

Elizabeth
Is there anything
in this?
Elena
cc: Bruce

Inmate Accused of Collecting Internet Child Pornography

By NINA BERNSTEIN

In a Federal grand jury indictment unsealed yesterday, a Minnesota prison inmate has been charged with conspiring to collect and trade child pornography via the Internet using a prison computer.

The inmate, George G. Chamberlain, 57, is a convicted pedophile whose role in the prison computer operation was reported in a front-page article in *The New York Times* on Nov. 18, 1996. The article dealt with a list of thousands of children that had been secretly compiled, annotated and stored along with a cache of child pornography in the prison computer used by Mr. Chamberlain.

The list of children does not figure

A case that raises extraordinary issues, prosecutors say.

in the indictment handed up by the Minneapolis grand jury, but it will be used as evidence in Mr. Chamberlain's trial, said a source close to the investigation who spoke on condition of anonymity.

The two-count indictment says that as early as January 1994, while he was a prisoner at the Minnesota Correctional Facility at Lino Lakes, Mr. Chamberlain used a prison computer and the Internet to obtain child pornography, used E-mail through an anonymous remailing service in Finland to discuss with others how to avoid detection, and possessed an optical disk containing 287 visual depictions of minors engaged in sexually explicit conduct.

"This case raises extraordinary and timely issues," United States Attorney David Lillehaug said yesterday. "The idea that a prisoner had this kind of access to the Internet, and was able to collect explicit child pornography and communicate with others on how to hide it is almost unbelievable."

Mr. Chamberlain has been in prison since 1979, but his adult history of sex-related felonies began when he was 18 and spans a 20-year period punctuated by prison terms in three

states and six failed attempts at treatment, according to court records. His youngest recorded victim was a 6-year-old girl. State law requires his release from prison in 2002, when he will have served two-thirds of his current sentence.

If convicted of the new charges, one count of conspiracy to distribute child pornography and one count of possession of child pornography, Mr. Chamberlain faces up to 15 years in prison, a \$250,000 fine or both.

After finding the pornography and the list of children, corrections officials brought him up on disciplinary charges, seeking to extend his prison stay. But Mr. Chamberlain denied involvement and blamed another inmate in the same prisoner-run computer programming and telemarketing business. An administrative hearing board found the case unproved.

Mr. Chamberlain has been the target of a Federal criminal investigation since the list and the child pornography were discovered during an outside audit of the computer operation in the fall of 1994.

"It takes a long time detect and analyze a hard drive when one false move can destroy critical evidence," Mr. Lillehaug said.

In the aftermath of the article about the list, the Minnesota Department of Correction was flooded with inquiries from worried parents asking if their children were on it, Shari Burt, a spokeswoman, said. The department set up a toll-free number and took from 10,000 and 15,000 calls, she said.

Children on the original list were mostly from small towns in northern Minnesota. They ranged from babies one month old to children in their early teens, but most were girls between 3 and 12. The list contained such personal details as "latchkey kids," "cute" and "Little Ms. pageant winner," apparently compiled from local newspaper articles.

An additional list of about 3,000 children in the Minneapolis-St. Paul area, including street addresses, was discovered in January on the computer hard drive. The department notified those parents by letter, Ms. Burt said, and sent them material with tips on keeping their children safe. There is no evidence that the list was distributed by inmates over the Internet or any other way, Ms. Burt stressed.

U.S. Is Asked to Take New Steps to Prevent Mad Cow Disease

By MARIAN BURROS

WASHINGTON, March 27 — A coalition of consumer groups, veterinarians and Federal meat inspectors asked the Federal Government today to improve its meat inspection system and to take other measures to help prevent the spread of mad cow disease.

Earlier this year, the Food and Drug Administration proposed a ban on using tissue from animals that chew their cud — cows, sheep, goats, deer and elk — in animal feed. The

coalition asked today that hogs be added to that list. There is strong evidence that mad cow disease, bovine spongiform encephalopathy, spreads through contaminated animal protein. Mink are included in the ban because they have carried a similar ailment.

Hogs have been excluded because the Government says it has no evidence that the animals have ever had the disease. But the coalition says there is evidence that hogs can develop a form of the disease. The

Safety and Inspection Service, said further measures were not necessary. He said that the agency was concerned about the possible risk in hogs used in feed but that there was "no evidence that argues for broadening what we are doing."

Dr. Stephen F. Sundlof, director of the center for veterinary medicine at the F.D.A., said the agency "would take the evidence presented today into consideration." The problem with a wider ban, he said, is that officials would have to find some way

to dispose of the animal renderings that are now used in feed.

In asking for a wider ban, Consumers Union and the Government Accountability Project, a Washington-based whistleblower support organization, cited evidence from 1979 suggesting unusual symptoms in the central nervous systems of young hogs. At the time, scientists were not aware of mad cow disease. But after the announcement by the British Government last year that there may be a link between mad cow disease and the human ailment Creutzfeldt-Jakob disease, which is always fatal, some Government scientists recommended a re-examina-

tion of the 1979 slides from the hogs. Although more recent readings of the slides have been inconclusive, experts in the field say that the evidence suggests transmissible spongiform encephalopathy. The coalition also noted that mad cow disease had been experimentally induced in pigs. It is asking the Agriculture Department to conduct further research and to step up inspections at hog slaughterhouses.

- Slides of one pig
- Inconclusive if TSE

- Haven't ruled out that they would do research on hogs - focused on cattle.

The New York Times

FRIDAY, MARCH 28, 1997

Table 1

BSE: ISSUES AND ACTIONS	
I. Actions to prevent the introduction of BSE into the domestic cattle herd.	
* Ban on the importation of live cattle and sheep from countries with native cases of BSE. (APHIS) * Restrictions on importation of meat from countries with native cases of BSE. (APHIS & FSIS) * Restrictions on importation of other bovine-derived products. (APHIS & FDA) * Surveillance of suspect animals — on-farm; antemortem slaughter, downers; diagnostic labs; practitioners and universities; zoos; UK imports; chronic wasting disease; mink TSE (APHIS & FSIS)	
II. Actions to prevent epidemic disease (BSE or TSE)	
* Proposed ruminant to ruminant ban (FDA) * Evaluation of rendering methods to find method for eliminating infectivity	
III. Actions to prevent potentially affected animals and tissues from entering the human food chain	
* Condemnation of cattle and sheep with CNS symptoms on antemortem (FSIS) * Condemnation of certain "downer" cattle on antemortem, such as those that are comatose. (FSIS)	
IV. Monitoring potential public health implications	
* Surveillance for incidence of CJD in this country; search for nv-CJD (CDC)	

Note: All categories include research and education activities.

