

IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF MONTANA
BILLINGS DIVISION

RANCHERS CATTLEMEN ACTION LEGAL FUND)
UNITED STOCKGROWERS OF AMERICA,)
)
Plaintiff,)
)
)
v.)
)
)
UNITED STATES DEPARTMENT)
OF AGRICULTURE,)
ANIMAL AND PLANT HEALTH INSPECTION)
SERVICE, et al.,)
)
Defendants.)

Cause No. CV-05-
06-BLG-RFC

SECOND DECLARATION OF WILLIAM D. HUESTON, GUY H. LONERAGAN,
AND SRINAND SREEVATSAN

We, William D. Hueston, Guy H. Loneragan, and Srinand Sreevatsan, declare and state the following:

1.1. We have previously submitted a declaration, which included a description of our qualifications and expertise. We will not repeat them herein.

1.2. We have reviewed the Plaintiff's Reply Memorandum of Points in Support of Plaintiff's Motion for Summary Judgment and Opposition to Defendants' Cross-Motion 28 June, 2005 (hereafter 'Reply Memorandum'), and the separate Declarations of Drs. Prusiner, Weaver, Cox, and Charnley filed on 28 June, 2005.

1.3. The purpose of our declaration is to correct specific errors and clarify misconceptions included in the documents prepared by the plaintiff or their declarants. These errors and misconceptions form the foundation of the plaintiff's flawed argument that the Final Rule establishing criteria for regions to be classified as Minimal Risk and, thereby, allowing importation of Canadian cattle less than 30 months of age into the USA, represents anything but minimal risk.

2. Canada and the USA have implemented science-based practices that effectively protect human and animal health from BSE exposure

2.1. BSE is a disease that has been extensively studied since its recognition as a clinical entity in 1986. A wealth of collective knowledge and experience exists worldwide concerning the causative agent, transmission, pathogenesis, and epidemiology of BSE. Most importantly, science-based strategies are available that effectively manage risk associated with BSE and thereby protect animal and human populations from BSE

exposure.¹ The science underpinning these mitigation strategies and their validated efficaciousness has been widely documented in scientific manuscripts, technical abstracts and presentations, governmental reports, and scientific opinions; the accumulated empirical data from experimental and epidemiological studies form the foundation of government rules and regulations. Effective and appropriately measured protective practices have been implemented in both Canada and the USA. These practices have been designed to effectively manage BSE risk and are affirmed by national and international organizations responsible for animal and human health such as USDA:APHIS, USDA:FSIS, CDC, FDA, OIE, and WHO.

2.2. Risk of BSE exposure to the cattle population is effectively managed by feed bans because the primary, if not exclusive way, cattle are infected is through consumption of contaminated feed. (AR 8366, 9770). Empirical data from the UK support the dramatic effectiveness of feed bans in protecting animal health.² In the UK, for example, the number of confirmed cases born in the first full year after the ban (1989) was approximately 65% lower than the number of cases confirmed in cattle born in the year immediately prior to the ban (1987).³ Furthermore, the number of confirmed BSE cases born in 1990 was 84% lower than the number of cases born in 1987. The profound

¹ World Health Organization, Understanding the BSE Threat, October, 2002, WHO/CDS/CSR/EPH/2002.6. Available at <http://www.who.int/csr/resources/publications/whocdscsreph20026/en/>.

² Brown et. al. Bovine Spongiform Encephalopathy and Variant Creutzfeldt-Jakob Disease: Background, Evolution, and Current Concerns. *Emerg. Infect. Dis.* 2001;7:6-16. AR11635; USDA:APHIS. Analysis of Risk—Update for the Final Rule: Bovine Spongiform Encephalopathy; Minimal Risk Regions and Importation of Commodities, December, 2004, AR08334.

³ Department for Environment, Food and Rural Affairs (DEFRA) available at <http://www.defra.gov.uk/animalh/bse/statistics/incidence.html>.

protective effect of the UK feed ban was plainly evident even though an overwhelming level of circulating infectivity existed at the time of implementation, and there were challenges with both producer compliance to and enforcement of the feed ban. (AR 8094, 8327, 8334, 12935).

2.3. Science-based practices, such as removing SRMs from the food supply, effectively manage risk of BSE exposure to humans because BSE infectivity has a limited and well-defined distribution in cattle.⁴ (AR 9965). Put simply, when tissues containing infectivity are prevented from entering the human food supply, public health is protected from BSE exposure. SRM removal adequately protects public health; empirical data from the UK support this claim as vCJD cases have declined subsequent to removal of SRMs from the human food supply.

3. TSE diseases of animals and humans differ in terms of their means of transmission, host range, genetic susceptibility, and other factors

⁴ USDA:APHIS. Analysis of Risk–Update for the Final Rule: Bovine Spongiform Encephalopathy: Minimal Risk Regions and Importation of Commodities, December, 2004, AR08333; European Union, Scientific Steering Committee. Opinion on TSE Infectivity Distribution in Ruminant Tissues (State of Knowledge, December 2001). Adopted January 10-11, 2002 AR11884; Comer and Huntly. Exposure of the human population to BSE infectivity over the course of the BSE epidemic in Great Britain and the impact of changes to the Over Thirty Month Rule. Over Thirty Month Rule (OTMR) review paper. 2003. Available at: <http://www.food.gov.uk/multimedia/pdfs/otmcomer.pdf>.

