CITIZEN PETITION BEFORE THE UNITED STATES

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Department of Health and Human Services

200 Independence Avenue, Southwest

Washington, D.C. 20201

HUMANE FARMING ASSOCIATION,

Post Office Box 3577,

San Rafael, CA 94912-8902,

et al.

Petitioners,

vs.

DONNA SHALALA,

in her official capacity as,

Secretary

Department of Health and Human Services

200 Independence Avenue, Southwest

Washington, D.C. 20201

GENE MATTHEWS

in his official capacity as,

Legal Advisor to CDC and ATSDR,

Center for Disease Control and Prevention

1600 Clifton Road N.E., Mailstop D35

Atlanta, GA 30333

Defendants.

PETITION SEEKING IMMEDIATE ACTION BY THE HHS AND CDC

TO COMBAT THE SPREAD

OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY (TSEs)

IN THE UNITED STATES

Petition Requesting Immediate Action by the

Department of Health and Human Services to Combat Transmissible Spongiform Encephalopathies in the United States

---

TABLE OF CONTENTS

I. Introduction

Pursuant to the Right to Petition Government Clause contained in the First Amendment of the United States Constitution,[(1)] the Administrative Procedure Act,[(2)] and the Health and Human

Services (HHS) implementing regulations, the undersigned submits this citizen petition for rulemaking and collateral relief and respectfully requests the Secretary to protect the public from Transmissible Spongiform Encephalopathies (TSEs) by establishing a national monitoring system for testing, studying and reporting on TSEs.

II. Petitioners

1. **The Humane Farming Association**, an I.R.S. Code 501(c)3 not for profit organization, with its principle offices located in the State of California and its mailing address at Post Office Box 3577, San Rafael, CA 94912-8902, 415-485-1495;

2. **The Center for Food Safety**, a project of the International Center for Technology Assessment, an I.R.S. Code 501(c)3 not for profit organization with its principle offices located in the District of Columbia at 310 D Street, N.E., Washington, D.C. 20002-5722; 202- 547-9359; 3. **Center for Media & Democracy, Inc.**, an I.R.S. Code 501(c)3 not for profit organization, with its principle offices located in the State of Wisconsin at 3318 Gregory St.; Madison, WI 53711-1725; 608-233-3346;

4. **R. Douglas and Tracie L. McEwen**, 4 East 270 South, Kaysville, Utah, 84037. Mrs. McEwen's husband is R. Douglas McEwen, 30, a territory manager for a brokerage until August of 1998, is terminally ill with CJD, in an extremely agonizing decline for him, her, and their two young daughters, ages 3 and 8. He was diagnosed on Nov. 25, 1998 at the University of Utah Hospital in Salt Lake City, Utah after a brain biopsy.

5. **Patricia C. Ewanitz**, 46-03 216th Street, Bayside, New York 11361; Ms. Ewanitz' husband, Andrew P. Ewanitz, Jr., a engineer and plant facilities manager, died of Creutzfeldt-Jacob Disease (CJD) at Jacob Perlow Hospice, 1st Avenue and 16th Street, New York City on April 9, 1997 after his initial diagnosis was mild stroke;

6. **Elizabeth A. Armstrong**, 205 S. Cherry Street, Lebanon, Illinois, 62254; Ms. Armstrong's father, Charles O. Butcher, a law enforcement officer, died of CJD on May 6, 1996, at Veterans Administration Medical Center, Richmond, Virginia, after an initial diagnosis of COPD then Parkinson's Disease;

7. **Melvin J. Steiger**, 5582 Cora Way, Salt Lake City, Utah 84118-2316. Mr. Steiger's wife, Eleanor M. Steiger, 57, an accounting clerk, was initially diagnosed with depression. Symptoms began in 1996 with severe leg muscle cramps. Symptoms eventually included changes in personality where she become less patient and more argumentative, loss of coordination, memory and ability to function. Admitted to the hospital for a possible stroke, Mrs. Steiger was positively diagnosed with CJD on the 19th of February, 1998. On February 23, 1998, a biopsy, an EEG and examination confirmed that diagnosis. She passed away at home on March 19, 1998

8. **Beverly Wilson Goodman**, 7308 Moody Court, Fort Worth, Texas 76180-6107; Ms. Goodman's father, Perry Marlin Wilson, died of CJD at Zale Lipshey a division of Parkland Hospital, Dallas, Texas, with a diagnosis of "unknown" on May 18, 1997;

9. **Mildred B. Campbell**, 2229 Hwy. 601-S, Mocksville, North Carolina; Ms. Campbell's husband, John D. Campbell, a retired law enforcement officer, died of CJD at Forsyth Memorial Hospital, Winston-Salem, North Carolina on March 18, 1997 with an original diagnosis of stoke and peripheral neuropathy. CJD was later confirmed by a brain biopsy;

10. **Dorothy E. Kraemer**, 901 S. Stanley Street, Stillwater, Oklahoma 74074; Ms. Kraemer's mother's cause of death from CJD was confirmed through a brain autopsy after her death at Stillwater Nursing Home on May 16, 1996. The original diagnosis was rapid onset Alzheimer's Disease;

11. **Cecile L. Sardo**, 17065 N.W. 22nd Street, Pembrook Pines, Florida 33028; Ms. Sardo's husband, Joseph Sardo died of CJD;

12. **Jim and Rebecca Goodman,** of E103 County Q, Wonewoc, Wisconsin 53968; The Goodman's make their living by farming in the State of Wisconsin;

13. **Bruce and Shelley Krug**, of Box 84 West Road, Constanbleville, New York 13325; The Krugs are dairy farmers.

III. Statement of the Facts

Transmissible Spongiform Encephalopathy (TSE) is a mysterious classification of diseases that are called by different names in different species. For instance some identified types of TSE are Creutzfeldt-Jakob Disease (CJD) in humans, scrapie in sheep, bovine spongiform encephalopathy (BSE) in British cattle, transmissible mink encephalopathy (TME) in mink in North America, and chronic wasting disease (CWD) in deer and elk in North America. There may be different strains of TSE within species, and new strains may be produced when TSEs move from one animal species to another.

There is vigorous scientific debate as to exactly what the TSE agent is, since it has never been completely isolated, but the leading theory seems to be that it is a mutant or infectious form of a naturally present protein probably present in all mammalian species called a prion protein; the name prion was created by Dr. Stanley Prusiner whose work was awarded a Nobel Prize in 1997.

The common characteristics of TSE diseases are that they are invariably fatal. They can be transmitted through iatrogenic or physician induced exposure, such as corneal transplants, use of dura mater during surgery, contaminated human growth hormone or contaminated probes. Additionally, infection may occur through exposure of infected material to mucous membranes or open cuts, and in some cases through ingestion of the infectious agent. The agent does not trigger an immune response, and the infected animal or human appears perfectly normal until at some point in life (usually the sixth decade in humans who develop sporadic CJD) the disease emerges as holes and amyloid plaque material spreading in the brain, causing symptoms of dementia, physical failure and death.

TSEs can be difficult to diagnose during life; confirmation of TSE disease is usually made post-mortem through autopsy analysis, or through a newly-developed spinal fluid test, and can be confirmed by passaging the infectious agent from the deceased specimen into the brain of a laboratory animal.

Prior to the outbreak of BSE in British cattle in the mid 1980s, and its spread to the human population as nvCJD which has claimed to date 35 lives, the best known TSE was sheep scrapie. It appeared in Spanish sheep in the 18th century, and has since appeared in most countries that raise sheep. Sheep scrapie appeared in U.S. sheep in the late 1940s, via Canadian sheep of British origins. After the failure of U.S. eradication efforts, sheep scrapie has spread across the United States and its actual occurrence is unknown.

