

Response to Public Comments

on the

Harvard Risk Assessment of Bovine Spongiform Encephalopathy Update, October 31, 2005

INTRODUCTION

The United States Department of Agriculture's Food Safety and Inspection Service (FSIS) held a public meeting on July 25, 2006 in Washington, D.C. to present findings from the *Harvard Risk Assessment of Bovine Spongiform Encephalopathy Update, October 31, 2005* (report and model located on the FSIS website: http://www.fsis.usda.gov/Science/Risk_Assessments/index.asp). Comments on technical aspects of the risk assessment were then submitted to FSIS. Comments were received from Food and Water Watch, Food Animal Concerns Trust (FACT), Farm Sanctuary, R-CALF USA, Linda A Detwiler, and Terry S. Singeltary. This document provides itemized replies to the public comments received on the 2005 updated Harvard BSE risk assessment. Please bear the following points in mind:

- i. Efforts were made to extract individual comments from among the documents sent to the docket. Thus, for example, opening statements about the commenter backgrounds, objectives and histories of organizations with which they are affiliated, etc. are not included below. To view the full text of all comments, see the transcript of the July 25, 2006 FSIS public meeting: http://search.usda.gov/search?q=20060011&btnG=Search&sort=date%3AD%3AL%3Ad1&output=xml_no_dtd&ie=UTF-8&oe=UTF-8&client=default_frontend&proxystylesheet=default_frontend&sit e=FSIS_DOCKET_COMMENTS
- ii. Individual comments were not edited for style or grammar. In some instances, accompanying material, such as references, is not included with the comments. Again, to view the full text of comments, see the transcript of the July 25, 2006 public meeting at the web site provided above.
- iii. The purpose of this document is to respond to comments on *technical* aspects of the risk assessment, not those regarding

policy. As such, in instances where comments addressed policy, they are not addressed here.

As part of the response to these comments, FSIS conducted additional simulations using the Harvard BSE simulation model. The results from the revised risk assessment, made in response to these public comments, are provided in the corresponding document, *Supplement: Results from the Revised 2005 Harvard BSE Risk Assessment Update in Response to Public Comments*, which can be found in the FSIS docket (Docket # FSIS-2006-0011) or on the FSIS website (http://www.fsis.usda.gov/Science/Risk_Assessments/index.asp).

RESPONSE TO COMMENTS FROM R-CALF USA

Comment #1: The Harvard BSE Update Overlooks a Known, Direct Pathway of BSE Infectivity.

The Harvard BSE Update, while it analyzes risks of mislabeling, contamination, and misfeeding events associated with feed ban compliance and enforcement, does not address the risks inherent to the feed ban's inadequacies. The Harvard BSE Update is silent on a known risk inherent to the U.S. feed ban – the risk of feeding poultry litter to cattle. This known risk was acknowledged but not addressed in the Revised Harvard Risk Assessment, which recommended that this risk be further investigated.¹ Despite recognition by the Food and Drug Administration (FDA) in 2004 that a ban on the use of poultry litter in cattle feed should be imposed, no such prohibition exists and poultry litter remains exempt from the U.S. feed ban.²

The Revised Harvard Risk Assessment stated:

“. . . [T]he use of chicken litter as a feed supplement could pose a risk (Public Citizen 2001) that should be investigated further. It is possible that cattle-derived protein feed supplements administered to chicken could contain BSE infectivity, and that BSE infectivity could pass through chicken [sic] and become available in cattle feed supplemented with chicken litter.”³

The omission of any analysis of risk associated with the ongoing practice of feeding poultry litter to cattle, a practice previously recognized by both the FDA and the Revised Harvard Risk Assessment as a pathway of BSE infectivity, is sufficiently profound to render the Harvard BSE Update fundamentally deficient.

The occurrence of mislabeling, contamination, or misfeeding can only occur if handlers knowingly or inadvertently violate the law, and, therefore, the Harvard BSE Update has determined that the frequency of such occurrences should be presumed isolated. This presumption is reflected in the Harvard BSE Update given that the rate factor for the worst case scenario for any of these violations is no higher than 4 percent.⁴ However, the potential pathway of BSE infectivity from chicken litter identified in the Revised Harvard Risk Assessment is subject to no such constraints nor can it be afforded any such presumption. The practice of feeding poultry litter is lawful and is ongoing. The risk to

¹ *Evaluation of the Potential for Bovine Spongiform Encephalopathy in the United States*, Joshua T. Cohen, Keith Duggar, George M. Gray, Sylvia Kreindel, Harvard Center for Risk Analysis, Harvard School of Public Health, Revised October 2003 (hereinafter “Revised Harvard Risk Assessment”), at 32.

² *Expanded “Mad Cow” Safeguards Announced to Strengthen Existing Firewalls Against BSE Transmission*, U.S. Department of Health and Human Services, News Release, January 26, 2006.

³ *Revised Harvard Risk Assessment*, at 32.

⁴ *Harvard Risk Assessment of Bovine Spongiform Encephalopathy Update, Phase IA*, Joshua T. Cohen, Ph.D., George M. Gray, Ph.D., Harvard Center for Risk Analysis, October 31, 2005, (hereinafter “Harvard BSE Update”) at 18.

cattle from BSE-infected poultry litter should be considered direct and BSE infection should be presumed to occur each time BSE-infected poultry litter is fed to cattle.

Importantly, the Harvard BSE Update found that the most influential assumption in its sensitivity analysis regarding animal feed was the misfeeding rate, which could lead to an R_0 of 1 or more with 5% probability if the most pessimistic value is used for the assumption.⁵ Given the direct pathway of infectivity associated with the practice of feeding poultry litter to cattle, there is the possibility this pathway could result in the R_0 exceeding 1, suggesting a potential for the spread of BSE if it were introduced into the United States.

Because the Harvard BSE Update does not include any analysis of risk associated with the practice of feeding poultry litter to cattle, which poultry litter is acknowledged by the Revised Harvard Risk Assessment as possibly containing BSE infectivity, the Harvard BSE Update is incapable of accurately or otherwise realistically assessing the potential risk associated with the introduction of BSE into the United States.

Response: The model has been revised to characterize this pathway explicitly. In brief, the base case now assumes that 40% of prohibited MBM produced by either mixed or prohibited-only renderers is used in poultry feed. The model has also been modified so that 1% of chicken litter is recycled back to cattle feed. This modification was made based: (i) on FSIS' communications with the U.S. Department of Agriculture, Animal Plant Health Inspection Service (APHIS), the Environmental Protection Agency and the Food and Drug Administration, Center for Veterinary Medicine (FDA/CVM); and (ii) data on estimated poultry litter used in cattle feed from the U.S. Poultry & Egg Association analysis of a 2000-2001 survey of 16,000 poultry growers (personal communication with Kevin Custer, American Protein Incorporated, November 15, 2005).

This explicit inclusion of the poultry litter pathway in the model (along with other changes to the base case, including less optimistic assumptions regarding ante mortem inspection) has increased the estimated mean total potential human exposure to the BSE agent over 20 years following the hypothetical introduction of 500 BSE-infected cattle into the U.S. from 3,800 cattle oral ID_{50} s (original base case described in the October 2005 report) to 6,600 cattle oral ID_{50} s in the revised base case prepared along with these responses to public comments. It increased the average number of new BSE cases in the U.S. from 180 over 20 years (original base case described in the October 2005 report) to an average of 200 new BSE cases in the U.S. over 20 years (base case prepared along with these responses to public comments).

A sensitivity analysis was conducted to further evaluate the impact of the effect of feeding poultry litter to cattle. As discussed above, there is a consensus among FDA, FSIS and APHIS that the estimated percentage of poultry litter fed to cattle nationwide, based on available data, is likely 1%. If however, it is assumed that 5% of chicken litter is recycled back to cattle feed, then the revised 2005 Harvard BSE Risk Assessment

⁵ *Harvard Risk Assessment of Bovine Spongiform Encephalopathy (BSE)*, Slide Presentation, Joshua T. Cohen, Ph.D., July 25, 2006, Slide 45.

Update predicts an increase in the number of cattle infected in the U.S. over 20 years from the base case mean value of 200 to 420. Total potential human exposure increases from a mean value of 6,600 cattle oral ID₅₀s to 7,500 cattle oral ID₅₀s over 20 years.

Under both the modified base case scenario and the sensitivity analysis, R₀ remains less than 1 with high probability (> 95%), indicating that it is very probable that the prevalence of BSE would decrease over time after its introduction into the U.S.

Comment #2: The Harvard BSE Update Understates the Significance of Cross-Contamination in the Spread of BSE.

The Harvard BSE Update acknowledges that both it and its predecessor studies suggest “. . . that cross-contamination of MBM and feed production lines is a relatively minor factor in the spread of BSE.”⁶ This statement suggests that the assumptions underpinning the base case, as well as the variables associated with subsequent sensitivity analyses, all presume that cross-contamination is a relatively minor factor in the spread of BSE. Recent facts, however, show that cross-contamination has been a significant factor in the spread of BSE in Canada.

The recent investigation completed by the Canadian Food Inspection Agency (CFIA) of the 50-month old BSE-infected Canadian cow that died on July 2, 2006, reveals that contamination has likely occurred between ruminant and non-ruminant feed in Canada. The investigation report states:

Considering the feeding regime on the farm and specific production records reviewed, the most likely source of exposure to BSE infectivity appears to be the heifer ration referred to above, which could have become contaminated by prohibited material from the non-ruminant ration produced immediately before it. Because of incomplete or absent documentation, the possibility of cross-contamination during transportation being a contributing factor could not be ruled out.⁷

Moreover, the CFIA news release accompanying the issuance of the foregoing report additionally stated:

Nonetheless, the extremely small infective dose of BSE means that even very limited opportunities for contamination may permit periodic cases. The emergence of such cases is common to almost every country reporting the disease.⁸

⁶ *Harvard BSE Update*, at 36.