3.1. A variety of differing phenotypes (e.g., Scrapie agent, BSE agent, chronic wasting disease (CWD) agent) and hosts (e.g., mice, hamsters, sheep, cattle, deer, monkeys) have been used in the extensive research of transmissible spongiform encephalopathies. It has become increasingly evident that their distinctive characteristics vary markedly depending on the specific phenotype-host combination. For instance:

3.1.1. CWD is contagious in deer and elk⁵ whereas BSE is not contagious in cattle;

3.1.2. Fatal familial insomnia (FFI) and Gerstmann-Straussler-Scheinker disease (GSS) are heritable diseases of people whereas BSE in cattle is not heritable;⁶

3.1.3. Certain genotypes of sheep are more susceptible to Scrapie than others whereas no clear genotype predilection to BSE in cattle has been documented;⁷

3.1.4. Prions have been found in the muscles of laboratory animals (mice and hamsters) infected with Scrapie whereas BSE has never been found in muscles of cattle.⁸

⁵ Miller and Williams. Prion disease: horizontal prion transmission in mule deer. *Nature*. 2003;425:35-6.

⁶ Wijeratne et al. A study of the inheritance of susceptibility to bovine spongiform encephalopathy. 1990. *Vet Rec*. 1990;126:5-8.

⁷ Hunter et al. Frequencies of PrP gene variants in healthy cattle and cattle with BSE in Scotland. *Vet Rec*. 1994 ;135 :400-3.

⁸ Bosque et al. Prions in skeletal muscle. *Proc. Natl. Acad. Sci. U.S.A.* 2002;3812-3817; Jeffrey and Gonzalez. Pathology and pathogenesis of bovine spongiform encephalopathy and scrapie. In: Mad cow disease and related spongiform encephalopathies. Ed: Harris,

3.2. Consequently, Dr. Prusiner's statement that 'there is no reason to believe that BSE prions in cattle behave differently from those in other mammals' (Declaration of Dr. Prusiner, 28 June, 2005, p6) is inconsistent with the current scientific knowledge of transmissible spongiform encephalopathies. Direct inferential generalization from research using Scrapie in mice or even vCJD in humans to BSE in cattle is scientifically unsound.

4. SRM removal protects human health; BSE infectivity has not been documented in bovine muscle tissue or blood

4.1. Contrary to the plaintiff's claims, all tissues with any documented potential to contain BSE infectivity, regardless of the level, are included as specified risk material⁹ as described in the Final Rule (p461 AR08045). SRM removal effectively protects public health from exposure to BSE infectivity. From a peak of 28 vCJD cases in the UK in 2000 (Figure 1), there were only 9 cases in 2004 and only 2 cases so far this year (to July 1, 2005¹⁰). While not identical, the UK control measures include practices similar to the

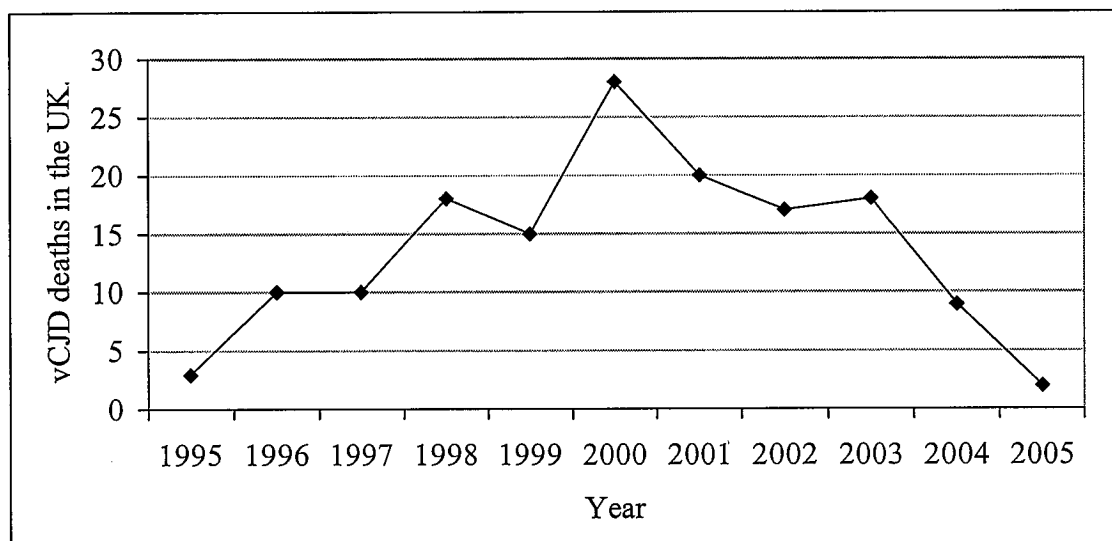
D. Springer, New York. 2004. pp 65-97; Wells, et al., Pathogenesis of experimental bovine spongiform encephalopathy: preclinical infectivity in tonsil and observations on the distribution of lingual tonsil in slaughtered cattle. *Vet. Rec.* 2005;401-7.

⁹ SRM; FSIS: Part V, 9 CFR Part 301, 309, et al. Interim Final Rules and Notice AR09958-09990.

¹⁰ <http://www.cjd.ed.ac.uk/figures.htm>.

