Overview of BSE in the United Kingdom: U.S. Response

Questions and Answers Regarding Bovine Spongiform Encephalopathy

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Introduction

In 1990, the government of Great Britain* established the Spongiform Encephalopathy Advisory Committee (SEAC), which consists of experts (outside of the British government) in neurology, epidemiology and microbiology, to provide scientifically based advice on the implications for animal and human health in regards to the spongiform encephalopathies. The main focus is bovine spongiform encephalopathy (BSE). In May 1990, the government of Great Britain also reinstituted active epidemiologic surveillance of Creutzfeldt-Jakob disease (CJD). The purpose of the surveillance is to identify changes in the pattern of CJD which might indicate an association with BSE.

On March 20, 1996, the SEAC advised the government of Great Britain that a newly recognized variant of CJD (V-CJD) had been identified in 10 patients in the United Kingdom** (UK). These cases had been identified through national surveillance and had an onset of symptoms between early 1994 and late 1995. In contrast to typical cases of sporadic CJD, this variant form has affected young patients (mean age, 26.3 years) with a relatively long duration of illness (mean, 14.1 months). The characteristic neuropathological profile in this variant consists of numerous widespread Kuru-type amyloid plaques with surrounding vacuolation and serve cerebellar lesions. A review of these patients' medical histories, genetic analysis (prion protein gene) and consideration of other possible causes of CJD failed to explain these cases adequately.

The SEAC concluded that although there was no direct scientific evidence of a link between BSE and CJD, based on current data and in the absence of any credible alternative, the most likely explanation at present is that these cases are linked to exposure to BSE before the introduction of control measures, in particular, the specified bovine offal (SBO) ban in 1989.

To date, V-CJD has been confirmed in 6 additional people -- 5 in the United Kingdom and one in France. There has also been another probable case identified in Great Britain.

An April 2-3, 1996, World Health Organization (WHO) consultation on Transmissible Spongiform Encephalopathies (TSEs) concluded that although a link has not yet been proven between V-CJD in the United Kingdom and the effect of exposure to the BSE agent, the most likely hypothesis for V-CJD is the exposure of the U.K. population to BSE. A follow-up May 14-16, 1996, WHO consultation on TSEs, concluded that the type of lesions and clinical presentation of the new variant CJD do not provide information on the possible origins of this disorder.

The result of a May 2-3, 1996 BSE meeting of the Office of International Epizootics (OIE) was a revised chapter on BSE for the OIE International Animal Health Code. This defines standards recommended for international trade in animals and animal products. In addition, OIE established minimum requirements for effective surveillance for determination of the BSE status of a country.

Below, by the following five topics, are some questions and answers related to this situation:

Topics:

1. Bovine spongiform encephalopathy (BSE)
2. Other human and animal transmissible spongiform encephalopathies (TSEs)
3. Creutzfeldt-Jakob disease (CJD) and variant-CJD (V-CJD)
4. USDA actions
   a. Surveillance
   b. Education
   c. Prevention
   d. Risk Assessment
5. Feeding of ruminant protein to ruminants

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1. Bovine spongiform encephalopathy (BSE)

What is bovine spongiform encephalopathy (BSE)?

BSE is a slowly progressing degenerative disease affecting the central nervous system of cattle. The disease was first diagnosed in Great Britain in 1986. BSE belongs to a family of diseases known as the transmissible spongiform encephalopathies (TSEs).

What are the clinical signs of BSE?

Cattle affected by BSE develop a progressive degeneration of the nervous system. Affected animals may display changes in temperament, such as nervousness or aggression, abnormal posture, incoordination and difficulty in rising, decreased milk production, or loss of body condition despite continued appetite. There is no treatment and affected cattle die.

The incubation period ranges from 2 to 8 years. Following the onset of clinical signs, the animal's condition deteriorates until it dies or is destroyed. This usually takes from 2 weeks to 6 months. Most cases in Great Britain have occurred in dairy cows between 3 and 6 years of age.