Prior to the appearance of nvCJD in Britain, the best known occurrence of a TSE in humans was the disease kuru, a TSE that appeared in the Fore tribe in New Guinea in the early 20th century, spread by rituals involving cannibalism until the recognition of that fact in work by Dr. Daniel Carleton Gajdusek and Dr. Clarence Joseph Gibbs of the U.S. National Institutes of Health. For this work Gajdusek received the first Nobel Prize awarded for TSE research.

When British BSE, dubbed 'mad cow disease', appeared in the mid 1980s, it stood out because of the madness symptoms of the animals in a rabies-free nation. From just a few animals the disease rapidly spread to infect hundreds of thousands. By 1988 epidemiology demonstrated that the disease was in fact being spread by the feeding of rendered animal

byproducts as a protein and fat feed supplement. British cattle were consuming the remains unfit for human consumption of sheep, cattle and other animals. While the high prevalence of sheep scrapie in Britain has led to presumptions that British BSE is sheep scrapie in cattle, there is

no proof of this. Others suspect that BSE may have emerged from the feeding of a small number of BSE infected cattle to other cattle. In any case, the feeding of infected cattle back to healthy cattle clearly amplified and spread BSE within British cattle, whatever the origins of the

TSE agent.

The practice of feeding rendered animal byproducts back to livestock began in the 19th century, and became a large widespread practice involving billions of pounds of rendered animal fat and protein from the 1960s to the present. The practice continues today, although with some recent restrictions due to the spread of BSE and the emergence of nvCJD.

Despite the British outbreak, the practice of feeding rendered livestock meat and bonemeal back to livestock has been most widely practiced in the United States, where no meaningful restrictions were even attempted until August, 1997. Today, the U.S. lags behind Britain and Europe in implementing safeguards in the feeding of animals, and allows practices that should be banned, such as feeding pigs to pigs, pigs to cattle, and cattle blood protein to calves. These practices are allowed despite evidence that TSE disease may already infect pigs and cattle in the U.S., and despite the existence of proven TSEs in U.S. sheep, deer, elk and mink.

Indeed, Dr. Clarence Joseph Gibbs theorizes that every mammalian species is likely to have individuals with a TSE disease at some low level, perhaps one in a million. Therefore, any cannibalistic feeding practices might amplify and spread the disease, as in Britain, possibly creating a TSE that can jump from one species to another, as has apparently happened in the

U.K. with the spread of BSE into humans as nvCJD. While 35 diagnoses of nvCJD to date seems a small number, the British government admits that the virtually invisible disease nvCJD may have an incubation period measured in decades, and therefore it will require the passage of many additional years to determine the extent of the disease and to predict the eventual death toll. Estimates have ranged from scores to hundreds of thousands eventual deaths from nvCJD in Britain.

CJD exists in the United States, but it is unclear at what level. Most CJD in the U.S. is considered 'sporadic' with an unknown cause. A smaller percentage appears to have a genetic factor. The U.S. has a large population of persons with various dementia diseases, the most often diagnosed being Alzheimer's (four million cases). There is no routine postmortem diagnosis of dementia deaths, although autopsy is the only sure way to determine what type of dementia a patient had. It appears that one of four dementia diagnoses made during life are actually incorrect, with another type of dementia causing the death.

Studies of people in the U.S. who have died of dementia suggest much higher levels of CJD than commonly suspected, with proven levels of anywhere from 1 to 13% of dementia victims. Individuals whose family members have died of CJD report great difficulties in receiving a correct diagnosis. Their recent activism through grassroots organizations such as CJD Voice, and the recognition that CJD seems much more common than thought, has led some states (Texas, Utah, Ohio and Kentucky) to begin making reporting of CJD mandatory. Without specific new programs to investigate and identify CJD cases, the true number cases and whether they are increasing will remain extremely difficult to document.

Given what is now known about the ability of TSE diseases to move between species and infect humans, the existence of animal TSEs in the United States, and the long and invisible incubation period during which a human or animal CJD strain might become epidemic, it is imperative that state and federal agencies implement programs for accurately determining

and monitoring the number of TSE cases in humans and animals in the United States.

One petitioner, R. Douglas McEwen, a thirty-year old Utah man is terminally ill with diagnosed CJD. Mr. McEwen's young age makes this an extremely unusual occurrence; British nvCJD came to public attention because of the similarly young age of its victims. Mr. McEwen was an avid consumer of deer and elk meat, and some areas of the western U.S., including the area where Mr. McEwen hunted, have been shown to have very high levels (1% to 6% in tested individuals) of chronic wasting disease (CWD) in deer and elk, a TSE apparently unique to North America.

It is possible that Mr. McEwen contracted CWD through contact with or consumption of infectious deer or elk. Whether or not this is the case, his CJD emphasizes the critical importance of taking all prudent steps to prevent the spread of TSEs in animals and people, and in determining and monitoring the existence of these diseases in the United States.

Human health concerns are amplified by the many unknowns surrounding CJD, including the invisibility of the agent and its infectious routes. For instance, contaminated medical instruments are impossible to adequately disinfect and have spread TSEs to healthy patients, as have cadaver-source tissue and organs. In laboratory tests blood can transmit TSEs, and in Britain the use of British plasma has been curtailed because of concerns that it might spread nvCJD. Mr. McEwen, the Utah CJD patient, was reportedly a plasma donor. Even in the United States, the FDA has recently been advised by scientists to limit blood donors to those who have not been exposed to Mad Cow Disease in England. This suggestion would mean that anyone who has visited Britain since 1980 could no longer give blood. Further, Health Canada has recently been advised to quarantine blood which may be contaminated with infectious CJD from Mr. McEwen who was dedicated to giving plasma on a regular basis.

In summary, the emergence of infectious TSEs that can move between species is a new and unanticipated public and animal health threat that is being inadequately studied and addressed in the United States. This petition seeks to remedy those failures.

IV. Procedural History

Considering the new emerging science of TSE as well as the new evidence presented in this petition, the petitioners request the HHS, including the FDA and the CDC, to immediately undertake the following actions to prevent the potential spread of transmissible spongiform encephalopathy (TSE), including bovine spongiform encephalopathy (BSE, also known as "Mad Cow Disease"), (CWD) Chronic Wasting Disease, and Creutzfeldt-Jacob Disease (CJD):

(1) initiate a significant epidemiological investigation to determine the incidence of transmissible spongiform encephalopathies among the human population of the United States; and

(2) develop an ongoing national monitoring and registry program utilizing autopsy examinations to determine any changes in the incidence of CJD-like diseases among the human population of the United States; and

(3) direct all state medical officers to engage in a reporting process, similar to the reporting requirements of other infectious and transmittable diseases, to the CDC for the purposes of establishing a national cumulative database and reporting system.

Petitioners incorporate by reference information submitted to the FDA through previous petitions dated June 23, 1993, December 21, 1993, March 26, 1996, March 27, 1996, and a petition dated January 6, 1999 to the FDA, copied to this agency. Petitioners also incorporate by reference the agency actions requested in those previous petitions. Petitioners also incorporate by reference information submitted to the agency in a previous petition dated March 27, 1997, by the Government Accountability Project which demanded that the laws and regulations that keeps pigs with TSEs and nervous system disorders from entering the food supply are fully enforced.

Petitioners further incorporate by reference information submitted to the agency in a previous petition and supplemental petition dated March 4, 1998 by Farm Sanctuary and Michael Baur that requested that the Food and Drug Administration (FDA) and the United States Department of Agriculture (USDA) immediately label all downed cattle and other livestock as adulterated pursuant to 21 U.S.C. § 342(a) in order to protect the health of the nation by reducing the potential of Transmissible Spongiform Encephalopathies (TSEs), bovine spongiform encephalopathy (BSE), also know as "Mad Cow Disease," (CWD) Chronic Wasting Disease and Creutzfeldt-Jakob Disease (CJD).