Talking Points
Food Safety and Inspection Service

- During 1978 and 1979, Dr. Doi, a USDA veterinary medical officer, became interested in pigs with symptoms of CNS disorders. He sent 100 tissue specimens to the USDA Eastern Laboratory. There was evidence of vacuoles in brain tissue of one animal.
- The carcass of the pig in question was destroyed and was not allowed to go into the food supply.
- In 1996, increased concerns about BSE caused Dr. Masuo Doi, now a circuit supervisor in USDA, to re-contact the EL and discuss the old finding.
- USDA pathologists were able to locate both records and slides remaining from 1979 and re-examined the brain slides for signs of TSE. One slide did have vacuoles but otherwise was not conclusively TSE, e.g. spongiform changes were not apparent.
- USDA EL requested independent evaluation of the slide(s):
 - 5 slides were sent to National Veterinary Services Laboratory in Ames Iowa, where they were tested for the presence of abnormal prion proteins. None were found.
 - Slides were also sent to the University of Georgia
 - The one slide that showed vacuoles was sent to Dr. W. J. Hadlow
 - These “outside” experts confirmed observations of the EL and, similarly, did not conclude that this was a TSE.
- FSIS presented a letter of commendation to Dr. Masud Doi for his vigilance in the area of CNS disorders in swine. This award was issued on January 14, 1997. Dr. Doi also received a letter of condemnation in 1980.
- There is no known naturally occurring Transmissible Spongiform Encephalopathy in pigs, even in the United Kingdom. The only known TSE in pigs was experimentally induced by injections, not feeding, of BSE infected tissue.
- Dr. Hadlow stated that the vacuoles he saw on the slide were located in a part of the brain where they might occur “as an incidental finding”.
- FSIS has not focused its limited resources on pigs since TSE has not been reported in that species at all..
- USDA is participating with other federal agencies in a cooperative effort to address potential public health problems associated with animal TSEs, specifically BSE. (see briefing for Sec, could include Table 1 here)

Statement in Response to Claims of Government Accountability Project

by

Thomas J. Billy

Administrator, USDA Food Safety and Inspection Service

March 27, 1997

USDA shares Government Accountability Project's concern about the possible risk of a Transmissible Spongiform Encephalopathy in pigs.

In 1979 and 1980 a Food Safety and Inspection Service veterinarian collected samples from pigs exhibiting abnormal behavior during antemortem observation. FSIS commended the veterinarian for his work at the time and responded to his request to retrieve the slide in 1996 after reports of Bovine Spongiform Encephalopathy in the United Kingdom. FSIS had the slide independently examined. Experts outside the Food Safety and Inspection Service did not conclude it was a Transmissible Spongiform Encephalopathy.

The independent confirmation results, coupled with no known occurrence of naturally transmitted TSE in pigs and no evidence that pigs have succumbed to oral exposure to BSE infected tissue in clinical studies, have convinced USDA to concentrate efforts on the detection and prevention of BSE.

Contrary to GAP's assertions, FSIS has intensified our training of veterinarians. USDA veterinarians are authorized to take the action necessary to make good determinations during antemortem inspection.

USDA would support adding pig tissue to FDA's proposed ruminant to ruminant ban if there was scientific evidence to support it. Further, FSIS has no intention to reduce antemortem inspection and is willing to refocus our efforts to respond to demonstrated risks.

INFORMATION PACKAGES
PRESS CONFERENCE
G.A.P.
MARCH 27, 1997

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 Food Chemincal News 202-544-1980
 Bloomberg 624-1832/624-1300
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 CNN 202-898-7633; #7604
 R. Scientist 331-0922; F#2082
 898-8319
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OTHER

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PRESS RELEASE

Coalition calls for USDA answers on "Mad Pig Disease"

Embargoed until Thursday, March 27

**For further information, call
Felicia Nestor or Tom Devine, GAP**

A coalition of veterinarians, inspectors and consumer organizations today called for the U.S. Department of Agriculture (USDA) and the Food and Drug Administration (FDA) to address long-dormant questions about whether public health concerns from England's "Mad Cow Disease" also apply to hogs. The British beef scare has sparked national policy reform proposals to protect American consumers and markets.

In a letter today to USDA Secretary Dan Glickman, the groups called for USDA to "take serious public health action this time before there is a tragedy." Groups included the Government Accountability Project, a whistleblower support organization, the National Association of Federal Veterinarians, leadership of the National Joint Council of Food Inspection Locals, Community Nutrition Institute, Humane Farming Association, National Consumers League, and Safe Tables Our Priority (S.T.O.P.), a national support organization for families victimized by food poisoning.

Consumers Union simultaneously recommended that the Food and Drug Administration include hogs in its proposed ban on animal protein in animal feed. The FDA's proposal is supposed to prevent spread of Transmissible Spongiform Encephalopathies (TSE), the scientific name for the disease. "Ignoring hogs could be the Achilles heel of U.S. efforts to protect Americans from TSE threats like Mad Cow Disease," warned GAP Legal Director Tom Devine. "The credibility of the President's Food Safety Initiative is at stake."

GAP's Food Safety Director Felicia Nestor explained that the group acted on concerns from government veterinarians that USDA has not followed through responsibly to resolve questions raised by a 1979 study, which suggests TSE's could exist in hogs. The vets renewed their concerns after the Mad Cow Disease outbreaks in Great Britain.

Nestor explained that current and planned policy has three public health loopholes – 1) inadequate ante-mortem inspection. The government reassures consumers that its inspection system will catch sick animals before slaughter. In reality up to 95% of livestock are uninspected for relevant disease symptoms, a rate which draft regulations threaten to increase; 2) inadequate post-mortem laboratory testing, which does not check for TSE in hogs; and 3) overly-narrow boundaries for feed bans, that could permit hogs to spread the disease. "These three public health loopholes are three strikes against consumers," warned Nestor. She added, "Diseased animals can still be a human health hazard. Until USDA publishes credible scientific research, it is inexcusable to cut back the government's early warning system for TSE or similar threats."

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March 27, 1997

Honorable Dan Glickman
14th and Independence Avenue, S.W.
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Dear Mr. Secretary:

In 1978, a United States Department of Agriculture (USDA) veterinarian began a study of young hogs with symptoms of central nervous system (CNS) disorders at a slaughterhouse in New York. This study has very significant implications for current food safety policy at USDA. It also is pertinent to the stated goal of the President's recently announced Food Safety Initiative ". . . to reduce, to the greatest extent possible, the incidence of foodborne illness."

