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Bovine Spongiform Encephalopathy: An Update

By Chris Clark, VetMB, MVetSc

The Canadian cattle industry changed on May 20th 2003. The effects of a diagnosis of bovine spongiform encephalopathy (BSE) in the Canadian cattle herd will remain for years to come. Although BSE continues to occasionally enter the news headlines, it is sporadic and there has been no attempt to critically examine what has happened over the past 4 years. This issue of *Large Animal Veterinary Rounds* reviews recent information about BSE around the world and examines some of the new scientific advances.

BSE around the world

According to the World Organization for Animal Health (OIE),¹ 25 countries around the world have reported homegrown cases of BSE and 2 countries have only found BSE in imported cattle (Table 1). The United Kingdom (UK) continues to be the world leader in BSE cases with more than 97% of the total cases. The Republic of Ireland is second with approximately 1% of cases. Despite enhanced surveillance programs, the incidence of the disease is decreasing in all countries where BSE has been identified and the decrease is thought to be due to the enforcement of ruminant feed bans. In almost every country, one of the first reported cases of BSE could be traced to an imported animal, typically from the UK.

BSE in the UK

The UK is the only country that has suffered a true epidemic of BSE (Table 2).² The first case was diagnosed in 1986. The epidemic peaked in 1992 with 36,680 cases diagnosed and, since that date, cases have steadily declined with only 33 cases confirmed this year (current to Aug 31st, 2007). In the UK, 81% of cases occurred in dairy animals and feed has always been the focus of research and control of BSE. Further, the majority of cases were identified as clinical suspects and only as the epidemic diminished has the routine testing of dead animals found significant numbers of cases.

The decline in the epidemic began 4-years after the implementation of the initial ruminant feed ban (note that the typical incubation period for BSE is 4 to 5 years) and this control measure can be credited with halting the epidemic. The feed bans in the UK and Europe have been tightened a number of times since the original ban. The most significant change occurred in 1996 when the feeding of ruminant protein to any species was banned. To date, 148 cases of BSE have occurred in animals born after the implementation of the ban; 46 of these animals were born in the year immediately following implementation.

Creutzfeldt-Jakob disease

The public perceptions of BSE are, for the most part, due to potential public health concerns associated with variant Creutzfeldt-Jakob disease (vCJD). In the 1990s, a number of commentators predicted nightmare scenarios of human infection. This has not occurred; the most recent statistics for vCJD incidence in the UK are 114 confirmed cases and 47 probable cases that were not confirmed by histopathology.³ There are currently a further 5 suspect clinical cases in the UK. To place these cases in perspective, there have been 920 sporadic cases of classic CJD in the UK since 1990 (Table 3).

Worldwide statistics for vCJD are not readily accessible, since the identification of the disease and the final diagnosis are very difficult. Data from the UK National CJD Surveillance unit are summarized in Table 4 and an additional 43 cases have been confirmed in 10 other countries.⁴ Most of these cases occurred in countries with a high number of BSE cases or in a patient who had spent a period of time



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Table 1: Total number of BSE cases by country. Data from the OIE. Some countries include data from 2007, imported cases may be classed by country of origin or country of diagnosis.

Country	Total number of BSE cases
Austria	5
Belgium	133
Canada	12
Czech Republic	26
Denmark	15
Finland	1
France	984
Germany	415
Greece	1
Ireland	1604
Israel	1
Italy	139
Japan	33
Liechtenstein	2
Luxembourg	3
Netherlands	82
Poland	55
Portugal	1029
Slovakia	23
Slovenia	8
Spain	681
Sweden	1
Switzerland	464
America	2
UK	181044
USA	2
Falkland Islands*	1
Omana	2

For more details see http://www.oie.int/eng/info/en_esbmonde.htm

* Imported cases only

living in a country with a high incidence of BSE. The true risk of BSE to human health remains unknown, but these statistics would suggest that the risk must be very low.