What causes BSE?

The causative agent of BSE as well as other transmissible spongiform encephalopathies is yet to be fully characterized. The agent has the following characteristics: (1) the agent is smaller than most viral particles and is highly resistant to heat, ultraviolet light, ionizing radiation, and common disinfectants that normally inactivate viruses or bacteria; (2) the agent causes no detectable immune or inflammatory response in the host; (3) the agent has not been observed microscopically.

The three main theories on the nature of the agent have been proposed:

1. An unconventional virus.
2. A prion- a partially protease-resistant protein, devoid of nucleic acid.
3. A virion or "incomplete" virus composed of naked nucleic acid protected by host proteins.

Proposed causes of TSEs with less supporting evidence are 1. retroviruses, 2. a spiroplasma, 3. organophosphates, and 4. peptide hormones.

Currently, the infectious protein or prion theory has gained acceptance among some in the science community. This theory suggests the infectious unit is a normal host protein (prion protein or PrP^{c}) that is posttranslationally transformed into the abnormal infectious unit or PrP^{sc}. The PrP^{sc} replicates or propagates by inducing the normal host PrP^{c} to become the partially protease-resistant form. It is postulated that transformation of the prion protein may occur from mutation of the prion gene or from contact with extraneous PrP^{sc}.

BSE seems to be caused by a single strain type. This BSE strain is different from at least 9 historical or contemporary isolates from sheep or goats with natural scrapie as determined by study of incubation periods and "lesion profiles" in mice.

Can BSE be confirmed in the live animal?

No. Currently, there is no test to detect the disease in a live animal.

The potential live animal tests, including some in development, are: (1) tests specific for the partially protease- resistant form of the prion protein: (a) a capillary electrophoresis test and (b) a western blot test with increased sensitivity; and (2) tests which identify unique substance of infected animals or humans: (a) a cyclic voltametric method which describes unique substances in urine and (b) an immunoblot test describing unique substances in cerebral spinal fluid. However, live animals tests have not been validated for practical use.

How is BSE diagnosed?

In the United Kingdom, diagnosis depends on the medical history and occurrence of clinical signs in combination with neurohistopathological examination.

In the United States, correlation between clinical diagnosis and histopathological confirmation may be complicated due to the existence of rabies. Therefore, supplemental tests must be done to confirm the diagnosis using immunobiochemical western blot straining and/or immunohistochemical (IHC) detection of protease resistant protein associated with infection and/or ultrastructural examination for the characteristic scrapie-associated fibrils (SAF) in the brain.

What caused BSE in Great Britain?

The epidemiologic data suggests that BSE in Great Britain is a common source epidemic involving feed containing contaminated meat and bone meal as a protein source. The causative agent is suspected to be from either scrapie-affected sheep or cattle with a previously unidentified TSE. Changes in rendering practices may have potentiated the agent's survival in meat and bone meal.

Has BSE been confirmed in countries other than Great Britain?

In addition to Great Britain, BSE has been confirmed in **native** cattle in Ireland, Northern Ireland, France, the Netherlands, Portugal, and Switzerland. Over 99% of all cases of BSE have occurred in the United Kingdom. BSE has also been identified in cattle **exported** from Great Britain to Oman, the Falkland Islands, Germany, Denmark, Canada and Italy.

Does BSE exist in the United States?

No. BSE has never been diagnosed in the United States.

Do we have BSE in North America?

No. There have been no cases in native cattle in North America. There has been one case of BSE confirmed on December 7, 1993, in western Canada. This case was a cow imported from Great Britain; the cow was euthanized and the carcass incinerated. In addition, Canadian authorities destroyed the herdmates of the infected cow and other cattle considered to be exposed.

The late Dr. Richard Marsh at the University of Wisconsin speculated that a transmissible spongiform encephalopathy (TSE) is already in U.S. cattle and takes the form of the "downer cow syndrome". What is APHIS's response to this?

