2.2.3.4 Autoimmune Disease

It has been proposed that BSE could be an autoimmune disease caused by exposure of cattle to bacteria containing proteins that induce immunologic cross-reactivity with central nervous system tissue (Ebringer et al., 1997, Tiwana et al., 1999). According to this theory, the BSE epidemic resulted from the production of feed rich in the specific bacteria with proteins that mimic brain tissue. Because of exposure to these bacteria, bovine anti-bacterial antibodies may have reacted with myelin proteins in the brain that were sufficiently similar to bacterial antigens. The damage caused by such an auto-immune disease would be chronic and would be consistent with some of the characteristics of BSE (SSC, 2000a). Autoimmune brain disease can be experimentally transmitted to animals but only by injecting large amounts of brain protein and adjuvants. In contrast, TSE can be transmitted by injecting only a few nanograms of brain without adjuvant (Horn et al., 2001). In addition, the neuropathology of autoimmune brain disease is different than that observed in TSE.

SSC has concluded that this theory is not consistent with the BSE epidemic for several reasons. First, the morphology and distribution of vacuolar or spongiform-like changes due to BSE are not the same as those caused by an auto-immune reaction. Second, lymphocytic infiltrates that are typical of and are a pathogenetically important component of auto-immune disease are atypical of TSE. Third, high infectious titers cannot be explained by the autoimmune hypothesis (SSC, 2000a). Fourth, as pointed out by the BSE Inquiry (Lord Phillips et al., 2000), mouse adapted-BSE can be transmitted i.c. to mice lacking a functional immune system. Fifth, common bacteria do not have the resistance to chemical and physical inactivation shown by the agents of transmissible spongiform encephalopathies, including bovine spongiform encephalopathy. Finally, this theory fails to explain why many thousands of animals suddenly became affected and why the BSE epidemic occurred predominantly in the UK (Horn et al., 2001).

2.2.3.5 Use of Pituitary Hormones

Posterior pituitary extracts obtained from oxen or other mammals were used as a source of oxytocin for veterinary purposes through the period 1950-1989. Use of anterior pituitary extracts as a source of follicle stimulating hormone or bovine growth hormone occurred but was not widespread (Horn et al., 2001). Recall that growth hormone, prepared from cadaver pituitary glands, has caused transmission of CJD in humans, and this theory suggests that a similar

phenomenon may have occurred in cattle (Airtime and Resources, 2001). This hypothesis suggests that pituitary hormones contaminated by BSE caused the epidemic in the UK but not in other countries. The source of the original infectivity (in humans it was one or more pituitaries from CJD cases) is not explained by this theory.

2.2.3.6 Wild African Antelope

A group of scientists from New Zealand postulated that BSE originated from wild African antelope, and that it spread into British cattle when an infected animal from a wildlife park was rendered into MBM that was fed to about 1,000 dairy cows in the southwest of England between 1975 and 1977 (ProMED-mail, 2001, April 18). They hypothesized that the disease probably spread through British cattle for about a decade before it was fully recognized. Kelly et al. (Kelly et al., 1980) suggested that the death of six white tigers in a British zoo was due to TSE but the histopathology was not consistent with BSE (Horn et al., 2001). This theory is not consistent with the absence of evidence that carnivores in zoos in the UK were dying from TSE-like symptoms before 1986. In addition, there is no evidence that any TSE exists in the wildlife of Africa (Horn et al., 2001).

2.3 Sources of BSE Infectivity

This section describes potential sources and pathways by which BSE infectivity could be introduced into the U.S., including the development of a spontaneous BSE case (Section 2.3.1), importation of an infected animal into the U.S. (Section 2.3.2), scrapie (Section 2.3.3), oral ingestion of chronic wasting disease infectivity (Section 2.3.4), horizontal or lateral transmission of chronic wasting disease (Section 2.3.5), transmissible mink encephalopathy (Section 2.3.6), TSEs in pigs (Section 2.3.7), TSEs in chickens (Section 2.3.8), and contamination from recycled products, including plate waste, gelatin, milk, blood and blood products, and tallow (Section 2.3.9).

