DECLARATION OF PHILIP B. STARK, PH.D.
8 June 2005

1. I am Professor of Statistics at the University of California at Berkeley. Appendix A lists my qualifications and recent testimony.

2. I was asked by the US Department of Justice to comment on Dr. Louis Anthony Cox's declarations in this matter.

OVERVIEW

3. Few risks are exactly zero. We live with many low-probability risks, including earthquakes, fire, flood, and bacteria in food. The government does not prohibit construction in areas subject to wildfire, in earthquake fault zones, or in flood plains. The University of California at Berkeley is on the Hayward fault, and the homes of many Berkeley faculty are in wooded areas with high fire risks. New Orleans is in the Mississippi flood plain. The government imposes building codes, maintains levees, and provides emergency services to mitigate consequences of earthquakes, fire, and flood. Similarly, the government does not ban rare hamburgers or eggs fried sunny-side up. But it inspects meat and poultry, and regulates commercial food preparation—measures that reduce but do not eliminate risk of foodborne disease.

4. Identifying a risk as low, without further quantification, is reasonable in many situations—because assigning a numerical value may require unreasonable assumptions. I believe that the risk from importing Canadian cattle under the new regulations is extremely small. I think it was reasonable for the Secretary to proceed without a numerical value for the risk.

5. BSE is Bovine Spongiform Encephalothopy. Dr. Cox says that some young Canadian cattle might test positive for BSE ("BSE-positive"). I agree. The rate of BSE-positives in Canadian cattle under thirty months old ("younger cattle") is crucial for estimating the risk of importing BSE, because only younger cattle may be imported under the new regulations. Empirically, the rate is zero: all four Canadian cattle discovered to have BSE were over six years old. The rate in younger cattle should be nearly zero, because feed bans like the 1997 ban in Canada markedly slow—if they do not entirely prevent—the spread of BSE. Canadian cattle now under thirty months old were born well after the feed ban. Moreover, younger cattle that are infected with the agent that causes BSE ("prions") create far less of a hazard than older ones. Their bodies generally contain much less of the infectious agent, because it has not had time to multiply. And the infectious material is largely confined to the "distal ileum" (part of the small intestine).

6. The primary, if not the only mode of transmission for BSE is through contaminated feed. Cattle are most susceptible in their first year of life. The time it takes BSE to manifest clinically depends on the dose of contaminated feed. In the UK, when BSE rates were highest, the median time to develop BSE was about four years. In the EU, where

infectious loads are much lower than in the UK at the height of the epidemic, the median ranges from six to nine years.

7. Dr. Cox claims that among young Canadian cattle, about 6.25 per million are BSE-positive, and that imports would be 1.7 million head of cattle under the new regulations. His calculations imply that the imports would probably include about eleven BSE-positive cattle each year:

$$1.7 \text{ million} \times 6.25 \text{ per million} = 11.$$

That could indeed create a hazard if other safeguards failed. However, his calculations depend on assumptions that contradict the data.

8. Dr. Cox assumes that BSE-positives are as common in younger cattle as in older cattle. That assumption does not take into account the feed ban or the rate of progression of the disease. Moreover, it contradicts experience: in the EU, about one BSE case in three thousand is observed in cattle under thirty months of age. The rate of BSE-positives among younger cattle is about three thousand times smaller than the rate in older cattle.

9. Even if the rate of BSE-positives among older Canadian cattle is 6.25 per million (it is smaller), the rate in younger Canadian cattle should be three thousand times smaller, i.e., 2 per billion. On this basis, Dr. Cox's calculations imply that the first mad cow is expected to cross the border around 2300 AD.

10. Dr. Cox's calculations are based on a particular statistical model for BSE. That model does not take into account differences in the rate of BSE by age or geography. It does not take into account the feed ban. It does not take into account the fact that cases are linked through contaminated feed. The model does not fit the data for Canada, or the UK, or France, or Switzerland.

11. Dr. Cox plugged the wrong numbers into the wrong statistical model. He used the wrong figures for the number of imports, the number of tested Canadian cattle, the median time to develop BSE in Canada, the ratio of the BSE-positive rate among high-risk animals to the BSE-positive rate among low-risk animals, and the ratio of the BSE-positive rate among older cattle to the BSE-positive rate among younger cattle. I will show what Dr. Cox's model implies if the right numbers are used, and why Dr. Cox's model is wrong. I will not offer my own quantitative risk assessment.

12. There is a minute risk that importing Canadian cattle will spread BSE into US herds or cause variant Creutzfeld-Jakobs Disease (vCJD) in humans. To me, this risk seems largely hypothetical. No case of vCJD has been traced to Canadian or US cattle. No case of BSE has been detected in native US cattle, despite substantial imports of live cattle from Canada prior to 2003. By contrast, we accept real risks—from earthquakes, fire, flood, and bacteria in food.