"Downer cow" is a common slang term used in the United States to refer to a cow that is down and cannot get up (i.e. non-ambulatory). "Downer cow" can describe animals that cannot rise because they have been affected with a metabolic disease, have broken limbs, joint problems, etc. In APHIS's view Dr. Marsh's hypothesis is based on considerable speculation and anecdotal evidence. In explaining his theory Dr. Marsh cited an outbreak of transmissible mink encephalopathy on one farm which had reportedly fed "downer cows" to mink. In response to this theory, APHIS adjusted its surveillance program to include sampling "downer cows" at slaughter, and to date have not identified a TSE in U.S. cattle.

Are all "downer cows" tested for BSE?

No. Most nonambulatory or "downer cows" are not true BSE-suspects, as recumbency is not a frequent clinical sign seen in BSE-infected cattle. Sampling of nonambulatory cattle began primarily in response to concerns that an undetected transmissible spongiform encephalopathy existed in U.S. cattle based on reports of a similar disease in mink that were reportedly fed "downer cows". Since 1993, the APHIS and FSIS have conducted a joint program in which brains of "downer" cattle have been randomly collected in slaughter establishments in Iowa, Pennsylvania, Texas, Michigan, California, and Wisconsin. Over 800 brains from this program have been analyzed with no evidence of BSE detected.

Dr. Joseph Gibbs of the National Institutes of Health believes that a cattle TSE may exist in the United States already. What is APHIS's position?

Dr. Gibbs has speculated transmissible spongiform encephalopathies (TSE) in many species may occur spontaneously at an extremely low level worldwide, and at a rate so low as to be very difficult to verify. We have no evidence to date to suggest that a TSE exists in our native cattle population despite actively searching for almost seven years. Our surveillance program is considered to be one of the most aggressive in the world, compared to other countries considered free of BSE.

What is the experimental host range of BSE?

BSE has been transmitted to cattle, sheep, goats, pigs, marmosets, mink, and mice by parenteral (intracerebral) inoculation.

Experimental oral transmission has been attempted in all these species except marmosets, and has been successful in all except pigs.

In addition, there is a recent report of intracerebral inoculation of 3 macaque monkeys which produced a brain disease with plaques "identical" to those of the V-CJD patients.

Chickens which have been inoculated both intracranially and orally with BSE still remain healthy after six years.

Has BSE affected other species?

Yes. In the United Kingdom, 7 species of captive wild ruminants have developed BSE; and 8 exotic (2 cases born in British zoos and exported) and 70 domestic cats have developed feline spongiform encephalopathy (FSE). There has also been one case of FSE in a domestic cat in Norway, one in Northern Ireland and one in Liechtenstein.

The agent isolated from several of these cases is indistinguishable from BSE in cattle using strain typing in mice, suggesting that FSE is actually BSE in exotic and domestic cats.

What tissues from BSE affected cattle are infective?

To date, brain, spinal cord, and retina from naturally infected animals have been found to be infective. The lower ileum (intestine) from experimental cattle inoculated was found to be infective.

What does "specified bovine offal (SBO)" include?

SBO (now specified bovine material [SBM] refers to a group of tissues specified by British regulations to be banned from animal and human consumption. SBMs include brain, spinal cord, spleen, intestines (duodenum to rectum), thymus and tonsil. Recently, an extension to SBMs includes the whole head of all cattle over 6 months (except for the tongue provided it can be removed without being contaminated) and spinal column.

What tissues from sheep naturally infected with scrapie are infective?

Based on bioassays of infectivity using mice injected intracerebrally, highest levels of infectivity are

found in the brain and spinal cord. Medium to minimal infectivity are found in a variety of tissues including lymphoreticular tissues, lung, liver, pancreas, adrenal, and pituitary glands.

Does horizontal or vertical transmission of BSE occur?