2.3.1 Spontaneous BSE

A potential way in which BSE could be introduced into the United States is the development of a spontaneous case of a BSE in a native animal. A "spontaneous case" is one that occurs in an animal with no known risk factors for development of BSE. The presumed mechanism by which a BSE could occur spontaneously is by the mutation of the PrP gene to a

form that codes for PrP^{sc}, and subsequent recruitment of PrP^{c} until disease is manifest (Prusiner, 1989); (for review see: (Chesebro, 1999). There is no direct evidence of this mechanism, although some argue that all mammals might have a low spontaneous rate of TSE (Hueston, 1997). In addition, a transgenic animal over-expressing the PrP gene has apparently replicated the human TSE GSS (Hsiao et al., 1991). Recent results, in which mice expressing the same point mutation but at normal levels failed to develop disease (Manson et al., 1999), suggest the mutations may increase susceptibility rather than directly cause the disease. Although at this time there is no scientific evidence suggesting that spontaneous BSE exists, the BSE Inquiry suggested that TSEs could possibly develop sporadically in other species, as they do in humans (BSE Inquiry, 2000). In contrast, the Review of the origin of BSE (Horn et al., 2001) concluded that although the spontaneous case hypothesis cannot be excluded, there is no evidence supporting the presence of sporadic form prion disease in cattle or sheep.

It is not possible to determine for any particular TSE whether the original cause was a mutation or transmission of disease from another species or from the same species. For example, transmissible mink encephalopathy (TME) has no known origin. There are a number of theories, most of which focus on transmission from another species (Marsh et al., 1991). In the case of BSE, there is little evidence from the epidemiology to suggest that cases arise without some exposure to infectivity (Wilesmith et al., 1991, Kimberlin and Wilesmith, 1994, Horn et al., 2001). On the other hand, there are a small number of cases for which there are no known risk factors (MAFF born after the ban, 2000 (MAFF, 2000a); Denmark born after the ban, 2000 (Tegtmeier et al., 2001)).

The existence of a spontaneous form of TSEs in animals is controversial. In humans, cases of CJD in persons with no known risk factors or exposure to the disease occur at an annual incidence of approximately one per million. The incidence appears to be relatively constant around the world, regardless of diet, environment, or other factors that may hypothetically influence disease rates. Cases in individuals with no known risk factors are often referred to as "sporadic CJD." The etiology of sporadic CJD is unknown. Sporadic CJD appears almost exclusively in humans more than 50 years old. Cases appear to occur without a predictable epidemiological pattern (Brown et al., 1994b, Will et al., 1986). Sporadic CJD accounts for 85% of all cases and these cases are characterized by a relatively rapidly progressive clinical course, although rare variants have shown an extensive duration of clinical illness (Brown et al., 1984). It is sometimes asserted that the rate of sporadic CJD in humans is likely to be representative of

the rate of spontaneous BSE in cattle (Biopharm, 1997), although the rate in cattle has never been directly measured and may in fact be zero.

2.3.2 Importation of BSE Infectivity into the United States

This section describes the potential for the importation of BSE infectivity into the United States, including the importation of live cattle (Section 2.3.2.1) and feed material (Section 2.3.2.2).

2.3.2.1 Importation of Live Cattle from the UK

The U.S. imported animals from the UK during the BSE epidemic. Between January 1, 1981, and July 1989, the United States imported 334 cattle from the UK. Ninety-six percent of these animals were beef breeding stock, while the remaining four percent were dairy cattle. In 1989, the US prohibited the importation of ruminants from countries affected with BSE.

Of the 334 UK imports, 161 were disposed of in a manner that eliminates the possibility that they could have contaminated either human food or animal feed. The remaining 173 cattle were imported before the peak of the epidemic, and none came from a birth cohort in which a BSE case is known to have developed (SSC, 2000d). Of these 173, 164 (94.8%) were beef breeding animals and nine (5.2 %) were dairy animals. It is possible that remains from some of the 173 cattle imported from the UK between 1980 and 1989 could have ended up in either animal feed, human food, or both. Section 3.4.3 describes our risk assessment of this scenario.