The study demonstrates that the frequency of antemortem inspection since at least 1979 is grossly inadequate to prevent animals with CNS symptoms from entering the human food supply. Further, the study revealed evidence of a disease condition in young hogs that has similarities to Transmissible Spongiform Encephalopathies (TSE's)¹, such as "Mad Cow Disease" which caused an epidemic in British cows.² The human health costs of BSE in

¹TSE's have been identified in a number of species, including cattle, sheep (scrapie), goats, deer, elk, mink, cats, zoo animals, and many primate species, including humans.

²The evidence of a spongiform encephalopathy in one of the animals in this study is especially significant because of the appearance of the "Mad Cow" epidemic in Europe. According to the Department of Health and Human Services, this form of TSE's in cows, known as Bovine Spongiform Encephalopathy, has been linked to the death of over ten people in Britain from a variant form of the TSE which exists in humans called Creutzfeldt-Jakob disease. Unfortunately very little is known for certain yet about this class of disease, except that there is a 100% mortality rate and no known cure. Although scientists have successfully created a TSE in hogs through inoculation, there has been no known evidence of natural development in swine. There are a number of reasons, however, why even if the disease exists, present methods may have been inadequate to detect it. The slides and/or the veterinarian's report of the behavior of animals in this study could be an evidentiary start.

Britain and from TSE's in the food supply in general are as yet unknown, but are potentially very high and a legitimate cause for concern.

To date, USDA largely has ignored the implications of this study. Instead, USDA should conduct a serious credible scientific assessment of the data's implications for future antemortem inspection policy. USDA should also investigate to learn the full extent of any CNS disease condition currently found in hogs which may be evidence of a TSE.

THE STUDY AND ITS FINDINGS

In late 1978, a Food Safety and Inspection Service (FSIS) veterinarian noticed unusual CNS symptoms in hogs being received at a slaughterhouse in New York. Because there was no single source of the animals³ and because the plant was not one which routinely dealt with diseased animals, the veterinarian believed the condition might affect animals being slaughtered nationwide. He began a study at the plant which included extended observation of the animals followed by histopathological analysis of tissues from various organs, after slaughter.

From November 1978 to February 1980, the veterinarian extended the usual two day observation period for animals suspected of having CNS disorders, to up to four days. During that time, the veterinarian took careful notes on the animals' behavior and other vital signs. After observation, animals still suspect were slaughtered and retained. Tissue samples from the brains and other organs were sent to the Eastern Laboratory in Athens, Georgia for confirmation of the diagnosis.

During the 16-month study from November 1978 to February 1980, over 70 young hogs (average age 4-8 months) were confirmed by laboratory analysis to have CNS symptoms. Though these condemnations were from only one plant, they represented 72% of CNS condemnations recorded nationwide by FSIS during the same time period.

Especially significant, in 1979 laboratory analysis for one of the animal's brain tissues (case #316074, laboratory #2709) found inexplicable similarities to TSE's in sheep and mink⁴.

³They were from a variety of sources in Indiana, Canada, New York, Illinois, Ohio and elsewhere in the Midwest.

⁴ The lab report notes:

'According to . . . Pathology of Domestic Animals, . . . "The degeneration of neurons, the reactivity of the glia . . . are the classical hallmarks of viral infection of the central nervous system and imply a sequence of viral cytopathogenicity and reaction to cellular degeneration." Scrapie of sheep, and encephalopathy of mink, according to the literature, all produce focal vacuolation of the neurons similar to the kind as described for this pig. I was unable to locate any lead as to the cause of this interesting phenomenon in other species including swine. . . '

At the time Bovine Spongiform Encephalopathy or "Mad Cow Disease" was unknown. USDA did no follow up studies on the specific brain or on hogs in general. Scientists involved with the case, however, mentioned it over the years to students and other scientists.

Once the characteristics of "Mad Cow Disease" were publicized, some of the veterinarians and scientists involved with this case salvaged and reviewed the scant evidence remaining from the 1979 analyses. Unfortunately most of the tissues had been discarded. These veterinarians and scientists also urged further review. In 1996, USDA sent the remaining slides to two experts on spongiform encephalopathies, Dr. Janice Miller of the Agricultural Research Service, and Dr. William Hadlow, a renowned TSE expert.⁵

Both scientists noticed similarities with other spongiform encephalopathies and found but were unable to conclude with certainty that the animal had a TSE. Many involved with the case urge still further study by USDA. Administrators have not yet authorized or allocated funds for any further research, although some studies could be devised which are not resource intensive.

POLICY ISSUES

The results of this study raise two significant concerns for consumers, scientists, and policy makers. First, USDA's frequency of antemortem inspection allows thousands of animals with CNS symptoms into the human food supply. This violates the law and severely departs from consumer expectations. Second, there may be a CNS disease in swine that we know very little about. This condition could have serious health significance for human and/or animal health. Additionally, the possibility that this study may have uncovered some evidence of a porcine TSE can not be ruled out. Further research is essential to know whether we have all necessary information for responsible public health decisions about the food supply or about measures necessary to prevent a spread of TSE's in this country.

The condemnation rate data from the study raises serious questions about the adequacy of the current antemortem inspection procedures, which are a critical part of the Food Safety and Inspection Service's (FSIS's) program for fulfilling its mandate to ensure the wholesomeness of the nation's meat and poultry supply. Central nervous system symptoms are detected by observing animal behavior. Therefore, inspection prior to slaughter is indispensable. Diseased animals are condemned and kept from the food supply. This has always been an important task because, by law, no animals with CNS disorders may be

316074. Report completed November 8, 1979.

⁵USDA sent fives slides to Dr. Miller and only one slide to Dr. Hadlow.

approved for human food.⁶ It is especially important since the recent outbreak of BSE. The BSE epidemic in Britain was first detected by a dairy farmer who noticed that one of his cows had become aggressive and nervous.⁷ If a TSE were to develop here in any of the livestock populations, antemortem inspection is one of the frontline measures for early detection. In fact, recently, in both public comments and documents, government representatives have identified our antemortem inspection procedures as a primary and reliable safeguard for protecting us from the ravages of a TSE in this country.⁸

Despite these critical implications of, and government assurances of safety based on antemortem inspection, this study strongly suggests that the vast majority of animals with CNS disorders are being passed for human consumption. During the 1979 calendar year, 57 of the approximately 350,000 swine sent for slaughter at the plant where the study was done, were determined to have a CNS disorder.⁹ The condemnation rate at that plant, therefore, was 0.01628%. Assuming a similar incidence of CNS disorders in swine being slaughtered nationwide¹⁰, there should have been 13,500 condemnations out of the 83 million hogs slaughtered during that year. According to USDA's Statistical Summary for the 1979 calendar year, there were only 79 swine condemnations for CNS disorders nationwide. Since the antemortem inspection procedures have remained about the same, it is reasonable to question

⁶"Ante-mortem inspection is to accept only animals (livestock or poultry) capable of producing products acceptable for use as human food. Such inspection removes from human food channels animals: . . . (2) with diseases or conditions difficult to detect on routine post-mortem inspection (central nervous system disorders, chemical poisoning). . .". Part 9 Ante-mortem inspection. Title: 9.1 PURPOSE.