⁷ *Report on the Investigation of the Seventh Case of Bovine Spongiform Encephalopathy (BSE) in Canada*, Canadian Food Inspection Agency, August 24, 2006, available online at <http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/ab2006/7investe.shtml>.

⁸ *BSE Investigation Reaches Conclusion*, Canadian Food Inspection Agency, August 24, 2006, available online at <http://www.inspection.gc.ca/english/corpaffr/newcom/2006/20060824e.shtml>.

This recent evidence shows that the risk of contamination should be considered a significant factor in the spread of BSE. This evidence is further reinforced by the epidemiological investigations conducted for the earlier BSE detections in Canada. The BSE infection of the 71 month-old cow detected in Canada in April 2006 was attributed to cross-contamination of ruminant and non-ruminant feed by the CFIA:

The findings of this investigation indicate that compliance with the 1997 feed ban regulations was largely achieved through adoption of dedicated manufacturing facilities. Despite this, it is evident that opportunities for cross-contamination remained where conveyances and equipment were cross-utilized.⁹

The CFIA also suspected cross-contamination as a factor in the BSE infection of the 69 month-old cow detected in Canada in January 2006:

However, the findings indicate that a particular calf grower ration could have become contaminated during either manufacture or distribution. Furthermore, investigators could not rule out the somewhat remote possibility of residual pre feed ban materials persisting on the farm.¹⁰

Cross-contamination was implicated as a source of infection for each of the three Canadian cows detected in 2006 that were born after Canada implemented its 1997 feed ban. Based on the nine BSE cases detected in indigenous Canadian cattle since 2003, cross-contamination was officially implicated as a source of infectivity in a full one-third of all Canadian cases. This fact belies the Harvard BSE Update's conclusion that cross-contamination is a relatively minor factor in the spread of BSE.

To the extent that the base case of the Harvard BSE Update and its predecessor reports are underpinned by the assumption that cross-contamination is a relatively minor factor in the spread of BSE, any output from the models, conclusions, and predictions drawn therefrom will grossly understate the significance of cross-contamination in the spread of BSE, as well as the overall risk that BSE will spread if introduced into the United States.

R-CALF USA is concerned that this erroneous conclusion, perpetuated in this and previous Harvard reports, has the affect of misleading policy-makers into falsely believing that scientific evidence suggests it is unnecessary to require either dedicated facilities or dedicated production lines for plants producing ruminant feed, either in the United States or in countries from which the United States imports cattle and beef.

R-CALF USA recommends that the Harvard BSE Update be revised to reflect that cross-contamination of ruminant and non-ruminant feed in feed production lines is a significant factor in the spread of BSE.

⁹ See *BSE in North America*, Latest News, Canadian Food Inspection Agency, available online at <http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/bccb2006/5investe.shtml>.

¹⁰ *Id.*

Response: The 2005 Harvard BSE Risk Assessment Update does not assume that cross-contamination is a minor factor in the spread of BSE. Rather, that statement is a conclusion implied by the risk assessment results. The role of cross-contamination identified in this risk assessment stems from assumptions regarding cross-contamination rates. Those rates are based on data described in section 2.2.4 of the 2005 Harvard BSE Risk Assessment Update.

The 2005 Harvard BSE Risk Assessment Update's conclusion that cross-contamination is a minor contributor to the spread of BSE is not the same as a conclusion that cross-contamination does not contribute to the spread of BSE. The 2005 Harvard BSE Risk Assessment Update conclusion that the quantitative contribution of cross-contamination is minor is not inconsistent with the conclusion that cross-contamination might be responsible for some of the specific cases of BSE detected in Canada.

Comment #3: The Harvard BSE Update's Prediction that Removing SRMs Reduces Human Exposure by 99 Percent on Average Is Not Supportable.

The Harvard BSE Update does not purport to lessen the risk factors associated with any specified risk material (SRM). In fact, the Harvard BSE Update has inputted additional at-risk tissues not previously contemplated by the Revised Harvard Risk Assessment. These new at-risk tissues include tonsils and bone-in cuts of beef from animals 24 months of age or over. Further, the Harvard BSE Update does not purport to change the effectiveness of the SRM removal procedures previously established in the Revised Harvard Risk Assessment. Consequently, the effectiveness of SRM removal should not change from the rate determined in the Revised Harvard Risk Assessment, and if it changes at all, it should be less effective given the addition of new, potentially infectious tissues.

The Revised Harvard Risk Assessment found that:

Prohibiting the rendering of animals that die, potentially from BSE, prior to being sent to slaughter (*i.e.*, animals that "die on the farm") substantially reduces the potential for contamination of cattle feed, decreasing the average predicted additional cases of BSE following introduction of ten infected cattle by more than 80%. Implementation of a UK style ban on specified risk material (*e.g.*, spinal cords, brains, vertebral columns) from both human food and animal feed reduces the predicted number of additional BSE cases in cattle by almost 90% and potential human exposure by 95%.¹¹

The Harvard BSE Update, however, concludes that "Removing high risk tissues, often called specified risk materials or SRMs, from animals over 30 months of age reduces potential human exposure by more than 99% on average."¹² This conclusion is suspect. Because potential human exposure is dependent on the predicted number of additional

¹¹ *Revised Harvard Risk Assessment*, at x.

¹² *Harvard BSE Update*, at 36.

BSE cases in cattle, and because the United States does not ban SRMs from animal feed, which would minimize infectivity in cattle caused by contamination, mislabeling and misfeeding, it is implausible that SRM removal alone could achieve a higher rate of effectiveness (99%) than that predicted when a more stringent SRM ban, which also includes a ban from animal feed, is in place (95%).

This incongruent finding is heightened by the Harvard BSE Update's related conclusion as to the effectiveness of modeling a "UK style" SRM ban that includes SRMs in cattle over 12 months of age and prohibits SRMs in animal feed. It states in regard to these additional measures, "Our evaluation suggests that this measure would reduce potential human exposure by more than 99% and the number of new cases by 80% relative to the base case."¹³ This stands in sharp contrast to the Revised Harvard Risk Assessment's conclusion above that contains a 90% to 95% relationship between the number of additional BSE cases in cattle and a reduction in human exposure. It is counterintuitive that a less effective reduction in additional BSE cases in cattle would improve the effectiveness in reducing human exposure.

Adding even further skepticism for the appropriateness of predicting that SRM removal would reduce human exposure by 99 percent is the recent study completed by the FSIS that evaluated mitigation options using the scientific findings of the Revised Harvard Risk Assessment.¹⁴ The FSIS concluded that the combined SRM and AMR (automated meat recovery) rules implemented by FSIS can reduce human exposure to BSE by about 80 percent.¹⁵

The three aforementioned studies provide contradictory conclusions regarding what is perhaps the most important question to be asked by policy-makers regarding the level of protection needed to prevent human exposure to BSE. Consequently, FSIS should carefully and thoroughly reexamine this conclusion and provide a full explanation that describes the differing assumptions used in each of the three studies that led to the three different conclusions, as well as a justification for any assumptions used to arrive at a new effective rate for SRM removal.

Response: The comment does not consider all the differences between the SRM bans evaluated in these two reports, including the 2005 SRM ban's exclusion from human food of vertebral bone from animals 30 months of age or older. That exclusion was not included in the SRM ban analyzed in the 2003 Harvard BSE Assessment. The detailed results from Appendix 2B of the 2005 Harvard BSE Risk Assessment Update indicate that more than 99% of the infectivity from beef on bone potentially available for human consumption comes from animals 30 months of age and older. In that report's base case, infectivity from beef on bone contributes more than 20% to potential human exposure to

¹³ *Ibid.*

¹⁴ See *Preliminary Analysis of Interim Final Rules and an Interpretive Rule to Prevent the BSE Agent from Entering the U.S. Food Supply*, USDA Food Safety and Inspection Service, at 47, attached hereto as Attachment A.

¹⁵ *Id.* at 56. 57, attached hereto as Attachment A.

the BSE agent. Thus, the restriction on use of beef on bone from animals at least 30 months old for human food is important.

In short, the 2003 Harvard BSE Assessment considered a ban on the use of SRMs in both human food and animal feed, whereas the ban considered in the 2005 Harvard BSE Risk Assessment Update (the “USDA B” scenario) restricts use of SRMs in human food only. Therefore, the SRM ban considered in the 2005 Harvard BSE Risk Assessment Update had a more limited predicted impact on the spread of disease among animals than did the SRM ban considered in the 2003 Harvard BSE Assessment.

The ban considered in the 2005 Harvard BSE Risk Assessment Update applies to a wider range of tissues. In particular, it applies to bone-in-beef, whereas the SRM ban considered in the 2003 Harvard BSE Assessment did not. It is the ban on bone, which is unique to the 2005 Harvard BSE Risk Assessment Update, which leads to a greater relative impact on human exposure.