USA's SRM rule;¹¹ these control strategies helped to limit the scope of the human epidemic. By selectively ignoring the experience of BSE and vCJD risk management in Europe and the UK in particular, the plaintiff infers these controls do not afford *any* protection for consumers and would have the USA needlessly 'wait years' (Reply Memorandum, 28 June, 2005, p5) to demonstrate the 'effectiveness [of SRM removal] in preventing vCJD in humans, or even the usefulness of the feed regulations in controlling the spread of BSE in cattle'.

Figure 1. Number of vCJD deaths in the UK for 1995 through 29 April, 2005.



Source of data: The National Creutzfeldt-Jakob Disease Surveillance Unit, Edinburgh, UK. Available at <http://www.cjd.ed.ac.uk/index.htm>

4.2. The plaintiff chooses to ignore substantial scientific data by claiming that current SRM-removal practices do not adequately protect public health (Reply Memorandum, 28

¹¹ Prohibition of tissue with documented potential to contain infectivity, prohibition of specific stunning techniques and mechanically separated beef recovery, antemortem inspections preventing diseased or downer animals entering the food supply, and the feed ban.

June, 2005, p12). For example, they contend that consumers may be exposed to BSE because residual tonsillar tissue has been found in some tongues. This grossly misrepresents data. While traces of infectivity were detected in *palatine* tonsils,¹² there is *no* documented evidence for the presence of BSE infectivity in residual lymphoid tissue in the tongue.

4.3. The plaintiff incorrectly states that USDA's argument is based on 'series of speculative, and often counterintuitive, assumptions' (Reply Memorandum, 28 June, 2005, p8). Despite extensive research, BSE has never been documented in bovine muscle tissue or blood; all tissues with demonstrated infectivity are included in the list of SRM in the Final Rule (p461 AR08045, 0808, 8368, 12941), and empirical evidence from the EU supports the effectiveness of SRM removal. To assume otherwise, as the plaintiff and their declarants do, is speculative and contradicts current scientific understanding.

5. BSE testing is for surveillance purposes; it does not protect public health

5.1. Dr. Prusiner's speculation that BSE is in bovine muscle tissue of affected animals because of findings in other species runs counter to the extensive scientific research of BSE in cattle.¹³ Dr. Prusiner extends his circular logic by stating that 'testing all

¹² Wells, et al., Pathogenesis of experimental bovine spongiform encephalopathy: preclinical infectivity in tonsil and observations on the distribution of lingual tonsil in slaughtered cattle. *Vet. Rec.* 2005;401-7.

¹³ European Union, Scientific Steering Committee. Opinion on TSE Infectivity Distribution in Ruminant Tissues (State of Knowledge, December 2001). Adopted

slaughtered animals is the *only* rational policy for adequately protecting the human food supply' (Declaration of Dr. Prusiner, 28 June, 2005, p9 emphasis added). This is just not so. BSE infectivity has not been documented in bovine muscle or blood and as described above, SRM removal effectively protects public health.¹⁴ Moreover, blanket testing of healthy slaughter cattle has never been recommended by the OIE and is seen as the least effective means of surveillance. (AR 8059).

5.2. Dr. Prusiner never contends that beef from Canadian cattle inherently possesses a greater BSE risk than beef from US cattle. In fact, Dr. Prusiner fails to mention Canada or the Final Rule in his entire declaration. The primary recommendation from his declaration is to immediately begin 'testing all slaughtered' cattle (Declaration of Dr. Prusiner, 28 June, 2005, p9). It is unclear to us if the plaintiff even realizes that Dr. Prusiner's recommendation is inclusive of cattle born, raised, and slaughtered in the USA regardless of age and would even include veal calves.¹⁵ Only Japan currently tests all slaughter cattle and their decision to do so was not based on science but rather to restore consumer confidence in the safety of beef. The substantial epidemiological data are clear: testing of healthy cattle less than 30 months of age is widely recognized as the most

January 10-11, 2002 AR11884; Wells, et al., Pathogenesis of experimental bovine spongiform encephalopathy: preclinical infectivity in tonsil and observations on the distribution of lingual tonsil in slaughtered cattle. *Vet. Rec.* 2005;401-7.

¹⁴ Bradley. BSE transmission studies with particular reference to blood. *Dev. Biol. Stand.* 1999;99:35-40; Wells, et al., Pathogenesis of experimental bovine spongiform encephalopathy: preclinical infectivity in tonsil and observations on the distribution of lingual tonsil in slaughtered cattle. *Vet. Rec.* 2005;401-407; European Union, Scientific Steering Committee. Opinion on TSE Infectivity Distribution in Ruminant Tissues (State of Knowledge, December 2001). Adopted January 10-11, 2002 AR11884.

¹⁵ InPro Biotechnology, Inc., News Release - *InPro Biotechnology, Inc. Urges Immediate BSE Testing in All U.S. Cattle*, 24 June, 2005, Attachment A.

grossly inefficient strategy to find BSE. The OIE takes a strong position by stating that testing healthy cattle less than 36 months of age is of 'relatively very little value'.¹⁶ Testing does not protect public health; SRM removal is the major factor protecting consumers from BSE exposure. (AR 8394)

6. Humans are substantially less susceptible to BSE than cattle

6.1. It is unclear what the plaintiff and their declarants believe concerning the species barrier that renders humans less susceptible to the BSE agent than cattle. Dr. Cox states that 'some [consumers] probably are' protected by a formidable species barrier (Declaration of Dr. Cox, 28 June, 2005, p43). Yet the plaintiff infers it is prudent for the USDA to ignore the most likely outcome, which is derived from the available empirical and scientific data, i.e., a substantial species barrier exists, and assume a 'worst-case', one-to-one level of susceptibility (Reply Memorandum, 28 June, 2005, p13).