There is no evidence that BSE spreads naturally by contact from cattle to cattle or from cattle to other species.

Interim results of British research show low levels of transmission of BSE from affected cows to their offspring. Preliminary results of research begun in 1989 suggest that, under field conditions, 1% of all calves born to cows which die of BSE will themselves die of BSE. The Spongiform Encephalopathy Advisory Committee, in considering this interim report, concluded that material transmission at this rate will not perpetuate the epidemic.

2. Other human and animal transmissible encephalopathies (TSEs)

Are there other similar diseases in humans and other animals?

Yes. Transmissible spongiform encephalopathies (TSEs) are caused by similar uncharacterized agents that produce spongiform changes in the brain. Specific examples of TSEs include: scrapie which affects sheep and goats; transmissible mink encephalopathy; feline spongiform encephalopathy; chronic wasting disease of mule deer, white-tail deer, black-tail deer, and elk; kuru, Creutzfeldt-Jakob disease, Gerstmann-Straussler syndrome, and fatal familial insomnia in humans.

What are the common characteristics of the transmissible spongiform encephalopathies (TSEs)?

The common characteristics of the TSEs are:

a. Long incubation periods of months to years.
b. The presence of scrapie associated fibrils in the brain.
c. The ability to transmit the disease to mice by an intracerebral inoculation of brain tissue from the host.

Have transmissible spongiform encephalopathies (TSEs) been identified in other animals in the United States?

Yes. Scrapie in sheep and goats; transmissible mink encephalopathy; and chronic wasting disease of mule deer, white-tail deer, black-tail deer, and elk have been identified in this country.

Have other TSEs been identified in other countries?

Yes. Scrapie is reported in most countries of the world. Transmissible mink encephalopathy has occurred on mink ranches in Finland, Russia, the United States, Canada and Germany. Chronic wasting disease has been diagnosed in elk, mule deer, white-tail deer, and black-tail deer in the United States and in one elk exported from the United States to Canada. Other cases of spongiform encephalopathy have been reported in kudu, eland, nyala, gemsbok, and domestic cats and a few exotic cats (none of these has been in the United States).

What are the USDA, APHIS's polices on the following animal TSEs?

Scrapie - Scrapie was first diagnosed in the United States in 1947. Since 1952 there has been some form of an eradication or control program. The latest program went into effect in 1992. It involves a Voluntary Flock Certification Program and interstate movement regulations which place restrictions on the movement of sheep and goats from infected and source scrapie flocks. The intent of the certification program is to monitor flocks over a period of 5 years or more and identify flocks which have not displayed evidence of scrapie. The program consists of four levels each with certain requirements. As a flock advances through the program, each level represents a lower risk of having scrapie.

TME - There is no official USDA program on transmissible mink encephalopathy (TME). We continue to monitor for reoccurrences of TME disease. The last known case of TME occurred in the United States in 1985. Prior to this one outbreak, several occurred prior to 1964.

CWD - Chronic wasting disease (CWD) in mule deer, white-tail deer, black-tail deer, and elk. There is no official USDA program on CWD. APHIS cooperates with state wildlife and diagnostic officials in Colorado and Wyoming in the limited areas where the disease has been reported.

3. Creutzfeldt-Jakob disease (CJD) and variant-CJD (V-CJD)

What is Creutzfeldt-Jakob disease (CJD)?

CJD is a slow degenerative human disease of the central nervous system with obvious dysfunction, progressive dementia, and vacuolar degeneration of the brain. CJD occurs throughout the world at a rate of 1-2 cases per 1 million people, per year. More rare are the related human TSE conditions of Gerstmann-Straussler syndrome (GSS), kuru, and Fatal Familial Insomnia (FEI).

Do we have Creutzfeldt-Jakob disease (CJD) in the United States?

Yes. The incidence in the United States (approximately 1 case per 1 million population per year) is similar to the incidence found in the rest of the world which includes Australia and New Zealand, countries which have not reported scrapie since the 1950s or BSE.