Results from the revised 2005 model (which explicitly includes the poultry litter pathway and less optimistic assumptions regarding ante mortem inspection, as previously discussed) show that a ban on the use of SRMs in human food from animals over 30 months of age reduces total estimated potential human exposure to the BSE agent over 20 years from 6,600 cattle oral ID₅₀s in the base case to 20 cattle oral ID₅₀s, consequent to a hypothetical introduction of 500 BSE-infected animals into the U.S. Again, it indicates that ban on the use of SRMs in human food almost completely eliminates potential human exposure.

Comment #4: The Harvard BSE Update Omits Significant Scientific Findings Regarding BSE Tissue Infection and Should be Revised.

The Harvard BSE Update does not incorporate or mention the additional bovine tissues that researchers have found to harbor BSE infectivity. A German study completed last year by Buschmann and Gruschup examined tissues from a cow naturally infected with BSE and found that the facial nerve and sciatic nerve of the BSE-infected cow contained sufficient BSE infectivity to cause BSE infection.¹⁶

The Animal and Plant Health Inspection Service (APHIS) was previously presented with this study but made a factual error when it improperly discounted its significance in its final rule on the importation of whole cuts of boneless beef from Japan. Therein APHIS stated:

Given these factors, APHIS has determined that the finding of BSE infectivity in facial and sciatic nerves of the transgenic mice is not directly applicable to cattle naturally infected with BSE. Therefore, we do not

¹⁶ See Anne Buschmann and Martin H. Gruschup, *Highly Bovine Spongiform Encephalopathy–Sensitive Transgenic Mice Confirm the Essential Restriction of Infectivity to the Nervous System in Clinically Diseased Cattle*, *The Journal of Infectious Diseases*, 192:934-42, September 1, 2005, Attached hereto as Attachment B.

consider it necessary to make any adjustments to the risk analysis for this rulemaking or to extend the comment period to solicit additional public comment on this issue.¹⁷

It was incorrect for APHIS to state that the infectivity was found in the facial and sciatic nerves of the transgenic mice. The facial and sciatic nerves were harvested from the cow naturally infected with BSE and the transgenic mice, which were used as a bioassay model, developed infectivity from those bovine tissues.¹⁸

Before FSIS uses the Harvard BSE Update as support for policy decisions that would either relax current BSE mitigations or forestall implementation of proposed mitigations, the recent scientific findings of BSE infectivity in the sciatic nerve and facial nerve of bovines naturally infected with BSE should be fully incorporated and integrated into the Harvard BSE Update.

Response: A sensitivity analysis conducted as part of the 2003 Harvard BSE Assessment showed that even an assumed doubling of infectivity in the carcass relative to the base case would increase disease spread and potential human exposure only modestly (see 2003 Harvard BSE Assessment, Appendix 3A, Section 2.2.1a and compare with Appendix 3A, Section 1). Therefore, the findings of this study would not alter the results of the 2005 Harvard BSE Risk Assessment Update.

In addition, the study in question refers to a finding in an animal “showing severe symptoms of late-stage clinical BSE, such as extreme nervousness, ataxia, and loss of balance” (Buschmann and Groschup article cited by commenter, p. 937). Because these clinical signs are likely to be detected on ante mortem inspection, such an animal is highly unlikely to enter the human food supply to contribute to potential human exposure.

Comment #5: The Harvard BSE Update Assumes that BSE Testing Will Be Used to Enhance Food Safety – A Proposition that is Inconsistent with USDA’s Practices and Policies.

The Harvard BSE Update suggests that its base case assumes that animals that have reached the clinical stage of disease and display clinical signs consistent with BSE would be tested for the BSE agent, and the carcasses of all animals testing positive would be destroyed.¹⁹ In regard to animals that have reached the clinical stage of the disease, the Harvard BSE Update states, “That is, as is effectively assumed in the simulation, the tissues from such animals could not be used in either human food or in animal feed.”²⁰

¹⁷ *Importation of Whole Cuts of Boneless Beef from Japan*, 9 CFR Part 94 [Docket No. 05-004-2] RIN 0579-AB93, Federal Register, Vol.70 No. 239 (December 14, 2005), at 73906.

¹⁸ See Anne Buschmann and Martin H. Gruschup, *Highly Bovine Spongiform Encephalopathy–Sensitive Transgenic Mice Confirm the Essential Restriction of Infectivity to the Nervous System in Clinically Diseased Cattle*, *The Journal of Infectious Diseases*, 192:934-42, September 1, 2005, Attached hereto as Attachment B.

¹⁹ *Harvard BSE Update*, at 10, 11.

²⁰ *Id.* at 11.

Thus it appears that the Harvard BSE Update relies upon BSE testing as a mitigation measure to support its assumption that the carcasses of all animals that have reached the clinical stage of BSE would be destroyed and completely removed from human food and animal feed, thereby presenting no risk of potential contamination to either human food or animal feed. However, the use of BSE testing as a mitigation measure is inconsistent with USDA's claim recently made in its final rule on the importation of whole cuts of boneless beef from Japan. In that rule, USDA stated, "A statistically and epidemiological valid surveillance plan is crucial to monitoring the success of risk mitigation measures, such as a feed ban, but surveillance is not a mitigation measure."²¹

USDA has even more recently reinforced its position that its BSE testing program is not a mitigation measure. The USDA stated in an agency news release on July 20, 2006:

BSE surveillance is not a food safety program. Human and animal health is protected by a system of interlocking safeguards, including the removal of specified risk materials - those tissues that studies have demonstrated may contain the BSE agent in infected cattle, along with the U.S. Food and Drug Administration's 1997 ruminant to ruminant feed ban.²²

In addition to USDA's opposition to the use of BSE testing for purposes of removing potentially infected carcasses from either human food or animal feed, i.e., as a risk mitigation measure, the USDA Office of Inspector General (OIG) found that there were inherent limitations in the USDA surveillance program in identifying and testing high-risk cattle.²³ The OIG report issued in January 2006 stated:

The U.S. program is voluntary and sampling is not random. The success of the program depends on the cooperation of industry and a variety of other conditions, including some that differ across geographical areas and other demographic attributes of the U.S. herd. Therefore, compared to the Europeans, USDA exerts less control over which animals can be tested for BSE, and is generally less able to assure that those tested represent the herd, their surveillance stream, or their age group within each surveillance stream.²⁴

Due to the combination of the voluntary nature of the USDA BSE surveillance program, along with USDA's opposition to using BSE as a mitigation measure, it cannot be assumed that carcasses of all animals that have reached the clinical stage of BSE would

²¹ *Importation of Whole Cuts of Boneless Beef from Japan*, 9 CFR Part 94 [Docket No. 05-004-2] RIN 0579-AB93, Federal Register, Vol.70 No. 239 (December 14, 2005), at 73914.

²² *USDA Announces New BSE Surveillance Program*, U.S. Department of Agriculture, Release No. 0255.06, July 20, 2006, available online at <http://www.usda.gov/wps/portal/usdahome?contentidonly=true&contentid=2006/07/0255.xml>.

²³ *Audit, Animal and Plant Health Inspection Service, Bovine Spongiform Encephalopathy (BSE) Surveillance Program – Phase II and Food Safety Inspection Service Controls over BSE Sampling, Specified Risk Materials, and Advanced Meat Recovery Products – Phase III*, U.S. Department of Agriculture, Office of Inspector General, (hereinafter "OIG Audit") at iii.

²⁴ *OIG Audit*, at 8.

be destroyed and completely removed from human food and animal feed. The use of such an unsupported assumption would grossly understate the risks associated with carcasses of animals that have reached the clinical stage of BSE.

R-CALF USA recommends that this assumption be revised downward to reflect the fact that the USDA does not use BSE testing to remove potentially infected animals from the food supply and that the voluntary nature of the USDA testing program does not ensure that all animals that have reached the clinical stage of BSE would be tested or otherwise diagnosed with BSE.

Response: This description is inconsistent with the model's operation. The model assumes that as part of the ante-mortem inspection process, slaughter facility inspectors attempt to identify animals showing clinical disease signs and that only animals detected visually as displaying signs consistent with BSE are further tested for the presence of the BSE agent. The model explicitly allows for the possibility that animals showing signs of disease will not be visually detected (and therefore will not be subject to further testing for the presence of the BSE agent). That is, the model does not assume that all clinical animals are necessarily prevented from entering the human food supply (see response to R-CALF Comment #6, below). Because the model does not assume any sort of active surveillance program, the statement that the USDA BSE surveillance program is voluntary and not random is not relevant.

Comment #6: The Harvard BSE Update's Base Case Overstates the Proportion of Animals With Clinical Signs That Would Be Detected By Inspectors.

