, reacted particularly strongly with beef demand falling by about 50 percent as manufacturers of processed meat products scrambled to reformulate with pork instead of beef. The immediate price effects were so great (see Table 3) that at one point in the subsequent months the trade pattern for beef within the EU was almost completely reversed—with Germany exporting beef to Ireland.

In the wake of the crisis, the EU took a series of measures designed to safeguard public health and restore confidence in beef (see Byrne). These included:

- BSE tests for all animals over 30 months intended for human consumption.³⁰ Animals not tested were eligible for a Purchase-for-Destruction scheme which ran from Jan 1 to June 30, 2001.
- BSE tests for all casualty animals over 24 months.
- Use of MBM in all animal feed was suspended and all MBM stocks to be destroyed.
- The list of SRM was extended to include vertebral column and the entire intestine.
- A ban on MRM.

BSE in Japan

The announcement of the first BSE case in Japan came on September 10, 2001. As of June 1, 2002, there had been 4 confirmed cases, all in dairy cows born in 1996. Initially, it was thought that the outbreak was caused by MBM imported from the U.K. during the early 1990's.³¹ If that

29 In 1999 for example, there were 31 cases in France, 91 in Ireland, 159 in Portugal, and 50 in Switzerland – compared to 2,254 in the U.K. where there had been a dramatic drop-off after a peak in 1992.

30 In 2001, approximately 8 million animals over the age of 30 months were tested with approximately 1,000 positive cases discovered.

31 Data from COMEXT, an EU trade database, for U.K. exports to Japan of flour, meals and pellets, of meat or

were true, the age of the affected animals would suggest that they were 2nd generation cases—infected from domestic MBM produced from animals infected by the original imports. That recycling scenario would suggest that the disease is widespread in Japan, but the slow pace of discovery of new cases suggests otherwise. An alternative explanation involved milk substitute manufactured with beef tallow imported from the Netherlands. However, infectivity has never been detected in tallow. What is now considered the most likely explanation is that the disease was introduced through inadequately sterilized MBM imported from Italy between 1995 and 1998.

Following the announcement, beef consumption fell dramatically. September retail sales were reported to be down 40 to 50 percent, while at regional markets wholesale prices were 30 to 60 percent lower than normal. According to a national survey by Asahi newspaper in mid-October, one out of four consumers had stopped eating beef altogether, and only 26 percent indicated that they had not altered their beef consumption pattern. Restaurants specializing in beef reported more than 50 percent loss in sales revenue during the first half of October, while school lunch programs around the nation altered their menus to exclude beef. Consumer confidence in beef received a further blow following revelations of deliberate mislabeling of imported Australian and U.S. beef as domestic beef in order to obtain government subsidies.

In its response to BSE, the Japanese government removed animals over 30 months from the human food chain and instituted BSE testing of all animals for human consumption.³² It also banned imports, processing and distribution of MBM for all uses. All livestock waste was to be processed into MBM and incinerated.³³ The total budget announced by the Ministry of Agriculture, Forestry, and Fishery (MAFF) for its response to BSE was 155.4 billion yen (approximately \$1.3 billion). Outlays included an income stabilization scheme for cattle farms, incineration costs of MBM, and the cost of a new electronic tagging system for all livestock. The government also decided to purchase and incinerate all beef in inventory prior to October 18th at a cost of over 10 billion yen (approximately \$100 million).

The government's handling of BSE was heavily criticized due to several administrative blunders. For example, MAFF first announced that the infected cow had been incinerated, but on Sep 14th acknowledged that it had in fact been processed into MBM. This resulted in an effort to track and incinerate 150 metric tons of MBM, a portion of which had already been transported elsewhere. The requirement to incinerate all MBM also caused problems; several slaughter facilities were forced to temporarily shut down because they had no means to dispose of their waste.