13. The Secretary's decision was reasonable.

MATERIALS RELIED ON

14. I reviewed documents filed in this matter and in the appeal of the preliminary injunction. I reviewed parts of the administrative record and parts of the scientific literature. I focused on materials that seemed most relevant to the health issues. I considered Plaintiff's exhibits and documents the Plaintiff highlighted. The paragraphs below summarize my understanding of the scientific basis for the Secretary's decision, some flaws in Plaintiff's arguments, and the reasons I find the Secretary's decision reasonable. Appendix B lists the materials I relied on.

IMPORTS

15. Dr. Cox assumes that 1.7 million head of Canadian cattle would be imported each year under the new regulations.[1] This Dr. Cox's first wrong number. I understand the correct number to be between 900,000—as estimated in February 2005 by the National Cattlemen's Beef Association—and 1.4 million, as estimated by the USDA.[2]

16. With the correct number of imported cattle, Dr. Cox's estimate for the number of BSE-positive cattle that would be imported each year should be revised downwards by 20% to 50%. That might seem like a small correction, but many others are described below. Some are much larger.

THE EPIDEMIOLOGY OF BSE

17. The primary, if not the only, means of transmission of BSE is through contaminated feed.[3] The UK data are evidence against cattle-to-cattle transmission by routes other than feed.[4]

18. Four dates are especially important in the progression of BSE: (i) the date at which the animal becomes infected, (ii) the date at which ingesting tissues from the animal could infect a human or another animal, (iii) the date at which BSE is detectable using diagnostic tests, and (iv) the date at which the animal has clinical symptoms.

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1 Declaration of Louis Anthony Cox, Jr., Ph.D., 28 January 2005, at 6; Supplemental Declaration of Louis Anthony Cox, Jr., Ph.D., 25 February 2005, at 2; Declaration of Louis Anthony Cox, Jr., Ph.D., 7 May 2005, at 21.

2 70 FR 18261 (AR 12582).

3 68 FR 62390 (AR 98); 70 FR 486 (AR 8070); J.A. Fox and H.H. Peterson, Risks and implications of bovine spongiform encephalopathy for the United States: insights from other countries, 29 *Food Policy* 45 (2004) at 47 (Plaintiff's exhibit 13).

4 J.W. Wilesmith, An epidemiologist's view of bovine spongiform encephalopathy, 343 *Philosophical Transactions of the Royal Society of London, Series B*, 357 (1994) at 359. See also paragraph 35.

19. The tonsils and distal ileum may present special problems. Experimentally, if calves are given high doses of infective material, their distal ileum can become infective in 6 months, and their tonsils can become infective in 10 months.[5] This is unlikely to be relevant in Canada, because the infectious load is low.[6] The infectivity of the tonsils seems to be very low.[7] Neither infectivity nor the presence of disease-specific proteins has been observed in the distal ileum of BSE cases that became infected with BSE naturally.[8] (Both experimental and natural data are limited.) Regulations require that the tonsils and distal ileum be removed from cattle of all ages at slaughter, and disposed of as inedible.[9]

20. BSE can only be detected by post mortem tests a few months before animals have symptoms.[10] The date at which an animal has detectable BSE and the date at which an animal has symptoms of BSE are the same, give or take a few months.[11] Bovine tissues (other than the distil ileum and tonsils) are rarely infectious until at least 32 months after exposure.[12] "Infected animals less than 30 months of age . . . are unlikely to have infectious levels of the prion protein (EU SSC 2002; Wells, et al.; 1994; Wells, et al.; 1998)."[13] All imported cattle would be slaughtered before 30 months of age, and often much younger.[14]

21. As noted in paragraph 20, tissues other than the distal ileum and tonsils generally do not show infectivity until at least 32 months after infection.[15] US and Canadian regulations require tissues that pose a hazard to be removed at slaughter. Tissues that must be removed from older cattle include brain, skull, eyes, trigeminal ganglia, spinal

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5 68 FR 62391 (AR 99); 70 FR 483, 512–13 (AR 8067, 8096-97); L.A. Terry, S. Marsh, S.J. Ryder, A.C. Hawkins, G.A.H. Wells, and Y.I. Spencer, Detection of disease-specific PrP in the distal ileum of cattle exposed orally to the agent of bovine spongiform encephalopathy, 152 *Veterinary Record* 387 (2003), at 387, 388, 390; G.A.H. Wells, M. Dawson, S.A.C. Hawkins, R.B. Green, I. Dexter, M.E. Francis, M.M. Simmons, A.R. Austin and M.W. Horigan, Infectivity of the ileum of cattle challenged orally with bovine spongiform encephalopathy, 135 *Veterinary Record* 40 (1994) at 40 (cited at AR 99); G.A.H. Wells, S.A.C. Hawkins, R.B. Green, A.R. Austin, I. Dexter, Y.I. Spencer, M.J. Chaplin, M.J. Stack and M. Dawson, Preliminary observations on the pathogenesis of experimental bovine spongiform encephalopathy (BSE): an update, 142 *Veterinary Record* 103 (1998) at 105 (AR 12504).

6 70 FR 513 (AR 8097).

7 69 FR 1862, 1865 (AR 9959, 9962); AR 8332.