⁷Richard Rhodes. "Mad Cows and Americans". Washington Post Magazine. March 9, 1997, p.13.

⁸In a Department of Health and Human Services FACT SHEET titled "BOVINE SPONGIFORM ENCEPHALOPATHY and CREUTZFELDT-JAKOB DISEASE, Public Health Service Actions to Ensure Against Health Risks", the section, "U.S. ACTIONS: Steps to Minimize any Possible Risk" states:

The United States Department of Agriculture (USDA) is responsible for the health of U.S. livestock. USDA's Food Safety and Inspection Service operates a substantial program for inspecting animals intended for human consumption . . . USDA examines all cattle before they can be approved for use as human food. Cattle with undiagnosed diseases of the central nervous system are not allowed for human food. USDA examines over 33 million head of cattle each year.

In FSIS's meeting for constituent groups on Advance Meat Recovery, in response to a question concerning BSE, Mr. Thomas Billy, FSIS Administrator, and Mr. Craig Reed, Deputy Administrator for Field Operations, identified FSIS's program of antemortem inspection as the barrier employed to keep animals with a TSE from entering the food supply.

⁹Freedom of Information Act Request #96-608, by Government Accountability Project on October 23, 1996 and received on November 20, 1996.

¹⁰This was a typical swine slaughterhouse that dealt with typical market hogs. See n.3, supra.

whether since at least 1979 USDA has been allowing 99.5% of animals with encephalitis, meningitis and other CNS disorders into the human food supply.

It is readily apparent how this could occur. First, the symptoms of CNS disorders are very subtle and the current antemortem procedure may not adequately them. Under current inspection, FSIS inspectors or veterinarians observe a group of approximately 200 hogs (or other livestock) while they are confined. They then observe only 5 - 10% of the animals while they are in motion. If the observers are inspectors, they segregate only the suspect animals for further observation by a federal veterinarian. **Subtle CNS symptoms are unlikely to be noticed by even experienced observers when the animals are at rest. This means that 90 - 95% of livestock animals are, therefore, virtually uninspected for CNS disease.**

Further problems include:

- *little program emphasis for federal inspectors on antemortem inspection training;
- *insufficient time allotted for this activity at slaughterhouses (typically, 15 minutes), preventing extended or repeat observation of suspect animals. The signs of CNS are so subtle that when such hasty judgements are forced upon inspectors and veterinarians, diseased animals will be forced into the food supply;
- *inability under the circumstances to detect nonroutine CNS symptoms.

It seems reasonable to conclude that a significant percentage of animals with CNS disorders are being passed for human food. This deficiency needs to be investigated fully and addressed.

The second important finding from this study involves the symptomatology of the hog identified as Laboratory #2709. This case was particularly interesting at the laboratory level in 1979. The diagnosing scientist reported that the only similarities he could find in the literature to the neuropathology of case 2709, were to sheep and mink TSE's. Other scientists later involved also noticed similarities but could not conclude that this was a TSE with so little evidence.

The possibility of a swine TSE has not yet been responsibly ruled out.¹¹ Unfortunately, policy with potentially far reaching human health implications is already being made on the assumption that there is little danger of a swine TSE. For example, the Food and

¹¹ Swine can manifest and transmit a spongiform encephalopathy. British researchers succeeded in creating a TSE in swine by injecting them with infective BSE material. (Phone conversation with Dr. Ray Bradley, 2/27/1997). Dr. Clarence Gibbs of NIH has proposed the theory that since all mammals have prion proteins [the currently-accepted cause of TSE's], it is theoretically possible that any of them could develop the prion mutation that leads to TSE's (Public comments of Public Voice to FDA Docket No. 96N-0135. February 18, 1997).

Drug Administration is currently considering the adoption of a ruminant-to-ruminant feed ban.¹² This would allow swine and other non-ruminant animals to be used as food for farm animals, and vice-versa. If a porcine TSE exists or were to develop here which crossed the species barrier into other livestock through refeeding, the currently proposed ban would be insufficient to protect animal and human health. Similarly, swine could be infected by suspect feed from non-porcine ruminants with TSE. Further study on the possibility of a swine TSE would reveal whether the proposed ban was sufficient.

Under another initiative, USDA has increased surveillance for TSE's only in cattle, based on an absence of conclusive evidence that dangerous and unknown TSE's exist in other food animals. It will not reassure consumers, if the absence of evidence merely reflects the government's refusal to look. Given the critical problems with antemortem inspection as currently practiced, and the fact that in the vast majority of CNS condemnations, no specimens are sent for lab analysis because of resource pressures, this possibility must be considered. This surveillance criterion is also problematic because it is circular – the government will not make any special effort to look for the disease until it is detected in the normal process of ante-mortem inspection, which needs to be intensified to catch it.

Finally, at a minimum, this study exposed symptoms which are still, unexplained. Further research would provide needed information on a potentially new central nervous system disorder which may have human health implications.

Given the significant implications of this study, we respectfully make the following recommendations:

* The law as well as consumer expectations require that animals with central nervous system disorders not be passed into the human food supply. USDA should upgrade antemortem inspection in its priorities. This should include increasing program emphasis, frequency and stepped-up continuing education on developing diseases and symptoms for inspectors and veterinarians, as well as increased time and opportunity to adequately inspect animals for CNS symptoms at slaughtering facilities. As recognized by the President's National Food Safety Initiative, new diseases are evolving or being imported every day.¹³ It is critical for human health that we retain an objective and trained veterinary and inspection force to detect the first signs of potentially lethal zoonotic disease in postmortem inspection procedures as well.