6.2. Dr. Prusiner suggests it is 'not sufficient to establish there is a species barrier' by comparing the magnitudes of the vCJD and BSE epidemics in the UK (Declaration of Dr. Prusiner, 28 June, 2005, p5). He makes this claim based on 1) speculation that the incubation period for vCJD may be longer than empirical data indicate; 2) speculation that vCJD is under-diagnosed; and 3) an apparent disregard for statistically-derived estimates quantifying the amount of infective tissue to which the UK population was

¹⁶ OIE Terrestrial Animal Health Code, Article 2.3.13.4.2. Appendix 3.8.4.2.4.

exposed. The plaintiff extols Dr. Prusiner's position and states that a human species barrier 'has not been proven' (Reply Memorandum, 28 June, 2005, p13). Since it is unethical to perform human challenge studies, absolute proof can never be established to satisfy everyone. In the absence of human studies, we must rely on empirical and scientific data; these data point to a substantial species barrier. (AR 8046, 8089). It is believed millions of infected cattle entered the human food supply in the UK prior to removal of SRMs (ban on Specified Bovine Offals for human consumption, 1989/1990); based on empirical data, it has been estimated that 54 million cattle infectious doses¹⁷ were consumed by the UK public.¹⁸ This tremendous exposure has resulted in a total of 150 vCJD deaths to date and the number of new cases per year has diminished substantially. When one considers the magnitude of the human exposure and the limited scope of the human epidemic, a striking discrepancy is obvious.

6.3. Dr. Prusiner provides an interesting perspective: the USDA should assume that 'one infectious unit (or dose) of prions is sufficient to cause vCJD in humans' (Declaration of Dr. Prusiner, 28 June, 2005, p5). It should be clarified that based on empirical and scientific data, the *infectious dose* required to successfully cause vCJD in humans is substantially greater than the *infectious dose* required to cause BSE in cattle. (AR 8383).

¹⁷ A 'cattle infectious dose' represents the dose required to infect a bovine 50% of the time (Comer and Huntley, Exposure of the human population to BSE infectivity over the course of the BSE epidemic in Great Britain and the impact of changes to the Over Thirty Month Rule. *J. Risk. Res.* 2004;523-543).

¹⁸ Comer and Huntly. Exposure of the human population to BSE infectivity over the course of the BSE epidemic in Great Britain and the impact of changes to the Over Thirty Month Rule. Over Thirty Month Rule (OTMR) review paper. 2003. Available at: <http://www.food.gov.uk/multimedia/pdfs/otmcomer.pdf>.

In other words, the data indicate humans are substantially less susceptible to oral challenges of the BSE agent than cattle.

7. The Canadian surveillance program is designed appropriately and includes sufficient animals to detect BSE and demonstrate BSE is rare in Canada

7.1. The surveillance programs of both the USA and Canada are principally designed to detect BSE within their adult populations at a given prevalence assuming the majority of cases would be in the high-risk population. (AR 9473). The USA has an estimated high-risk population of approximately 450,000 (roughly 1.0% of the adult population) whereas Canada's high-risk population is estimated to be 80,000 (roughly 1.2% of the adult population). To detect at least one of the expected cases present in the high-risk population within the USA, at least 268,500 samples need to be tested.¹⁹ Canada has appropriately defined their high-risk population,²⁰ estimated the number of BSE cases present within it, and calculated a statistically appropriate sample size sufficient to detect at least one of these cases when present within their high-risk population. The sample size does not represent an absolute number²¹ or fixed proportion of any population but rather a statistically sufficient and efficient sample of the targeted (high-risk) population

¹⁹ USDA:APHIS. Bovine Spongiform Encephalopathy (BSE) Surveillance Plan, March, 2004 AR09473.

²⁰ Canadian Food Inspection Agency, BSE enhanced surveillance program AR09557.

²¹ The sample size clearly cannot be an absolute number as it would be impossible for Canada to sample 268,500 high-risk animals per year if there are only 80,000 high-risk animals available.

to detect BSE.²² Canada, like the USA, has substantially exceeded their stated targets and both programs provide excellent sensitivity to detect BSE.

7.2. In the 12 months to June 30, 2005, Canada has tested 53,243 high-risk animals and detected 2 cases.²³ This provides a surveillance yield of $2/53,243 = 0.38$ confirmed BSE cases per 10,000 tests performed on high-risk animals, i.e., those animals most likely to have BSE. The surveillance yield among high-risk cattle within the EU is at least 15 to 20 times greater than Canada's; in 2002 and 2003 the EU's high-risk surveillance yield was 9.2 and 6.0 confirmed BSE cases per 10,000 tests, respectively.²⁴ Moreover, all Canadian cases were restricted to cohorts born around the time of the feed ban and exposed to feed produced prior to or shortly after the ban. While additional details on the ages and geographic distribution of cattle tested in the surveillance program would further clarify the situation, the finding that all the positive Canadian cattle are tightly clustered within animals born between 1996 and 1998 suggests that age-specific BSE prevalence differs, with those cattle born around the time of the feed regulation implementation being the most highly exposed, and consequently, the most affected. Empirical data from the European surveillance documents decreasing proportions of BSE test-positive cattle in birth-cohorts born after the implementation of the feed regulations.

²² Cannon and Roe. Livestock disease surveys. A field manual for veterinarians. Bureau of Range Science, Department of Primary Industry. Australian Government Publishing Service. 1982.