In cutting back on beef, Japanese consumers made no distinction between imported and domestic product. Sales of imported U.S. and Australian beef, although those countries remain BSE free,

meat offal unfit for human consumption: 1988—0, 1989—0, 1990—132 metric tons, 1991—62 metric tons, 1992—43 metric tons, 1993—31 metric tons, 1994—64 metric tons, 1995—0. It was not until 1991 that the U.K. banned exports of SBO to non-EU countries. Furthermore, the definition of SBO at that time did not include several tissues later recognized to carry infectivity including eyes, skull, and vertebral column.

32 Japan is the only country that tests all slaughtered cattle for BSE, regardless of age.

33 Approximately 1.7 million metric tons annually, which is processed into about 400,000 metric tons of MBM.

were equally adversely affected (Figure 4). According to the U.S. Meat Export Federation, the value of U.S. beef exports to Japan for the first quarter of 2002 was 42 percent below the previous year (\$190 million vs. \$329 million). To restore consumer confidence in domestic beef, the Japanese government launched an aggressive marketing campaign centered on the claim that Japanese beef is “the safest in the world” (Seng). Because this cast doubt on the safety of imported beef, agencies acting on behalf of Australian and U.S. producers were forced to respond with their own promotions.

The situation in the United States.

To date there have been no cases of BSE in the U.S. and the only cases in North or South America have been in cows imported from the U.K. to Canada (1993) and the Falkland Islands (1989). Other animal TSE’s are found in the U.S. including scrapie in sheep, transmissible mink encephalopathy (TME), and chronic wasting disease (CWD) in deer and elk. TME was first found in the 1960’s in rancher mink and was attributed to the feeding of scrapie-infected sheep tissue (McKenzie *et al.*). Scrapie was first found in the U.S. in 1947 and since then has been reported in over 1000 flocks. The Federal Government has implemented a program designed to eradicate scrapie by 2017.

CWD was first identified in the 1960’s in captive deer at a research station in Colorado. During the past decade, it has spread to wild deer in several mid-western states and Canada, and to farmed elk. In Wisconsin, testing in 2001 indicated that up to 3 percent of the deer population might have the disease. Much remains unknown about CWD—in particular its modes of transmission and original source. At this time there is no evidence to suggest that CWD can be transmitted to humans. Nevertheless, the WHO advises that tissue from deer or elk with CWD should not be used in animal or human food.

To prevent BSE, the U.S. banned imports of live animals from countries with BSE in 1989, imports of MBM from those countries in 1991, and extended those restrictions to countries at risk for BSE (i.e., all of Europe) in 1997. A ban by the Food and Drug Administration (FDA) on the feeding of most mammalian protein to ruminants was implemented in 1997. A recent report by the Harvard Center for Risk Analysis and the Tuskegee University Center for Computational Epidemiology (Cohen *et al.*) has been widely quoted to support the idea that the risk of BSE in the U.S. is extremely low.³⁴ However, as is stated in that report, its purpose was to evaluate the robustness of measures to prevent the spread of BSE **if** it were to occur, and it goes on to state that “BSE is extremely unlikely to **become established** in the U.S.” (emphasis added).

The EU Scientific Steering Committee classifies the U.S. as a Geographical BSE-Risk (GBR) level II country - one in which the presence of BSE is ‘unlikely, but not excluded.’³⁵ While U.S. meat exports now carry a label stating, “The United States meets or exceeds all of the

34 “The Harvard Risk Analysis showed that the risk of BSE occurring in the United States is extremely low...,” stated by Secretary of Agriculture Ann M. Veneman regarding the GAO Report on BSE. February 26, 2002.

35 The U.S. is classified at level II due to imports of live animals from the U.K. and Ireland prior to 1990. In December 2001, three GBR-II countries - Austria, Finland, and Slovenia - detected their first domestic cases of BSE.

international guidelines to be considered free of BSE,” the EU requires that GBR level II countries remove SRM from any beef exported to the EU.

Adequacy of U.S. measures.

