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FSIS Docket Room
Rm 102
300 12th Street SW
Washington, DC 20250-3700

01-027N
01-027N-5
Gene Bauston

RE: Harvard Study on Bovine Spongiform Encephalopathy Risk in U.S.

Dear Madam or Sir:

I am writing on behalf of Farm Sanctuary, a national non-profit organization that works to stop irresponsible agricultural practices. While we commend the USDA for commissioning a study by Harvard University on the risk of Bovine Spongiform Encephalopathy ("BSE") transmission in the U.S., we write today to express concern regarding the conclusions reached by the Harvard Study.

The Harvard Study fails to consider important research that brings to light possible differences of great consequence between BSE in the United States and the BSE that has wreaked havoc in many European countries. A study conducted by Dr. Richard Marsh of the Department of Animal Health and Biomedical Sciences at the University of Wisconsin, Madison provides compelling evidence that downed (non-ambulatory) cattle in the US may harbor a variant of BSE which produces different symptoms from those commonly associated with the disease. This theory is supported by independent research conducted by R.C. Cutlip. Failure to consider this possibility led the Study to underestimate the potential risk to the U.S. food supply by the practice of processing downed cattle for human consumption.

Missing Research

Dr. Marsh's research involved mink infected with the BSE-like disease transmissible mink encephalopathy ("TSE"). Since 1947, 11 U.S. mink farms are known to have been affected by TSE. In 1985, Dr. Marsh studied one such outbreak at a mink farm in Stetsonville, WI. The meat portion of the Stetsonville mink diet was composed almost exclusively of downer dairy cows. To test the possibility that cattle may have been the source of this TME outbreak, Marsh inoculated two Holstein cattle with the affected mink brain. Within two years, both animals became downed, and a brain examination revealed spongiform encephalopathies. Marsh then fed brain tissue from these cattle to minks, and found the minks exhibited a TME-like disease within seven months. Marsh concluded "[t]hese findings are compatible with the Stetsonville incident of TME being caused by feeding mink infected cattle tissue and they **suggest the presence of an unrecognized BSE-like disease in the United States** (emphasis added)." The Harvard study dismisses this finding, stating "there is no consensus over the

source of disease initiating [the mink farm TME] outbreaks."

Marsh's research does not stand alone as proof that U.S. downer cattle may harbor a BSE-variant that produces different symptoms from the European strain. This theory is bolstered by independent research showing that scrapie-infected U.S. sheep cause cattle to become "downed" rather than "mad." When "mad cow disease" (BSE) broke out in England, scrapie-infected sheep were widely suspected to be the cause. Scientists in the U.S. tested this theory by infecting U.S. cattle with scrapie-infected U.S. sheep. R.C. Cutlip et al reported, "[t]hus, undiagnosed scrapie infection could contribute to the 'downer cow' syndrome [in the U.S.]. . ." Cutlip's research is noted in the Study, but the possibility that a U.S. BSE strain produces different systems than the European strain is not taken into account in the Study's base case assumptions or even in its "worst case scenario" assumptions.

The significance of the Study's failure to recognize this demonstrated possibility is that the potential risk to the U.S. food supply posed by downer cattle is grossly understated.

Questionable "Assumptions"

The Study purports to calculate the number of cattle oral ID50s (the amount of infectious tissue that would on average cause 50% of cattle exposed to develop BSE) that would reach the U.S. food supply based on different numbers of BSE-infected cattle involved in a U.S. outbreak. However, this calculation is highly suspect, based on its questionable underlying assumptions.

The Study developed a simulation model, which it used to estimate the impact of introducing BSE into the U.S. cattle population, in terms of both animal health and human expose to contaminated food products. The model relies on numerous "base line assumptions" including birth rate, slaughter rate, rate of death by other causes, contents of cattle feed, the incubation period for BSE, and so on. Many of these assumptions are grounded in fact. USDA-FSIS and NASS collect statistics on birth rates and slaughter rates, for example. However, by necessity some assumptions have little or no factual basis, and the Study fails to adequately explain the rationale behind some of these chosen numbers.