8 AR 8332; Wells et al. (1998) at 105 (AR 12504); Terry et al. (2005) at 387, 390, 391.

9 70 FR 461, 465, 466, 496 (AR 8045, 8049, 8050, 8080).

10 69 FR 1871 (AR 9968); 70 FR 475 (AR 8059); 69 FR 1864 (AR 9961); AR 8331-32.

11 See also Wells et al. (1998) at 103 (AR 12502).

12 AR 8332.

13 68 FR 62391 (AR 99). See also 70 FR 483, 512–13 (AR 8067, 8096–97).

14 70 FR 512 (AR 8096); AR 8342.

15 68 FR 62391 (AR 99); 69 FR 1862, 42296 (AR 9959, 9939); 70 FR 483, 502 (AR 8067, 8086).

cord, most of the vertebral column, dorsal root ganglia, and small intestines.[16] These tissues are banned from entering the human or bovine food chain.[17] Also see paragraph 19.

22. Bovine muscle tissue—meat—has never been found to be infective.[18] Importing Canadian meat, especially from younger cattle, is safe.

THE FOUR CANADIAN COWS WITH BSE

23. Four cows born in Canada were diagnosed with BSE. These cows are clustered in time and space. They were 6 to 8 years old at the time of diagnosis (median 6 1/2 years). They were diagnosed within 19 months of each other, in May 2003, December 2003 (the cow found in Washington State), January 2005, and January 2005. They come from a small area in northeastern Alberta—a region 280 miles across. At least 3 of the 4 cows were exposed to meat and bone meal processed before the feed ban by the same renderer, and shipped between January 30 and February 28, 1997.[19] It is likely that all 4 cases were caused by exposure to contaminated feed when the animals were young, before or shortly after the Canadian feed ban.[20] Dr. Cox acknowledges the clustering: "It appears plausible that at least one such area of higher risk exists in Canada, centered in Alberta Province."[21]

16 69 FR 1862, 1868–69, 10635, 42292 (AR 9959, 9965–66, 3839, 9935); 70 FR 461, 466, 18256 (AR 8045, 8050, 12577).

17 69 FR 42292 (AR 9935); 70 FR 465–66, 493 (AR 8049–50, 8077).

18 AR 8332–33; 68 FR 62391 (AR 99); 69 FR 1865 (AR 9962); Fox and Peterson (2004), at 48 (Plaintiff's exhibit 13).

19 U.S. Department of Agriculture, Summary of the Epidemiological Findings of North American Bovine Spongiform Encephalopathy Positive Cattle, April 2005 (Plaintiff's Motion for Summary Judgement, 9 May 2005, Exhibit 11), at 16–17, 25.

20 USDA, April 2005, at 17.

21 Cox Decl, 7 May 2005, at 17.

INCUBATION TIME

24. Infection is most common during the first year of life.[22] The time from infection to clinical BSE ("incubation time") depends on the dose of contaminated feed:[23] when the dose is low, the incubation period is long. "It is well known that as the force of infection increases over time, the average age at infection decreases and thus the average age at onset of clinical signs decreases . . ."[24]

25. At the height of the UK epidemic, median incubation time was about 4 years.[25] Because most BSE cases occurred in the UK when the epidemic was severe, the median incubation time would still be about 4 years if we pooled all the available data. However, for reasons mentioned in paragraph 24, the median incubation time will be substantially longer when the infectious load is low, as in Canada.

26. There is not much data for Canada. In the UK and Europe, where infectious loads are now much lower than they were in the UK at the height of the epidemic, the median incubation period ranges from 6 to 9 years.[26] I estimate the incubation time in Canada around the time of the feed ban to be 6 to 8 years.

27. The four Canadian cows with BSE showed clinical signs at a median age of about 6 1/2 years. See paragraph 23. If the cows were infected at age 6 months, their median incubation period would be about 6 years. This supports the estimate in paragraph 26.

28. The Secretary's language sometimes suggests that the median incubation period in Canada could be as short as 4 or 5 years.[27] However, the Secretary's argument remains cogent even if the median is 6 to 8 years. Canada has had a feed ban in place since 1997.[28] That is 8 years ago, so a large fraction of the infected cattle in Canada already should have shown clinical signs of BSE. Moreover, cattle born before the feed ban are well over 30 months old by now, and thus could not be imported. (Under the proposed regulations, none of the 4 infected cows could have been imported.)

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22 AR 8341; M.E. Arnold and J.W. Wilesmith, Estimation of the age-dependent risk of infection to BSE of dairy cattle in Great Britain, 66 *Preventive Veterinary Medicine* 35 (2004), at 35–36, 45.

23 AR 8329–31, 8335.

24 C.A. Donnelly, N.M. Ferguson, A.C. Ghani, M.E.J. Woolhouse, C.J. Watt, and R.M. Anderson, The epidemiology of BSE in cattle herds in Great Britain. I. Epidemiological processes, demography of cattle and approaches to control by culling, 352 *Philosophical Transactions of the Royal Society of London, Series B*, 781 (1997) at 788.

25 70 FR 470 (AR 8054).