In February 2002, the General Accounting Office (GAO) responded to a congressional request to evaluate the effectiveness of measures taken to prevent BSE. The GAO concluded that measures adopted in the U.S. did not sufficiently ensure that BSE would be prevented, citing weaknesses in import inspections, inadequate testing of animals that die on farms, and non-compliance with the FDA feed ban. The report also pointed out that the U.S. still allowed cattle brains and other central nervous system tissue to be sold as human food and that consumers were not adequately informed about foods that contained such tissue. The GAO recommended increasing import inspections, strengthening the enforcement of the feed ban, and informing consumers about products that could contain central nervous system tissue.

The Feed Ban

The FDA feed ban, adopted in 1997, is not particularly restrictive in that it allows protein from non-ruminants such as pigs and horses in ruminant feed. To compare the FDA feed ban with those adopted elsewhere, we can classify bans as follows:

- Level 1 Prohibits feeding *ruminant* protein to *ruminants*
- Level 2 Prohibits feeding mammalian protein to *ruminants*
- Level 3 Prohibits feeding mammalian protein to all farm animals (and fish)
- Level 4 Prohibits feeding mammalian protein to all animals (including pets)

In this classification, the U.S. feed ban is in fact less restrictive than that described at Level 1 because it permits certain ruminant proteins (milk, blood) to be included in ruminant feed. The ban adopted in the U.K. in 1988 was at Level 1, and was not fully effective due to cross contamination between ruminant and non-ruminant feed. In fact, over 43,000 cases of BSE (almost 25 percent of the total to date) have been confirmed in animals born after that ban was implemented. The feed ban adopted by the EU in 1994 was at Level 2. In 1996, the U.K. enhanced its ban to Level 3 by banning the use of MBM for all farm animals and fish. In 2001, the EU adopted, initially temporarily, a complete ban on the use of MBM. Japan has recently taken a similar measure.

The GAO found weaknesses in FDA enforcement of the feed-ban; in particular in its efforts to re-inspect firms that had not complied with requirements to label feed containing prohibited protein. The GAO also found major weaknesses in the FDA database of inspection records, which led to underreporting of the number of firms not in compliance. Given the extent of cross-contamination problems experienced in the U.K. and the inadequacies in enforcement reported

by the GAO, there can be little assurance at present that ruminant feed does not contain prohibited material.

Specified Risk Material (SRM)

The Harvard/Tuskegee report identified additional measures that would reduce the risk from BSE. These included prohibiting the rendering of animals that die on farm, and the implementation of a European type ban on SRM (brain, spinal cord, vertebral column) from human and animal feed. A ban on SRM was estimated to reduce the predicted number of BSE cases in an outbreak by 80 percent, and reduce potential human exposure by 95 percent.

For countries at risk for BSE, the WHO recommends removal from the food chain of tissues likely to contain the BSE agent,³⁶ and, as noted above, the EU requires removal of SRM from product exported from the U.S. At present, the U.S. does not have a ban on human consumption of bovine brain or other central nervous system (CNS) tissue. According to the GAO, products such as beef stock, beef extract, and beef flavoring can contain CNS tissue, and since 1997, U.S.DA has found CNS tissue in 12 of 63 samples of meat derived from advanced meat recovery (AMR) systems. In its response to the GAO recommendation that consumers be informed about products that might contain CNS, the U.S.DA said that labeling and warning statements should be reserved for known hazards. Because the U.S. does not have BSE, there is no hazard.

Surveillance

Testing for BSE in the U.S. began in 1990. The surveillance program targets cattle diagnosed with central nervous system disorders at slaughter and "downer" animals—those unable to walk at the time of slaughter. As of mid 2001, over 13,000 tests had been conducted. The GAO report noted that very few animals that died on farm, a relatively high-risk population, were included in the testing program. Animals that die on farm may be buried or collected for rendering—unlike the situation in Europe where deaths of all animals over 24 months must be notified, the animal tested for BSE, and the carcass collected for incineration. The Harvard-Tuskegee analysis showed that excluding such animals from the rendering process would significantly reduce the potential for BSE to spread.