²³ Canadian Food Inspection Agency, available at <http://www.inspection.gc.ca/english/animas/heasan/disemala/bseesb/surv/surve.shtml#num>.

²⁴ European Commission. Report on the monitoring and testing of ruminants for the presence of transmissible spongiform encephalopathy (TSE) in the EU in 2003. May 2004.

7.3. As stated in our previous declaration (Declaration of Drs. Hueston, Loneragan, and Sreevatsan, 10 June, 2005, p31), the Canadian surveillance program has exceeded the previous OIE recommendations since 1996.²⁵ The OIE recently changed their surveillance guidelines to reflect increased epidemiological understanding of the distribution of BSE in cattle populations.²⁶ Samples are now assigned ‘points’ based on their epidemiological value to surveillance.²⁷ For example, a sample collected from a 4- to 7-year-old *Casualty Slaughter*²⁸ animal is worth 1.6 points whereas a sample collected from a healthy slaughter animal less than 2 years old is valued at 0.01 points (Table 1). This implies that BSE is 160-fold more likely to be detected in a sample from the former type of animal than the latter type of animal. Using the OIE values from Table 1, tests performed on a 5-year-old *Clinical Suspect*²⁹ provides an equivalent epidemiological value to a surveillance program as tests performed on 75,000 healthy yearling cattle at routine slaughter. By extension, testing 25 to 26 5-year-old *Clinical Suspects* provides as much surveillance value as testing approximately 1.9 million healthy slaughter yearlings. The assignment of points, or surveillance value, represents the current extensive epidemiological understanding of BSE population dynamics.

²⁵ OIE Terrestrial Animal Health Code, Article 2.3.13. Appendix 3.8.4.2. AR10077.

²⁶ OIE Terrestrial Animal Health Code, Article 2.3.13. Appendix 3.8.4.

²⁷ In other words, samples are assigned points relative to the likelihood of being BSE positive.

²⁸ ‘Cattle over 30 months of age that are non-ambulatory, recumbent, unable to rise or to walk without assistance; cattle over 30 months of age sent for emergency slaughter or condemned at ante-mortem inspection (casualty or emergency slaughter, or downer cattle’; OIE Terrestrial Animal Health Code, Article 2.3.13. Appendix 3.8.4.2.2.)

²⁹ ‘Cattle over 30 months of age displaying behavioural or clinical signs consistent with BSE’ (OIE Terrestrial Animal Health Code, Article 2.3.13. Appendix 3.8.4.2.1.)

7.4. For a country such as Canada, the OIE-assigned target is to accumulate 300,000 points over a maximum of 7 consecutive years. Based on the new OIE surveillance guidelines, Dr. Cox correctly calculates Canada can accumulate sufficient surveillance points (i.e., 300,000) by sampling 187,500 *Casualty Slaughter* animals that are 4 to 7 years of age (i.e., $300,000/1.6 = 187,500$).³⁰ However, the Canadian surveillance program also includes *Fallen Stock*³¹ and *Clinical Suspects*.³² It would be equally true that Canada could achieve its surveillance points target by sampling 400 *Clinical Suspects* between 4 and 7 years of age (i.e., $300,000 \text{ points}/750 \text{ points per animal}=400$; Table 1). The OIE suggests that countries should derive their surveillance sample from at least 3 of the 4 cattle subpopulations, a practice that Canada has implemented.³²

Table 1. Surveillance point values for samples collected from animals in the given subpopulation and age category.

Age	Surveillance subpopulation			
	Routine Slaughter	Fallen Stock	Casualty Slaughter	Clinical Suspect
≥ 1 year and < 2 years	0.01	0.2	0.4	-
≥ 2 years and < 4 years	0.1	0.2	0.4	260
≥ 4 years and < 7 years	0.2	0.9	1.6	750
≥ 7 years and < 9 years	0.1	0.4	0.7	220
≥ 9 years	0.0	0.1	0.2	45

Table adapted from OIE Terrestrial Animal Health Code, Article 2.3.13. Appendix 3.8.4.4.2.

³⁰ In the declaration of Dr. Cox, he states that Canada can achieve 300,000 surveillance points by sampling '187,500 consecutive targeted cattle' (Declaration of Dr. Cox, 28 June 2005, p24). It is unclear what Dr. Cox means by 'consecutive'. Nowhere does the OIE recommend testing consecutive cattle. Statistical sampling infers that each animal has a probability less than 1.0 of being sampled. As such, it would appear that Dr. Cox is advocating a census approach to sampling as desired by Dr. Prusiner, an approach not scientifically justifiable.

³¹ 'Cattle over 30 months of age which are found dead on farm, during transport or at an abattoir' (OIE Terrestrial Animal Health Code, Article 2.3.13. Appendix 3.8.4.2.3.)

³² Canadian Food Inspection Agency, BSE enhanced surveillance program AR09557.