R-CALF USA agrees with the recommendations presented by Peer Reviewer 1 regarding the necessity to significantly reduce the assumption that ante-mortem inspectors will detect 90% of animals with clinical signs. As indicated by Peer Reviewer 1, this detection rate is inconsistent with data from the United Kingdom (UK) and the European Union (EU). In addition, this detection rate is inconsistent with data from the cases of BSE detected in indigenous Canadian cattle. Both the Canadian BSE case detected in Canada in May 2003 and the Canadian-origin BSE case detected in the U.S. in December 2003 were presented for, and were slaughtered, without being identified as suspects at ante-mortem inspection. The CFIA stated in its report of the latter case only that: "Like the first Canadian case, the animal exhibited signs that placed it in one of the surveillance classes recommended by the OIE."²⁵

Based on Harvard's response to Peer Reviewer 1 regarding this issue, it appears that Harvard acknowledges that its base case may overstate the ability of ante-mortem inspectors to detect animals with clinical signs for BSE. Harvard suggests that Sensitivity Analysis 5 addresses this issue because it lowers the detection rate to reflect that 50% of ambulatory clinical cases and 25% of non-ambulatory clinical cases are detected. The results of this sensitivity analysis increases the mean number of BSE infections from 180

²⁵ *Canada's Assessment of the North American BSE Cases Diagnosed from 2003 to 2005 (Part II)*, Canadian Food Inspection Agency, January 23, 2006, available online at <http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/eval2005/evale.shtml#app>.

to 190 animals over 20 years and increases human exposure by about 50%. Harvard suggests that this changed assumption could be applied to other scenarios by increasing the projected human exposure in each by approximately 50 percent.²⁶

Given the validation of the concern that the base case overstates the detection rate of animals with clinical signs as well as the substantial increase to projected human exposure resulting from the lower rates, the Harvard BSE Update's base case should, itself, be revised to reflect the lower, i.e., 50% and 25%, rates of detection and the sensitivity analysis should likewise be revised to reflect the possibility of even more pessimistic probabilities. This important revision to the base case will add credibility and realism to the Harvard BSE Update and will prevent potential misunderstandings on the part of policy-makers about the predicted risks of introducing BSE into the United States.

Response: The base case has been revised. It now assumes that ante mortem inspection detects 50% of ambulatory animals with clinical BSE signs, and 25% of non-ambulatory animals with clinical BSE signs. A revised sensitivity analysis investigates the impact of assuming ante mortem inspection fails to detect any animals with clinical signs of BSE (see the results for the simulations conducted as part of the response to these comments).

Comment #7: The Harvard BSE Update Improperly Assumed a Likelihood of Smaller Exposures to BSE Infectivity than that Evidenced in the United Kingdom.

In the closing paragraph of the Harvard BSE Update, the authors offer remarks inferring that the BSE risks identified in the base case may be overstated based on a theory that cattle in the U.S. could be subject to much smaller exposures to BSE infectivity, which would lead to longer BSE incubation periods. Presumably, the theoretical conclusion that BSE incubation periods would be longer in the U.S. than in the UK influenced the development of Sensitivity Analysis 6, which doubled the incubation period from about 50 months in the base case to about 100 months. The Revised Harvard Risk Assessment postulated a median incubation period of 4.2 years, which is comparable to the mean incubation period of 4.2 years calculated from UK data.²⁷

The theory that the incubation period would be longer in the U.S. if additional BSE infectivity were to be introduced is contradicted by the incubation periods of the BSE cases detected in Canadian-origin cattle. Of the nine Canadian-origin BSE cases detected since 2003, seven died before reaching the age of 99 months. The ages of these seven cases at death were 50, 69, 70, 71, 80, 81, and 98 months.²⁸ The Revised Harvard Risk Assessment references studies that estimate that susceptibility to BSE infection peaks

²⁶ *Harvard BSE Update*, Appendix 4 – Revisions and Responses to Peer Review Comments, at 7.

²⁷ See *Bovine Spongiform Encephalopathy; Minimal-Risk Regions and Importation of Commodities, Final Rule*, U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Federal Register, Vol. 70, No. 2, January 4, 2005, at 470, 474.

²⁸ See *Canada's Assessment of the North American BSE Cases Diagnosed from 2003 to 2005 (Part II)*, Canadian Food Inspection Agency, January 23, 2006, available online at <http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/eval2005/evale.shtml#app>; See also *BSE in North America, Latest Information*, Canadian Food Inspection Agency, available online at <http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/situatione.shtml>.

when cattle reach the age of 1.31 years and between 0.5 and 1.5 years of age.²⁹ Subtracting 12 months from the age of each of the seven Canadian BSE cases referenced above, to factor the period during which these animals may have become infected, reveals potential incubation periods of 38, 57, 58, 59, 68, 69, and 86 months.

Given that over 75% of the Canadian-origin cattle with BSE died before reaching the age of 99 months, and the potential that their incubation periods were likely closer to 50 months than 100 months, the theory that the U.S. should expect a longer BSE incubation period than in the UK is unfounded. The Harvard BSE Update should be revised to eliminate any inference that the risks of BSE may be less than what its base case predicts because incubation periods would be longer in the U.S. than in the UK.

Response: R-CALF's comment refers to a remark made in the conclusion of the 2005 Harvard BSE Risk Assessment Update. That remark discussed the implications of one of the sensitivity analyses. In particular, Sensitivity Analysis #6 showed that if the incubation period for BSE were assumed to be longer than what is assumed in the base case, then the spread of BSE would be less than what the model predicted for the base case. The last paragraph in the 2005 Harvard BSE Risk Assessment Update stated that it is plausible that the incubation period in the U.S. is relatively long because BSE exposure levels in the U.S. are limited (compared to the UK) and there is evidence that incubation period is inversely related to exposure level.

It is important to note that we did not infer this possibility from the results of our analysis, as suggested by R-CALF's comment. Moreover, this line of reasoning does not at all affect the base case assessment described in the 2005 Harvard BSE Risk Assessment Update.

Finally, the data presented by R-CALF are not sufficiently compelling to dismiss the possibility that incubation periods for the U.S. are longer than they were in the UK. The R-CALF data are based on an extremely small sample of nine animals from Canada. The quantitative analysis offered by R-CALF does not support the hypothesis that a longer incubation period for the U.S. is implausible. Even for the seven animals that died before the age of 99 months, the incubation periods estimated by R-CALF have an average value of 62 months, or more than five years. Thus, retention of Sensitivity Analysis #6 remains justified.

Comment #8: The Harvard BSE Update Appears to Incorporate an Industry Practice into the Base Case that is Not Required in the U.S.

The Harvard BSE Update purports to revise the original BSE simulation model used in the Revised Harvard Risk Assessment with the assumption that the SRM ban would apply to dead stock as well as to cattle that went to slaughter. It is unclear if this means that the model would assume that SRMs from dead stock could not be included in the production of non-ruminant animal feed, such as feed for poultry. If the BSE simulation model does adopt this assumption, then the model's output regarding the potential spread

²⁹ *Revised Harvard Risk Assessment*, at 10.

of BSE would be understated because the USDA does not ban the use of SRMs from dead stock in the production of non-ruminant animal feed.

The FDA proposed a rule on October 6, 2005 that would ban SRMs obtained from dead stock for use in animal food or feed.³⁰ However, this proposed rule has not been finalized or otherwise implemented.

The implication of incorporating a ban on the use of SRMs from dead stock for animal feed within the Harvard BSE Update when such a ban is not required is that the simulation model would understate the level of infectivity available from missfeeding, contamination, mislabeling, and the more direct pathway of BSE infectivity – the practice of feeding poultry litter to cattle.

If R-CALF USA’s interpretation regarding this issue is correct, the Harvard BSE Update should not be used to support policy decisions that would relax existing BSE mitigation measures or forestall the implementation of additional BSE mitigations. Moreover, and again if R-CALF USA’s interpretation is correct, the Harvard BSE Update should contain a clear and prominent disclaimer that its base case inputs contain mitigation measures not presently implemented in the United States.

Response: This interpretation of the model is incorrect. The base case does not assume removal of SRMs, and only one of the scenarios in the analysis (Int Comm 1) assumes that SRMs are removed from dead stock. Therefore, the assertion that the model incorporates a practice that is not required in the United States is not accurate.

The comment applies to one of the scenarios included in this report, i.e., the “Int Comm 1” scenario described in Section 2.3.3 of the report. As explained in that section, the Int Comm 1 scenario represents a proposal made by the International Review Subcommittee of the Secretary’s Advisory Committee on Foreign Animal and Poultry Diseases. That proposal does indeed recommend the removal of SRMs from all animals, including those that die prior to being sent to slaughter.

Importantly, the analysis did not assume removal of SRMs from dead stock for any of the other scenarios, including the base case and the “USDA B” scenario. The USDA B scenario, which is relevant because it corresponds to measures implemented by FSIS in its 2004 interim final rule, excludes SRMs from human food, and only applies to animals 30 months of age and older slaughtered for human food. In particular, this scenario does not affect the handling of SRMs or any other materials from animals that die prior to slaughter.

Comment #9: The Scope of the Harvard BSE Update is Entirely Too Narrow to Support the Overreaching Conclusion of its Authors.

³⁰ *Substances Prohibited From Use in Animal Food or Feed; Proposed Rule*, Federal Register, Vol. 70, No. 193, October 6, 2005, at 58580.

According to the transcript accompanying the issuance of the Harvard BSE Update, the purpose of the Harvard BSE Update was *limited* to assessing risk “. . . associated with the introduction of BSE into the U.S. and to assess the impact of various risk management strategies.”³¹ Among their responses to concerns raised by Peer Reviewer 1, the authors further qualified the limited scope of the Harvard BSE Update stating, “Keep in mind that the purpose of this analysis has been to evaluate how different measures affect the spread of BSE in the U.S. following its introductions. It is not the purpose of this analysis to evaluate specific introduction scenarios.”³² And, again, when Peer Reviewer 1 recommended that a spatial risk assessment be conducted regarding risks that arise from previous imports of Canadian cattle and meat-and-bone meal, the authors stated, “Such an analysis is beyond the scope of this report.”³³

Thus, the scope of the Harvard BSE Update is much narrower than that of the original Harvard Risk Assessment, which was commissioned for the purpose of conducting a comprehensive investigation of the BSE risk in the United States.³⁴

Despite its much narrower scope, as substantiated by the authors’ acknowledged limitations and qualifications, the Harvard BSE Update nevertheless concluded:

Qualitatively, our finding here are the same as in our earlier analysis, with the results indicating that the spread of BSE in the U.S. cattle population would be limited, that BSE would be eradicated from the U.S. over time, and that potential human exposure to BSE-contaminated food would be low.³⁵

This conclusion overreaches the limited scope of the Harvard BSE Update. The Revised Harvard Risk Assessment identified two key mitigation measures that are most effective at reducing the spread of BSE in the United States.³⁶ These measures include 1) the ban on the import of ruminants from countries known to have BSE and 2) the feed ban.³⁷ However, the Harvard BSE Update does not factor in any risk associated with the United States’ relaxation of its ban on imports from countries with BSE. In 2005, the United States imported over 1 billion pounds of beef from Canada, a BSE-affected country; and since the 2005 reopening of the Canadian border to Canadian live cattle, approximately 1.2 million head of live cattle have been imported into the United States.³⁸ The United States also imports beef from Japan, another BSE-affected country. Any BSE risks

³¹ *Harvard Bovine Spongiform Encephalopathy (BSE) Risk Assessment Technical Meeting*, U.S. Department of Agriculture, Food Safety and Inspection Service, July 25, 2006, at 14.