In February 2002, U.S.DA announced that it would increase, from 5,000 in 2001 to 12,500 in 2002, the number of cattle brains tested for BSE. However, with between 5 and 6 million cows slaughtered each year, those numbers represent a small fraction of a higher-risk category of animals. Compared to Europe, where almost 8.5 million animals out of a total adult cattle population of 40.8 million were tested in 2001,³⁷ the level of testing in the U.S. is miniscule. Of the animals tested in Europe, 770,000 were designated "at risk"—a category including animals that died on-farm or those found sick or injured at slaughter. In the "at-risk" category, 760 animals tested positive for BSE, an incidence of about 1 per thousand. If the U.S had a similar

36 Recommendation of the joint WHO/FAO/OIE Technical Consultation on BSE, Paris, June 2001: "Whenever the possibility that slaughtered animals may be infected with BSE cannot be excluded, all tissues that have been proved capable of carrying BSE infectivity should be removed and destroyed, i.e. an SRM ban should be imposed."

37 Data available at: http://europa.eu.int/comm/food/fs/bse/testing/bse_results_en.html

incidence in “at-risk” animals, 12,500 tests per year in that category provide a 99.999 percent probability of detecting at least one positive case. But with no clinical cases to date, the incidence of sub-clinical BSE in the U.S. herd, if it is present, has to be at a far lower level. If, for example, it were present in U.S. “at risk” animals at the same level as in “healthy” animals in Europe (279 positives in 7.6 million tests, or approx 1 per 25,000), the current level of testing provides only a 39 percent probability of detection, and it would require almost 120,000 tests to increase the likelihood of detection to 99 percent. In short, the fact that BSE has not yet been found provides little assurance that it is not present.

It could be reasonably be argued that if the disease is present at an extremely low level, the U.S. would be better off not detecting it—especially if there is little chance of it spreading due to the controls already in place. However, the infectivity of sub-clinically infected animals (as demonstrated by the failure of the U.K.’s feed ban and SBO controls) is a characteristic of this disease that suggests the potential peril of ignoring its presence - even at an extremely low level.

Possible routes of exposure facing the U.S.

1. Import of infected animals or contaminated feed: Currently the U.S. bans imports of live animals and animal feed from all countries at risk for BSE. According to the GAO, the U.S. imported about 1,000 cattle during the past 20 years from countries in which BSE was later found. These included 334 animals from the U.K. between 1980 and 1989, 443 animals from other EU countries between 1983 and 1997, and 242 animals from Japan between 1993 and 1999. It is likely that material from several hundred of these animals has been used for animal feed and, given the incubation period of the disease, the possibility that some of those animals were infected with BSE cannot be ruled out.
2. Lateral transmission of CWD: Scientists do not yet fully understand the transmission mechanism for CWD but lateral transmission appears very likely. It is not known whether CWD can spread from deer to cattle or if so, what form it would take in cattle.
3. Transmission of scrapie from sheep/Spontaneous appearance in cattle: One of these two pathways is regarded as the likely origin for BSE. Since scrapie occurs in the U.S., transfer of the disease from sheep was a possible route of infection prior to the feed ban.
4. Deliberate introduction: There have been instances in Europe in which farmers have been prosecuted for attempting to deliberately introduce animal diseases, including BSE, into their herds in an effort to take advantage of government compensation. Because the BSE agent is stable, infectious in small amounts, apparently harmless unless ingested, likely relatively easy to obtain (compared for instance to anthrax), and capable of having a very significant economic impact, one cannot discount the possibility of it being used as a biological weapon.

Consequences of BSE in the U.S.

It is clear from the experience in Europe and Japan that BSE would have disastrous consequences for the beef industry. With a single confirmed case, exports, which account for

about 12 percent of production, would cease and domestic demand would inevitably fall. If the response mirrors that of EU and Japanese consumers, beef consumption would decline by 30 percent.³⁸ Combined with the elimination of exports, the industry could face an overall reduction in revenue of 40 percent in the short- to intermediate-run. If demand recovers over a period of 4-5 years, as happened in the U.K., the cumulative loss to the beef sector would be on the order of \$30 billion.