2.3.2.2 Importation of Cattle from Continental Europe

Between 1983 and 1987 397 breeding cattle were imported from Switzerland, France, Italy, and Belgium. These animals were beef breeds except for ten dairy animals imported from France in 1984. From 1996 to 1997, there were also 46 animals imported from Belgium, Germany, Austria and Italy. Because the vast majority of the imports from continental Europe occurred before 1988, they pose only a limited risk to the U.S. All animals imported after 1996 have been traced and their movements controlled. There is therefore virtually no risk that these imports introduced BSE into the US cattle population. Finally, two head of cattle imported from Belgium in 1996 are also under quarantine.

2.3.2.3 Importation of Cattle from Non-European Countries

It is estimated that since 1986, the U.S. has imported from Canada and Mexico between 750,000 and 2.5 million animals annually. The vast majority of the cattle (at least 80%) are animals for feeding or slaughter. The cattle imported from Canada for immediate slaughter are sent to slaughterhouses in sealed trucks. All identification is collected at the time of slaughter and they are noted as animals of Canadian origin. These imports are extremely unlikely to pose a risk of introducing BSE to the U.S.

2.3.2.4 Imported MBM and Feed

There are no reliable data documenting the type and composition of feed imported into the U.S. between 1980 and 1990. Current APHIS regulations prohibit the import of ruminant meat and edible products produced from animals in regions where BSE has been documented.

USDA reports that no MBM was imported from the UK between 1980 and 1990. Since 1989, MBM has been imported from New Zealand, Canada, Chile, Peru, and Australia. Most MBM came from Canada (an average of 25,000 metric tons per year). Chile exported to the U.S. only 3,000 metric tons of MBM in 1989. Peru exported 15,000 metric tons of MBM to the U.S. in 1989 and 4,000 metric tons in 1990. Panama exported 7,000 metric tons of MBM in 1989 and 4,000 tons in 1990. It is important to note that: 1) the U.S. accounts for approximately 60% of the rendering materials produced globally (Rudbeck, 1999); 2) the U.S. is mainly an exporter of MBM; and 3) shipping MBM to the U.S. from overseas is likely to be economically non-competitive (Don Franco, personal communication).

The geographical assessment of BSE in the U.S. (SSC, 2000d) reported the export of mammalian meal and flour from the UK in 1981 (10 tons), 1984 (2 tons), 1989 (20 tons), and 1997 (37 tons). The U.S. does not have corresponding import statistics for 1989 (USDA-APHIS, 2000d). It is likely that the materials imported in 1997 included non-mammalian protein because the UK Overseas Trade statistics did not specifically break out separate values for MBM, but instead subsumes this material in the category, "*flours and meals of meat and offals, unfit for human consumption, greaves*". In addition, since the adoption of the Commission Decision 93/239/EC on March 27, 1996, it has been illegal to export from the UK meat meal, bone meal, and MBM derived from mammals. MBM imported prior to the peak of the epidemic in the UK is not thought to pose a substantial BSE risk. Most importantly, MBM is used for pet food and

other products and poses little exposure risk to cattle. Finally, although it is possible that MBM from the UK entered the U.S. *via* a third country, there is no evidence that has occurred. In summary, past MBM imports pose little risk of exposing U.S. cattle to BSE.

2.3.3 Domestic Scrapie

The first case of scrapie diagnosed in the U.S. occurred in 1947. Various control and eradication programs have existed since 1952 when the disease became nationally reportable. At that time, when the disease was confirmed in a flock, the flock was quarantined and then depopulated. In 1957, the regulations were amended to include location and subsequent depopulation of the of source flocks. Different surveillance approaches have been instituted since that time (*e.g.*, the bloodline surveillance program, reporting incentives, *etc.*). The Voluntary Scrapie Flock Certification Program, approved in 1997 was designed to monitor flocks and certify the status of the animals enrolled in the program (USDA-APHIS, 2000c). This program is still in effect.