On June 23, 1993, petitioning attorneys submitted an "Amended Petition Requesting the Food and Drug Administration to Halt the Feeding of Ruminant Animal Protein to Ruminants" on behalf of several individuals and a non-profit organization. The petition requested the Food and Drug Administration and the United States Department of Agriculture to undertake agency actions that would address the potential health threat posed to U.S. animal herds, especially cattle, from transmissible spongiform encephalopathies (TSEs) and the threat posed to U.S. meat consumers, especially beef consumers, through the potential zoonotic development of Creutzfeldt-Jakob Disease (CJD) as a result of eating TSE-contaminated meat.

On behalf of the same individuals and organizations, petitioning attorneys submitted a "Supplemental Petition Requesting the Food and Drug Administration to Halt the Feeding of Ruminant Protein to Ruminants." On December 21, 1993, the supplemental petition was filed as result of, *inter alia*, the FDA's failure to respond to the June 23, 1993 petition within the one hundred and eighty (180) days mandated by 21 C.F.R. § 10.30(e)(2).

In a much belated response to the two petitions, in 1994 the FDA issued a proposed rule in the Federal Register stating that specified offal from adult sheep and goats (of more than 12 months of age) is not generally recognized as safe for use in ruminant feed and is an unapproved food additive when added to ruminant feed.[(3)] In June 5, 1997, a limited rule was promulgated by the FDA providing that a few certain types of animal proteins would not be used as raw materials in the rendering industry.[(4)]

Despite petitioners continuing requests, the FDA has failed to promulgate a rule that will adequately protect animal and human health from TSEs in general and BSE in particular. **V. Statement of the Law**

A. The Food and Drug Administration, Department of Health and Human Services, Subchapter L; Regulations Under Certain Other Acts Administered by the Food and Drug Administration, Part 1240, Control of Communicable Disease, Subpart B, Administrative Procedures states:

Whenever the Commissioner of Food and Drugs determines that the measures taken by health authorities of any State or possession (including political subdivisions thereof) are insufficient to prevent the spread of any of the communicable diseases from such State or possession to any other State or possession, he [sic] may take such measures to prevent such spread of the diseases as he [sic] deems reasonably necessary, including inspection, fumigation, disinfection, sanitation, pet extermination, and destruction of animals or articles believed to be sources of infection.

21 C.F.R. § 1240.30 (1998).

B. Title 42. The Public Health And Welfare, Chapter 6a, The Public Health Service, General Powers And Duties, Research And Investigations, 42 U.S.C.S. § 241 (1998), Research and Investigations Generally:

(a) Authority of Secretary. *The Secretary* ***shall*** *conduct in the Service, and encourage, cooperate with, and render assistance to other appropriate public authorities*, scientific institutions, and scientists in the conduct of, and promote the coordination of, research, investigations, experiments, demonstrations, and studies *relating to the causes, diagnosis, treatment, control, and prevention of* physical and *mental diseases and impairments of man*, including water purification, sewage treatment, and pollution of lakes and streams. In carrying out the foregoing the Secretary is authorized to--

(1) collect and make available through publications and other appropriate means, information as to, and the practical application of, such research and other activities;

(2) make available research facilities of the Service to appropriate public authorities, and to health officials and scientists engaged in special study;

(3) make grants-in-aid to universities, hospitals, laboratories, and other public or private institutions, and to individuals for such research projects as are recommended by the advisory council to the entity of the Department supporting such projects and make, upon recommendation of the advisory council to the appropriate entity of the Department, grants-in-aid to public or non-profit universities, hospitals, laboratories, and other institutions for the general support of their research;

(4) secure from time to time and for such periods as he [sic] deems advisable, the assistance and advice of experts, scholars, and consultants from the United States or abroad;

(5) for purposes of study, admit and treat at institutions, hospitals, and stations of the Service, persons not otherwise eligible for such treatment;

(6) *make available, to health officials, scientists, and appropriate public and other nonprofit institutions and organizations, technical advice and assistance on the application of statistical methods to experiments, studies, and surveys in health and medical fields*;

(7) enter into contracts, including contracts for research in accordance with and subject to the provisions of law applicable to contracts entered into by the military departments under title 10, United States Code, sections 2353 and 2354, except that determination, approval, and certification required thereby shall be by the Secretary of Health, Education, and Welfare;

(8) adopt, upon recommendations of the advisory councils to the appropriate entities of the Department or, with respect to mental health, the National Advisory Mental Health Council, such additional means as the Secretary considers necessary or appropriate to carry out the purposes of this section.

The Secretary may make available to individuals and entities, for biomedical and behavioral research, substances and living organisms. Such substances and organisms shall be made available under such terms and conditions (including payment for them) as the Secretary determines appropriate.

(b) *Testing for* carcinogenicity, teratogenicity, *mutagenicity, and other harmful biological effects*; consultation.

(1) *The Secretary **shall** conduct and may support through grants and contracts studies and testing of substances for* carcinogenicity, teratogenicity, *mutagenicity, and other harmful biological effects*. In carrying out this paragraph, the Secretary shall consult with entities of the Federal Government, outside of the Department of Health, Education, and Welfare, engaged in comparable activities. The Secretary, upon request of such an entity and under appropriate arrangements for the payment of expenses, may conduct for such entity studies and testing of substances for carcinogenicity, teratogenicity, mutagenicity, and other harmful biological effects. (Italics and bold added for emphasis.)

C. Title 42. The Public Health And Welfare Chapter 6a. The Public Health Service General Powers And Duties Federal-State Cooperation, 42 U.S.C.S. § 243 (1998), General grant of authority for cooperation:

(a) Enforcement of quarantine regulations; *prevention of communicable diseases*. The Secretary is authorized to accept from State and local authorities any assistance in the enforcement of quarantine regulations made pursuant to this Act which such authorities may be able and willing to provide. *The Secretary **shall** also assist States and their political subdivisions in the prevention and suppression of communicable diseases and with respect to other public health matters*, shall cooperate with and aid State and local authorities in the enforcement of their quarantine and other health regulations, *and **shall** advise the several States on matters relating to the preservation and improvement of the public health.*

(b) Comprehensive and continuing planning; training of personnel for State and local health work; fees. *The Secretary shall encourage cooperative activities between the States with respect to comprehensive and continuing planning as to their current and future health needs, the establishment and maintenance of adequate public health services, and otherwise carrying out public health activities.* The Secretary is also authorized to train personnel for State and local health work. The Secretary may charge only private entities reasonable fees for the training of their personnel under the preceding sentence.

(c) *Development of plan to control epidemics and meet emergencies* or problems resulting from disasters; cooperative planning; temporary assistance; reimbursement of United States. (Italics and bold added for emphasis.)

D. Title 42. The Public Health And Welfare Chapter 6a. The Public Health Service General Powers And Duties Quarantine And Inspection, 42 U.S.C.S. § 264 (1998), Regulations to control communicable diseases:

(a) Promulgation and enforcement by Surgeon General. *The Surgeon General, with the approval of the Administrator [Secretary], is authorized to make and enforce such regulations as in his judgment are necessary to prevent the introduction, transmission, or spread of communicable diseases from foreign countries into the States* or possessions, or from one State or possession into any other State or possession. *For purposes of carrying out and enforcing such regulations, the Surgeon General may provide for* such inspection, fumigation, disinfection, sanitation, pest extermination, *destruction of animals or articles found to be so infected or contaminated as to be sources of dangerous infection to human beings*, and other measures, as in his judgment may be necessary. (Italics added for emphasis.)