Diagnosing BSE

Despite much media hype, there has been no official evaluation of any live animal test for BSE. The only recognized diagnostic test is postmortem evaluation of brain tissue. A number of rapid tests using proteolytic digestion of brain material followed by either enzyme-linked immunosorbent assay (ELISA) or Western blot analysis for digestion (protease)-resistant prion protein (PrP) are currently in use. The “gold standard” for diagnosis remains immunohistochemistry (IHC) or Western blot analysis. IHC has the advantage of possibly visualizing the pathology in the brain stem. However, the Western blot can be used on samples that have lost their tissue architecture due to freezing or autolysis and also to distinguish between different strains of BSE.

BSE in Canada

When discussing BSE in Canada, it is often forgotten that the first case of BSE in Canada was actually identified in a beef cow in 1993. The cow had been imported from the UK in 1987 and it was assumed that the disease was probably con-

Table 2: Cases of BSE in the UK. Passive surveillance cases are clinically affected animals that are considered BSE suspects. Active surveillance programs include; fallen stock, cohorts of clinical cases, and animals tested at slaughter. The vast majority of the active surveillance cases are animals >30 months of age that have died on farm.

Year	Passive surveillance cases	Active surveillance cases
Pre 18/7/1988	727	
1988	2180	
1989	7133	
1990	14181	
1991	25026	
1992	36680	
1993	34370	
1994	23943	
1995	14301	
1996	8013	
1997	4310	
1998	3179	
1999	2256	18
2000	1311	44
2001	781	332
2002	445	594
2003	173	375
2004	82	227
2005	39	164
2006	15	89
2007	4	29

tracted from contaminated feed the cow received as a calf while in the UK.⁵ This assumption, coupled with the limited information available at the time about the possible transmissibility of BSE to humans, meant that the event passed with minimal impact on the Canadian cattle industry. The initial response was to identify all other British imported cattle and slaughter them. All animals having contact with the index case were also slaughtered. Since the disease cannot spread laterally, this aggressive response was somewhat excessive and because the affected animal was identified, destroyed, and the carcass disposed of appropriately, there was no risk to other animals. The only potential risk at that time would have been the off-spring of the index case or other British imported cattle.

The discovery of this case had two other effects on the management of BSE in Canada. First, there was the very real possibility that BSE had not only entered Canada but, potentially, was also circulating within the Canadian cattle herd (see below). Second, if BSE was present within Canada, pre-emptive measures could be used to ensure that the possibility of an outbreak, such as that seen in the UK, could be prevented. This was eventually achieved through the implementation of the 1997 ruminant feed ban that prohibited the feeding of mineral bone meal (MBM) derived from ruminants back to ruminants.

The spread of BSE in Canada

Any discussion of the spread of BSE in Canada includes some presumptions because complete information does not exist to absolutely confirm the hypothesis. Based on available

Table 3: British cases of human spongiform encephalopathies. (Data current to September 4, 2007) <http://www.cjd.ed.ac.uk/figures.htm>

Year	Sporadic CJD	Iatrogenic CJD	Familial CJD	GSS	vCJD	Total deaths
1990	28	5	0	0	–	33
1991	32	1	3	0	–	36
1992	45	2	5	1	–	53
1993	37	4	3	2	–	46
1994	53	1	4	3	–	61
1995	35	4	2	3	3	47
1996	40	4	2	4	10	60
1997	60	6	4	1	10	81
1998	63	3	3	2	18	89
1999	62	6	2	0	15	85
2000	50	1	2	1	28	82
2001	58	4	3	2	20	87
2002	72	0	4	1	17	94
2003	79	5	4	2	18	108
2004	51	2	4	1	9	67
2005	65	3	7	6	5	86
2006	64	1	6	3	5	79
2007	26	2	2	1	3	34
Total	920	54	60	33	161	1228