26 Report on the monitoring and testing of ruminants for the presence of transmissible spongiform encephalopathy (TSE) in the EU in 2003, Table 17. http://europa.eu.int/comm/food/food/biosafety/bse/annual_reps_en.htm This report is referenced below as "EU;" it is cited in Cox Decl 7 May 2005, at 6.

27 See, e.g., 70 FR 468, 470, 475 (AR 8052, 8054, 8059).

28 70 FR 467, 512 (AR 8051, 8096).

29. Dr. Cox compares 4.2 years—the mean incubation period for the UK[29]—with the ages of the 4 Canadian cows with BSE when they were diagnosed. He concludes that the cows became infected after the feed ban.[30] The 4.2 is Dr. Cox's second wrong number. It would be more appropriate to compare the ages of the cows to the range 6 to 8 years, the median incubation period for Canada. See paragraph 26. The cows were infected either before the feed ban, or just after—when stores of contaminated feed had not yet been exhausted. It is unlikely that the cows became infected when they were 2 or 3 years old, as Dr. Cox's argument suggests. See paragraphs 23 and 24.

YOUNG CANADIAN CATTLE ARE SAFE

30. Under the proposed regulations, the US would import only cattle less than 30 months of age, or beef from such cattle. Imported live cattle would be slaughtered before reaching 30 months of age.[31] According to Dr. Cox, "It is the prevalence rate defined as *the fraction of BSE-positive cattle among all imported cattle* that is relevant for risk assessment purposes"; moreover, "excluding cattle over 30 months old presumably greatly reduces the prevalence of detectable BSE cases."[32] By "BSE-positive cattle," he means cattle with BSE that can be detected by current surveillance and testing procedures; he does not mean cattle infected with BSE.[33] I follow his usage throughout this document. In my judgment, the prevalence of BSE-positives among younger Canadian cattle is now essentially zero.

31. BSE-positive young cattle are extremely rare, even in countries with high rates of BSE-positives among older cattle. "While some cases of BSE have been found in animals less than 30 months of age, these are relatively few and have occurred primarily in countries with significant levels of circulating infectivity. . . . Infected animals less than 30 months of age . . . are unlikely to have infectious levels of the prion protein . . . "[34] The administrative record reports that in the UK, only 1 in 10,000 BSE-positives has been in cattle under 30 months old.[35] The last was in 1996.[36] (I believe the factor of 10,000 may be high; I think Table 1 of AR 8330 was not interpreted correctly.) Heavy exposure is required to cause BSE-positivity in younger cattle.[37] There is no evidence of BSE in Canadian cattle under 30 months old; all 4 diagnosed cases were cows at least 6 years old.

29 70 FR 470 (AR 8054).
30 Cox Decl, 28 January 2005, at 7.
31 70 FR 548–49 (AR 8132–33).
32 Cox Decl, 7 May 2005 at 22–23, 24.
33 Cox Decl, 7 May 2005 at 24.
34 68 FR 62391 (AR 99). See also Wells et al. (1998) at 103, 105–06 (AR 12502, 12504–05).
35 AR 8330.
36 Department for Environment, Food and Rural Affairs, BSE: Statistics – Youngest and oldest cases by year of onset – GB, 11 May 2005. http://www.defra.gov.uk/animalh/bse/statistics/bse/yng-old.html
37 69 FR 1864, 42296 (AR 9961, 9939); AR 8335.

See paragraph 23.

EVIDENCE THAT FEED BANS WORK

32. Feed bans prevent infectious tissue from being recycled to feed other cattle. Feed bans can slow or stop the spread of BSE, even in epidemics as heavy as the one in the UK.[38]

33. Although the caption to Figure 1 at 70 FR 462 reads "Confirmed cases in UK cattle born after feed ban implementation," the figure actually shows confirmed cases in UK cattle by year of birth, both before and after the feed ban. The first UK feed ban was in 1988. The epidemic grows until the feed ban. Then the infection rate plummets as contaminated feed is used up and compliance with the ban improves. The feed ban cannot protect animals born before the ban and exposed to contaminated feed when they were calves. It can—and does—protect cattle born after the ban. The figure is compelling evidence that feed bans work.

34. Figure 2 in Dr. Cox's Declaration of 7 May 2005 is similar to Figure 1 at 70 FR 462, but the horizontal axis shows year of clinical onset rather than year of birth. At the height of the UK epidemic, clinical onset was typically about 4 years after infection. See paragraph 25. The figure demonstrates that the bulk of cases with clinical onset after the feed ban were among cattle born before the feed ban. A feed ban cannot prevent infection in animals born before the ban; they were exposed as calves, when infection is most likely to occur. See paragraphs 24 and 33. Dr. Cox's figure displays the data in a way that obscures the effectiveness of the feed ban.

35. The effectiveness of feed bans is part of the evidence that the only important means of transmission of BSE is through contaminated feed.[39]

36. So far, I have discussed evidence that shows that feed bans work, and that young cattle are safe. Dr. Cox disagrees with these conclusions. The following paragraphs examine his reasoning.