³² *Harvard BSE Update*, Appendix 4 – Revisions and Responses to Peer Review Comments, at 9.

³³ *Id.* at 13.

³⁴ *Harvard Risk Assessment of Bovine Spongiform Encephalopathy (BSE) Update*; Notice of Availability and Technical Meeting, Federal Register, Vol. 71, No 133, July 12, 2006, at 39282.

³⁵ *Harvard BSE Update*, at 24.

³⁶ See Revised Harvard Risk Assessment at viii.

³⁷ See *Id.* at viii; See also *Id.* at 99.

³⁸ Livestock, Dairy, and Poultry Outlook, Cumulative U.S. Meat and Livestock Trade, Updated October 12, 2006, available online at http://www.ers.usda.gov/Publications/LDP/xlstables/cumulative_US_livestock_meat_trade.xls.

associated with these imports would be constant and ongoing, meaning the risks will persist year-after-year. Thus, the conclusions that BSE would be eradicated from the U.S. over time and that potential human exposure to BSE-contaminated food would be low cannot be supported by the Harvard BSE Update that does not consider this potentially persistent and cumulative risk.

R-CALF USA recommends that the conclusions and predictions contained in the Harvard BSE Update be revised to more accurately reflect the limited scope of the analysis. It is detrimental to the interests of the U.S. cattle industry to have a BSE risk analysis that draws overreaching conclusions as they tend to undermine our industry's credibility when such overreaching conclusions are later proven false. Already, empirical evidence has disproved a number of overreaching conclusions contained in the Revised Harvard Risk Assessment. For example, the ongoing BSE epidemic in Canada has rendered the following Harvard conclusions erroneous:

- “Our analysis finds that the U.S. is highly resistant to any introduction of BSE or a similar disease.”³⁹ However, BSE was introduced as evidenced by the December 2003 detection of a BSE-infected cow imported from Canada and the detection of two BSE cases in the United States.
- “These imports [referring to previously imported Canadian cattle] are extremely unlikely to pose a risk of introducing BSE to the U.S.”⁴⁰ However, the first case detected in the United States was an imported Canadian cow detected in 2003.
- “Because APHIS has banned the import of cattle and feed from countries in which the presence of native BSE has been documented (see Section 2.3.2), the import of even a single infected animal is not highly likely.”⁴¹ However, the detection of an imported Canadian cow with BSE in 2003 raises the question of whether adequate surveillance is being conducted in all countries from which we continue to import cattle or beef.

R-CALF USA recommends that the conclusions and predictions contained in the Harvard BSE Update be revised to reflect a strict, conservative standard regarding the interpretation of the risks associated with the introduction of BSE into the United States. Research on and understanding of the BSE disease and its epidemiology is quite nascent and the potential impact of underestimating BSE risks could be devastating to the domestic live cattle industry.

Response: This comment takes the statements in the 2005 Harvard BSE Risk Assessment Update out of context. The report is clear that it considers only a one-time finite introduction of BSE into the U.S. The conclusion quoted in the comment regarding the 2005 Harvard BSE Risk Assessment Update refers to that report's finding that following a one-time introduction, the prevalence of the disease would decrease over time, leading eventually to its elimination. The 2005 Harvard BSE Risk Assessment Update did not attempt to model the impact of continual introductions of BSE into the U.S. The U.S.

³⁹ Revised Harvard Risk Assessment, at vii.

⁴⁰ *Id.* at 23.

⁴¹ *Id.* at 84.

Department of Agriculture used a one-time introduction scenario because it determined that hypothesizing a continuous, low-level entry of BSE infectivity was unnecessary for evaluating BSE control measures within its purview.

Comment #10: R-CALF claims that the “empirical evidence has disproved a number of overreaching conclusions contained in the [2005] Revised Harvard Risk Assessment.” To support this claim, R-CALF identifies three statements in the 2003 Harvard BSE Assessment. R-CALF identifies three quotes to support the group’s contention. First, R-CALF claims that the introduction of BSE into the U.S. from Canada that was discovered in December 2003 contradicts the statement in the Executive Summary of that report that “Our analysis finds that the U.S. is highly resistant to any introduction of BSE or a similar disease.”

Response: This comment misrepresents the nature of the statement from the 2003 Harvard BSE Assessment. The text that follows the sentence quoted in the comment reads, “BSE is extremely unlikely to become established in the U.S. For example, in a hypothetical scenario in which ten cattle infected with BSE are imported into the U.S., on average only four new cases of BSE would occur. Moreover, the disease is virtually certain to be eliminated from the country within 20 years after its introduction.”

Comment #11: R-CALF claims that the following statement from the 2003 Harvard BSE Assessment has been subsequently shown to be inconsistent with empirical observations: “These imports [referring to previously imported Canadian cattle] are extremely unlikely to pose a risk of introducing BSE to the U.S.”

Response: This statement is not based on the analysis conducted in the report using the BSE simulation model. The statement, which was taken from Section 2 of the 2003 report, is part of the report’s literature review. It is not based on the analysis in that report. Any inconsistency between this claim and subsequent observations therefore does not cast doubt on the analytical approach used, risk assessment outputs, or corresponding conclusions reached in the 2003 risk assessment report that “... measures taken by the U.S. government and industry make the U.S. robust against the spread of BSE to animals or humans should it be introduced into this country.”

Comment #12: R-CALF claims that the following statement from the 2003 Harvard BSE Assessment has been subsequently shown to be inconsistent with empirical observations: “Because APHIS has banned the import of cattle and feed from countries in which the presence of native BSE has been documented (see Section 2.3.2), the import of even a single infected animal is not highly likely.”

Response: This statement is not based on the analysis conducted in the risk assessment report using the BSE simulation model. The statement is qualitative and was made to provide context for the introduction scenario used in the report’s base case.

RESPONSE TO COMMENTS FROM TERRY S. SINGELTARY SR.

Comment #1: SINCE the first Harvard BSE Risk Assessment was so flawed and fraught with error after the PEER REVIEW assessment assessed this fact, how do you plan on stopping this from happening again, will there be another peer review with top TSE Scientists, an impartial jury so-to-speak, to assess this new and updated Harvard BSE/TSE risk assessment and will this assessment include the Atypical TSE and SRM issues?

Response: The original (October 2003) and the revised (October 2005) Harvard BSE risk assessments underwent external peer review. Subsequently, revisions were made to the analysis. In the most recent review, the most significant revisions have been: 1) the addition of explicit modeling of the poultry litter pathway for the potential recycling of bovine protein into cattle feed; and 2) a decrease in the assumed effectiveness of *ante mortem* inspection in the identification of animals with BSE.

Comment #2: WITH A RECENT NATION WIDE MAD COW FEED BAN RECALL in the past few months that consisted of some 10,878.06 TONS, then another Mad Cow feed ban warning letter in May, IT should seem prudent to ask why our feed bans continue to fail in 2006, and continue to fail today?

Response: This question about feed bans is a matter for policy. As such, it is not addressed in this response.

Comment #3: WHY still now only partial ruminant feed ban, with the fact that now we seem to have 3 cases of nvCJD to humans i.e. human bovineTSE that were responsible from blood, and the fact the last 2 mad cows documented in the USA were that of an Atypical strain, would it not seem prudent to remove blood as well from ruminant feed? WOULD it not seem prudent to improve and expand the SRM list now? as per your own thinking; If transmission occurs, tissue distribution comparisons will be made between cattle infected with the atypical BSE isolate and the U.S. BSE isolate. Differences in tissue distribution could require new regulations regarding specific risk material (SRM) removal.

Response: This comment pertains to policy. As such, it is not addressed here.

Comment #4: WHAT does USDA/FDA ET AL intend to do about the risks of atypical BSE/TSE in cattle now that infectivity shows in tissue samples other than CNS in Japan, the fact now that the last Texas mad cow and that last mad cow in Alabama were indeed of the atypical strain, the fact that the studies long ago in Mission, Texas of USA sheep scrapie transmission to the USA bovine, which proved an 'atypical tse' in the USA bovine, the fact also that USDA/FDA are still floundering on the last SRM regulations, but with the BASE strain now in cattle that is not similar to nvCJD, but very similar to the sporadic CJD, and sporadic CJD has tripled in the last few years in the USA. WHAT do you plan to do to protect human health from these atypical strains of TSE, in relations to SRMs ?