GSS = Gertmann-Straussler-Scheinker disease;
vCJD = variant Creutzfeldt-Jakob disease

records and epidemiology, we can be relatively confident about the spread of BSE within Canada. Between 1986 and 1993, 169 cattle were imported from the UK (animals were not imported prior to 1986 and after the BSE case in 1993). At the time of the 1993 case, 100 of these animals that were still alive were all slaughtered and disposed of appropriately (69 animals had either died or already been slaughtered). In tracing back to the UK, it was revealed, that of these 69 animals, 10 came from farms that had subsequently developed at least one homegrown case of BSE. In fact, 3 of the animals came from the same farm as the index case. It is assumed that at least one of these 10 animals must have been incubating BSE at the time of its death and that some of the specified risk materials (SRMs) from the carcass were rendered into MBM that was then used as a component of cattle feed for calves.

It should be noted that the majority of UK cattle imported into Canada came to Alberta. Since rendering and feed mixing are both relatively local businesses, it is reasonable to expect the majority of subsequent cases to occur within Alberta or to have a trade link to Alberta.

Canadian cases of BSE⁵

Since the imported index case in 1993, there have been 10 cases of BSE diagnosed in Canada and one case in a Canadian animal exported to the United States (US).

Case 1: Reported May 20, 2003

The first Canadian case was 6–8 years old at the time of its death in January 2003. The cow had passed through several herds during her lifetime. Genetic testing was used to try and identify or rule-out possible herds of origin. Eventually, 2,700 animals were destroyed during the investigation. The case was

Table 4: Human cases of vCJD on a worldwide basis. Data from UK National CJD Surveillance unit. <http://www.cjd.ed.ac.uk/vcjdworld.htm> (July 2007)

Country	Total number of primary vCJD cases
UK	163 (5)
France	22 (2)
Republic of Ireland	4 (1)
Italy	1 (0)
USA	3 (0)
Canada	1 (0)
Saudi Arabia	1 (1)
Japan	1 (0)
Netherlands	2 (0)
Portugal	2 (1)
Spain	1 (0)

() = number alive

reported on May 20, 2003. Undoubtedly, the delay between the death and the diagnosis created significant problems for the Canadian Food Inspection Agency (CFIA), particularly, to ensure appropriate carcass disposal. Unfortunately, the animal's carcass had been rendered and MBM had been prepared. The CFIA had to identify the eventual disposition of any contaminated feed. All subsequent cases were identified rapidly and the carcasses were immediately controlled and disposed of in an approved manner confirming that no portion of the animal entered either the human or animal feed chain. Consequently, the investigations of the CFIA focused on three specific areas:

- offspring of female animals born within 2 years of the onset of clinical signs
- animals born on the farm of origin within 12 months of the birth date of the case (birth cohorts)
- feed history of the animal within the first year of life.

These avenues of investigation were those prescribed by the OIE. Since 2003, requirements have changed somewhat; in particular, it is no longer necessary to destroy and test all cohort animals and calves.

Case 2: December 17, 2004 in Northern Alberta

The second case was a Holstein cow born October 5, 1996. The cow had been purchased and both of the two most recent calves were dead. The birth cohort comprised 135 animals of which 9 were still alive and all tested negative for BSE; 4 animals could not be traced. Feed records from the farm of origin revealed that the cow was fed a ration containing MBM in the spring of 1997 and again in the fall of 1997. Both feeds contained 15% MBM of ruminant origin and the feed given in the fall had been produced in March 1997 before the institution of the feed ban in August of that year. This feed was assumed to be the source of infection.

Case 3: January 4, 2005 in Innisfail, Alberta

The third case was a Charolais cow born March 21, 1998 on the index farm (the farm the animal was born on). One of the cow's most recent calves was already dead, the other was slaughtered and tested negative for BSE. The birth cohort contained 349 animals, of which 41 were traced and slaughtered;

3 animals were untraceable. None of the feed given to the animal contained prohibited material; however, the potential exists that improper flushing procedures were used that allowed contaminated materials to enter another feed preparation leading to infection.