PREVALENCE OF BSE IN CANADIAN CATTLE OVER 30 MONTHS OLD

37. Dr. Cox estimates that, in Canada, 6.25 per million cattle are BSE-positive.[40] There

38 69 FR 42289 (AR 9932); 70 FR 462 (AR 8046); AR 8335; R. Bradley and P.P. Liberski, Bovine Spongiform Encephalopathy (BSE): The end of the beginning or the beginning of the end? *Folia Neuropathologica, Supplement A* 55 (2004), at 55, 59, 66 (cited in Cox Decl, 7 May 2005, at 14, 20)

39 Wilesmith (1994) at 361.

40 Cox Decl, 7 May 2005, at 24. Elsewhere, Dr. Cox estimates the rate to be 5.5 cases per million, based on the number of tests in Alberta alone. Cox Decl, 28 January 2005, at 5–6. The assumptions underlying these two estimates differ. See footnotes 41 and 52.

are 4 BSE cases among Canadian cattle. Dr. Cox divides 4 by 40,000, which is supposed to be the number of BSE tests that have been performed on Canadian cattle. That gives a rate of 1 in 10,000 among tested cattle.[41]

38. The 40,000 is Dr. Cox's third wrong number. Far more than 40,000 Canadian cattle have been tested for BSE. Indeed, 16,318 Canadian cattle were tested through 2003.[42] Another 23,550 Canadian cattle were tested in 2004. From January 2005 through May 2005—the most recent data available to me—Canada tested 28,569 cattle.[43] Thus, over 68,000 Canadian cattle have been tested. The rate of BSE-positives among tested animals in Canada is currently about

$$4/68{,}000 = 6 \text{ per } 100{,}000,$$

which is 40% below Dr. Cox's most recent estimate.

39. Canada's inspection program targets animals at high risk. To estimate the rate of BSE-positives among all cattle, we must compare the rate in the high-risk population to the rate in the low-risk population. Using data from the EU, Dr. Cox estimates that BSE-positivity is 16 times more common in the high-risk population.[44]

40. The 16 is Dr. Cox's fourth wrong number. Table 2 of Dr. Cox's declaration contains an error. Dr. Cox lists the number of targeted cattle tested in Belgium as 349,888.[45] In the original EU report, the value is 34,988.[46]

41. If we correct this error, the ratio for Belgium changes from 0.51 to 5.09. The overall ratio for the 15 EU countries listed by Dr. Cox is 19.88, not 15.99.[47] This agrees with the ratio of 20 mentioned in the EU report.[48] Dr. Cox quotes the ratio of 20 from the EU report, but uses 16.[49]

42. The ratio Dr. Cox calculated for Belgium was 0.51, implying that BSE-positives are

41 Cox Decl, 7 May 2005, at 23. As mentioned in footnote 40, Dr. Cox also estimates the rate to be 5.5 cases per million. That estimate involves dividing 3 cases by the number of tests in Alberta alone, which he indicates is about 9,000. Cox Decl, 28 January 2005, at 5–6.

42 Fax from Frank Fillo, USDA, 16 May 2005, Table 1.

43 Canadian Food Inspection Agency, BSE Enhanced Surveillance Program, Sample Status and Testing Results. http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/surv/surve.shtml#num

44 Cox Decl, 7 May 2005, at 6, 23.

45 Cox Decl, 7 May 2005, at 6.

46 EU, May 2004, at Table 13.

47 EU, May 2004, at Tables 13, 14.

48 EU, May 2004, at 22.

49 Cox Decl, 7 May 2005, at 23. Dr. Cox refers to "targeted" animals, which correspond to "risk" animals in the EU report.

less common among high-risk animals than among low-risk animals.

43. Paragraph 38 gives the infection rate among tested animals in Canada as 4/68,000. The ratio from the EU report is 20. Dr. Cox's estimate of the prevalence in older Canadian cattle should be

$$(4/68{,}000) \times (1/20) = 3 \text{ per } 1{,}000{,}000.$$

This is less than half of Dr. Cox's latest estimate.[50]

44. Even the ratio of 20 in paragraph 43 is problematic. The EU report warns against combining figures for different countries, as Dr. Cox has done in constructing his estimate, because different EU countries have different monitoring programs.[51] Canada's monitoring program is different yet again.

45. Elsewhere, Dr. Cox takes the ratio (of the BSE-positive rate among high-risk animals to the rate among low-risk animals) to be 60.[52] If 60 is correct, the estimated prevalence of BSE-positive older cattle drops to

$$(4/68{,}000) \times (1/60) = 1 \text{ per } 1{,}000{,}000.$$

A SUBTOTAL

46. Dr. Cox's estimate of the number of imported cattle should be revised downwards by 20% to 50%. See paragraph 16. Dr. Cox's estimate for the rate of BSE-positives among high-risk cattle in Canada should be revised downwards by 40%. See paragraph 38. Dr. Cox's estimate for the ratio of the BSE-positive rate among high-risk cattle to the rate among low-risk cattle should be revised upwards by 25% to 275%. See paragraphs 41 and 45. Without the corrections, his argument implies that the US can expect to import 11 BSE-positive cases per year. After the corrections, the expected number ranges from just under 1 to just over 4 cases per year.[53]

PREVALENCE OF BSE IN CANADIAN CATTLE UNDER 30 MONTHS OLD

47. BSE-positivity is extremely rare in younger cattle. The administrative record reports that in the UK epidemic, only 1 in 10,000 BSE-positive animals was under 30 months old. See paragraph 31.