Should the U.S. take additional precautions?

The response to BSE can be viewed in terms of finding an appropriate balance between costs (to industry) and benefits (to consumers) of actions that mitigate the risk. The problem is that the level of risk is difficult to quantify. If one believes that BSE is inevitable or perhaps already present in U.S. cattle, then a case can be made for taking additional measures to protect human health and eradicate the disease. But if one takes the position that BSE is not here and is extremely unlikely to occur, current measures are more than adequate (see Murphy for an example of this viewpoint). However, the absence of BSE is not so reassuring given the low level of detection efforts. In light of the EU's assessment of the risk level for the U.S., and the discovery of the disease in Germany, Japan, and other countries where it was thought not to exist,³⁹ a case can be made for both modifying the "official line" on BSE and enhancing preventive measures.

Discovery of BSE in Germany and Japan illustrated two points: i) the fact that BSE has not been detected does not imply its absence, and ii) promoting consumer confidence in a country's "BSE free" status when there is any risk may be unwise. Official assurances that those countries were free of BSE were unwarranted, contributed to loss of trust in government when BSE was discovered, and likely exacerbated the extent to which consumers moved away from beef. The "official" U.S.DA position at present appears very similar to that taken in Japan and Germany prior to BSE. The emphasis is on the fact that the disease has not been detected and the Harvard/Tuskegee report is cited to support the idea that the risk is very small. Because significant economic consequences would result from the discovery of just a single case, it would seem prudent for the U.S. to explicitly acknowledge the possibility that BSE may be discovered, while at the same time enhancing some counter measures.

Particular consideration should be given to measures that reduce the potential for human exposure. In the event of a discovery, having such measures in place would provide more credibility to reassurances about the safety of consuming beef. As already noted, for countries that cannot exclude the possibility of BSE the WHO recommends an SRM ban.⁴⁰ If BSE were

38 Pennings *et al.* report that the response to BSE by U.S. consumers would likely be similar to that of German consumers, since for both groups the consumption decision was driven more by risk attitude than by their perception of the level of risk. For Dutch consumers, who have not responded quite as dramatically to BSE as have Germans, the consumption decision was more closely related to risk perception than to risk attitude.

39 Israel, which had been one of the first countries to ban imports of MBM and live animals from countries with BSE, discovered its first case in June 2002.

40 Adoption of the human SBO ban in the U.K. in 1989 is probably the only example in the BSE story of a government going beyond expert opinion in taking a precautionary measure. It turned out to be the correct decision, and likely saved thousands of people from exposure to the disease.

discovered, the fact that bovine brain and other CNS tissues are not banned for human consumption could be very damaging given the tendency for the media to sensationalize risk issues. Limiting human exposure to bovine CNS tissue might also involve restrictions on AMR.⁴¹ Different AMR systems likely result in different rates of CNS contamination—but until it can be established that one or more systems result in an acceptably low level of contamination, a ban on the use of vertebral column for AMR may be the only way to guarantee absence of CNS in meat.

Other options include extending the feed ban to pigs and poultry. The U.K. experience with cross-contamination suggests that a U.S. style feed ban is inadequate, particularly if enforcement is weak. Eliminating animals that die on-farm from rendering would remove a high risk source of infectivity, while increasing the level of surveillance testing would provide greater assurance about the continued absence of the disease. Another potential measure is the introduction of animal tracing systems. In the event of BSE being detected, U.S. export markets would be compromised indefinitely because there is no way to trace an animal to its farm of origin and then forward-trace its cohorts or offspring. However, given the substantial costs, it would be difficult to obtain support for a broad range of measures to counter a theoretical risk. Any enhancement in BSE measures at this stage should focus on human health protection in an effort to minimize the market disruption that would result from a discovery.