The precise prevalence of scrapie in sheep in the U.S. is unknown. It is likely that changes in the national scrapie control and eradication program affected the reporting of potentially infected sheep. Statistics from the period between 1947 and 1992 indicate that that during this period, there were a total of 1,117 affected sheep from 657 flocks located in 39 states (Wineland et al., 1998). The number of scrapie-positive flocks increased slightly between 1965 through 1992, probably reflecting changes in reporting protocols and changes in incentives offered to farmers to report affected animals. One hundred sixty-eight rams and 949 ewes were also reported to be scrapie-positive during the study period. Annual mortality due to scrapie in a flock is usually low (three to five percent), although higher mortality rates of up to 50% have been reported (Detwiler, 1992). In Great Britain, the prevalence of detectable scrapie infection in the slaughter population was estimated to be 0.11% in 1997/1998. This detectable rate probably corresponds to a true infection prevalence in the same population of up to 11% (Webb et al., 2001). Table 2-1 details the number of sheep condemned with scrapie in the U.S.

Table 2-1
Sheep Condemned With Scrapie in the United States:
Fiscal Year 1998

Animal Category	Number Slaughtered	Number Condemned	Percent Condemned

Mature Sheep	181,615	3,480	1.92
Lambs and Yearlings	3,272,844	3794	0.12
Goats	396,473	1505	0.38

The epidemiology of scrapie in the U.S. has reflected changes in the surveillance of the disease, reporting requirements, and in reporting incentives. The geographic concentration of scrapie-positive flocks in certain states has been attributed to the level of surveillance conducted within those states. There has been no identification of a seasonal incidence pattern associated with scrapie.

The BSE epidemic in the UK influenced the processing of animal proteins in the United States. In 1989, the National Renders Association (NRA) and the American Protein Producers Industry (APPI) recommended a voluntary ban on the processing of dead sheep. Consequently, the proportion of renderers processing inedible sheep offal also declined (Eastern Research Group, 1996). Later, the FDA prohibited the use of all ruminant proteins in ruminant feed (Food and Drug Administration, 1997).

2.3.4 Chronic Wasting Disease: Oral Exposure

Chronic-wasting disease (CWD) is a spongiform encephalopathy recognized for the first time in captive deer at a Colorado research facility in 1967 and later in Rocky Mountain elk at a Wyoming research facility in 1978 (Williams and Young, 1980). Additionally, CWD was confirmed in mule deer in 1977 (Williams and Young, 1980), and in free-ranging deer and elk in a five county region in Colorado and Southeastern Wyoming (Williams and Young, 1982). In high-risk areas, the prevalence of the disease is estimated to be between five and six percent, and in the surrounding areas, the prevalence is estimated to be around one percent (Miller et al., 2000). Outside the U.S., CWD has been diagnosed on game ranches in Saskatchewan, Canada since 1996. In 2001, the Canadian Food Inspection Agency (CFIA) confirmed that a wild mule deer in Saskatchewan had tested positive for CWD (Venter, 2001), representing the first case of CWD in the wild animal population in Canada (Canadian Food Inspection Agency Animal Products Animal Health and Production, 2001).

CWD is characterized by a progressive loss of body condition and by neurologic changes. It is believed that the infection is passed between animals *via* horizontal transmission. Most of the animals dying naturally from the disease are between the ages two and seven years.

Polymorphism in the PrP gene in elk has been associated with changes in the susceptibility to infection (O'Rourke et al., 1999). Extensive nationwide surveillance started in 1997 in an effort to better understand the geographic location and magnitude of the problem (USDA-APHIS, 2000a).

A small number of deer are commercially slaughtered for human consumption every year (USDA-FSIS, 1998). These animals are usually younger than 1.6 years and are not likely to be recycled into ruminant feed (L Floyd, personal communication). Hunters often process the cervids they kill themselves or use local butchers. In these cases, high-risk materials may be further processed at a prohibited rendering facility. Game deer are fed with protein supplements that contain small amounts of vegetable supplemental protein (L Floyd, personal communication).