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50 Cox Decl, 7 May 2005, at 24.

51 EU, May 2004, at 10, 22, 32.

52 Cox Decl, 28 January 2005, at 6. Dr. Cox also uses the number 2%=1/50. Cox Suppl Decl, 25 February 2005, at 4. Each of Dr. Cox's declarations proposes a different value for this ratio.

53 The smaller figure is $(4/68{,}000)\times(1/60)\times 900{,}000 = 0.9$. The larger figure is $(4/68{,}000)\times(1/20)\times 1.4 \text{ million} = 4.1$.

48. From 1997 to 31 March 2005, there were 14,712 BSE-positive cattle found in the UK.[54] None was under 30 months old.

49. Two BSE-positive cattle under 30 months old were found in the EU15 countries from 2001 to 2003.[55] In the same period, 5,657 BSE-positive cattle were found in the EU15 countries.[56] The ratio is

$$2/5{,}657 = 1 \text{ in } 2{,}800.$$

50. Surveillance practices might make BSE-positives appear less common among younger cattle. On the other hand, more cattle are under 30 months old than over. And as the epidemic wanes, the rate of BSE-positives among younger cattle should drop faster than it does in older cattle. Empirically, the median age of BSE-positive cases in the EU is growing,[57] in part because new infections are falling now that feed bans are in place, and in part because the infectious dose is lower.[58] The feed ban drastically reduced—if it did not completely eliminate—the source of infection for younger cattle.

51. The amount of infective material circulating in Canada is low.[59] It takes a large dose of infected feed to cause cattle to become BSE-positive at a young age.[60] It is therefore unlikely that many young cattle in Canada are BSE-positive.

52. Paragraphs 47 to 51 suggest that the rate of BSE-positive younger Canadian cattle is probably between 1/2,800 and 1/10,000 of the rate of BSE-positive older Canadian cattle. Therefore, Dr. Cox's estimate of 6.25 BSE-positives per million imported cattle should be divided by a number between 2,800 and 10,000. Dr. Cox's fifth error is to ignore this divisor. The corrected estimate is the range 6.25 per ten billion to 22.3 per ten billion.

53. Dr. Cox says that we should expect to import one BSE-positive animal every 5 weeks.[61] But if we correct the rate of BSE-positives as in paragraph 52 and adjust the other factors as in paragraph 46, Dr. Cox's calculations imply that we should expect to import one BSE-positive animal every 7 to 110 centuries.[62] This is what Dr. Cox's model

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54 DEFRA, BSE: Statistics – Confirmed cases of BSE reported worldwide, Data as at 31 March 2005, 11 May 2005. http://www.defra.gov.uk/animalh/bse/statistics/bse/worldwide.htm

55 EU, May 24, 2004, Chart 31, Table 34. Chart 31 has data for 2001-2003 only. In 69 FR 1864 (AR 9961), the number of cases is cited as 3. I think that number is wrong.

56 EU, May 2004, Table 6.

57 EU, May 2004, Charts 7 to 9.

58 AR 8335.

59 70 FR 513 (AR 8097).

60 AR 8331, 8335.

61 Cox Decl, 7 May 2005, at 24.

62 One end of the range is the reciprocal of

$$(4/68{,}000)\times(1/20)\times(2/5{,}657)\times 1.4 \text{ million per year,}$$

which is 6.9 centuries. The other end of the range is the reciprocal of

gives, if we plug in the right numbers.

54. Dr. Cox ignores the effect of the feed ban. He assumes that the BSE-positive rate is the same among cattle born after the feed ban as among cattle born before the feed ban. But the rate among younger cattle must be much lower. Moreover, the incubation period for BSE in Canada is long, and getting longer. See paragraphs 24 and 26. This too makes the BSE-positive rate in younger cattle much lower than the rate in older cattle. Finally, the amount of infective material in a young infected animal is likely to be much lower than the amount in an old infected animal.[63] See paragraphs 20 and 31. Dr. Cox ignores these very large factors, all of which reduce the risk.

DR. COX'S STATISTICAL MODEL

55. Dr. Cox assumes that BSE-positive Canadian cattle can be described by a "Poisson distribution."[64]

56. Many of Dr. Cox's risk estimates ride on this assumption, including his formula for the expected number of BSE-positive cattle that would be imported each year,[65] the probability that one or more BSE-positive cattle would be imported in a given time period,[66] and the estimate that it would take 5 weeks to import the first BSE-positive animal.[67]

57. The calculation in paragraph 7 is an application of the "f × N" formula Dr. Cox states for the Poisson model.[68] The result in paragraph 9, that the first BSE-positive cattle would cross the border around 2300 AD, follows from the Poisson model. (The expected waiting time to the first event in a Poisson process is the reciprocal of the expected rate at which events occur.) The same goes for the result in paragraph 53, that it would take 7 to 110 centuries to import the first BSE case.