Acknowledging the possibility of BSE and enhancing measures to safeguard human health should help engineer, in political/economic parlance, a “soft (or at least a softer) landing” if the disease is discovered. It may allow whatever additional measures are taken in response to a discovery to be more proportionate to the risk that is presented. The goal should be to avoid the extreme inefficiency of the Japanese situation in which massive costs are being incurred in response to what now appears to be an extremely low threat. Achieving that goal entails additional up-front costs, but the rate of return on ‘two stitches in time’ is still favorable.

⁴¹ AMR provides around 10 pounds of meat per animal, most of which is used in processed meat products such as hotdogs. The EU banned the process in 2001.

References.

Bosque, P. J., C. Ryou, G. Telling, D. Peretz, G. Legname, S. J. DeArmond, and S. B. Prusiner. "Prions in Skeletal Muscle." *Proc. Natl. Acad. Sci.* 99:6,3812-3817, March 19, 2002

Byrne, D., EU Commissioner for Health and Consumer Protection. "Speaking Note on the Olsson Report on BSE." Starsbourg. February 5, 2002. Available at www.defra.gov.U.K./animalh/bse/index.html

Cohen, J. T., K. Duggar, G. M. Gray, S. Kreindel, H. Abdelrahman, T. Habtemariam, D. Oryang, and B. Tameru. "Evaluation of the Potential for Bovine Spongiform Encephalopathy in the United States." Harvard Center for Risk Analysis and Center for Computational Epidemiology, Tuskegee University. November 2001.

DEFRA. BSE Homepage. U.K. Department for Environment, Food, and Rural Affairs. www.defra.gov.U.K./animalh/bse/index.html

Food Standards Agency. "Review of BSE Controls: Final Report." December 2000 [http://www.bsereview.org.U.K./](http://www.bsereview.org.U.K/)

Henson, S. and M. Mazzocchi. "Impact of BSE on Agribusiness in the U.K.: Results of an Event Study of Equity Prices." *American Journal of Agricultural Economics* 84(2002):370-386.

Horn, G. *et al.* "Review of the origin of BSE." July 2001. www.defra.gov.U.K./animalh/bse/bseorigin.pdf

McKenzie, D., J.C. Bartz, and R.F. Marsh "Transmissible mink encephalopathy" *Seminars in Virology*, 7(1996):201-206.

Murphy, D. "Commentary: GAO's report on BSE a bunch of BS." www.meetingplace.com March 1, 2002.

Pennings, J., B. Wansink, and M. Meulenber. "A note on modeling consumer reaction to a crisis: The case of mad cow disease." *Intl. Journal of Research in Marketing*. 19(2002):91-100.

Phillips, N. A., Bridgeman, J. & Ferguson-Smith, M. "The Inquiry into BSE and variant CJD in the United Kingdom." October 2000. [www.bseinquiry.gov.U.K./](http://www.bseinquiry.gov.U.K/)

Seng, P., President and CEO, U.S. Meat Export Federation. Presentation to the American Farm Bureau Federation Convention, January 6, 2002.

Table 1. List of Abbreviations

AMR	advanced meat recovery
BAB	born after ban
BSE	bovine spongiform encephalopathy
CJD	Creutzfeldt-Jacob Disease
CNS	central nervous system
CWD	chronic wasting disease
FAO	Food and Agriculture Organization of the United Nations
FDA	Food and Drug Administration
GAO	General Accounting Office
GBR	geographical BSE-risk
MAFF	Ministry of Agriculture, Fishery, and Forestry, Japan
MBM	meat and bone meal
MRM	mechanically recovered meat
OIE	Office International des Epizooties
SBO	specified bovine offal
SRM	specified risk material
TME	transmissible mink encephalopathy
TSE	transmissible spongiform encephalopathy
vCJD	variant Creutzfeldt-Jacob Disease
WHO	World Health Organization