E. The Public Health and Welfare regulations Chapter 6A, Public Health Service, General Powers and Duties Injury Control, 42 U.S.C.S. § 280b Research (1998), states

(b) *The Secretary, through the Director of the Centers for Disease Control and Prevention,* ***shall*** *collect and disseminate, through publications and other appropriate means, information concerning the practical applications of research conducted or assisted under subsection (a).* In carrying out the proceeding sentence, the Secretary shall disseminate such information to the public, including elementary and secondary schools. (Italics and bold added for emphasis.)

F. Administrative Procedure Act, 5 U.S.C. Section 706, Scope of Review.

To the extent necessary to decision and when presented, the reviewing court shall decide all relevant questions of law, interpret constitutional and statutory provisions, and determine the meaning and applicability of the terms of an agency action. The reviewing court shall -

(1) compel agency action unlawfully withheld or unreasonably delayed; and

(2) hold unlawful and set aside agency action, findings and conclusions found to be -- (A) arbitrary, capricious, an abuse of discretion, or otherwise not in accordance with law.

VI. Argument

A. The Department of Health and Human Services Should Implement a National System for Monitoring all New Strains of TSE, Including "British BSE-nvCJD" in Humans Because the Current Scientific Evidence Shows That TSEs are Transmissible to Humans.

1. The Current Scientific Evidence

TSE is not the first animal disease to cross the species barrier and affect humans. Many other human infectious diseases started in animals.[5] For example, measles is related to canine distemper and influenza is related to a porcine disease.[6] Usually, related diseases exist between humans and domesticated animals.[7] The call for agency action to protect the United States public has come from science and policy experts who have studied TSEs around the world.

The number of hypothetical risks from these novel disease agents seems endless. They could pop up in medicines, organ transplants, in gelatine (which is used in everything from dessert mixes to medicine gel-caps), or in garden fertilizer made from rendered bone meal. Government and industry officials worry that public discussion of hypothetical risks could trigger unnecessary panic . . . What we need is good data, and in the meantime we need serious implementation of measure to prevent the disease from spreading - *not* just surveillance that will only alert us to tragedy after it has already arrived. We need the precautionary principle.[8]

Just this year, the largest scale case control epidemiological study of sporadic CJD (not nvCJD) comprised of 405 CJD patients linked sporadic CJD to consumption of animal products, especially brains.[9] Although this is a European study, there is no reason to suspect that the results would be different in the U.S., particularly in light of the small case control epidemiological studies done in the U.S. Those smaller U.S. studies also link sporadic CJD to the eating squirrel brains and pork products. This means the FDA policy that only "British BSE-nvCJD" poses a health risk to humans and animals may be incorrect. In fact, sporadic CJD may also be linked to consumption of American animal products as well.

Dozens of people have died in England because of this disease.[10] In fact, several people died before the English government admitted the "mad cow's" disease and human nvCJD were related.[11] To date, there is no other infectious disease agent like TSE; it is an entirely new contagion. Although viruses build up over hours, days, or months until the body's immune system is activated, TSE does not illicit an immune response or any outward symptoms of infection until the patient is terminal. As the TSE causative agent is not conclusively known, the agency should use the precautionary principle to protect the public by putting into effect a cumulative TSE monitoring and reporting system until the scope of the disease is known and the extent of a United States epidemic is understood.[12] The FDA has been advised by some of the world's leading TSE scientists, including Dr. Stanley Prusiner who won the Nobel Prize for his scientific discoveries involving TSEs, to bar blood donations from people who lived in or visited Britain.[13] "The [scientific advisors] said blood banks should survey donors to find out if they lived or spent up to a year in Britain from 1980 to the present."[14] Health Canada has advised the Canadian Blood Services and Hema-Quebec to put a temporary hold or quarantine on blood products.[15] At present, Canada is being told not to distribute any of the designated blood products on their shelves and it is possible that the blood that has not already been used on patients might be withdrawn and destroyed.[16]

Public health agencies have been cautioned by scientists and have been "put on notice" that mere surveillance for one brain disease or pathology is not sufficient.

Last summer the Yale researcher Laura Manuelidis showed the CJD can evolve into more virulent strains by being passed from human beings to mice, from there to hamsters, and finally to rats. Her paper, published in the July 4, 1997, issue of the journal *Science*, concluded that when the agent changed, it provoked a variant disease, and warned that public-health agencies should be on the watch for diverse signs

of brain pathology in human beings - not only those symptoms known to be associated with the new variant of CJD. Manuelidis's concern is that if a new variant of CJD has appeared in the United States, we will be slow to realize it, because we are looking for the variant that appeared in Great Britain.[17]

While "sporadic CJD" has been around for some time, a new variant dubbed, "nvCJD", has been linked to the consumption of TSE-infected cattle products in England ("British BSE-nvCJD"). The scientific evidence shows that agencies must be on the watch for *different and new variations* of CJD.

B. TSE is an Infectious Transmissible Disease

1. Humans May Contract TSE from Wildlife

TSEs are undisputably present in wildlife in the United States.[18] A specific variation of wildlife TSE, called Chronic Wasting Disease (CWD), has been found in various deer and elk for at least the past 30 years.[19]

CWD is insidious because the method of spread is unknown and it has a long incubation period. Furthermore, there is no test available to detect infection in a live animal; only long quarantines and postmortem tests can be used. The recent rash of cases in captive elk, coupled with the complex web of animal traffic that has been associated with the rapid growth of elk ranching, has created a strong possibility that things are going to get worse with CWD. [20]

The United States Animal Health Association Wildlife Diseases Committee has recommended resolutions and recommendations for a Model Program for Surveillance, Control, and Eradication of CWD in Domestic Elk.[21] However, the HHS has no such model recommendations for CJD in humans. In 1996, in portions of Colorado where hunters were required to turn over the heads of mule deer and elk, four- to six-percent of mule deer and one-percent of the elk population killed by hunters where shown to have indications of CWD.[22] These are extremely high numbers for what was once considered a rare disease. These numbers indicate that the disease is at an epidemic proportion in some areas of the Rocky Mountains. Since the disease can only be detected at the end of a long incubation period, the true incident of CWD in the mule deer and elk population is ultimately much higher. The Wyoming Game and Fish Department has produced a video titled "Chronic Wasting Disease of Deer and Elk" which cautions hunters against consuming visibly ill animals. Animals in the early stages of infection, not exhibiting symptoms, will test negative for signs of CWD; this does not mean they are not infected and infectious, it merely shows that the disease is not being detected by test. In June of this year, CWD was diagnosed in a captive elk in Oklahoma.[23] In 1998 alone, three additional states have discovered CWD: Nebraska, Oklahoma and South Dakota.[24]

One case control epidemiology study has suggested that exposure to deer might be associated with a health risk to humans.[25] Specifically, exposure to deer via hunting leads to an increased risk of contracting CJD. This study indicates that those exposed to deer were nine times more likely to later develop CJD. Indeed, one petitioner from Utah, currently suffering from CJD, is 30-year old Utah man who has hunted elk in Southern Wyoming where CWD in elk is known to occur. This is an unusually young age for classic, sporadic CJD. This tragic situation must be addressed by the agencies through monitoring and reporting procedures as well as possible case studies. Petitioners plea for the agencies to recognized these problems of CWD in light of the larger issues of TSEs and begin a significant epidemiological investigation to determine the incidence of TSE in U.S. humans and animals.