¹²This would prohibit feeding of ruminant animals (cud-chewing animals, such as sheep and cows) back to other ruminants. Such "refeeding" is a common practice in the livestock industry as a way of providing supplemental protein to developing livestock. The basis for the ban is FDA's analysis of current scientific literature which suggests that ruminant-to-ruminant feeding could spread or amplify any TSE which developed in the ruminant population, potentially leading to the development of a silent epidemic in this country.

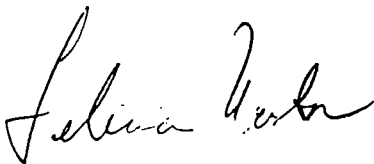
¹³"Discussion Draft and Current Thinking/ A National Food Safety Initiative." pg 20

* The USDA should reopen its dormant study and initiate controlled studies to see if an unknown CNS disorder exists. If so, the potential human health implications should be researched.

* The USDA should include swine in appropriate research protocols on TSE's. For example, brains from swine with CNS symptoms should be collected and analyzed as part of the collection and analysis of brains from cattle with CNS currently being administered by APHIS.

Mr. Secretary, we are writing, because USDA has been aware of this issue for almost a year but has not acted. It would be premature to give a clean bill of health for hogs, simply because USDA has refused to look since the TSE threat resurfaced. We request USDA to take serious public health action this time before there is a tragedy.

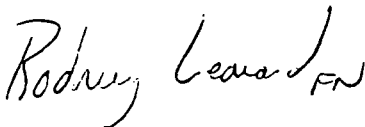
Sincerely,



Felicia Nestor
Food Safety Director
Government Accountability Project



Elizabeth Dahl
Staff Attorney, Food Safety Program
Center for Science in the Public Interest



Rodney Leonard
Executive Director
Community Nutrition Institute



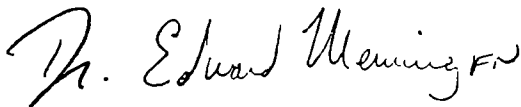
Diana Neidle
Public Policy Associate
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Bradley Miller
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Arthur Hughes
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Elaine Dodge
Safe Tables Our Priority (S.T.O.P.)

S.T.O.P. - Safe Tables Our Priority

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Victim hotline 1-800-350-STOP Phone 619-439-2969 Fax 619-967-0612

I am Heather Klinkhamer, board member of S.T.O.P. -- Safe Tables Our Priority. S.T.O.P. is the only national, grassroots foodborne illness victims organization. It was founded in 1993 by families and friends of people who became ill or died from exposure to E. coli 0157:H7.

S.T.O.P. commends the Government Accountability Project's food safety program for its work with USDA whistle blowers. The investigative work on the 1978 hog study is superb. It raises serious issues and legitimate concerns.

Some may think our request to reopen the 1978 study encourages the government to be over protective -- to investigate a flimsy lead that may not provide results relevant to public health. S.T.O.P. disagrees.

In 1994, an outbreak of E. coli 0157:H7 was linked to sausage produced at one of the most responsible and state of the art sausage companies in the United States. Initially, people responded to the outbreak with surprise. Until the outbreak happened, everyone thought that E. coli 0157:H7 was destroyed in the curing process. Well, almost everyone.

A 1992 industry study indicating that E. coli 0157:H7 could survive the curing process surfaced soon after the outbreak. Apparently, the implications of the 1992 study were viewed as counterproductive to business concerns and were, therefore, ignored. Unfortunately, in 1994 many individuals and a good company suffered not only from a deadly pathogen, but also from apathy and wishful thinking.

Consumers, the government, and industry simply cannot afford to ignore the possible repercussions of the 1978 hog study. S.T.O.P. implores USDA to pursue the study to inform us of the possible risks linked to pork products or to inform us that there are no risks and put our minds at ease.

A good scientist has an unbiased, inquisitive, and critical mind. A good public health program based on sound science and preventing illness will encourage scientists to pursue research with significant public health repercussions, regardless of the potential impact on business concerns.

USDA is saddled with the conflicting roles of promoting and inspecting meat and poultry. However, USDA and the new National Food Safety Initiative have repeatedly assured consumers that they are committed to preventing foodborne illness and to detecting emerging foodborne hazards before they threaten public health. The government can begin demonstrating this commitment by pulling its head out of the sand and immediately pursuing the 1978 study.

Consumer Policy Institute

Consumers Union

March 24, 1997

Stephen F. Sundlof, D.V.M., Ph.D.
Center for Veterinary Medicine
Food and Drug Administration
7500 Standish Place, Room 482, HFV-1
Rockville, MD 20855

Dear Dr. Sundlof:

We are writing to you to submit information that has recently come to our attention which suggests that a TSE-like disease (transmissible spongiform encephalopathy) might exist in pigs in the U.S. We believe this new information calls for intensive research and makes it urgent to ban the use of all mammalian proteins, including swine, in the feed of all food animals, until better answers are found.

The evidence for the potential PSE (porcine spongiform encephalopathy) is as follows. In 1979, an FSQS veterinarian, Dr. Masuo Doi, noticed some unusual central nervous system (CNS) symptoms in young (about 6 months old) hogs coming into a slaughter plant in Albany, New York. Since the plant received hogs from a wide variety of sources (New York, Canada, Indiana, Illinois, Ohio, and other Midwestern states) and was not a plant used to dealing with diseased animals, Dr. Doi thought that the problem might be affecting hogs slaughtered nationwide. So, he decided to conduct a detailed study on central nervous system (CNS) symptoms/disease in young hogs coming into that slaughter plant. The study ran for 15 months (January, 1979 to March, 1980) and consisted of extended observations of the behavior of animals with suspected CNS symptoms at the plant, followed by pathological, histopathological, and microbiological work on tissues from various organs of particular animals after slaughter.

For his behavioral observational work, Dr. Doi extended the usual two-day observation period to three to four days, during which he took careful notes on the animals' behavior and other vital signs. During the 15 month period of the study, some 106 animals exhibiting CNS symptoms were retained during antemortem inspection.