Response: The BSE risk assessment simulation model characterizes the disease history of BSE, including the agent's spread within the body of the animal over time. It also quantifies the agent's persistence during the feed manufacture process, and ultimately the agent's ability to cause disease in other exposed animals. There is no definitive evidence that these properties differ substantially for atypical BSE strains, compared to the typical BSE agent.

Comment #5: THE 2004 Enhanced BSE surveillance program, that tested all those cows, but then we found just how terribly flawed the program was, from testing protocols, to testing the most likely to have BSE i.e. high risk, to the geographical distribution of the testing and high risk areas, to letting the tissue samples of one mad cow sit on a shelf for 7+ months and then having to have an act of Congress to ever get that cow finally confirmed, to that other Texas mad cow they decided to not even bother testing at all, just rendered that very suspect cow, too suspect to test evidently, back to that Alabama mad cow that they could only give a guess as to age with dentition where we all know that the age of that cow was so close to 10 years it could have been 9 years 7 months to 10 years 3 months, thus possibly being an BAPB i.e. USA 'born after partial ban', to all those rabies suspect cows that did not have rabies, and DID NOT get tested for BSE/TSE in that June 2004 enhanced surveillance program, even though the common lay person knows the suspect rabies negative cows are suppose to be BSE/TSE tested, how does one correct all these blatant failures and will they be corrected?

Response: This comment pertains to policy. As such, it is not addressed here.

Comment #6: WHAT happened to the test results and MOUSE BIO-ASSAYS of those imported sheep from Belgium that were confiscated and slaughtered from the Faillace's, what sort of TSE did these animals have?

Response: It is not clear how the test results referred to in this comment are relevant to the Harvard BSE Risk Assessment Update. Sheep were not considered in the risk assessment.

Comment #7: WHY is it that the Farm of the Mad Sheep of Mad River Valley were quarantined for 5 years, but none of these farms from Texas and Alabama with Atypical TSE in the Bovine, they have not been quarantined for 5 years, why not, with the real risk of BSE to sheep, whom is to say this was not BSE ?

Response: This comment pertains to policy. As such, it is not addressed here.

RESPONSE TO COMMENTS FROM FOOD ANIMALS CONCERNS TRUST (FACT)

Comment #1: FACT believes that the updated model has not incorporated the latest information on infectivity to BSE at very low doses. The United Kingdom Department for Environment Food and Rural Affairs (DEFRA) infectious dose experiment has shown that cattle can be orally infected with doses as low as 0.001 grams. As reported in the Hill Report 2005, one of fifteen calves (7%) fed a 0.001 gram dose was later shown to be infected. The Harvard Risk Assessment Model never explicitly states how large in grams is one ID₅₀. From the sources listed in the review, it appears that 0.1 gram of infected brain includes one ID₅₀. Based on this assumption about the size of an ID₅₀, using the Harvard Risk Assessment Model's linear dose-response function predicts only 0.5% risk of infection at the 0.001 gram dose (.01ID₅₀). Thus the model underestimates the likelihood of infection at the .001 gram level by 13%. The .001 gram infectious dose is the lowest level tested so it is unclear at what dose level no infectivity will be detectable. FACT recommends that the model be modified to include a dose-response function in four parts. For exposure to doses below 0.0001 gram (.001ID₅₀), there is no risk of infection. This is one order of magnitude smaller than the lowest known level of infectivity. For exposure from 0.0001 gram (.001ID₅₀) to 0.01 gram (.1ID₅₀) the chance of infection is 7%. For exposure greater than .01 gram, the chance of infection equals that of the current model increasing linearly to 100% at 2 ID₅₀s. For doses higher than 2 ID₅₀s, the risk would remain the same. This modified dose-response function would more accurately model the existing information on infectivity at very low doses.

The model's underestimation of the likelihood of infection by very low doses may explain why the model's results are inconsistent with the evidence from Europe indicating that "commingling" between prohibited and non-prohibited meat and bone meal can be a significant cause of new BSE cases after a ruminant feeding ban is in place (SSC 2000). Peer reviewer #1 pointed this out in his comments to the updated model (Appendix 4, page 7) stating "I consider this element to continue to be underestimated." In their response, the reports authors stated that even with pessimistic assumptions about commingling examined in the October 2003 risk assessment, the effects on additional BSE cases were minimal. FACT believes that this points to a flaw in the model not that the risk from commingling is low. The current model does not accurately reflect expert scientific opinion on how BSE can continue to spread after a ruminant feed ban is in place. This model flaw may be resolved by correcting the dose-response function at very low doses. The model's unrealistic assumptions about the risk of infection from low doses is also likely to lead to an underestimation of the impact of requirements for dedicated production lines as examined in the 2005 update.

Response: The comment claims that although the current model's dose response function is realistic at exposures exceeding 0.01 cattle oral ID₅₀s, the report of 1 in 15 BSE-infected animals exposed to 0.001 g of brain from a clinical BSE case indicates that the model's linear dose response relationship understates infection risk at lower levels of exposure. This deviation is unlikely to be important for the following reasons:

- First, FSIS has evaluated the impact of using an alternative dose-response relationship that assumes a higher risk of infection at low doses (10,000 iterations). This alternative scenario assumed that infection risk increases linearly from zero at no exposure to 7% at 0.001 cattle oral ID₅₀s (0.0001 g brain). Above that level of exposure, this scenario assumed that risk increases linearly until it reaches unity at an exposure of 2 cattle oral ID₅₀s (0.2 g brain). The number of new BSE cases increased from the base case mean of 200 to 280 in this alternative scenario. Total human exposure increased from the base case mean of 6,600 cattle oral ID₅₀s over 20 years to 6,700 cattle oral ID₅₀s in the alternative scenario. R₀ remains less than 1 with high probability (>95%), indicating that it is very probable that the prevalence of BSE would decrease over time after its introduction into the U.S.

- Second, the low dose data from the experiments cited by the commenter also indicate that exposures in these ranges are also associated with much more extended incubation periods. For example, while the mean incubation period for animals exposed to 1 ID₅₀ was 60 months, the corresponding duration for animals exposed to 0.01 ID₅₀s was 283 months (Gerald Wells, unpublished data). Because the quantity of the infective agent remains at several percent or less of its clinical total until relatively close to the end of the incubation period, cattle exposed to very low levels of infectivity are unlikely to ever pose a large risk. The alternative scenario described in the preceding paragraph did not reflect this difference in the incubation period.

- Third, it is not true that the Harvard risk assessment results are inconsistent with the experience with BSE in Europe. The Harvard analysis does not claim that the risks associated with commingling are zero, only that they are small relative to other potential sources of contamination (in particular, misfeeding). The commenter does not present any quantitative data indicating that the commingling risks predicted by the Harvard model are inconsistent with data from Europe.

- Finally, as described in the October 2003 Harvard BSE risk assessment report, model predictions for Switzerland during the 1990s were within a factor of two of the observed results (predicted BSE cases were approximately 50% of the observed number of cases). Given the substantial data gaps inherent in characterizing the Swiss agricultural system, this concordance provides comfort that the model's predictions are plausible.

Comment #2: FACT appreciates that FSIS has placed the actual model on the website as a DOS executable file. While this is commendable there is no associated documentation explaining how the model could be used. FACT recommends that some minimal instructions on running the model and interpreting results be included.

Response: Documentation for how to use of the revised 2005 Harvard BSE risk assessment update simulation model is available on the FSIS website: http://www.fsis.usda.gov/Science/Risk_Assessments/index.asp.

RESPONSE TO COMMENTS FROM FOOD AND WATER WATCH

Comment #1: While the conventional wisdom has been that BSE prions are found only in the central nervous system of infected animals, recent research indicates that BSE prions can migrate to other organs such as the liver and spleen. Limiting the risk assessment discussion to SRMs may be too restrictive to truly assess the magnitude of the risk to both the animal and human populations.

Response: Assuming some of the infectivity in an animal is present in the liver or spleen, rather than elsewhere would not change the total amount of infectivity that is recycled back to cattle.

In addition, the significance of the results from the article cited in the comment is limited. The article does not make claims regarding the amount of BSE potentially present in the spleen or liver; it is based on results from a mouse model of prion distribution; and it does not describe any measurements in cattle. The assumptions in the Harvard BSE model are based on measurements from cattle (see the October 2003 report).

Comment #2:

There are mixed signals coming from USDA on the policy of banning non-ambulatory cattle from being slaughtered for the human food supply. Secretary Mike Johanns has indicated in several different forums over the past 20 months that he is re-evaluating the policy of banning downer cows from being accepted at slaughter facilities. As recently as July 20, 2006, the Secretary stated that he is still studying the issue. Yet, his Under Secretary for Food Safety has argued that the downer ban has been instrumental in removing the risk of BSE entering the human food supply. This continued ambivalence is very worrisome and a change in policy could punch a major hole in the Harvard Risk Assessment model, which assumes that the downer ban will continue.

Response: The base case does not assume that non-ambulatory cattle cannot be used in food (see Section 2.2.2 of the October 2005 Updated Harvard BSE Risk Assessment).

Comment #3: The recent announcement by Secretary Johanns that the agency would drastically scale back the BSE surveillance program is very alarming for a number of reasons. Even when the agency was doing significantly more tests, the program was far from adequate. Because the testing program is voluntary, it does not give a representative picture of how prevalent the disease is in the U.S. herd. Another weakness has been documented by the USDA's Inspector General's office, which criticized the program's sampling protocols. Finally, we are very concerned about the lack of consistent protocols used to test tissues from suspect animals, and the influence exercised by top administrators at the Animal and Plant Health Inspection Service (APHIS) to dissuade agency scientists from using all available tests to confirm BSE in tissue samples. These design flaws, combined with a reduced amount of testing, significantly weaken the ability of the surveillance program to provide an accurate picture of the extent of BSE in the U.S. The updated risk assessment should reflect the limited ability of the surveillance program to detect the disease, but currently does not.