Indirect evidence exits that TSEs occur in other wildlife as well. In a 1984 case control epidemiological study, four CJD victims had eaten wild animal brain: one patient has eaten wild goat and three other patients had consumed wild squirrel brain; all four developing CJD.[26] More recent evidence exits that squirrel brains might be particularly risky. "In the last four years, 11 cases of a human form of transmissible spongiform encephalopathy, called Creutzfeldt Jakob disease, have been diagnosed in rural western Kentucky . . . 'All of them were squirrel-brain eaters.'"[27] Another article indicates this squirrel disease is being linked as a possible cause to the infection of five different people who ate squirrels in different areas of Kentucky.[28] "In a letter to The Lancet, [Dr. Joseph Berger, a neurologist at the University of Kentucky] . . . advise[d] that 'caution might be exercised in the ingestion of this arboreal rodent.'"[29] That doctor went on to bemoan the lack of clear data available on the disease. "CJD has been a reportable disease in Kentucky only for the last year and a half, so we do not have enough data to judge whether the incidence of CJD in the state is higher than elsewhere, though that is my impression."[30] Petitioners request that the agencies begin a national reporting and monitoring system so that different parts of the country can begin to assess whether the incidence of the disease is higher in one region or another so that the infection can be traced.

CJD researchers have determined that as the disease-causing substance infects different species, the actual substance itself might change.[31] Furthermore, some species may just be "carriers" for the disease, which may mutate into new variations.[32] Due to the fact that there is so little certainty even amongst the experts in this field, it is imperative that the Federal agencies compile data on TSEs. The identification of the possible risk between CWD in cervids, such as elk and deer, and TSE in squirrels, and the relationship between human nvCJD to BSE indicates that the disease is of extreme importance to the public health community. Therefore, United States health agencies need to monitor not just "sporadic CJD" or "British BSE-nvCJD", but must monitor all TSEs.

2. Humans May Contract TSE from Pork

Epidemiological evidence exists that TSE in pork is linked to CJD in humans. This has been alleged by three studies, two from the U.S. and one from Europe. The first study involved 38 CJD patients and stated that one-third of those CJD patients had eaten animal brains.[33] Of all patients who consumed brains, those who consumed brains frequently with a preference for hog brains were at a higher risk of CJD compared to a control group who also ate brains.[34] The second study involved 26 victims of CJD. That study found that nine out of 45 food items were statistically linked to the risk of CJD infection. Of those nine, a total of *six* came from pigs. "An increased consumption among [CJD] patients was found for roast pork, ham, hot dogs . . ., roast lamb, pork chops, smoke pork and scrapple."[35] The scientists cautioned "[t]he present study indicate[s] that consumption of pork as well as its processed products (e.g., ham, scrapple) may be considered as risk factors in the development of Creutzfeldt-Jakob Disease."[36]

The third study indicated a possible link between brain consumption and *sporadic* CJD, not nvCJD. In this case control epidemiology study, over four hundred CJD cases were studied and researchers discovered a link between animal consumption and sporadic CJD.[37] This link from animal consumption to sporadic CJD[38] is worthy of notice because it has been presumed that only nvCJD, not *sporadic* CJD, was caused by animal consumption. In light of the risk of CJD from the consumption of pork and pig products, federal health agencies must begin a pro-active policy of protecting the public from this disease.

3. Humans May Contract TSE from "Downer Cows"

An undiagnosed TSE may occur in cows in the U.S. cattle population called "downer cows." In 1979, at the USDA field station in Mission, Texas, researchers inoculated cattle with scrapie to see if cattle were susceptible to it.[39] Some of the affected animals developed a TSE different from British-style BSE. The symptoms were more similar to those seen in "downer cow" syndrome (a stiff-legged gait, incoordination, abnormal tail position, disorientation, and terminal recumbency). A leading expert in a TSE study stated that this evidence suggested that a bovine TSE was present at a low level in U.S. cattle and that "downer cows" should be tested. "Susceptibility of cattle to scrapie further suggests the possibility that sporadic cases of BSE may have occurred in the United States under the clinical picture of the downer cow syndrome."[40] While there is a small BSE monitoring program begun in 1990, that program did not look at brains from downer cows until 1993.[41] Although now included in the study, very few downer cows have been tested.[42] As of January 23, 1997, only around six hundred brains of downers were studied out of some 5,342 cattle brains tested. There are approximately 100,000 downer cows each year in the U.S.[43] If just one-half of one percent (0.5%) of these cows had a TSE, then there are 500 infected cows in the U.S.[44] Even though the agencies are testing for TSEs in cattle, they are only testing for one strain and the program is ineffective. Even though the agencies are testing for BSE in cattle, they are only looking for British-style BSE. The petitioners demand that the agencies begin a national reporting and monitoring system to address this problem.

4. Humans May Contract TSE from Other Humans

Aside from the concern of humans being infected through consumption of meat, a threat now exists of human to human transmission. In September 1998, a forty-year-old Colorado woman died of CJD.[45] Experts struggled to determine whether she died of the same disease which has now claimed 35 people in England, "British BSE-nvCJD".[46] After the autopsy, experts decided that although she had many symptoms of nvCJD, she did not have nvCJD.[47] Rather, this individual was infected with a TSE disease when doctors used a CJD contaminated medical product made of dura mater (the tissue that covers the brain).[48] The Colorado woman was actually infected with a TSE during an otherwise successful surgery.[49] While in England a special task force has been set up to address this problem, the United States has not undertaken any plan to address it. England is now assessing the costs and benefits of destroying *all* the equipment used in surgery, including operating tables and light switches.[50] Utilizing a precautionary analysis, the chair of the English working group stated, "Even if it leads to a small reduction in the numbers of who contract CJD it will be worth it."[51]

Clearly, the U.S. public is not safe from this disease. In addition to possible infection through food vectors, growing evidence of iatrogenic TSE transmission warrants the establishment of studies to understand the scope of the problem.[52] The State of Utah has decided to begin the process to make CJD a reportable disease after R. Douglas McEwen, a petitioner in this action and a 30-year old Kaysville man, has come down with the disease.[53] Since Mr. McEwen was a regular plasma donor and that plasma was pooled, it is uncertain just how far this contagion has spread.[54] Other governments continue to call for additional research while noting the inadequacy of current studies.Most of the laboratory and epidemiological studies of blood infectivity and disease transmissability have been made on the sporadic form of CJD, and clinical and neuropathological observations suggest that nvCJD may have distinctive biological features. *Further studies of new variant cases are needed in order to determine* whether or not the tissue distribution of infectivity in nvCJD differs from that of classical CJD, and, in particular, *whether the infectious agent might be present in blood more frequently or in greater amounts than in blood of patients with other forms of CJD*.[55]

Such a study needs to be included within a larger system for discovering, reporting, and compiling information on TSEs to protect the public.

C. The Department of Health and Human Services Should Implement a National System for Monitoring all new Strains of TSE, Including "British BSE-nvCJD" in Humans because it is Necessary to Protect Human Health

1. Health and Human Services' Statutory Mandate Requires the Monitoring of TSEs

The petitioners request that the Department of Health and Human Services establish and implement a reporting procedure in concert with other agencies to report the incidences of CJD and combat this infectious disease in the United States. The scientific evidence shows that TSEs have been identified by scientists as a human health threat in the United States. Although TSEs are a grave risk to human health, neither state nor federal health agencies have any type of uniform or coordinated programs for testing, monitoring, reporting, compiling, or maintaining information on this disease. Thus, there is a scientific need for such a system to aid the public and other agencies in tracking this disease to protect human health.