A 1980 paper (copy attached) that summarized Dr. Doi's findings on the clinical symptoms and incidence of the "disease," contained descriptions of CNS symptoms that sound remarkably similar to the symptoms noted for bovine spongiform encephalopathy (BSE): "Excitable or nervous temperament to external stimuli such as touch to the skin, handling and menacing approach to the animals is a common characteristic sign among swine affected with the disease. . . . In the advanced stage of the disease, manifestation of neurological signs are evidenced in the form of general ataxia . . . Many animals have been found to be "downers" at first observation; if the hindquarters of these downers are raised they may be able to walk one or two steps and then fall to the ground" (Doi et al., 1980: 2, 4). Indeed, a

table of symptoms includes, for the early stage: "excitability and nervousness (squealing, smacking of lips, grinding of teeth, chewing, gnawing and foaming at mouth); stiffness of limbs . . . 'tic'; weakness of hindquarters; focal tremors of skeletal muscles"; and for the advanced stage: "depression; ataxia; crossing over of limbs . . . kneeling posture . . . crawling". In addition to his clinical observations, Dr. Doi also made an 8 mm film of thirteen of the affected animals; film of two of the pigs was shown at the MPI National Pathology Meeting in Seattle, Washington on July 20, 1979.

Dr. Doi sent tissue samples from suspect cases to the USDA's Eastern Laboratory in Athens, GA for pathological, histopathological and microbiological work. Known infectious diseases were ruled out. As Dr. Doi points out, "Histopathological studies of tissue collected from the brain and spinal cord of these animals in the early stage of the disease show congestion, hemorrhage and neuronal degeneration. All animals in the advanced stage of the disease have been confirmed to have Encephalitis or Meningitis by MPI laboratory" (Doi et al., 1980: 5). Eventually some 60 animals were confirmed by the MPI Laboratory to have encephalitis or meningitis, with no identifiable cause. As pointed out in a paper presented at the 1979 MPI National Pathology Meetings, "Since January, a number of hogs in this establishment have been found, in antemortem, to show what appears to be CNS. Sets of tissue samples were sent to the laboratory for examination. Various tests were done which include histological study (E-H stain), fluorescence antibody technique, virus neutralization and viral and bacteriological isolation. Differential diagnosis was also done to exclude vitamin B deficiency, post vaccination reaction, chlorinated hydrocarbon, arthritis, and transport stress" (Doi et al., 1979).

The brains of the 60 animals were examined. The brain of one of these pigs, on histopathological analysis, exhibited signs reminiscent of a TSE. This histopathological work was performed by Dr. Karl Langheinrich, Pathologist-In-Charge at USDA's Eastern Laboratory in Athens, Georgia. According to the USDA FSQS laboratory report, dated early November, 1979, Dr. Langheinrich noted:

"Microscopic examination of the barrow tissues revealed a encephalopathy and diffuse gliosis characterized by vacuolated neurons, loss of neurons and gliosis in a confined region (nucleus) of the brain stem (anterior ventral midbrain). Only an empty sometimes divided vacuole was present instead of the normal morphology of a nerve cell. Occasionally a shriveled neuron was seen. According to . . . *Pathology of Domestic Animals*, . . . 'The degeneration of neurons, the reactivity of the glia . . . are the classical hallmarks of viral infection of the central nervous system' *Scrapie of sheep, and encephalopathy of mink, according to the literature, all produce focal vacuolation of the neurons similar to the kind as described for this pig.* I was unable to locate any lead as to the cause of this interesting phenomenon in other species including swine" (Langheinrich, 1979). (Italics added).

Indeed, Dr. Langheinrich's main diagnosis was, "1. Encephalopathy and diffuse gliosis of undetermined etiology." Portions of the brain were sent for microbiological testing to a neurologist at the University of Georgia, where they

came up negative for pseudo-rabies. The brain was unique enough that USDA scientists, such as Dr. Langheinrich and Dr. Doi, mentioned it to students and scientific colleagues over the years.

In 1979-1980, BSE was completely unknown. However, both the behavior of the pigs, as well as the histopathology on at least one pig, both showed signs consistent with a porcine TSE. This raises particular concern because the affected animal was only 6 months old; in an animal this young, one would not expect to see *any* physical signs of TSE in the brain. Histopathology of TSEs can be very variable, so that spongiform appearance (i.e. vacuolated neurons) are not always present. Behavioral changes can be seen in TSE-infected animals *before* any changes in brain morphology are visible. Dr. Clarence Gibbs, in testimony before a Congressional hearing on the TSE issue on January 29, 1997 made just this point:

"In the mid-1960s, we demonstrated with our French and English collaborators that *during the early incubation of the TSEs, when the virus titer in the brain was very low, there were already marked functional changes, even though no pathology was yet detectable, even ultrastructurally.* A month or two later, polynucleation of neurons appeared in spider monkeys, incubating kuru, and somewhat later, microvacuolation and membrane changes visible only by electron microscopy. *This preceded the first appearance of astrogliosis and spongiform change.* It was only much later that the classical scrapie-TSE pathology appeared with virus titers in brain of 10^{-5} or higher" (italics added) (Gibbs, 1997: pg. 4).

Given that TSEs can cause behavioral changes in infected animals before any physical changes in the brain can be seen, that the manifestation of TSE in the brain can be quite variable, and that changes in brain morphology are not usually seen in 6 month old animals, we are concerned that the brain of one pig actually showed physical evidence consistent with a TSE.

Following the announcement in March, 1996 of ten cases of new variant CJD (Creutzfeldt-Jakob Disease) in the United Kingdom and their possible connection to BSE, Drs. Doi, Langheinrich and others urged reinvestigation of this case.

In August, 1996, the USDA sent five slides, one of which was a histopathology slide, to Dr. Janice Miller of USDA's Agricultural Research Service¹. Dr. Miller stained four of the slides for prion protein (she didn't stain the H&E slide). Dr. Miller told Consumers Union that Dr. Patrick McCaskey, USDA/FSIS, in charge of the Research Center at Athens, GA, called her, told her that he had five slides that all showed "problems" and asked her to stain four of them. The H&E slide, which clearly show vacuoles in the neurons (one sign of TSE), wasn't stained because to stain for PrP entails removing the slide cover, baking the slide to destain it and then restaining it for PrP; they didn't want to risk destroying the H&E slide.

¹ Dr. Doi had kept frozen samples of the brain and spinal chord of the suspect PSE pig in case the Eastern lab wanted more material for analysis. Unfortunately, these samples were discarded when the packing plant in Albany, NY closed in 1991. It appears that the brain material sent to the University of Georgia may have been discarded. [pers. com., Dr. Doi 3/13/97]

Dr. Miller found that the PrP stained in the four pig slides was found only on the inside of neurons, while a positive control slide from a scrapie sheep showed massive amounts of extraneuronal staining. In a letter summarizing her results (copy attached), she concludes that the PrP stained in this pig was normal: "in the pig sections you will see a small particulate type of staining that is confined to neurons and as I indicated on the phone, I would interpret as normal PrP. It is in marked contrast to the massive amount of extraneuronal staining seen in the scrapie section" (Miller, 1996).