Response: The updated risk assessment model does not assume the implementation of any active surveillance program. It only assumes that animals discovered during normal *ante mortem* inspection displaying clinical signs consistent with BSE will be further evaluated. If follow-up testing reveals the presence of BSE, the model assumes material from that animal will not enter animal feed or human food supply.

Comment #4: Despite an announcement in January 2004 by the Secretary of Health and Human Services and Commissioner of the Food and Drug Administration (FDA) that the agency would tighten the restrictions on animal feed, nothing has been done by FDA reduce the risk of BSEcontaminated feed entering the animal food supply. While the agency did propose a rule in October 2005 to provide a “90% solution” for mitigating the remaining risk in the animal feed supply, no final rule has been issued – even though FDA officials promised the publication of that rule by July 1, 2006. Consequently, the U.S. cattle population is being exposed to a greater risk of BSE with regulations that have not been updated since 1997. The updated risk assessment does not factor in the persistent weakness in the feed rules.

Response: The assumed potential for commingling and/or mislabeling cattle feed used in the updated Harvard risk assessment is based on FDA compliance data and not on stated policy goals (see Section 2.2.4 of the October 2005 Revised Harvard risk assessment).

Comment #5: We believe that USDA has been premature in re-opening cattle trade with Canada, especially since it is becoming increasingly apparent that Canada has had difficulty in enforcing its feed ban. Within the past year, Canada has reported two cattle with BSE that were born after 1997, when the Canadian feed ban was instituted. The most recent case is most troublesome since the cow was only 50 months old – born in 2002, years after the rules that would supposedly prevent the disease went into effect. USDA re-opened trade for live cattle under 30 months of age in 2005; it was on the verge of re-opening trade for cattle over 30 months of age until the most recent BSE case was discovered in Canada. Until Canada can prove that it can effectively enforce its animal feeding regulations, we believe that the United States should stop all cattle trade with that country. The updated risk assessment does not adequately address the risk presented by cattle from Canada.

Response: The purpose of this risk assessment is to evaluate the impact of risk management measures on the spread of BSE in the U.S. *after it is introduced*. The source of BSE-infected cattle entering the U.S. is not relevant to how the risk assessment model functions.

Comment #6: According to scientists in Great Britain, Europe could experience a “second wave” of vCJD cases in the future since the disease can lie dormant in humans for decades after they have consumed BSE-contaminated meat. While there have been no reported cases of indigenous vCJD cases in the United States, we believe the possibility remains since the food safety net has been riddled with holes for decades.

Response: The 2005 Harvard BSE Risk Assessment Update quantifies the impact of various food safety risk management measures on human exposure to the BSE agent. While this comment provides useful background, it does not further add to the information provided in the 2005 Harvard BSE risk assessment update and it is not clear how this comment is relevant to further refine the assessment.

RESPONSE TO COMMENTS FROM FARM SANCTUARY

Comment #1: The Harvard-Tuskegee Study identified three pathways that could allow transmission of the BSE agent to humans:

- Non-compliance with the FDA ban on ruminant to ruminant feed.
- Rendering of animals who die on the farm and use of the rendered product in ruminant feed.
- Inclusion of high-risk tissue from cattle in products for human consumption.

Farm Sanctuary recommends the following Specified Risk Materials (SRM) be excluded from both human and animal foods: brain and spinal cord of all cattle, skull and vertebral column of all cattle, and intestines from pylorus to anus from all cattle.

A total ban on SRM, regardless of age of the animal, would best protect the public since a blanket ban would significantly improve enforcement of the prohibition and eliminate the need to determine the age of each animal. The intestine should be considered a primary source of infectivity since infection with BSE has come from cattle ingesting contaminated feed, according to the Scientific Steering Committee of the European Union. In classifying the entire intestine as SRM, the EU Steering Committee noted that because slaughterhouse contamination of other intestinal areas with matter from the ileum can't be avoided, it is prudent to remove the entire small and large intestine.

Consuming meat products contaminated with BSE has been linked to more than 150 human deaths worldwide from variant Creutzfeldt-Jakob Disease (Associated Press, 2006). Japan's national policy is for every cow to be tested for BSE (Lempert, 2006). The U.S. should do the same to protect the health and well-being of the American people. Currently, the USDA tests less than one percent of the cattle population and is in the process of initiating a reduction in BSE surveillance. Instead of reducing surveillance, Farm Sanctuary urges the USDA to increase the number of BSE tests conducted nationwide.

We urge the FDA to ban all animal protein except milk and eggs from use in feed for any animal that enters the human food chain, an action that has already been taken by the European Union (Bonné, 2004). There is evidence that Transmissible Spongiform Encephalopathy (TSE) diseases, of which BSE is one, are capable of crossing the species barrier. Some scientists theorize Britain's BSE outbreak occurred when cows ate feed containing parts of sheep and goats infected with scrapie (another TSE). In addition to stopping the practice of feeding mammals to other mammals, it should also be illegal to feed mammal remains to chickens and then feed the chicken litter back to mammals. It is not known whether prion infectivity is reduced or eliminated by passage of infected feed through the chicken's intestinal tract.

Regardless of whether the feeding of mammals to birds, or the feeding of birds to mammals or to other birds, poses a risk of direct transmission of BSE, these practices should be banned to prevent accidental feed contamination. Chicken feed containing cattle protein may spill on the barn floor and then become mixed with poultry litter that is then fed back to cattle. The Report on Measures Relating to BSE in the United States, produced by the international panel of experts convened by the USDA, noted that ruminant derived protein contained within the lumen of porcine or avian intestines at slaughter may be included in ruminant feed. Furthermore, the rendering process is not exact. Prohibited cattle feed can be mixed with other feed at rendering plants, feed mills, ranches, and other facilities where mixing takes place. With animals routinely being rendered and fed to other animals, there is no certainty that intentional or accidental contamination won't take place somewhere in the process. The only sure way to guard against the accidental feeding of poultry or pig-feed to cattle, or vice versa, is to not allow any avian or mammal meat or bone meal to be processed into animal feed.

Farm Sanctuary also supports prohibiting the use of animal blood in animal feed and milk replacer. Currently in the U.S. cow's blood collected at the slaughterhouse is used to supplement the colostrum replacer given to young calves. Dairy producers use milk replacer made from cattle blood protein as a cheaper alternative to milk. This practice enables intensive farming operations to remove calves from their mothers immediately after birth, which contributes to physical and behavioral problems for the animals. Cattle blood may also be sprayed directly on the feed of weaned calves and young pigs. A number of published studies have shown prion transmission through blood (Vojvodic S, 2002; U.S. Food and Drug Administration, 2002; BBC News, 2005) and the European Commission report on the assessment of BSE risk in the U.S. specifically condemned the practice of "intraspecies recycling of ruminant blood and blood products."

An extension of the current animal feed ban must be accompanied by the enforcement of measures to prevent cross contamination. The USDA's panel of international experts on BSE have recommended enforcement through an inspection program including sampling and testing of feed. The advance notice states that current compliance with the 1997 feed ban rule by feed mills, renderers, and protein blenders is very high. However, we understand that this assessment is based on feed businesses' reporting of their own practices and not on on-site government inspections and testing of animal feed. We encourage the FDA to include on-site inspections and feed testing in any new regulations related to animal feed.

It is generally agreed that non-ambulatory cattle are more likely to be infected with BSE than healthy cattle and therefore pose a greater risk to public health. In fact, all three cows found to have BSE in the U.S. (Washington - 2003, Texas - 2005, Alabama - 2006) were downed and non-ambulatory. Therefore, Farm Sanctuary strongly supports the USDA's ban on the slaughter of downed cattle, and urges that this ban be made permanent and expanded to other livestock species.

The Harvard-Tuskegee Study has also suggested that the risk of BSE transmission could be further reduced by prohibiting the rendering of animals who die on the farm, an approach we support.

In addition, we request that the following concerns be taken into consideration and addressed by any on-farm surveillance program of live non-ambulatory animals:

- It is important that examination of live animals be conducted in a timely manner so that animal suffering is minimized.
- Animals must be humanely euthanized in a timely manner by properly trained personnel and only by approved methods.

Response: We thank Farm Sanctuary for their comments. Because these comments pertain to policy, they are not addressed here.

RESPONSE TO COMMENTS FROM LINDA A. DETWILER, D.V.M.

Comment #1: Consideration of new research: By the end of 2004 there was increasing evidence in species other than cattle that peripheral nerves and muscle have infectivity. In some of these species, studies indicate that the agent migrates to the brain and spinal cord, replicates to high levels in the central nervous system (CNS) and then spreads centrifugally from the spinal cord back down through the spinal neurons to the neuromuscular junction into the muscle cells themselves. (Bosque et al., 2002; Glatzel et al., 2003; Bartz et al., 2002; Androletti et al., 2004; Mulcahy et al., 2004; Thomzig et al., 2003; Thomzig et al., 2004)

A recent German study (Buschmann and Groschup, 2005) examined nerves and muscle from a **cow naturally infected with BSE** and found that infectivity was present in several peripheral nerves. The method of detection was bioassay in bovinized transgenic mice that show the same or greater sensitivity to transmission of BSE as cattle. It must be pointed out that the amount of infectivity in the peripheral tissues appears to be at significantly lower levels than which is found in the CNS.