What is the variant Creutzfeldt-Jakob disease (V-CJD)?

V-CJD has a characteristic clinical and pathological phenotype. The characteristic neuropathological features are the presence of large numbers of kuru-type PrP amyloid plaques surrounded by a halo of spongiform change and severe cerebellar lesions. These cases share an early age at onset of symptoms, an unusual clinical course, with early psychiatric features and a prolonged duration of illness.

Do we have the variant CJD in the United States?

The Centers for Disease Control and Prevention (CDC) recently updated its previous review of national CJD mortality and began conducting active CJD surveillance in five sites in the United States. These reviews did not detect evidence of the occurrence of the newly described variant form of CJD in the United States.

Are U.S. farmers and veterinarians at increased risk for contracting CJD?

No. There is no scientific evidence to suggest they are at higher risk than the general U.S. population.

What is the issue causing the concern in Great Britain?

On March 29, 1996 the SEAC advised the government of Great Britain that a newly recognized variant of CJD (V-CJD) had been identified in 10 patients in the United Kingdom. These cases had been identified through national surveillance and had an onset of symptoms between early 1994 and late 1995. In contrast to typical cases of sporadic CJD, this variant form has affected young patients (mean age, 26.3 years) with a relatively long duration of illness (mean, 14.1 months). The characteristic neuropathological profile in this variant consists of numerous widespread Kuru-type amyloid plaques with surrounding vacuolation and severe cerebellar lesions. A review of these patients' medical histories, genetic analysis (prion protein gene) and consideration of other possible causes of CJD " failed to explain these cases adequately."

The SEAC concluded that although there was no direct scientific evidence of a link between BSE and CJD, based on current data and in the absence of any credible alternative, the most likely explanation at present is that these cases are linked to exposure to BSE before the introduction of control measures, in particular, the specified bovine offal (SBO) ban in 1989.

Why did the Spongiform Encephalopathy Advisory Committee (SEAC) make the announcement of a possible link between BSE and V-CJD at this time?

When attempting to take responsible actions in response to an emerging disease, one runs the risk of either acting prematurely and causing an unnecessary crisis or not acting soon enough resulting in increased risk to a certain population. Considering the seriousness of the situation, the Committee felt it scientifically, ethically, and morally necessary to state what was know, what was not known, and what is their best science-based hypothesis. Hence each consumer, regulatory official, or scientist could then take whatever action they feel is appropriate.

Is there any geographical clustering of these U.K. V-CJD cases?

No geographical clustering is evident, but the number of cases is small.

How many cases of V-CJD have been confirmed?

As of April 3, 1997, there were 15 confirmed cases and 1 probable case in the United Kingdom and 1 in France.

How do we know these V-CJD cases were not caused by the scrapie agent?

Scrapie has existed in the sheep population in the U.K. for 250 years, and has never been shown to be a human health risk. There is no reason to suppose that this has changed.

Is the incidence of CJD higher in the United Kingdom than elsewhere?

No. The disease, first diagnosed in the 1920's occurs with roughly the same frequency as in other countries at the present time.

4. USDA Actions

What is the USDA policy in regards to BSE and what actions has the USDA taken?

The USDA policy has been to be proactive and preventative. As BSE is not know to exist in the United States, the measures taken have been in surveillance, prevention, education, and response. Import restrictions have been in place since 1989 and active surveillance efforts began in 1990. The USDA continually monitors and assesses all ongoing events and research findings regarding spongiform encephalopathies, as new information and knowledge may lead to revised conclusions and prevention measures. APHIS has also created a Transmissible Spongiform Encephalopathy (TSE) Working Group to analyze risks of BSE to the United States, disseminate accurate information about the TSEs, and act as a reference source for responding to questions about TSEs.

Is APHIS working with other agencies and groups to coordinate efforts?