The Health and Human Services Secretary, Donna Shalala, is directed by the Congress to protect the public health by controlling communicable or transmissible diseases. This congressional mandate, cited *supra*, under "Statement of the Law," directs the Secretary to use her power to research, investigate and work cooperatively with state and local health authorities to prevent infectious diseases from spreading as well as allowing her to prevent the introduction of transmissible diseases into the United States. Additionally, the Secretary is directed to collect and disseminate information through the Director of the Centers for Disease Control and Prevention. Petitioners demand the agencies start using this power to test, study and report on Transmissible Spongiform Encephalopathies (TSEs).

Petitioners have also shown that TSE is a communicable disease. Failure of the agencies to control TSE through a national monitoring program is contrary to the statutory mandate under Title 21, part 1240 directing the Secretary to control communicable diseases. The law mandates under Title 42, part 241 that the Secretary *shall* use her power to research and investigate diseases to protect the public from physical and mental impairments; all the scientific evidence indicates that the public needs protecting from a communicable disease such as TSE. Congress has allowed the Secretary the authority to cooperate with state and local health authorities for the prevention of communicable diseases under Title 42, part 243, in order for there to be an effective understanding of the current spread of the disease a uniform national reporting procedure is necessary. As it relates specifically to iatrogenic and matters involving "British BSE-nvCJD" the Secretary is authorized under Title 42 part 264 to use her authority to prevent the introduction of transmissible diseases into the United States. Further, the Secretary is directed to collect and disseminate information through the Director of the Center for Disease Control and Prevention, examples of which are the tests, studies and reports requested under Title 42 part 280b. Failure for the Secretary to execute a national monitoring program in light of the significant extent and depth of scientific information on TSEs is arbitrary and capricious.

An agency's action is reviewed by the judiciary under the arbitrary and capricious standards of law. Under this standard, an agency must show a "rational connection between the fact found and the choices made."[(56)] An agency action is arbitrary and capricious when an agency

has relied on factors which Congress has not intended it to consider,

entirely failed to consider an important aspect of the problem, offered

an explanation for its decision that runs counter to the evidence before

the agency or is so implausible that it could not be ascribed to a difference

in view or the product of agency expertise.[57]

In determining whether an agency decision was "arbitrary and capricious," a reviewing court considers whether the decision was based on a reasoned evaluation of the relevant factors and whether there has been a clear error of judgment.[58]

Given the significant human health risk involved and the agencies knowledge of the extent and breadth of the scientific evidence agency failure to initiate a national TSE monitoring program would clearly be arbitrary and capricious under this standard.

2. A National Monitoring Program is Necessary Since CJD is Commonly Misdiagnosed

Alzheimer's disease is a neurological disease with symptoms similar to those of CJD. It is considered one type in the classifications of dementias. Dementias are difficult to clinically diagnose and there is typically a 25% error rate in diagnoses.[59] The GAO has projected the prevalence of Alzheimer's in this country to be 5.7% (1.9 million) of Americans 65 years old and older by the Government Accounting Office.[60]

a. The University of Pittsburgh Study:

A University of Pittsburgh study indicates that CJD is commonly misdiagnosed as Alzheimer's disease or other senile dementia.[61] In the study, autopsies were done on 54 demented patients who had been diagnosed with a non-CJD dementia or Alzheimer's disease.[62] Of those 54 patients, scientists found 3 cases of CJD.[63] This study represents a 5.5% misdiagnosis rate.[64]

b. The Yale University Study:

A Yale University study also indicates that CJD is commonly misdiagnosed as Alzheimer's disease.[65] In the study, 46 patients diagnosed with Alzheimer's were autopsied.[66] The autopsy showed that 6 patients actually had CJD.[67] This study represents a 13% misdiagnosis rate.[68]

c. Other Evidence:

CJD Voice is a support group for people who have had a case of CJD in their families or among their friends.[69] As of July, 1998, CJD Voice had 223 members, and 91 out of 99 members reported an initial misdiagnosis.[70] Those false diagnoses were stroke (18), depression (13), dementia (10), unknown (6), and Alzheimer's disease (5) instead of the correct diagnosis of CJD.[71]

Among the problems in tracking CJD and TSE diseases is that autopsies are rarely performed. In a recent report, the Congressional Research Service noted this problem:

An autopsy is required to confirm a CJD diagnosis, and the number of autopsies performed in the United States has declined over the past 25 years to *less than 10%* of non-homicide-related deaths. There probably is an under-reporting of CJD in the elderly due to the autopsy decline and the similarity with the symptoms of Alzheimer's disease. One study found that 13% of clinically diagnosed Alzheimer cases were proven to be CJD at autopsy. Younger persons with these neurological symptoms are more likely to be autopsied. Pathologists may be reluctant to perform an autopsy on a CJD victim because of the potential risk of contracting the disease.[72]

While autopsies are very rare overall, they are even more rare in the case of suspected CJD. Medical workers are understandably reluctant to put themselves at risk of catching an infectious disease in order to correct a misdiagnosis. While it is generally believed that only one-in-a-million people suffer from sporadic CJD, scientists know this disease is being grossly under reported in clinical practice. One leading scientist expressed her concern with the lack of good empirical data on the disease.

Fewer than 10% of all deaths are investigated with an autopsy, and even a smaller percentage of victims of dementia. Alzheimer's disease was rarely diagnosed prior to the 1940s, but now we diagnose all sorts of people with the disease. But Alzheimer's is a heterogeneous disease with many different causes. One cause for some people could be an infectious agent. And we really have no idea how much CJD there is. The one-in-a-million figure may be an underestimate.(73)

Another scientist has suggested that monitoring CJD and TSE will play a key role in protecting the public from this health threat.

Many were wrong about [BSE], including me. We didn't imagine that it could pass from cows into humans. But now we think it can, and it has the potential to be terrifying. Perhaps the best analogy is to the AIDS epidemic. Although it's almost certain that TSE doesn't transmit as readily as HIV, it's similar in a number of other ways. It can remain in the body for long periods without obvious symptoms, and it is fatal. But what I'm thinking about is how we regarded AIDS in the early days, before we really understood it. We underestimated the threat. Perhaps we should avoid making that mistake again.(74)

The prevalence of misdiagnosed CJD infection and the scientific community's need for information on this health issue indicates that a national testing and monitoring system should be implemented immediately to protect public health.

3. New Lab Tests are Available for Testing TSE

In September of 1996, Drs. Kelvin H. Lee, Clarence Joseph Gibbs and others announced that they had discovered a way to determine whether a patient was suffering from CJD or from Alzheimer's disease or other dementia.(75) This test along with a case study is a useful tool to determining ante-mortem whether a patient is suffering from CJD. Dr. Larry Schonberger, a medical epidemiologist at the Centers for Disease Control and Prevention, in Atlanta, Georgia, stated, "This is an important step forward. We've been hungry for a test for this disease. A lot of people have dementia, and it's unclear as to what it might be, and we're very hesitant to do a brain biopsy. This would be a much easier way to make a diagnosis while the patient is still alive."(76)

A distinguished leading expert on this disease, Dr. Clarence Joseph Gibbs, reported to Congress that the accuracy of this cheap and simple spinal fluid test rates at 96%-99%.(77) The New York Times reported that the test correctly identified 96 percent of the patients with TSE and correctly ruled the disease out in 96 percent of those patients with other types of dementia.(78) This test is especially important as it will confirm for family members whether a patient is not expected to survive a year, as in the cases of TSEs, or whether a patient's suffering will be a "long, drawn-out affair" as in the case of Alzheimer's.(79)

Aside from the benefits to the individual patients and their families, this test allows agencies to determine actual numbers of sufferers of TSEs among the public. Petitioners believe that this test can be used to assist in the establishment of a national reporting system by the Centers for Disease Control and Prevention. This test provides the methodology needed to get accurate data to the agencies, which can then inform the public about the risk of the disease. Although these tests can be used while the human or animal is alive, these tests only detect the disease toward the end of a long incubation period. Before a human or animal exhibits symptoms there is no way to test for the disease even though the human or animal is infected and

contagious. Given the long incubation period, and the fact that the disease can be transmitted and undetected and the epidemiological evidence that consumption of animal products especially brain consumption has been linked to sporadic CJD, a precautionary approach must be utilized by the agencies. Therefore, the petitioners demand action from the agencies to begin epidemiological tests with corresponding reporting and monitoring systems.