Table 2. BSE Chronology

Date	Event
Dec. 1984	First recorded case of the new disease
June 1987	BSE confirmed
Dec. 1987	Epidemiologists link BSE to MBM
July 1988	U.K. bans <u>ruminant derived</u> MBM in ruminant feed
Nov. 1989	SBO (brain, spleen, spinal cord, thymus, tonsils, intestines) banned <u>for human consumption</u>
Sep. 1990	Ban on use of SBO in <u>all animal feed</u> (including pet food)
Mar. 1991	First BSE case in animal born after the July 1988 feed ban
<i>June 1994</i>	<i>EU bans feeding of <u>mammalian</u> protein to ruminants</i>
Aug. 1995	SBO definition expanded (entire skull incl. brains, eyes)
Dec. 1995	Ban on use of spinal column in MRM
Mar. 1996	Government announces probable link between BSE and CJD
Aug. 1996	Enhanced MBM feed ban (<u>all farm animals</u>) enforced
Jan. 1998	Introduction of EU-wide Specified Risk Material regulations
June 2000	First confirmed case born after the enhanced feed ban
<i>Nov. 2000</i>	<i>Germany, Spain report first domestic cases (Italy in Jan. 2001)</i>
<i>Jan. 2001</i>	<i>EU wide ban on MBM for <u>all farm animals</u></i>
Sep. 2001	First case born outside Europe (Japan)

Table 3. Beef Prices in Selected Countries: 2000-01^a

	<u>Aug. 2000</u>	<u>Aug. 2001</u>	<u>Change</u>
Ireland	202	178	-12%
U.K.	224	213	- 5%
France	230	178	-22%
Italy	252	195	-22%
Germany	214	161	-25%
Australia	118	137	+16%
U.S.	204	238	+16%

^a Units are Irish pounds per kilogram of dead weight. Compiled by the authors from various national sources.

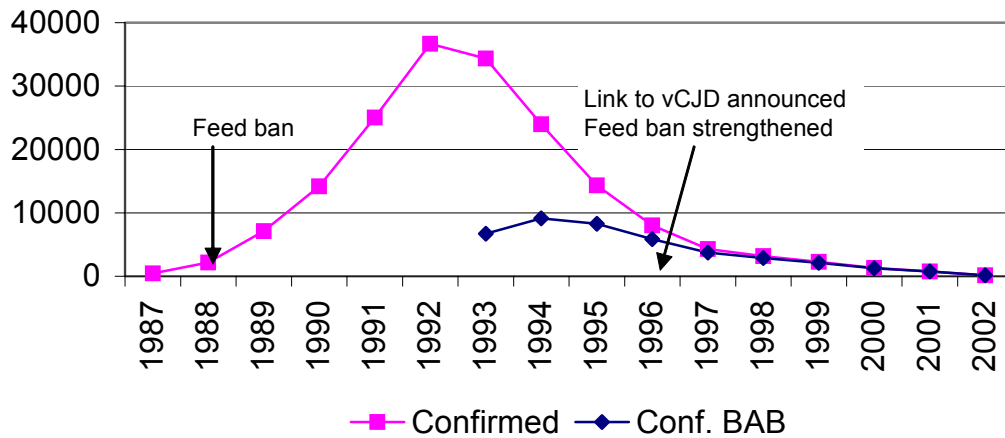


Figure 1. BSE in the U.K. Cases detected by passive surveillance as of May 10, 2002. Data from U.K. Department for Environment, Food, and Rural Affairs.