Case 4: January 12, 2006 in north central Alberta

A Holstein-Hereford cow born April 15, 2000 on the index farm was case 4. One of its calves was dead and the other traced and slaughtered. The birth cohort consisted of 156 animals, of which 38 were alive and all tested negative. The entire cohort was accounted for. As in the previous case, no prohibited material was fed to the animal as a calf; however, the potential exists for contamination of prepared feed products following the failure of standard operating procedures (SOPs) for segregation or flushing.

Case 5: April 8, 2006 in the Fraser Valley, British Columbia

Case 5 was a Holstein cow born April 29, 2000 on a different farm. Both calves of this cow were already dead. The birth cohort consisted of 146 animals, of which 23 were alive and 33 were untraceable. The feed history was similar to previous descriptions, with no obvious source of MBM in the diet, but the existence of several routes through which cross-contamination may have occurred.

Case 6: June 15, 2006 in Manitoba

A commercial beef cow approximately 16-years-old was case 6. This case differed from previous Canadian isolates of BSE, since it was the so-called H-type of BSE previously identified in Europe (see below). The two most recent calves from this cow had both entered the feedlot and were presumed to be dead. The source farm could not be identified and, therefore, the cohort could not be traced. The age of the cow meant that exposure to ruminant MBM in feed was possible.

It should be noted that, at the time of this case, the CFIA policy regarding tracing associated with BSE cases was altered in response to the change in OIE recommendations. In particular, it was no longer necessary to trace and slaughter the offspring of a positive female.

Case 7: July 2, 2006 in Northern Alberta

Case 7 was a Holstein cow born April 22, 2002 on the index farm. Only one calf had been produced. The birth cohort consisted of 172 animals, of which 38 were still alive (8 animals were untraceable).

Case 8: August 9, 2006 in Northern Alberta

A Charolais crossbred beef cow approximately 10-years-old was case 7. The animal had been purchased at a sale and the origin farm could not be identified. Feed tracing was also not possible, but the animal was likely born before the implementation of the 1997 feed ban.

Case 9: January 20-22, 2007 in Northern Alberta

Case 9 was an Angus bull born in the spring of 2000 on the index farm. The birth cohort consisted of 536 animals of which 64 remain alive and quarantined on the original farm (42 were untraceable).

Case 10: April 24, 2007 in the Fraser Valley, British Columbia

A dairy cow born November 10, 2001 on the index farm was case 10. The birth cohort consisted of 156 animals of which 41 were alive; 5 have been destroyed and the remainder are currently quarantined (23 animals were untraceable). Feed used on the dairy farm came from a feedmill that also prepared feeds for nonruminants containing ruminant MBM. The potential for cross-contamination cannot be ruled out.

Cases of BSE in the United States⁶

Case 1: December 9, 2003 in Washington State

The first case was a Holstein cow born April 9, 1997 on a farm in Alberta. The herd of 113 animals was dispersed in September 2001. The birth cohort consisted of 57 animals, of which 25 were exported to the US, 3 animals remained in Alberta, and 2 were untraceable. The trace was complicated by the fact that there was a second group of 57 animals from a separate operation fed the same feed. They were on the farm, 2 were untraceable and 4 animals remain in a group of 150, but were unidentifiable. The entire group was tagged for close surveillance and follow-up.

The US identified 255 animals of interest, including 28 that were directly associated with the importation of the BSE case. All animals were destroyed and they tested negative for BSE. An investigation of the feeding practices on the original Canadian herd revealed the use of a dairy ration until June 1997 that contained ruminant MBM. This is presumed to be the source of the infection.

Case 2: November 15, 2004 in Texas (reported June 10, 2005)

The animal was a Brahman cross, approximately 12-years-old that had been born and raised on the index farm; 67 animals were identified as being in the cohort on the farm. A further 200 animals had left the farm and 20 were considered untraceable. Given the age of the animal, it is considered likely that the animal was infected through feed prior to the implementation of the ruminant feed ban in 1997. The strain of BSE affecting this animal has since been classified as H-type (see below)

Case 3: February 28, 2006 in Alabama

This case was approximately 10-years-old based upon dentition and was a crossbred cow. The cow had been on the index farm for approximately 2 years. One calf was alive and a calf from the previous year had died.