7.5. Dr. Weaver misinterprets OIE surveillance guidelines and the number of samples proposed by Dr. Cox. Dr. Weaver states ‘[t]he criterion requires a country with Canada’s demographics to test about 187,000 cattle *per year*’ (Attachment A of the Declaration of Dr. Weaver, 28 June, 2005, p5 emphasis added). This is simply incorrect. The OIE Code plainly states that ‘[t]he total points for samples collected may be accumulated over a period of a maximum of 7 consecutive years to achieve the target number of points.’³³ Dr. Weaver also chooses to ignore the value assigned to other subpopulations of animals and that Canada’s high-risk population includes *Clinical Suspects* and *Fallen Stock*. Prior to 2001, the Canadian surveillance program focused exclusively on *Clinical Suspects* (Noel Murray, Personal communication 5 July, 2005³⁴). For the 3 years prior to 2001, 2,841 animals were tested for BSE. Since samples from *Clinical Suspects* are assigned 220 to 750 points, these tests account for at least 625,020 points (2841 samples*220 points per sample; Table 1) but potentially as many as 2,130,750 points (2841 samples*750 points per sample).

7.6. As the Canadian program expanded to include *Casualty Slaughter* and *Fallen Stock* categories, *Clinical Suspects* also continued to be tested. For example, in 2002, Canada tested 3,377 high-risk animals³⁵ including 451 *Clinical Suspects*. Based on the current OIE recommendations, these samples from *Clinical Suspects* alone potentially accounted

³³ OIE Terrestrial Animal Health Code, Article 2.3.13. Appendix 3.8.4.4.2.

³⁴ Noel Murray, BVSc (Hons), MACVSc (Epidemiology), Animal Health Policy Analyst, Canadian Food Inspection Agency, Ottawa, Ontario, Canada.

³⁵ Canadian Food Inspection Agency, Technical overview of BSE in Canada, 24 March, 2005. (AR 12905).

for as many as 338,250 points (451 samples*750 points per sample; Table 1).

Consequently, Dr. Weaver's assertion that 'Canada's ongoing BSE surveillance program falls well short of the surveillance levels recommended by the OIE' (Declaration of Dr. Weaver, 28 June, 2005, p2) is not consistent with *any* OIE guidelines (past or present). Moreover, the OIE does not require that all 300,000 points be achieved prior to attaining Controlled Risk status. Rather, the surveillance program to achieve the necessary points should be 'in place' as it is in Canada.³⁶

7.7. Canada has implemented a surveillance program that appropriately samples from the population in which BSE is most likely to occur and substantially exceeds both old and new OIE recommendations. Canada tests sufficient numbers of high-risk animals to detect BSE *and* empirically demonstrates that BSE is rare in Canada. The burden of BSE in Canada is at least 15 fold less than that in the EU and is restricted to a few cohorts of older animals born around the time of the feed ban.³⁷ It is clear the plaintiff and their declarants choose to ignore the empirical data demonstrating the extremely low and age-restricted burden of BSE in Canada and that the Canadian surveillance program satisfies all the requirements of the OIE.

8. *Testing of healthy cattle less than 30 months of age serves no scientific purpose*

³⁶ OIE Terrestrial Animal Health Code, Article 2.3.13.4.2.

³⁷ On one hand, Dr. Cox questions the meaning of 'rare' (Declaration of Dr. Cox, 28 June, 2005, p42) yet he justifies the use of his statistical model based on the assumption that 'BSE cases are rare' in Canada (Declaration of Dr. Cox, 28 June, 2005, p30).

8.1. The plaintiff falsely accuses us of ‘assuming away the 21 and 23 month old BSE cases found in Japan’. They contend, ‘[t]here is simply no reason in do so, other than that they do not fit the USDA’s preconceived notions’. This is simply not true. There is sufficient reason to believe these animals were in fact BSE negative rather than positive. Both animals were weak reactors on the BioRad ELISA³⁸ and have failed to be confirmed using an OIE-validated confirmatory test.³⁹ The plaintiff contends that Dr. Prusiner has ‘no doubt that these two [Japanese] cows had BSE’ (Reply Memorandum, 28 June, 2005, p11) even though no such proclamation of his doubtlessness is ever included in Dr. Prusiner’s declaration.

8.2. Despite millions of tests performed worldwide, BSE has not been confirmed in an animal less than 30 months of age since 2001. The most recently confirmed BSE cases in cattle less than 30 months of age were 2 animals in Germany in 2001; during that year, there were 2,198 confirmed cases worldwide.⁴⁰ In Dr. Prusiner’s declaration, he referenced 84 confirmed BSE cases in the UK that were 30 months of age or younger

³⁸ Yamakawa et al. Atypical proteinase K-resistant prion protein (PrP^{res}) observed in an apparently healthy 23-month-old steer. *Jpn. J. Infect. Dis.* 2003;56:221-2.

³⁹ Both were negative on histological and immunohistochemical analyses. No evidence of transmissibility has been detected using a mouse bioassay despite allowing more than twice the time typically required for disease to manifest. The basis of their classification as positive is a questionable reaction using a modified Western blot procedure that has not been internationally validated. Even so, no samples have been subjected to independent confirmation at a recognized TSE-reference laboratory (e.g., Veterinary Laboratories Agency in Weybridge, UK). The OIE states that following a positive reaction on a rapid screening test such as the ELISA, ‘confirmation of a diagnosis of BSE requires examination of fixed brain by histopathology and/or [immunohistochemistry]’ or presumably the OIE SAF Western blot (2004 OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals Chapter 2.3.13.). This requirement has not been fulfilled.

⁴⁰ Department for Environment, Food and Rural Affairs (DEFRA) available at <http://www.defra.gov.uk/animalh/bse/statistics/incidence.html>.