Yes. APHIS has actively shared information and

met with state and federal agencies including CDC, FDA, FSIS, NIH, and stakeholders to assure we are taking the proper actions in response to changing knowledge and information concerning BSE.

Is BSE a notifiable disease in the United States?

Yes. Under Title 9 Code of Federal Regulations, Parts 71 and 161, BSE is a reportable disease by accredited veterinarians.

Is there a risk to humans from cattle products, other than meat, from BSE-affected countries?

In the United States the use of such materials for human products such as pharmaceuticals, cosmetics, medical devices is regulated by the Food and Drug Administration (FDA). The FDA should be contacted regarding their recommendations. There have been publications in the Federal Register where the FDA has advised against using cattle-derived materials from other countries affected with BSE. APHIS regulations since 1989 restrict the importation of most ruminant materials from a BSE affected country.

4a. Surveillance

What types of BSE surveillance are we doing?

USDA-APHIS, in cooperation with USDA-FSIS, has a comprehensive surveillance program composed of two main types, active and passive surveillance.

Active Surveillance:

* USDA-APHIS educates veterinary practitioners, veterinary laboratory diagnosticians, industry and producers on the clinical signs and pathology of BSE.
* USDA-APHIS monitors the remaining cattle imported from the United Kingdom.
* Since 1990, more than 60 veterinary diagnostic laboratories across the United States and USDA's National Veterinary Services Laboratories continue to examine hundreds of cattle brains each year submitted from adult cattle displaying neurologic signs either at slaughter or on the farm. As of March 31, 1997 a total of 5,552 brains from 47 states and Puerto Rico have been examined with no evidence of BSE detected.

Passive Surveillance:

* The network of private veterinary practitioners that refers unusual cases to veterinary schools or State diagnostic laboratories around the United States provides an extensive informal but important surveillance system.
* USDA has trained over 250 State and Federal field veterinarians located throughout the United States in the recognition and diagnosis of foreign animal diseases, including BSE.
* The Veterinary Medical Data Base maintained by Purdue University compiles diagnoses submitted by 27 U.S. veterinary schools, including many neurological cases.
* USDA-FSIS performs antemortem slaughter inspection at all Federally-inspected slaughter establishments, and inspectors are alert for central nervous system (CNS) disorders. They also maintain a data base on these and other conditions.
* The Veterinary Diagnostic Laboratory Reporting System (VDLRS) maintains a data base on selected disease conditions submitted by 29 State and university veterinary diagnostic laboratories throughout the U.S., including the results of histologic examinations for BSE. The VDLRS is a cooperative effort of the American Association of Veterinary Laboratory Diagnosticians, the United States Animal Health Association, USDA:APHIS:VS' Centers for Epidemiology and Animal Health, and the 29 Laboratories mentioned above.
* Veterinary pathologists at zoos in the United States routinely conduct post mortem examinations on the brains of zoo animals exhibiting neurologic signs since BSE-like encephalopathies have been diagnosed in seven species of exotic Bovidae at zoos in England.

How we imported cattle from the United Kingdom?

Yes. Between 1981 and 1989, there were 496 cattle imported from the United Kingdom. These U.K. imports have been traced and there are only 25 cattle still alive in the United States (as of April 14, 1997). All of these animals have been under quarantine since April, 1996. APHIS is currently attempting to purchase these cattle for diagnostic research purposes. In July of 1989, the importation of live ruminants from the United Kingdom was banned.

Can we account for all of the U.K. imported cattle?

All but 33 animals have been traced. APHIS estimates that, based on their ages, only 9 of these 33 animals would still be alive. All cattle of unknown status would be greater than 8 years of age and would have a reduced likelihood of developing BSE at this late date.

4b. Education

What proactive initiatives are underway to educate farmers, veterinarians, extension agents, etc.?