The lack of a definitive identification (ID) meant that the cow's origins could not be traced, although 35 farms were investigated. This case was also found to be due to the H-type of BSE.

The effectiveness of feed bans in controlling BSE

All the scientific evidence points to the fact that BSE was spread in MBM as a result of the infectious agent's ability to survive the temperatures and pressures that are typically used in the rendering process. Theoretically, a feed ban should be all that is required to control BSE; however, of the 10 plus 1 domestic cases of BSE seen in Canada, 7 were born after the introduction of the August 1997 feed ban. BSE is still an exceptionally rare disease in Canada and it is hard to assess the impact of the disease and the control measures implemented in this country. It is more instructive to examine the British situation.

There have actually been 3 separate feed bans introduced in the UK. The first was introduced in 1988 and was a simple ban prohibiting the use of ruminant protein in ruminant feed. This feed ban was credited with the decrease in the epidemic from 1993 onward; however, the feed ban was certainly not 100% effective. Analysis of the BSE cases by date of birth reveals that 45,290 cases have been born since the introduction of that first feed ban. Half of those animals were born in the 18 months following the introduction of the feed ban.

The explanation of the failure of the feed ban to completely control BSE actually relates to two interconnected issues. Recent studies have now demonstrated that infection can occur when young calves are experimentally dosed orally with 1 mg of brain material from an animal that died of BSE. This is an incredibly low dose.

For a number of months after the introduction of the feed ban, there was almost certainly feed still in circulation that was prepared before the ban. Although the use of ruminant protein was banned in ruminant feed, all portions of cattle carcasses could still be rendered to MBM for inclusion in poultry and swine rations. It is likely that most rendering plants continued to process ruminant and nonruminant carcasses, and that feed mills continued to use both MBM from ruminants and non-ruminants. Although the streams used in the preparation and processing of the materials were separated, it seems likely that some degree of cross-contamination did occur due to a lack of SOPs. It is possible that insufficient flushing of equipment between batches allowed residue to pass from one batch to another. Given the low infectious dose for BSE, only minimal contamination would be required to potentially infect a calf.

A more substantial feed ban was introduced in 1990. This feed ban ensured that tissues of the carcass most closely associated with BSE infectivity (now termed SRMs) had to be removed from all carcasses prior to processing. This is very similar to the enhanced feed ban introduced in Canada in July 2007. It is difficult to

appreciate the effect of this feed ban on the number of BSE cases in the epidemic curve. There was no marked reduction in the number of cases in 1995; however, if you look at the birth date of BSE cases, there is a reduction in the number of cases born after 1990. To date, there have been 20,000 cases in cattle born since 1990.

The final feed ban was introduced in 1996 following the evidence that linked BSE and vCJD. This was a complete mammalian feed ban in which no protein derived from mammals could be used in the production of any animal feeds. Once again, it is hard to see a direct effect in the number of BSE cases on the epidemic curve; however, the effect on the number of cases born after the introduction of the ban is very obvious. Thus far, only 148 animals born after the feed ban have been affected. Explaining these cases is difficult, if one fully accepts the contaminated feed hypothesis for the mechanism of BSE transmission. Most of the cases were born immediately following the introduction of the 1996 feed ban. The possibility of residual contaminated feed exists. It is also possible that trace amounts of contaminated feed were left in feed bins, etc. that carried over into other batches and were able to cause disease, given the small infectious dose. When considering such small details, it is easy to forget the larger picture. Specifically, the British epidemic is very obviously coming to an end and this outbreak was certainly controlled by the introduction of feed bans.