58. The clustering of BSE cases in Canada is not compatible with the Poisson model. If the Poisson model were correct, there would be far less clustering. The Poisson model requires BSE cases to be nearly independent of each other.[69] This contradicts the epidemiology of BSE: cases are not independent, because they are linked through sources of contaminated feed. BSE-positives in Canada are not even approximately independent: at least 3 of the 4 BSE-positive Canadian cows were exposed to contaminated feed from

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(4/68,000)×(1/60)×(1/10,000)×900,000 per year,
which is 113 centuries. See also paragraphs 55–57.

63 AR 8331.

64 Cox Decl, 7 May 2005, at 21; Cox Supp Decl, 25 February 2005 at 3.

65 Cox Decl, 7 May 2005, at 21–22, 27.

66 Cox Decl, 28 January 2005, at 6; Cox Supp Decl, 25 February 2005, at 2, 3, 5; Cox Decl, 7 May 2005, at 21–22, 24–26

67 Cox Decl, 7 May 2005, at 24.

68 Cox Decl. 7 May 2005, at 21.

69 Cox Supp Decl, 25 February 2005, at 3.

the same renderer. See paragraph 23. Dr. Cox's sixth error is to use the wrong statistical model.

59. Dr. Cox claims that if his model is wrong, the true risks are higher than his estimates.[70] There is no technical justification for this claim. And there are numerous examples to the contrary. One has been illustrated here in detail: if the risk is concentrated in older animals, and we import younger animals, the true risk will be far lower than he estimates.

60. Clustering of BSE cases is not unique to Canada. "In every case, the [UK] results indicate significant deviation from the Poisson assumption indicating aggregation of cases within natal holdings."[71] Dr. Cox also cites papers that find the clustering of BSE cases to be inconsistent with a Poisson distribution in the UK, France, and Switzerland: "The reality of spatial heterogeneity of BSE risks is now well documented in multiple countries . . ."[72]

61. Dr. Cox has written about clustering, under the rubric of "heterogeneity."[73] Dr. Cox acknowledges heterogeneity. See paragraphs 23 and 60. Dr. Cox warns: "an important requirement for more accurate and predictively valid BSE risk modeling is that the effects of *spatial heterogeneity* of BSE risk should be better understood and more fully incorporated into risk predictions."[74] "Risk assessment models that do not consider these effects of spatial heterogeneity of BSE risk may yield unrealistic predictions."[75] His risk assessment model does not consider heterogeneity in Canada.

MISCELLANY

62. When the proposed regulations were announced in the Federal Register in November 2003, only one case of BSE in cattle born in Canada had been discovered.[76] Plaintiff

70 Cox Decl, 28 January 2005, at 6; Cox Supp Decl, 25 February 2005, at 3–4.

71 Donnelly et al. (1997) at 791.

72 Cox Decl, 7 May 2005 at 16. The papers are M.A. Stevenson, J.W. Wilesmith, J.B.M. Ryan, R.S. Morris, A.B. Lawson, D.U. Pfeiffer and D. Lin, Descriptive spatial analysis of the epidemic of bovine spongiform encephalopathy in Great Britain to June 1997, 147 *Veterinary Record* 379 (2000) at 379, 382; D. Abrial, D. Calavas, N. Jarrige, C. Ducrot, Spatial heterogeneity of the risk of BSE in France following the ban of meat and bone meal in cattle feed, 67 *Preventive Veterinary Medicine* 69 (2005) at 70, 77–78, 80; M.G. Doherr, A.R. Hett, J. Rüfenacht, A. Zurgbriggen and D. Heim, Geographical clustering of cases of bovine spongiform encephalopathy (BSE) born in Switzerland after the feed ban, 151 *Veterinary Record* 467 (2002) at 467, 469–70. These papers are cited in Cox Decl, 7 May 2005, at 4, 16–17.

73 Louis Anthony Cox, Jr., Mathematical Foundations of Risk Measurement, Ph.D. Dissertation, MIT (1986), Chapter 8.

74 Cox Decl, 7 May 2005, at 15.

75 Cox Decl, 7 May 2005, at 18.

76 68 FR 62386–87 (AR 94–95).

argues that the Secretary should have revised the risk assessment when more Canadian BSE cases were found.[77] As of May 2003 when the first infected cow was diagnosed, about 10,500 Canadian cattle had been tested for BSE.[78] The rate of BSE-positives was 1/10,500 among tested cattle. This is just under 10/100,000, which is also Dr. Cox's current number.[79] As noted in paragraph 38, the rate at the end of May 2005 is 6/100,000. The rate in January 2005 was scarcely different from the rate in May 2003. If anything, the rate has declined from May 2003 to May 2005. Plaintiff's argument is puzzling.