An important part of the USDA's active surveillance program is the training of veterinary practitioners in the clinical signs, diagnosis and sample submission for BSE. Videotapes of cattle showing clinical signs of BSE have been distributed to veterinarians in Federal and State governments, veterinary diagnostic laboratories, and pathology departments of veterinary colleges. Microscope slides showing typical BSE lesions have been distributed to the above diagnostic laboratories, and Federal Foreign Animal Disease (FAD) diagnosticians have trained in Great Britain in BSE recognition. BSE fact sheets, risk assessments, and reviews have also been sent to State and Federal veterinarians, private practitioners, other industries, and to producers. In addition, APHIS personnel have given numerous presentations to various animal health groups. Finally, over 250 Federal and State veterinarians throughout the U.S. have been trained in the recognition of FADs including BSE.

4c. Prevention

What measures has USDA, APHIS taken to prevent the introduction of BSE?

To prevent BSE from entering the United States, APHIS has restricted the importation of live ruminants and certain ruminant products from countries where BSE is known to exist.

On July 21, 1989, APHIS banned the importation of all ruminants and restricted the importation of most cattle products from the United Kingdom.

On December 6, 1991, APHIS formally restricted the importation of ruminant meat and edible products and banned most byproducts of ruminant origin from countries known to have BSE (56 FR 63868, 56 FR 63869).

> Certain products cannot be imported into the United States, except under special permit for scientific, educational or research purposes, or under special conditions to be used in cosmetics. These products include serum, meat and bone meal, bone meal, blood meal, offal, fat, glands, and collagen.
>
> Importation requests for ruminant material are considered individually and authorization is granted only to those materials that would not have allowed exposure to ruminants in the United States.
>
> In addition, the regulation requires that imported meat and edible products for human or animal consumption from ruminants in the bovidae family be deboned, with visible lymphatic and nervous tissue removed; that it be obtained from animals which have undergone a veterinary examination prior to slaughter; and that it be obtained from ruminants which have not been in any country which BSE has been reported during a period of time when that country permitted the use of ruminant protein in ruminant feed.

What actions are taken at USDA federally-inspected slaughter establishments to ensure that cattle with BSE would not enter the human food supply?

All cattle presented for slaughter in the United States are inspected before slaughter by a trained inspector for signs of central nervous system impairment. Any animals exhibiting neurological signs during this inspection are condemned, and the meat is not permitted for use as human food. The brains from these animals are submitted to the USDA, National Veterinary Services Laboratories for analysis.

Have we imported beef or beef products from the United Kingdom?

FSIS reports that there have been no beef imports from the United Kingdom since 1985. Although current APHIS regulations allow for the importation of ruminant meat under certain conditions, there have not been any importations of British beef under these regulations. The regulations which went into effect in 1989 require that the meat must be deboned, with all visible lymphatic and nerve tissue removed, from animals receiving antemortem inspection and who were born after the ruminant to ruminant feed ban. Prior to 1989 other restrictions prevented the entry of beef from the United Kingdom.

Did we import beef prior to the ban in the United Kingdom?

No. Prior to 1989, there were no FSIS approved establishments eligible to ship U.K. beef to the United States. Therefore, no beef was imported.

Did the United States import any ruminant proteins (meat and bone meal) from the United Kingdom prior to the 1989 regulations?

Yes. Prior to 1986, there was a small amount (approximately 14 tons) of ruminant protein imported. No further importations in the 1980s were made. Our current regulations prohibit the importation of ruminant proteins from all countries considered to be affected with BSE.

4d. Risk Assessment

What epidemiologic and risk analyses has USDA conducted in response to the BSE outbreak?

USDA continues to analyze and report epidemiologic findings and potential risks to the United

States. In 1991, USDA issued two reports analyzing risk factors associated with BSE in the United Kingdom based on the British hypothesis of the disease occurring as a result of feeding scrapie contaminated meat and bone meal. Because of some similarities in the animal industries across the two countries the possibility of BSE occurring in the United States could not be eliminated, however, the probability of occurrence was determined to be very low as the amount of sheep offal was determined to be very low as the amount of sheep offal was determined to be 0.6% of all rendered product.