VII. Environmental Impact & Certification

The enforcement actions here requested will not cause the release of any substance into the environment. They are categorically excluded from the requirement of environmental documentation under 21 C.F.R. § 25.33(g).

The undersigned certify that, to the best knowledge and belief of the undersigned, this petition includes all information and views on which the petitions relies, and that it includes representative data known to the petitions which are unfavorable to the petition. Except as described above, petitioners know of no other similar issue, act, or transaction to this petition currently being considered or investigated by any HHS office, other federal agency, department, or instrumentality, state municipal agency, court or any law enforcement agency consistent with FDA regulation 21 C.F.R. § 10.30(e)(2).

VIII. Agency Action Requested

The petitioners request the HHS and its departments, the FDA and the CDC, to take the following immediate action to prevent the potential spread of transmissible spongiform encephalopathy (TSEs):

(1) initiate a significant epidemiological investigation to determine the incidence of transmissible spongiform encephalopathies among the human population of the United States;

(2) develop an ongoing national monitoring and registry program utilizing autopsy examinations to determine any changes in the incidence of CJD-like diseases among the human population of the United States; and

(3) direct all state medical officers to engage in a reporting process, similar to the reporting requirements of other infectious and transmittable diseases, to the CDC for the purposes of establishing a national cumulative database and reporting system.

Petitioners are requesting a response to this petition within one hundred eighty (180) calendar days. In the absence of an affirmative response, the Petitioners will be compelled to consider litigation in order to achieve the full and complete action required to address this violation of federal law.

Dated this 6th day of January, 1999.

On behalf of all petitioners,

_____

Andrew Kimbrell

Executive Director

_____

Joseph Mendelson, III

Legal Director

CC: Jane Henney, Commissioner

United States Food and Drug Administration

c/o Dockets Management Branch

12420 Parklawn Drive, Room 1-23

Rockville, MD 20857

1. The right to petition for redress of grievances is among the most precious of the liberties safeguarded by the Bill of Rights. United Mine Workers of America, Dist. 12 v. Illinois State Bar Association, 389 U.S. 217, 222, 88 S. Ct. 353, 356, 19 L. Ed. 2d 426 (1967). It shares the "preferred place" accorded in our system of government to the First Amendment freedoms, and has a sanctity and a sanction not permitting dubious intrusions. Thomas v. Collins, 323 U.S. 516, 530, 65 S. Ct. 315, 322, 89 L. Ed. 430 (1945). Any attempt to restrict those First Amendment liberties must be justified by clear public interest, threatened not

doubtful or remotely, but by clear and present danger." Id. The Supreme Court has recognized that the right to petition is logically implicit in, and fundamental to, the very idea of a republican form of government. United States v. Cruikshank, 92 U.S. (2 Otto) 542, 552, 23 L. Ed. 588 (1875)

2. 5 U.S.C. § 553(e) (1995).

3. 59 Fed. Reg. 44,584 (Aug. 29, 1994).

4. 21 C.F.R. § 589, 62 Fed. Reg. 30,936 (1997).

5. William H. McNeill, Plagues and People, 1977.

6. Id.

7. Id.

8. Sheldon Rampton and John Stauber, Mad Cow U.S.A. at 218-219 (1997).

9. C.M. van Duihn, N. Delasnerie-Lauprêtre, C. Masullo, I. Zerr, R. de Silva, D.P.W.M. Wientjens, J.-P. Brandel, T. Weber, V. Bonavita, M. Zeidler, A. Alpérovitch, S. Poser, E. Graneri, A. Hofman, R.G. Will for the European Union (EU) Collaborative Study Group of Creutzfeldt-Jacob Disease (CJD), Case Control Study of CJD in Europe During 1983- 1985, 351 The Lancet 1081-1085 (Apr. 11, 1998).

10. See, Sheldon Rampton and John Stauber, Mad Cow U.S.A. (1997) and <www.prwatch.org>.

11. Id.

12. The "precautionary principle" urges agency action regulating activities which may be harmful to the public health, welfare and safety *even* if conclusive scientific evidence of their harmfulness is not yet available. At its most profound , the precautionary principle dictates the institutionalization of precaution, which entails the shifting of the burden of proof from those encouraging agency action to those engaged in the challenged industry. See e.g., Philippe Sands, ed., Greening International Law at 118 (1994).

13. Reuters, U.S. Could Block British Blood Over Mad Cow, (Dec. 18, 1998).

14. Id.

15. Mark Kennedy, U.S. Donor Sets Off Alarm in Canadian Blood System: Mad Cow Link: Thousands of Blood Units in Quarantine, National Post at A6 (Dec. 19, 1998).

16. Id.

17. Ellen Ruppel Shell, Could Mad-Cow Disease Happen Here?, 81 The Atlantic Monthly, 92 at 106 (Sept., 1998).

18. Michael W. Miller, Epidemiology of Chronic Wasting Disease in Northeastern Colorado Deer and Elk Populations, Presented at the Wildlife Diseases Conference, 1998, Madison, Wisconsin. This scientists presented information that over 2,500 brain samples from deer and elk. Also see, Resolution: Committee on Captive Wildlife and Alternative Livestock and Wildlife Diseases, Chronic Wasting Diseases, United States Animal Health Association (USAHA), Minneapolis, Minnesota, October 2-9, 1998; This committee formally resolved that because "CWD is an emerging disease with serious negative repercussions for

livestock and wildlife" they will develop educational campaigns, provide personnel for epidemiological investigations, fund research, pay for post-mortem tests and provide personnel to support herd surveillance.

19. E. S. Williams and S. Young, Chronic Wasting Disease of Captive Mule Deer: Spongiform Encephalopathy (16) 1 Journal of Wildlife Diseases 89-98 (Jan. 1980).

20. Id. "The CWD agent probably enters an elk via oral exposure to infectious secretions or excretions (e.g., saliva, feces, urine)." Michael W. Miller, Margaret A. Wild and Elizabeth S. Williams, 34(3) Epidemiology of Chronic Wasting Disease in Captive Rocky Mountain Elk, Journal of Wildlife Disease 532-538 (1998)

21. Correspondence from Victor F. Nettles to USAHA Wildlife Disease Committee Members dated October 12, 1998 and Resolutions, supra, note 18.

22. Juliet Wittman, Mad All Over: This Family of Killer Diseases Hits Close to Home. Too Close. <http://www.westword.com/1998/111298/news3.html>.

23. CWD in Oklahoma 14(2) SCWDS Briefs, A Quarterly Newsletter from the Southeastern Cooperative Wildlife Disease Study College of Veterinary Medicine at the University of Georgia at 1 (July, 1998).

24. Id.

25. Z. Davanipour, M. Alter, E. Sobel, D.M. Asher, and D.C. Gajdusek, Transmissible Virus Dementia: Evaluation of a Zoonotic Hypothesis, Vol. 5, No. 4, Neuroepidemiology (1986).

26. Marc Kamin and B.M. Patten, Creutzfeldt-Jacob Disease: Possible Transmission to Humans by Consumption of Animal Brain, 76(1) American Journal of Medicine 142-145 (1984).