Results from a collaborative study between scientists in Japan and the United Kingdom concur with these findings. The testing which was completed in Japan found PrP^{res} in peripheral nerves. There is increasing evidence that the pathogenesis of BSE might not be entirely different from TSEs in other species, in that at the point of clinical disease or just prior to the onset of clinical signs, there may be peripheral involvement. (International Conference -Prion Diseases of Domestic Livestock; The Radisson Edwardian Hotel, Heathrow, London, United Kingdom, 28-30 May 2006)

The current USDA regulations prohibiting certain SRMs from food for humans do not remove these peripheral tissues and tests are not conducted on cattle passing ante mortem and postmortem inspection. Thus, if BSE infected cattle near or at the end of the incubation period are passed for slaughter and there is infectivity in peripheral nerves edible product may be contaminated. This new research is not considered in the model.

Response: A response to this comment has been previously provided. Please see the FSIS response R-CALF comment #4.

Comment #2: Ability to detect BSE by clinical observation alone: The updated Harvard Risk Assessment assumes that USDA inspectors would be able to detect 95% of the cases of BSE which can still walk and 85% of those which are down by visual inspection alone. These cattle would then be prevented from being passed for slaughter (http://www.fsis.usda.gov/PDF/BSE_Risk_Assess_Report_2005.pdf). The assessment did not provide data to support these assumptions.

The percentages of detection seem optimistically high. Unless FSIS has hard data to support such assumptions, they should consider the comments from Peer Reviewer One and other sources of information as provided below to adjust these figures accordingly:

Comments from Peer Reviewer One

Peer reviewer one provided actual data from the United Kingdom and observations from other European countries. These data suggest that the US assumptions considerably overestimate the ability of inspectors to identify BSE. In the United Kingdom, the country with the most cases of BSE in the world, inspectors have only identified half of the BSE cases prior to slaughter. Testing has identified the other half. Reviewer one states, “So it seems as if only approximately 50% of clinical cases may be detected at ante-mortem inspection at abattoirs in the UK at present, and even then they are still only identified as “risk animals” rather than as BSE suspects.” (http://www.fsis.usda.gov/PDF/BSE_Risk_Assess_Appendix_4_2005.pdf)

Past performance of clinical BSE detection in North America

Past performance indicates that the system of clinical examination of the nonambulatory bovines was not adequate for determining a disposition regarding BSE. This was clearly illustrated by the first two native cases of BSE in North America in 2003. Both cases of BSE (May and December) were observed by veterinarians prior to slaughter. Neither was specifically set aside as a BSE clinical suspect. The Washington State case was passed for human consumption because she was determined to have a calving injury (injuries may be a result of the manifestations of BSE rather than the primary cause of illness). Meat from this animal was sold into the retail grocery system.

The May 2003 case detected in Canada was submitted for slaughter in January but condemned as a case of pneumonia. The carcass went to rendering but because it was not identified as an actual BSE suspect, it was not tested until May. By this time, the meat and bone meal produced from this cow was disseminated. In fact three farms with cattle were depopulated as a result of this exposure.

As per CFIA, the other 6 Canadian cases were all nonambulatory. None presented with classical clinical signs of BSE thus none were “BSE suspects”. They were picked up under the testing program for nonambulatory cattle (3D/4D).

The subtlety of clinical BSE

In many cases the clinical signs of BSE are so subtle as to go unnoticed especially if the observer is not familiar with the normal behavior of the animal. In addition, a number of clinical signs may be easily mistaken for other conditions. As indicated by data from the United Kingdom and Europe, even veterinary inspectors with experience are not able to detect the majority of cases by clinical examination alone. To further illustrate this point, I have attached some clips which are from a CD on the clinical signs of BSE. This was produced by the United Kingdom’s Veterinary Laboratories Agency (VLA).

Performance in detecting scrapie by ante mortem inspection

In the United States certain sheep are tested for scrapie at slaughter. If they have recognizable clinical signs they are not passed for human consumption. If they appear to be “normal healthy” sheep and pass ante mortem inspection samples are taken to be screened for scrapie. I believe that looking at the data regarding the ability of inspectors

to detect scrapie by visual inspection alone may be a useful tool when estimating this number for BSE.

Since its inception in April 2003 until July 2006, the USDA, APHIS scrapie slaughter testing program has identified 258 sheep that have passed ante mortem inspection but subsequently tested positive for scrapie. (<http://www.aphis.usda.gov/vs/naahps/scrapie/>) I could not calculate the total percentage of scrapie suspect sheep which may have been removed from the system prior to slaughter by visual inspection alone as I did not have data from FSIS going back to April 2003.

The number of scrapie positive samples collected at slaughter and tested positive by APHIS in fiscal year 2005 was 100 and in fiscal year 2006 (Sept 2005-July 2006) was 52. In 2005 FSIS inspectors condemned 2 mature sheep as nonambulatory and 3 mature sheep which were recorded as having signs of a central nervous system disorder. In 2006 (until July 27) FSIS inspectors condemned 1 mature sheep as non ambulatory and none as having a central nervous system disorder. I could not calculate a performance rate as APHIS tallies data by fiscal year and FSIS by calendar year. Despite not being able to calculate a percentage, the total figures suggest that the vast majority of scrapie infected sheep are identified at slaughter not by clinical observation, but by testing.

Accurate assumptions regarding the ability of inspectors to recognize BSE by clinical signs alone are extremely important as they affect a number of outputs of the model. If inspectors were to be able to identify the majority of the BSE positive animals prior to slaughter as the model suggests, any risk to humans from SRMs, other tissues and cross contamination is significantly reduced because the animals never enter the processing system. This assumption is reflected in the conclusion which states that the removal of nonambulatory cattle for human consumption decreases the risk to the public by only 3%. However, if the rate of performance is as low or lower than data provided by peer reviewer 1, the risk to humans from down cattle increases by at least 50%. This may also affect the other conclusions.

I strongly urge that FSIS not use this data as part of a justification to allow nonambulatory cattle to be passed for human consumption. The combination of the new research findings as stated above and the difficulty in determining BSE status by inspection alone warrants the continual ban on this class of high risk animals.

Response: The base case in the 2005 Harvard BSE Risk Assessment Update has been revised. It now assumes that ante mortem inspection detects 50% of ambulatory animals with clinical BSE signs, and 25% of non-ambulatory animals with clinical BSE signs. A revised sensitivity analysis investigates the impact of assuming ante mortem inspection fails to detect any animals with clinical signs of BSE. This change, in addition to the explicit characterization of the poultry litter pathway, has increased the estimated mean human exposure to the BSE agent from 3,800 cattle oral ID₅₀s (original base case described in the October 2005 report) to 6,600 cattle oral ID₅₀s in the revised base case prepared along with these responses to public comments. It increased the average number of new BSE cases in the U.S. from 180 over 20 years (original base case described in the

October 2005 report) to an average of 200 new BSE cases in the U.S. over 20 years (base case prepared along with these responses to public comments). R_0 remains less than 1 with high probability (>95%), indicating that it is very probable that the prevalence of BSE would decrease over time after its introduction into the U.S.

Sensitivity Analysis #5, prepared along with these responses to public comment, evaluates the impact of assuming that ante mortem inspection does not detect any animals with BSE. There is a mean of 210 new BSE cases in this sensitivity analysis (compared to 200 in the new base case), and a mean of 9,700 cattle oral ID_{50} s to which humans are exposed over 20 years (compared to 6,600 cattle oral ID_{50} s in the revised base case). Under the sensitivity analysis, R_0 remains less than 1 with high probability (>95%), again indicating that it is very probable that the prevalence of BSE would decrease over time after its introduction into the U.S.

Comment #3: Compliance with the FSIS regulations: The Harvard model ran the all of the calculations assuming 100% compliance in regard to SRM removal. FSIS ran simulations at 99, 98, 97, 96 and 95% levels of compliance. They found that for each percent drop in compliance there was an increase of 1 percent risk to the public which illustrates the importance of full compliance with the SRM removal regulations.

The FSIS regulations state that specified risk materials are inedible and cannot be used in human food. The plants must develop and implement plans to remove, segregate and dispose of the SRMs. Furthermore, they must take corrective action when either the establishment or FSIS deems that the procedures are not effective in keeping specified risk materials out of human food. On the surface this sounds very straight forward.

It is my opinion that these requirements are difficult for FSIS to monitor unless the standard operating procedure designates a point prior to boning where SRMs are completely prohibited. For example, if the plant HACCP program merely states that SRMs are to be removed and not included in product, this allows for the possibility that spinal cord may be allowed into the boning room. Unless an inspector observes the cord in edible product or observes it fall into edible product this may not be recognized as a deficiency.

I would argue that once spinal cord or other central nervous system tissue enters the boning room and contaminates the tables and equipment, the potential risk from BSE is already there and removal at this point is not completely sufficient. The properties of the causative agent require more than sanitation at 180° F for inactivation. One suggestion would be to require the removal of spinal cord, and other such SRMs on the slaughter floor using dedicated equipment. This approach reduces the risk of cross contamination and provides a defined point from which one could audit. In my current position as a consultant, I have visited as well as audited numerous slaughter establishments both in the United States and in other countries.

Response: FSIS appreciates these comments and will continue to consider a number of risk management options, including those guided by the updated 2005 Harvard BSE Risk Assessment.