Since 1991, USDA has closely followed scientific findings and has updated the BSE risk factor analysis, first in 1993 and as recently as April 1996. Changes within each of the risk factors have been evaluated and since there has either been no change or a decrease in the magnitude of risk factors, the overall risk of scrapie-induced BSE in the United States is believed to have decreased.

What are the differences between the United States and Great Britain sheep and cattle industries?

A 1991 USDA report on the Bovine Spongiform Encephalopathy outbreak in Great Britain contrasted animal industries in the United States and Great Britain. Both countries are actively involved in the rearing of cattle and sheep however substantial differences do exist. Great Britain has approximately 40 million sheep versus 8.5 million in the United States. Conversely, Great Britain has 11.8 million cattle versus 103.8 million cattle in the United States. Thus the proportion of sheep to cattle is substantially higher in Great Britain versus the United States.

5. Feeding of Ruminant Protein to Ruminants

Does the United States feed meat and bone meal to ruminants?

Yes. The U.S. rendering industry produces ruminant derived animal protein products. However, substantial differences exist between the United States and Great Britain concerning the abundance and availability of plant based proteins as an animal feed source. The United States is a major producer and user of plant based protein products, such as soybean meal. Plant based proteins are the main protein components of complete animal feeds in the United States.

Does the United States ban the feeding of ruminant derived protein to cattle?

No. There are currently no government restrictions in the United States regarding the feeding of ruminant derived meat and bone meal to cattle. Animal feed ingredients are regulated by the Food and Drug Administration (FDA).

In 1989, as a direct result of the BSE epidemic in the United Kingdom and its putative scrapie agent etiology, the National Renderers Association and the Animal Protein Producers Industry recommended to their members a voluntary ban on rendered sheep offal for use in cattle feed. In a 1992 FDA survey, half of the sheep renderers surveyed were selling product for use in cattle feed.

A proposed FDA rule in 1994 to ban sheep and goat offal from ruminant feed was made, but was not enacted due to the costs of implementing the regulation and doubts over the risk posed (only 0.6% of rendered protein is of sheep origin).

In view of recent developments in the United Kingdom, on March 29, 1996, national livestock organizations and professional animal health organizations, supported by the USDA and the U.S. Public Health Service, called for a voluntary ban on feeding any ruminant-derived meat and bone meal to ruminants.

In the Federal Register of May 14, 1996 (61 FR 24253, Advanced Notice for Proposed Rulemaking), the FDA requested public comment and information on all aspects of TSEs, including BSE, and the potential consequences of a prohibition on the feeding of ruminant protein to ruminants. Comments received in the ANPRM were considered in the development of a proposed rule on the feeding of ruminant protein to ruminants. On January 3, 1997, the FDA published a proposed rule to prohibit the feeding of ruminant and mink proteins to ruminants. The comment period closed on February 18, 1997. On April 17, 1997, the FDA published a proposal to enact regulations which would prohibit the inclusion of certain mammalian tissues in ruminant feed.

Were the rendering processes in the United States and Great Britain similar at the onset of BSE?

Yes.

Changes in rendering practices in Great Britain may have potentiated the agent's survival in meat and bone meal. During the late 1970s and early 1980s, two critical steps were removed from the rendering process: the prolonged exposure to organic solvents to extract fat from meat and bone meal and the treatment of meat and bone meal from super-heated steam to remove the residual solvent. These procedures could have provided sufficient additional inactivation of the infective agent to keep its level in meat and bone meal below the threshold at which it could infect cattle. It is believed that this combination of factors allowed the infective agent to reach a clinical disease threshold in cattle being fed ruminant meat and bone meal.

Similar rendering practices, i.e., elimination of solvent extraction, in the U.S. have existed since the 1970s.