27. Sandra Blakeslee, Kentucky Doctors Warn Against a Regional Dish: Squirrels' Brain: a Southern Variant of Mad Cow Disease is Suspended, New York Times, at A10 (Aug. 29, 1997).

Squirrels are a popular food in rural Kentucky, where people eat either the meat or the brains but generally not both . . . Those who eat only squirrel meat chop up the carcass and prepare it with vegetables in a stew called burgoo. Squirrels recently killed on the road are often thrown into the pot. Families that eat brains follow only certain rituals. 'Someone comes by the house with just the head of a squirrel," Dr. Weisman said, "and gives it to the matriarch of the family. She shaves the fur off the top of the head and fries the head whole. The skull is cracked open at he dinner table and the brains are sucked out.' It is a gift giving ritual. The second most popular way to prepare squirrel brains is to scramble them in white gravy, he said or to scramble them with eggs. In each case, the walnut-size skull is cracked open and the brains are scooped out for cooking. Id.

28. Id.

29. Id. referring to the following scientific letter, Joseph R. Berger, et al., Creutzfeldt-Jakob Disease and Eating Squirrel Brains, 350 (9078) Lancet (Sat., Aug. 30, 1997). Dr. Berger is in the Department of Neurology at the University of Kentucky.

30. Id.

31. Laura Manuelidis, et al., Evolution of a Strain of CJD That Induces BSE-Like Placques, 277 Science, at 94-98 (July 4, 1997).

32. Adriano Aguzzi, Protein conformation dictates prion strain, 4(10) Nature Medicine at 1125-1126 (Oct. 1998).

33. A.R. Bobwick, J.A. Brody, M.R. Matthews, R. Roos, D.C. Gajdusek, Creutzfeldt-Jakob Disease: A Case-Control Study, 98 American Journal of Epidemiology 381-394 (1973).

34. Id.

35. Z. Davanipour, M. Alter, E. Sobel, D.M. Asher, D.C. Gajdusek, A Case-Control Study of Creutzfeldt-Jakob Disease: Dietary Risk Factors, 122 American Journal of Epidemiology 433-451 (1985).

36. Id. at 448.

37. C.M. van Duihn, N. Delasnerie-Lauprêtre, C. Masullo, I. Zerr, R. de Silva, D.P.W.M. Wientjens, J.-P. Brandel, T. Weber, V. Bonavita, M. Zeidler, A. Alpérovitch, S. Poser, E. Graneri, A. Hofman, R.G. Will for the European Union (EU) Collaborative Study Group of Creutzfeldt-Jacob Disease (CJD), Case Control Study of CJD in Europe During 1983- 1985, 351 The Lancet 1081-1085 (Apr. 11, 1998).

38. Id. at 1083.

39. Clarence Joseph Gibbs, et al., Experimental Transmission of Scrapie to Cattle, 335 Lancet 1275 (1990).

40. Id. at 1275.

41. M. Hansen, Consumers Union's Comments on Docket No. 96N-1035, Prosed Rule: Substances Prohibited for Use in Animal Food or Feed; Animal Proteins Prohibited in Ruminant Feed, February 14, 1997.

42. Id.

43. Id.

44. Id.

45. Juliet Wittman, Mad All Over, This family of killer diseases hits close to home. Too Close., Westword News Website, <http://www.westword.com/1998/111298/news3.html>, Ann Schrader, Woman Shows Symptoms of Rare Disease, Denver Post, at 1B, (Sept. 15, 1998).

46. Tillie Fong, Federal experts summoned in case of rare brain disease, Rocky Mountain News, Sept. 17, 1998.

47. Id.

48. Id.

49. Wittman, *supra*, note 45. A California women, who wishes to remain anonymous, relayed that her husband was infected with CJD when he had reconstructive eye surgery. I.

50. Michael Prescott and Steve Farrar, Surgical Instruments Could Pass CJD Between Patients, Sunday Times of London, (Nov. 8, 1998).

51. Id. "One scientist said . . . that the number of people incubating CJD (in England) could run to millions, meaning many operations were being carried out on infected people, and infected implements then reused." Id.

52. David Brown, Medicines "Greater BSE Risk than Beef", The Telegraph (Dec. 16, 1998).

53. Lois M. Collins, Donor Illness Spurs Blood Quarantine, Deseret News (Dec. 16, 1998).

54. Id.

55. Report of a WHO Consultation on Medicinal and other Products in Relation to Human and Animal Transmissible Spongiform Encephalopathies, Section 2.2.2 Measures to minimize risks to humans from human derived material <http://www.who.int/emc/diseases/bse/

tse_9703.html>. (Emphasis added.)

56. Motor Vehicle Manufacturers Association v. State Farm Mutual Automobile Insurance Co., 463 U.S. 29 (1983).

57. Id. at 43.

58. Marsh v. Oregon Natural Resources Council, 490 U.S. 360, 378 (1989).

59. W.A. Marks, et al., Cerebral degenerations producing dementia: importance of neuropathologic confirmation of clinical diagnoses, 1(4) Journal of Geriatrics-Psychology-Neurology, at 187-98 (Oct./Dec. 1988).

60. United States General Accounting Office, Alzheimer's Disease: Estimates of Prevalence in the United States, GAO/HEHS-98-16 at 6 (Jan. 1998).

61. François Boller, Oscar L. Lopez, John Moossy, Diagnosis of Dementia: Clinicopathologic Correlations, 38 Neurology at 76-79 (Jan., 1989).

62. Id.

63. Id.

64. Id.

65. E.F. Manuelidis and L. Manuelidis, Suggested links between different types of dementias: Creutzfeldt-Jakob disease, Alzheimer disease, and reteroviral CNS infections, 2 Alzheimer Disease and Associated Disorders 100-109 (1989). "[T]he new BSE agent has acquired an enhanced ability to evade host defenses. . . . Because human CJD infections can be undetectable for more than 20 years, it is likely that we will see more BSE-linked cases." Laura Manuelidis, et al., supra, note 31 at 94.

66. Id.

67. Id.

68. Id.

69. Testimony of M. Hansen, Ph.D, to the FSIS/APHIS Meeting on the Harvard BSE Risk Analysis Project, (Sept. 28, 1998).

70. Id.

71. Id.

72. CRS Report for Congress, Congressional Research Service, The Library of Congress, Judith A. Johnson and Donna U. Vogt, "Mad Cow Disease" or Bovine Spongiform Encephalopathy: Scientific and Regulatory Issues, (July 9, 1997) (Emphasis added.)

73. Ellen Ruppel Shell, Could Mad-Cow Disease Happen Here?, 81 The Atlantic Monthly, 92 at 106 (Sept., 1998) quoting Dr. Laura Manuelidis, a Yale researcher.

74. Id. quoting Dr. Stephen Morse, Director of the Program in Emerging Diseases at the Columbia University School of Public Health.

75. Gary Hsich, Kimbra Kenney, Clarence Joseph Gibbs, Kelvin H. Lee, Michael G. Harrington, The 14-3-3 Brain Protein in Cerebrospinal Fluid as a Marker for Transmissible Spongiform Encephalopathies, 335 New England Journal of Medicine 924-930 (1996).

76. N.Y. Times News Service, New Lab Test May Detect Mad-Cow Disease (Sept. 26, 1996).

77. Dr. Clarence Joseph Gibbs, Statement to Congress, Committee on Government Reform and Oversight, Subcommittee on Human Resources and Intergovernmental Relations, Jan. 29, 1997.

78. Id.

79. Daniel Q. Haney, AP Medical Editor, New Test for Mad Cow Disease, (Sept. 26, 996).