One important base line assumption pertains to the likelihood that BSE will be detected in infected cows that are presented for slaughter. The Study's calculations assume that if there is a BSE outbreak, FSIS inspectors will almost always recognize affected cattle. The Study's assumption is that clinical BSE cases will be detected at ante-mortem inspection 90% of the time. This extraordinarily high detection rate is assumed in spite of scientific research demonstrating that BSE may already exist in the U.S., and that BSE-infected cattle may exhibit "downer" behavior rather than the European "mad cow" behavior. The U.S. does not test all downer cattle for BSE before processing them for human consumption. Only downers exhibiting traditional "mad cow" behavior such as head tilting, having head in abnormal position, staggering, circling, muscular tremors, etc. are tested. The Study admits that its estimate of ante-mortem detection is "highly uncertain." According to the Study, the probability that an animal will pass ante-mortem inspection depends upon the animal's age and on whether it has clinical signs of BSE. No consideration is given to the possibility that US BSE infected cattle will not display the classic "clinical signs" of BSE.

Given the fact that ante-mortem identification of BSE will depend solely upon visual inspection of the animal, and given the demonstrated possibility that the US strain of BSE does not produce the classic "mad cow" symptoms of the European strain, the Study's base line assumption that 90% of BSE cases will be spotted at the slaughterhouse door seems extraordinarily high.

The Study takes note of the presence of TSE variants in sheep; according to the study "sheep in the UK are known to carry several 'strains' of scrapie." However, the Study does not consider the possibility, demonstrated in Marsh's and Cutlip's work, that there is a different variant of BSE present in the US that produces different symptoms than those exhibited by BSE-infected cows in Europe. As a result, the Harvard model incorrectly assumes current US inspection methods are adequate to detect the disease's presence in the US. Also relevant to the likely rate of detection is the finding of a recent USDA survey of 61 US meat processing plants that the FSIS veterinary inspectors at 9 of the 61 plants were not even *aware* of FSIS policies and sampling requirements for central nervous system disorders in cattle.

Any calculation of the number of oral ID50s that would enter the human food supply in a BSE outbreak should take into account the possibility that a considerable number of infected downer cattle would likely be passed for slaughter. In recognition of the unreliability of its 90% detection estimate, the Study does include a "Best Case" and "Worst Case" scenario in addition to its "Base Case." However, the Study still assumes that at a *worst case scenario* inspectors would visually identify 50% of all BSE cases. By contrast, the best case scenario lists a 99% identification probability.

Inadequate Explanation of Assumptions

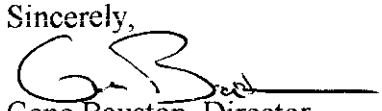
As previously stated, although some of the Study's base line assumptions are grounded in statistical fact, other assumptions, such as the ante-mortem inspection rate described above, have an inadequately explained factual basis. Another such assumption worth mentioning involves the Study's estimate of "mis-splitting frequency."

Mis-splitting is an incomplete cutting of the spinal column, which may leave pieces of spinal cord encapsulated in the vertebral column. It is relevant in estimating possible human impact because the spinal column is a known source of BSE infectivity, and mis-splitting allows the spinal cord from an infected animal to contaminate human food. According to the Study, the likelihood of a mis-splitting occurring depends upon an animal's size and weight, "calves are more likely to be mis-split than bulls or cows" and the proficiency of the person operating the saw. The Study uses base line mis-splitting assumptions are 5% and 8% for old and young cattle respectively. The only basis for these estimates, explained on p. 31-32 of Appendix 1, is "personal communications" with three industry officials and three FSIS officials. More explicit information regarding the basis of such an important estimate would be helpful. And, unlike the base line assumption regarding detection of BSE-infected cows on ante-mortem inspection, no best and worst case scenario cases varying this rate are considered. However, the Study does note in its Sensitivity Analysis (p. 88-89) that its conclusions are "sensitive to assumptions about contamination due to carcass splitting process."

Conclusion

The Harvard Study's failure to analyze the documented possibility of a BSE variant in the U.S. that produces symptoms of downer, rather than "mad," cows means the risk that downer cattle pose to the U.S. food supply is grossly understated. The Study overestimates the likelihood that BSE cases will be identified in ante-mortem inspection. Consequently, USDA policies developed in reliance upon this Study may fail to prescribe sufficient safety precautions, such as banning the use of downed animals for human consumption.

Sincerely,



Gene Bauston, Director
Farm Sanctuary, Inc.