63. Plaintiff claims Secretary was particularly unreasonable for not updating the risk assessment in January 2005, when 2 cases were discovered. [80] Through January 2005, 45,062 Canadian cattle had been tested.[81] So, at that time, the rate of BSE-positives among tested cattle was 4/45,062, which is about 9/100,000—compared with 10/100,000 in May 2003. See paragraph 62.

64. The prevalence calculations in paragraphs 37, 38, 62 and 63 suffer from a potential flaw: the cattle population changes fairly rapidly over time. Animals tested at different times are therefore from different populations. For this reason, many experts prefer to look at the "incidence rate," i.e., the rate at which new cases are discovered per year.[82] Consider the year ending in January 2005. Two new cases of BSE were discovered, among 28,358 animals tested.[83] The incidence rate is 2/28,358, or 7 per 100,000 per year. Incidence rates and prevalence rates are stable or declining.

65. Dr. Cox suggests that importing Canadian beef may cause 29 cases of vCJD in US consumers each year.[84] This estimate is based on the assumption that 1 billion pounds of Canadian beef would be imported into the US annually. Canadians consume about 2.2 billion pounds of Canadian beef each year.[85] Dr. Cox's arithmetic, applied to Canada,

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77 Memorandum of Points and Authorities in Support of Plaintiff's Motion for Summary Judgement, 9 May 2005, at 13–14.

78 Canadian Food Inspection Agency, BSE Enhanced Surveillance Program, General Information. http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/surv/infoe.shtml

79 Cox Decl, 7 May 2005, at 23.

80 Memorandum of Points and Authorities in Support of Plaintiff's Motion for Summary Judgement, 9 May 2005, at 13.

81 Canadian Food Inspection Agency, BSE in North America, BSE Enhanced Surveillance Program. http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/surv/surve.shtml See also footnote 42.

82 See, e.g., OIE, Annual incidence rate of bovine spongiform encephalopathy (BSE) in OIE Member Countries that have reported cases, excluding the United Kingdom, 19 May 2005. http://www.oie.int/eng/info/en_esbincidence.htm The OIE computes incidence rate by dividing the number of BSE cases by the number of cattle over 24 months old instead of the number of tests.

83 http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/surv/surve.shtml

84 Cox Decl, 28 January 2005, at 11.

85 US Department of Agriculture, Livestock and Poultry: World Markets and Trade,

implies that we should expect 2.2 × 29 = 64 new cases of vCJD in Canada each year. Not a single case has been observed. The number of recorded cases of vCJD in the world—for all time—is less than 200. Most cases are attributable to the UK BSE epidemic.[86]

66. There have been over 180,000 cases of BSE in the UK.[87] There have been 4 BSE-positive Canadian cattle. See paragraph 23.

67. I have responded to what I believe to be the main arguments in Dr. Cox's declarations. The bulk of Dr. Cox's declarations are devoted to showing that the risk of importing BSE is not exactly zero. I agree. However, most of his arguments are irrelevant to (i) deciding whether the Secretary's decision was reasonable, or
(ii) determining the magnitude of the risk of importing BSE. For example, Table 3 of his 7 May 2005 declaration points out that SRM (specified risk material) has been found in beef imported to the UK from Spain, Denmark, and France. The table shows that SRM removal is not perfect. The table does not report the fraction of imported beef that contains SRM, because it reports only contaminated shipments. The table does not report how much SRM was found. It does not report whether any of the SRM contained infectious material. It does not report how much SRM ended up in the food chain. It is not useful for determining the magnitude of the risk of importing BSE.

68. The scientific literature seldom speaks with a single voice, even when there is general consensus. The present case is no exception. For example, Bradley and Liberski (2004) suggest that North America may be at the beginning of a BSE epidemic. They also suggest that the epidemic is at a low level, and unlikely to become large if there is compliance with the feed ban and SRM removal policy.[88] Wilesmith et al. (1997) study the risk of maternal transmission of BSE. In their view, their data do not demonstrate maternal transmission, due to confounding by genetics and time of birth. They find some aspects of their data inconsistent with maternal transmission.[89] On the other hand, using the same data, Donnelly et al. (1997) suggest that maternal transmission plays some role.[90]

SUMMARY

69. I find the Secretary's decision reasonable. Moreover, I think the Secretary is correct: under the proposed regulations, the risk that imports from Canada will introduce BSE into the US herd is negligible—even though it is not exactly zero. The risk that imports will

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Circular Series DL&P 1-05, April 2005, at 11. http://www.fas.usda.gov/dlp/circular/2005/05-04LP/dlp5-03LP.pdf

86 70 FR 18259 (AR 12580).

87 EU, May 2004, Table 6.

88 Bradley and Liberski (2004) at 55, 64.

89 J.W. Wilesmith, G.A.H. Wells, J.B.M. Ryan, D. Gavier-Widen, and M.M. Simmons, A cohort study to examine maternally-associated risk factors for bovine spongiform encephalopathy, 141 *Veterinary Record* 239 (1997) at 239, 241–43 (AR 12506, 12508–12510).

90 Donnelly et al. (1997) at 783.

cause vCJD in humans is still more remote.

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

_____ Dated ____ June 2005.

Philip B. Stark