Making direct comparisons with the Canadian situation is not easy. Feeding rendered MBM to Canadian animals, especially beef animals, was not as prevalent as in the UK. The feed ban introduced in Canada in 1997 was equivalent to the first British feed ban (1988), but was introduced as a preventive measure, not an epidemic control measure. It is likely that a feed ban of any type will reduce the significance of an epidemic. The comprehensive scope of the feed ban will simply be proportional to how fast the disease can be eliminated.

Can BSE be eradicated?

In theory, since BSE is known to spread through contaminated feed, an effective feed ban should be all that is required to eradicate the disease. Unfortunately, we do not know where BSE originated and without that knowledge, it is not possible to be sure that the disease can be eradicated.

One of the most likely scenarios to explain the origins of BSE is that it originated as a naturally-occurring transmissible spongiform encephalopathy (TSE) of cattle in the same way that classic CJD is found in humans. The disease is thought to occur when the naturally-occurring prion protein (PrP) is randomly refolded in an alternative tertiary structure. This process results in an abnormal, but very stable structure that is able to catalyze the refolding of other PrP, which accumulate within cells. If the disease did occur naturally, it would have been extremely rare and unlikely to ever be diagnosed in cattle.

It is assumed that the cycle of rendering and feeding MBM to dairy cows in the UK artificially amplified the disease in the cattle population. It is also possible that repeated passage of PrP in cattle may have resulted in a more virulent strain of infection with a much shorter incubation period (the same effect is seen in laboratory strains of mice that are repeatedly infected with any type of spongiform encephalopathy).

If BSE is a naturally-occurring disease, it will not be possible to completely eradicate the disease, but control measures should prevent amplification in the future. Until recently, all cases of BSE appeared to be the same strain. This is unusual, since TSEs in other species often have more than one strain. Strains can be distinguished based on a number of features through the use of Western blot analysis and the use of specific antibodies to detect portions of the PrP, as well as the different glycosylated isoforms. It is also possible to biotype TSEs based on the incubation period and the characterizations of the pathology in species of inbred mice.

Recently, two atypical strains of BSE have been identified.⁷ The first strain is classified as L-type and is unglycosylated with a lower molecular mass. This form has been found in Italy, Japan, Belgium, and Germany. The H-type has a higher molecular mass and has been found in France, Germany, the US, and Canada. In addition, a retrospective analysis of all British BSE cases identified one case of H-type BSE that died in 2005.

The atypical strains of BSE are most commonly found in older animals. The pattern of pathology in the brain appears to be different and the disease in laboratory animals is different from classic BSE. Recent research has demonstrated that the characteristics of these atypical strains of BSE are preserved after passage through experimental animals. The significance of these atypical strains is currently unknown and is an area of ongoing investigation.

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Dr. Clark has stated that he has no disclosures to announce in association with the contents of this issue.

Abstract of Interest

Case-control study on feed risk factors for BSE cases born after the feed ban in France.

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In France, after the ban on meat and bone meal (MBM) in cattle feeding in June 1990, cases of Bovine Spongiform Encephalopathy (BSE) have continued to be detected in bovines born after that ban (called BAB cases). A case-control study was therefore carried out to determine the way these cases were contaminated. A multivariate conditional model was built adjusting for the production type of the animals and taking into account the herd size. The results confirmed that feeding cattle with proprietary concentrates was at risk for BSE, with an adjusted odds ratio of 6.8 (2.5; 18.7) for the consumption of less or three different proprietary concentrates and 17.6 (5.7; 54.8) for more than three, when comparing with no consumption of proprietary concentrates, considering feeding of bovines before the age of two. The results suggest that cross-contaminations by MBM in bovine concentrates have occurred after 1990. To a lesser extent, on-farm cross-contaminations, i.e. consumption by cattle of feedstuffs initially dedicated to other animals and which could legally contain MBM, have probably also existed, since the presence on farms of poultry fed purchased feed involved an increased risk of BSE with an odds ratio of 1.8 (1.1; 3.0). The use of milk replacers, which often incorporates animal fats, was also at risk with an odds ratio of 1.8 (1.0; 3.1).

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