(Declaration of Dr. Prusiner, 28 June, 2005, p9). However, 32 of the 84 cases were 30 months of age; hence, only 52 animals were less than 30 months of age.⁴⁰ These 52 cases⁴¹ were identified among 180,780 total cases in the UK. In other words, of every 10,000 confirmed BSE cases, *only* 3 were less than 30 months of age. To put this in perspective, a grand total of 5 confirmed BSE cases, 3 in Canada and 2 in the USA (1 of which was Canadian in origin), have been found in North America. The science and empirical data categorically demonstrate that the likelihood of a case in an animal less than 30 months of age is rapidly approaching zero. Of more than 20 million tests in the EU during 2002 and 2003, not a single BSE case was confirmed in an animal less than 30 months of age.⁴² Further, no animal younger than 30 months of age has been confirmed with BSE in the UK since 1996. (AR 8329).

⁴¹ The majority of the young cases detected in the UK were infected at a time when circulating infectivity and exposure doses were greatest and consequently, incubation periods were shortest. Circulating levels of infectivity in Canada and the USA never reached that of the UK. Furthermore, the data indicate feed bans dramatically reduce in-feed levels of infectivity. Therefore, it is highly improbable that North American cattle will demonstrate short incubation periods similar to those observed in the UK during the peak of the epidemic. The plaintiff challenges the existence of a relationship between dose of BSE agent and incubation period (Reply Memorandum, 28 June, 2005, p7). The data presented in our previous declaration represent right-truncated data (Declaration of Dr. Hueston, Loneragan, Sreevatsan, 10 June, 2005, Table 2, p22), i.e., studies have to be stopped at some stage and in the case of BSE studies, they are invariably stopped while some animals are still incubating the disease. For example, 10 of 15 animals exposed to 100 g developed BSE 33 to 61 months post exposure whereas 7 of 15 animals developed BSE 45-75 months post exposure to 1 g. Right-truncated data imply that up to 5 of the 100-g group and 8 of the 1-g group have incubation periods greater than 110 months (the time post exposure at which the study was terminated). Therefore, we can conclude that lower doses result in longer incubation periods and low attack rates.

⁴² European Commission. Report on the monitoring and testing of ruminants for the presence of transmissible spongiform encephalopathy (TSE) in the EU in 2002. June 2003; European Commission. Report on the monitoring and testing of ruminants for the presence of transmissible spongiform encephalopathy (TSE) in the EU in 2003. May 2004.

8.3. Despite the empirical data suggesting a detectable BSE prevalence approaching zero in animals less than 30 months and Dr. Cox's acceptance that 'BSE prion contamination cannot be detected in younger cattle except in rare cases' (Declaration of Dr. Cox, 28 June, 2005, p37), he subsequently recommends testing 'cattle that would be imported into the United States' all of which would be less than 30 months of age (Declaration of Dr. Cox, 28 June, 2005, p39).

8.4. While the collective worldwide experience of BSE distills down to the plain realization that testing healthy slaughter cattle less than 30 months of age provides little or no surveillance value and is a substantial burden on finite resources, the plaintiff and Dr. Cox extol testing Canadian animals eligible for importation to the US. Without any direct supporting evidence, Dr. Prusiner recommends testing *all* animals. Dr. Matthews, TSE Programme Manager, Veterinary Laboratories Agency at Weybridge,⁴³ UK, and internationally recognized BSE expert, summarized the current epidemiological understanding of BSE and the accumulated successes of control strategies when he succinctly described the role and future of BSE testing:⁴⁴

'Inevitably just like Europe [the USA is] going to have to consider at some point whether [they] maintain this level of surveillance or you actually reduce the number of samples that you're collecting.'

'[Testing] is not there as a primary consumer protection tool only in the sense that you may detect BSE and thereby enforce new controls. But the primary measure that protects consumers is the SRM rule. The second measure is the feed ban...'

⁴³ Veterinary Laboratories Agency at Weybridge, UK, is the international reference laboratory for BSE.

⁴⁴ Comments presented at the USDA's teleconference of 24 June, 2005, to announce the results of additional testing in Dr. Matthew's laboratory of the 18 November, 2004, inconclusive case.

Dr. Matthews continued by stating that at some point, we will have to
*'learn to live with extremely low risk worldwide from BSE without
necessarily having to find every last case that exists in any country'*.

***9. The Final Rule effectively protects animal and human health; refusal to allow
importation of Canadian cattle less than 30 months of age is not justified by current
scientific or empirical data***

9.1. The plaintiff declares that the USDA's argument, and therefore, the Final Rule is dependent on speculative assumptions (Reply Memorandum, 28 June, 2005, p8). This is simply not so. The Final Rule is consistent with current scientific and empirical data concerning the etiology, pathogenesis, transmission, and epidemiology of BSE. The Final Rule, coupled with the existing control strategies such as SRM removal, slaughter controls, and the Feed Ban, effectively protect animal and human health by appropriately managing risk. Moreover, empirical data collected in Europe demonstrates conclusively that implementation of feed bans and increasing compliance result in decreased circulation of BSE infectivity. Based on these data and the effectiveness of the Canadian feed ban, the likelihood of exposure of Canadian cattle to BSE is decreasing with each passing year. By extension, empirical data support the argument that the likelihood of new BSE infections in Canadian cattle is decreasing with each consecutive birth cohort. Dr. Cox continues to ignore the impact of feed bans on the rate of new infections when developing his statistical models.

9.2. The wishful claim that 'R-CALF's position does not depend on a series of speculative, and often counterintuitive, assumptions' (Reply Memorandum, 28 June,

2005, p8) is belied by their Reply Memorandum and the declarations of their declarants.