Unfortunately, Dr. Miller's finding does not conclusively rule out a TSE. We are concerned that while British BSE and scrapie create a massive amount of extraneuronal staining, there are TSEs where this isn't the case. Three experiments were done in the U.S.—in Mission, TX (APHIS work), Pullman, Washington (ARS work), and Ames, Iowa (ARS work)—to see whether sheep scrapie can possibly infect cows. In all the experiments, cattle were inoculated with tissue from scrapie-infected sheep—primarily by intracranial injection, but in the case of the Texas and Iowa studies also by oral feeding—to see if cattle were susceptible to scrapie at all. In all three experiments, the majority of cows injected in the brain with scrapie-infested sheep material (usually brains) also developed a fatal spongiform encephalopathy. However, in all three examples, the symptoms of the spongiform encephalopathy differed from "mad cow" disease in England, as did the appearances of slides from their brains. The brain lesions seen in all these animals were more variable than those seen in England. When Dr. Miller did similar staining for PrP from these brains (what she called "bovine scrapie,") she only found PrP stains on the *inside* of the neurons, not the massive extraneuronal staining seen in BSE (Miller, pers. comm., March 7, 1997). Thus, Dr. Miller's finding of PrP stains only *inside* the neurons in the suspect pigs is not particularly reassuring.

In November, 1996, USDA sent the single histopathology slide to Dr. William Hadlow, one of the foremost spongiform encephalopathy pathologists in the world. (For unknown reasons, Dr. Hadlow was only sent the one slide; he was not told of the existence of the other slides, nor of Dr. Miller's findings, nor was he told or given the behavioral report from Dr. Doi or the morphology work by Dr. Langheinrich, or shown film of the affected pigs [Dr. Hadlow, pers. com., 3/13/'97]). From this single slide, Dr. Hadlow found some evidence consistent with TSEs but not enough for a conclusive diagnosis. He noted that the slide contained vacuoles inside neurons, one of the signs of a TSE (Dr. Langheinrich had noted this as well). However, since such vacuoles occasionally occur normally in pigs, he thought that was not something special: "About twelve (12) neurons in the parasympathetic nucleus have unilocular optically empty vacuoles in the perikaryon. This is the site where such vacuolated neurons have been seen in the swine (as well as in cats and sheep) as an incidental finding. So I do not think such cells have any significance in this pig" (Hadlow, 1996). However, he did see evidence, including changes in astrocytes, that suggested a TSE, but without examining other parts of the brain to look for other evidence of TSE, he couldn't be sure:

"I am impressed, though, with what seems to be an increase in the number of astrocytes in the section. Some astrocytes are in clusters, some are enlarged and vesicular. Where they are most numerous, a few rod

cells (activated microglia) are seen. These findings suggest some perturbation of the nervous tissue. *Although such a glial response occurs in the transmissible spongiform encephalopathies, I do not know its significance in this case without examining other parts of the brain for changes characteristic of these diseases.* Thus, from looking at this one (1) section of brain, I cannot conclude that the pig was affected with a scrapie-like spongiform encephalopathy" (italics added) (Hadlow, 1996).

In sum, Dr. Hadlow's letter does not rule out the possibility of a TSE. He says that there is suggestive evidence, but that he would need to look at other slides/sections of the brain, to make a conclusive diagnosis.

In our view, the implications of this data are extremely serious. Experiments in the United Kingdom have shown that pigs are susceptible to BSE. Pigs inoculated with BSE develop a TSE (Dawson et al., 1990). Feeding experiments are underway in the UK to see if BSE can be orally transmitted to pigs; as of March, 1997, some 6+ years after the start of the experiment, none of the pigs fed BSE brain have come down with a TSE. Unfortunately the design of this experiment severely limits what we will learn from it, and will most likely not tell us conclusively if pigs can get BSE from feed. It turns out that the pigs were not fed BSE brain continuously. Rather, the pigs were only fed BSE brain material on three days, over a three week period (i.e., one day each week). Following these three doses, the pigs were *never* fed contaminated material again. The total amount of infective material given to the pigs was therefore quite small. Thus, a negative finding would be hard to interpret and would *not* mean that BSE is not orally active in pigs.

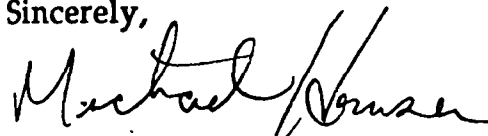
We believe that as a top priority USDA should conduct follow-up studies to look for potential CNS/PSE cases in pigs (we plan to communicate about this to USDA separately). In brief, we feel that the following kinds of studies need to be done:

- i) TSE pathology experts should examine *all* the slides from the suspect pig (2709). To our knowledge, at least 12 separate slides exist.
- ii) Determine if any brain material from the suspect pig (2709) still exists at the University of Georgia. If so, this material should be retrieved and used for transmission studies. In particular, suckling pigs should be inoculated with the material and then permitted to live until they die of a disease or old age, at which point their brains should be examined for physical signs of a TSE as well as for immunohistochemical evidence (i.e. staining looking for the abnormal PrP).
- iii) Increase antemortem inspection for CNS symptoms at hog facilities. Inspectors should be trained to detect the subtle CNS symptoms seen in the Doi et al. study. At a select number of slaughter facilities, animals exhibiting CNS symptoms should be removed and held for observation until they die, at which time their brains should be examined for evidence of a TSE.

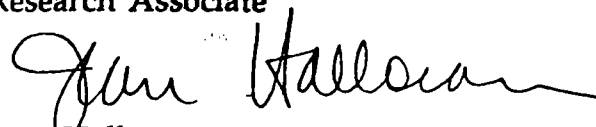
- iv) Research on CNS symptoms among the 6,000 or so breeding sows which are permitted to live for 3+ years. Sows exhibiting CNS symptoms should be removed and held for observation until they die, at which time their brains should be examined for evidence of a TSE.

While such work is underway, given the above information, we believe that as a precautionary measure the FDA *must* expand the proposed ruminant plus mink-to-ruminant feed ban to prevent protein from any mammal, including hogs, being fed to *any* food animal.