Although CWD has been transmitted to cattle *via* i.c. inoculation, transmission by natural routes to cattle is very unlikely to occur (Hamir et al., 2001). For example, cattle orally inoculated with CWD infected brains have not shown any evidence of infection (Beth Williams, personal communication). *In vitro* experiments investing the conversion of bovine PrP^{c} by PrP^{CWD} suggest evidence of a molecular barrier limiting the susceptibility of cattle (Raymond et al., 2000). Moreover, even if CWD could be transmitted to cattle, it is not clear it could cause BSE. For example, preliminary analysis of intracerebral inoculation of cattle with CWD revealed no typical TSE lesions in brain but did reveal the presence of PrP^{sc} by immunuhistochemistry, Western Blot, and electron microscopy in three animals 22-27 months post-inoculation (Hamir et al., 2001).

Even if CWD exposure can cause BSE, it is likely that the species barrier is substantial. *In vitro* assays have suggested that homology between an infectious and endogenous PrP molecule influences the rate and likelihood of conversion from PrP^{c} to PrP^{sc}, allowing the TSE disease to develop in the host (Priola et al., 1994). *In vitro* studies have also shown differences between bovine PrP and PrP^{CWD} and these differences may represent a substantial species barrier. For example, Raymond et al. (Raymond et al., 2000) reported that the cell-free conversion efficiency between bovine PrP^{c} and PrP^{CWD} is between five and twelve orders of magnitude weaker than inter-cervid transmission. This finding suggests that the species barrier may be between 10^{5} and 10^{12} (Raymond et al., 2000).

Ascertaining the potential risk posed by oral exposure to CWD is further complicated by the following sources of uncertainty. First, there are no accurate statistics documenting the number or type of deer and elk killed by hunters. Second, the type of deer and elk that can be hunted in different geographic areas varies. Third, the disposition of deer and elk remains after slaughter is uncertain. Finally, the prevalence of the disease in all but the highest risk areas is unknown.

2.3.5 Chronic Wasting Disease: Lateral Transmission

Direct contact between cattle and cervids in regions where CWD is prevalent may provide another pathway by which cattle may become infected. Epidemiologic modeling suggests that among cervids, environmental contact provides a pathway for the spread of CWD (Miller et al., 2000). Moreover, the prevalence of the disease under experimental conditions appears to be extraordinarily high. For instance, when deer within the endemic research facilities have been introduced into CWD negative deer herds, the disease quickly reaches a prevalence of 50% and 60% (Food and Drug Administration, 2001a).

Nonetheless, there is no evidence that CWD can cause TSEs in cattle. As noted in Section 2.3.4, any such transmission would be limited by what appears to be a substantial species barrier. Moreover, cattle cohabiting with CWD infected deer and elk in a research facility in the endemic area have shown no evidence of infection (Beth Williams personal communication). Finally, targeted surveillance of cattle brains (by immunohistochemistry and histopathology) from endemic areas have failed to reveal the presence of CWD or any other TSEs (Daniel Gould, personal communication; James Voss, personal communication, (Gould, 2000)).

2.3.6 Mink

Transmissible Mink Encephalopathy (TME) is a rare disease known to occur only in farm-raised mink. Epidemiological studies have suggested that TME is a foodborne disease with an incubation period of between seven months and a year (Hartsough and Burger, 1965). The disease is characterized by a long incubation period, a clinical course of several weeks, and neurological changes. Mink experience increased aggressiveness, hyperexitability, ataxia, and hyperaesthesia. Cases in Wisconsin and Minnesota were recognized on mink ranches as early as 1947. Outbreaks have been reported in Ohio, Canada, Finland, Germany, and Russia. Five outbreaks have been recorded in the U.S., affecting a total of 23 mink ranches (Hartsough and

Burger, 1965, Hadlow et al., 1987, Marsh et al., 1991). Three of the outbreaks in Wisconsin were associated with the use of fallen or sick cattle in mink feed (Hartsough and Burger, 1965, Marsh et al., 1991). However, there is no consensus over the source of disease initiating these outbreaks. The rancher involved in the Stetsonville, Wisconsin outbreak claimed that his mink were fed only dead stock and that they were never fed sheep.