For instance:

9.2.1. The plaintiff would have the USDA ignore substantial empirical and scientific data. For example, the plaintiff and Dr. Prusiner would supplant all current data concerning a species barrier with Dr. Prusiner's speculations and disregard for statistically based estimates of exposure risk.

9.2.2. The plaintiff relies on many conflicting statements of their declarants. For example, Dr. Cox recommends testing 'cattle that would be imported into the United States' (i.e., cattle under 30 months of age; Declaration of Dr. Cox, 28 June, 2005, p39) and Dr. Prusiner would have us test 'all slaughtered animals' (Declaration of Dr. Prusiner, 28 June, 2005, p9) yet Dr. Cox apparently understands that 'BSE prion contamination cannot be detected in younger cattle except in rare cases'⁴⁵ (Declaration of Dr. Cox, 28 June, 2005, p37). Elsewhere in Dr. Cox's declaration, his recommendation for Canadian surveillance changes in that he says Canada should test 'at least 187,500 to 1.9 million consecutive *at-risk* [cattle]... and [all these animals should] be BSE-free before resuming imports'

⁴⁵ Young BSE cases are extraordinarily rare indeed. Based on data from around the world, we would expect to confirm 1 BSE case less 30 months of age for every roughly 3,000 confirmed BSE cases. At present, 5 BSE cases have been detected in North America despite approximately 500,000 tests performed on those cattle most likely to have BSE.

(Declaration of Dr. Cox, 28 June, 2005, p24).⁴⁶ If Dr. Cox cannot reconcile his own arguments, it is not surprising that conflicting, counterintuitive positions are adopted by the plaintiff.

9.2.3. The plaintiff erroneously concludes that Canada does not qualify for OIE Controlled Risk status. They base their conclusion on the declaration of Dr. Weaver even though it contains a wholly impressive collection of errors concerning the OIE guidelines as described above.

9.2.4. The plaintiff relies on misrepresentations and grossly illogical statements. Because prions have been detected in blood of some species other than cattle, they reason that bovine blood must contain infectivity (Reply Memorandum, 28 June, 2005, p10). While the former is true, the later grossly misrepresents scientific data. Even though Dr. Prusiner is a well-known scientist, infectivity has never been detected in bovine blood no matter how 'convinced' he is of its presence there.

9.2.5. The plaintiff infers the Final Rule does not effectively manage risk because 'a BSE-infected cow has already entered the U.S. from Canada' (Reply Memorandum, 28 June, 2005, p6). This is plainly illogical as the Final Rule was

⁴⁶ There are an estimated 80,000 high-risk cattle in Canada each year. Of these, Canada will test approximately 60,000 high-risk cattle this year. At this rate, Dr. Cox would make Canada wait 3.1 to 31.7 years without finding a BSE case. If Dr. Prusiner's speculation that BSE can occur spontaneously is true (Declaration of Dr. Prusiner, 28 June, 2005, p10), then Dr. Cox would hold Canada to an unreasonable and impossible goal, a goal that the USA could not even achieve.

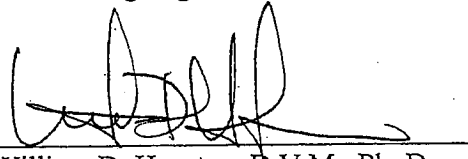
developed and promulgated years *after* the infected animal entered the USA. Moreover, the infected animal would have been ineligible for importation under the Final Rule because it was greater than 30 months of age at the time of entry and was not a slaughter animal yet the plaintiff chose irrational arguments over reason and logic.

9.3. The Canadian surveillance program strongly demonstrates that BSE is rare in Canada and restricted to a small cohort of animals born around the time of the feed ban, the time at which infectivity in feed was greatest. The USDA does not assume zero risk of BSE in Canada but appropriately concluded from its risk assessment that Canada represents Minimal Risk for BSE.⁴⁷ To clarify, the risk that has been assessed is the hypothetical potential for generation of new BSE cases in cattle or vCJD cases in humans in the USA as a consequence of importing Canadian cattle. The Final Rule, coupled with the sequential, interlocking, and overlapping control strategies implemented in the USA, effectively manages any risk associated with Canadian cattle eligible for import under the Final Rule. Because of effective BSE risk management in the USA and Canada, Canadian cattle imported into the USA under the Final Rule represent no measurable increased risk to human or animal health than USA-born cattle.

⁴⁷ USDA:APHIS. Analysis of Risk–Update for the Final Rule: Bovine Spongiform Encephalopathy; Minimal Risk Regions and Importation of Commodities, December, 2004, AR08333.

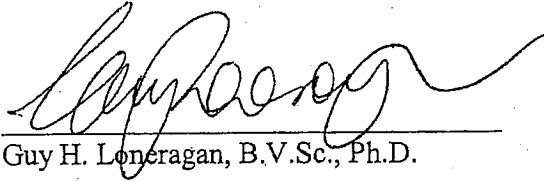
We declare under penalty of perjury that the foregoing is true and correct.

July 13, 2005
Date



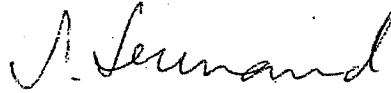
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Diplomate, A.C.V.P.M.

July 13, 2005
Date



Guy H. Loneragan, B.V.Sc., Ph.D.

July 13, 2005
Date



Srinand Sreevatsan, D.V.M., M.P.H., Ph.D.