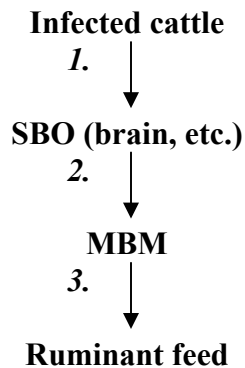
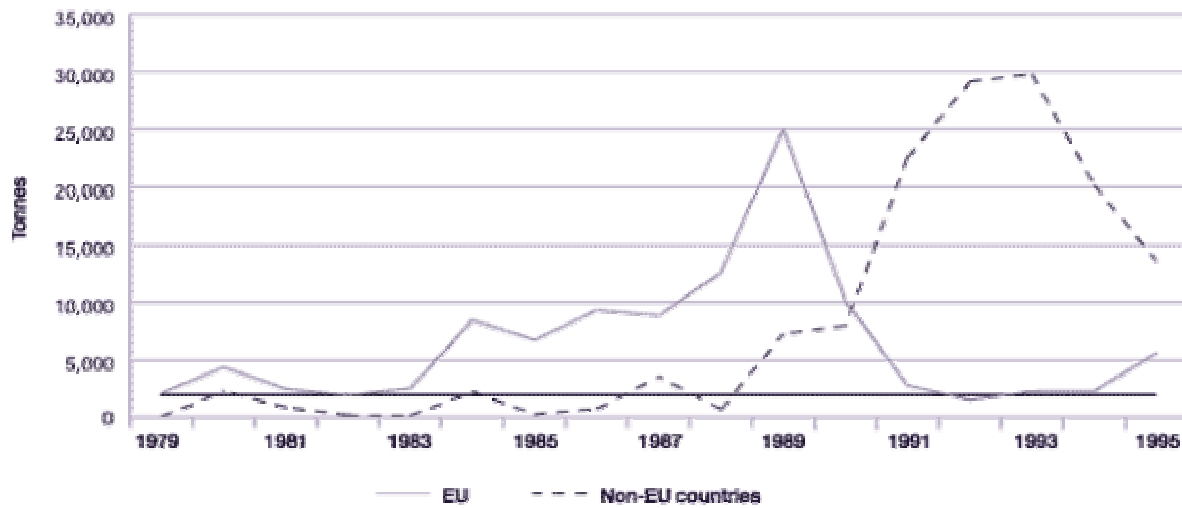


Figure 2. BSE infection route.



Source: HM Customs and Excise

Figure 3. MBM exports from the U.K. Figure 7.1 from the BSE Inquiry: U.K. exports of flours, meats and pellets of meat or meat offal, unfit for human consumption (greaves), 1979-95.

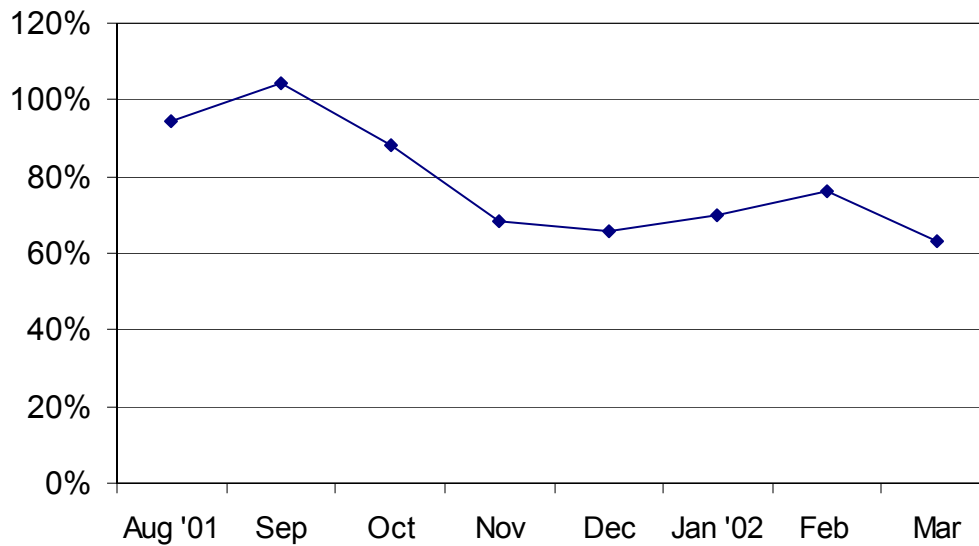


Figure 4. U.S. beef exports to Japan (percent of previous year). Data from U.S. Meat Export Federation.