In theory, transmission of TME to a bovine could cause BSE. Inoculation of cattle with TME *via* i.c. administration resulted in spongiform encephalopathy a short time later (Marsh et al., 1991, Robinson et al., 1995). In addition, cattle passage of TME remains pathogenic to mink when administered either orally or *via* i.c. (Marsh et al., 1991). According to Marsh, these results suggest that the species barrier between mink and cattle may not be substantial. To protect against the possibility that TME might be transmitted to cattle, the FDA prohibits use of mink protein in ruminant feed. Mink are considered prohibited materials. The relatively small number of farmed mink, their small size, and recycling prohibitions make transmission of a prion disease from mink to cattle extremely unlikely.

2.3.7 Pigs

There is a theoretical risk that cattle could be exposed to a TSE as the result of consuming feed supplemented with porcine-derived protein. Moreover, the fact that federal regulations classify protein from pigs as non-prohibited increases the potential for cattle to be exposed to any infectivity they may harbor.

There are two potential sources of this exposure: a natural TSE that infects pigs (Section 2.3.7.1), and BSE-contaminated feed in the gut at the time the pig is slaughtered (Section 2.3.7.2). In practice, neither infectivity source will make a substantial contribution to cattle exposure because only a small portion of porcine-derived MBM is used as cattle feed. One reason for the limited use of porcine-derived protein in cattle feed derives in part from its price. For example, in May, 2001, the price of porcine-derived protein was $238/ton, compared to $177/ton for soy protein (Southern States Cooperative, 2001). In addition, much rendered porcine protein is used in feed for pigs

The remainder of this section outlines additional factors that influence the importance of this potential source of TSE exposure among cattle. Because these sources are unlikely to be significant, we do not address it quantitatively in our risk assessment (Section 3).

2.3.7.1 Potential Infectivity in Pigs due to TSE Infection

Pigs might become infected with a TSE as the result of any of the following possibilities:

- The existence of a porcine-specific TSE agent;
- A nonspecific TSE agent not yet adapted to pigs that has an incubation period that is longer than the life of the pig; or
- The spontaneous misfolding of the prion protein leading to a spontaneous TSE case in porcine.

Consumer groups in the U.S. have expressed concern that it may be possible for a pig to become infected with a TSE. This concern stems from a 1979 incident in which one of 60 pigs presented with clinical neurologic signs, neurological degeneration, and gliosis on histopatological examination (Hansen, 1999). However, further testing showed that the lesions were not patognomonic of spongiform encephalopathy (Linda Detwiler, personal communication). Moreover, the animal in question was young, a factor that is inconsistent with the TSE diagnosis. SSC (SSC, 1999b) concluded that these factors argue against the presence of an unrecognized spongiform encephalopathy in pigs in the U.S.

Other evidence also suggests that the existence of a porcine-specific TSE agent is unlikely. No naturally occurring TSE has ever been reported in pigs (MAFF, 2000b). Moreover, pigs inoculated orally with BSE have not developed disease (MAFF, 2000b). Experimental inoculation of pigs with different strains of Kuru *via* parenteral administration did not lead to spongiform encephalopathy 52 to 76 months post inoculation (Gibbs et al., 1979). A similar result was reported for pigs challenged with scrapie (SSC, 1999b) up to 63 months after inoculation. Pigs have also been challenged with brain material from cattle naturally infected with BSE by combined i.c., i.p., and i.v. routes (Dawson et al., 1990). This experiment is still ongoing but preliminary results show that seven of ten pigs developed spongiform encephalopathy (Ryder et al., 2000).

In an ongoing experiment in which ten pigs were subject to oral challenge with large amounts of brain from cattle naturally infected with BSE, none of the animals have developed clinical disease or neuropathologic changes. The oral challenge consisted of homogenized brain from confirmed cases of BSE. Each pig received a dose of 1.2kg of brain divided into three doses at intervals of one week. Mouse bioassays of neural and non-neural tissues from pigs killed at 84 months post inoculation were initiated in October and November, 1997 and were completed in May, 2000. As of the drafting of this report, there is no evidence of residual infectivity in any of the tissues. These findings may indicate that the species barrier between pigs and cattle is higher than the species barrier between cattle and humans or between cattle and other animals that have developed spongiform encephalopathy after exposure to BSE-contaminated MBM.

The fact that a naturally occurring spongiform encephalopathy has never been reported in pigs may indicate that pigs are particularly resistant to this type of disease. That is, the species barrier between pigs and other species may have prevented the transmission of natural disease to pigs. For example, it is very likely that pigs in the UK were exposed to substantial doses of contaminated MBM before the implementation of the feed ban. Although most pigs are slaughtered at a very young age, there are a significant number of sows and boars that usually live until age four. The fact that parenterally challenged animals developed disease 17 months post inoculation suggests that these four year old animals were sufficiently old to develop disease. Other species serve as examples. Marsh et al. (Marsh et al., 1969) noted the recovery of TME from the spleen of one chicken, and from spleen, caecom, tonsils, and bursa of Fabricius after i.v. inoculation. Race and Chesebro (Race and Chesebro, 1998), have shown that after inoculation of mice that either did or did not express the prion protein (PrP) gene with the hamster scrapie strain 263K, no clinical disease was produced in mice. However, infectivity found in the brain and spleen of mice expressing the prion protein was capable of causing disease in hamsters but not in mice. Hill et al. (Hill et al., 2000), have shown the possible presence of subclinical TSE in certain animals by demonstrating that a strain of hamster prions thought to be nonpathogenic for conventional mice leads to high levels of prion replication in such mice without causing clinical disease. Alternatively, it is possible that cases in pigs in the UK have gone unnoticed.

Finally, even if pigs could become infected with a TSE, most are slaughtered at a young age, making it unlikely that the disease would have time to generate more than a small amount of infectivity. For example, in 1998, more than 95% of the pigs slaughtered in the U.S. were no older than six months (USDA-FSIS, 1998).

2.3.7.2 Potential Infectivity in Materials Consumed by Pigs

Even if pigs do not become infected with a TSE, contaminated material may be present in their digestive tract when they die. In particular, if feed administered to pigs contains cattle-derived MBM that is contaminated with BSE, pigs could harbor BSE in their alimentary tract. The following discussion outlines several factors suggesting that the potential is limited for BSE to be recycled through the guts of pigs.

First, most pigs are not exposed to cattle-derived MBM because there are many other economical sources of protein. For example, in the U.S., soybean meal is usually the most economical source of high quality protein available for porcine diets. It is comparable to animal proteins in terms of the quality of its amino acid components and can be used as the only protein source in most swine diets. Other sources of proteins fed to pigs include porcine MBM, peanut meal, fish meal, cottonseed meal, canola meal, sunflower meal, and raw soy beans. The amount of protein added to feed varies based on the specific needs of the animal as it grows. MBM, blood meal, and plasma can comprise between 2.5 and 5 percent of feed for pigs between weaning and 60 days of age and during the animal's growing and finishing stages. Because it is not uniformly used, it is likely that approximately 80% of the pigs grown in the United States never receive MBM.

Second, even among pigs that do receive cattle-derived MBM, it is likely that little if any feed would remain in the GI system at the time of processing because pigs are usually sent to slaughter after restricting feed intake for 14 to 16 hours.

Third, due to the high water content of the GI tract, contents are unlikely to be rendered.

Finally, if the gut contents are rendered, any BSE-contaminated material that does make its way back to cattle will have gone through rendering twice, thus providing an additional opportunity for infectivity to be destroyed by this treatment.