Sincerely,



Michael Hansen, Ph.D.
Research Associate



Jean Halloran
Director

cc: Bert Mitchell

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Consumer Policy Institute

Consumers Union

Consumers Union's Comments on Docket No. 96N-0135, Proposed Rule: Substances Prohibited for Use in Animal Food or Feed; Animal Proteins Prohibited in Ruminant Feed

February 14, 1997

Summary

Consumers Union¹ commends the U.S. Food & Drug Administration (FDA) for considering steps to prevent the possible occurrence and spread of transmissible spongiform encephalopathies (TSEs) in ruminants. However, FDA's current proposed action—prohibiting the use of ruminant or mink proteins in ruminant feed—is not sufficient to protect the public from the risk of TSEs entering the food supply. In fact, none of the six options being looked at by the FDA are, in Consumers Unions' opinion, sufficiently protective of public health.

We urge FDA to prohibit the use of rendered *mammalian* protein in feed of *any food animals*, as has been done in the United Kingdom (UK). We also urge FDA to conduct research on whether poultry protein should be included in the feeding ban.

All mammals are thought to be susceptible to TSEs. One type, Bovine Spongiform Encephalopathy (BSE) has been experimentally induced in pigs. Furthermore, TSEs are believed to be transmissible from food animals to humans, and are invariably fatal.

We therefore urge FDA to act immediately and take the most prudent course and to ban feeding of mammal protein, including swine, to all food animals. Furthermore, we urge FDA not to exclude gelatin and blood from coverage under the proposed rule.

Background

As the FDA notes in its proposed rule, published January 3, 1997 in the Federal Register, the cause of TSE has not yet been definitively established. However, the favored theory at present maintains that the agent responsible for TSEs, of which BSE in the United Kingdom is one example, is an abnormal form of

¹Consumers Union is a nonprofit membership organization chartered in 1936 under the laws of the State of New York to provide consumers with information, education and counsel about goods, services, health, and personal finances; and to initiate and cooperate with individual and group efforts to maintain and enhance the quality of life for consumers. Consumers Union's income is derived solely from the sale of *Consumer Reports*, its other publications and from noncommercial contributions, grants and fees. In addition to reports on Consumers Union's own product testing, *Consumer Reports*, with approximately 5 million paid circulation, regularly carries articles on health, product safety, marketplace economics and legislative, judicial and regulatory actions which affect consumer welfare. Consumers Union's publications carry no advertising and receive no commercial support.

a naturally occurring protein, called a prion. The normal prion protein occurs on the surface of cells, primarily nerve cells, but also on white blood cells (lymphocytes). Its normal function is unknown.

The gene that results in animals producing prion protein has been identified and the prion gene has been found in all mammals looked at so far, as well as in salmon and *Drosophila* (fruit flies) (Gibbs, 1997). TSEs have been found, or experimentally induced in, many mammals—including sheep, pigs, goats, cattle, deer, elk, mink, mice, hamsters, guinea pigs, domestic cats, puma, cheetah, eland, kudu, Arabian oryx, nyland, marmosets, macaques, chimpanzees and humans (Manuleidis et al., 1985; Dawson et al., 1991; Baker et al., 1993; Kimberlin, 1994; Lasmesis et al., 1996). In addition, a TSE has been reported in ostriches in a German zoo (Schoon et al., 1991).

TSEs are characterized by long latency periods, the fact that they are invariably fatal and the fact that the infectious agent is incredibly resistant to deactivation. Animals can contract the disease by eating infected animals or parts of infected animals. The portion of the animal that is most infectious is the brain and spinal chord, the portion that has the most prion protein (Kimberlin, 1994). Cooking the protein, or even rendering it at high temperatures, does not completely eliminate infectivity.

There are at least four known human prion diseases—kuru, Creutzfeldt-Jacob disease (CJD), Fatal Family Insomnia (FFI), and Gerstmann-Straussler-Scheinker syndrome (GSS). Some of these are inherited diseases, and appear to be related to a mutation in the human prion gene. However, some 85% of all CJD cases are spontaneous and occur at the stable rate of 1-2 per million throughout the world. Perhaps the best theory to explain the stable rate of spontaneous CJD is that these cases are caused by a spontaneous mutation in the prion gene² (DeArmond and Prusiner, 1995).

Since the cause of human and animal TSEs is similar, and since all mammals have prion genes, it is not unreasonable to expect that there is a spontaneous mutation rate that will lead to TSEs in all mammals that contain prion genes. If this is true, then we could expect that cattle, pigs, sheep, etc. will all spontaneously develop a TSE at some low rate.

This possibility raises grave concerns for our food supply, given our current and comparatively recent practice of feeding animal protein back to food animals on a massive scale. Since the TSE infectious agent is so resistant to deactivation, the agent could remain active in rendered animal protein and could infect animals of the same or other species that eat this rendered protein. Through animal feed one infected animal could infect hundreds of others. Work with scrapie and other TSEs has shown that there is a dosage response to this agent in terms of the incubation time for the disease. Low levels of the infective agent can infect an animal, but the

²Actually, this rate represents the combined probabilities of a somatic (i.e. not in the germ line) mutation in the prion gene which leads to the synthesis of the abnormal form of the prion which, in turn, causes the normal form of the prion to switch to the abnormal form at a rate fast enough to cause clinical disease before the patient dies.

time lag between infection and appearance of disease symptoms (the incubation period of the disease) could exceed the lifespan of the animal. But if infected animals are fed back to others, levels in the animals will slowly build up over generations (Kimberlin, 1996). When the level of infective material in the diet reaches a certain threshold, the incubation period will become shorter than the lifespan of the organism and the disease will finally manifest itself (i.e. symptoms will occur). Thus, by the time that the disease is noticed in animals, it will already be widespread.

The process of feeding rendered animal protein could result in the spread of a TSE, regardless of its origin. One theory about the origin of BSE in Britain is that it arose from feeding scrapie-infested meat and bone meal to cattle. Another theory is that a particularly virulent strain arose spontaneously in one cow that was subsequently rendered and fed back to other cows (Kimberlin, 1996). The process of feeding cattle rendered cattle protein gradually spread the strain until the outbreak appeared in Britain in the mid-1980s. This strain that appeared in Britain is particularly virulent, as it has been shown to fairly readily cross species barriers—the occurrence of a spongiform encephalopathy (SE) in over 70 domestic cats, and various exotic animals in zoos in the United Kingdom has been linked to animal feed.

There is also fairly strong suggestive evidence that the new strain of CJD (called new variant CJD) that recently appeared in the UK, and has taken 15 lives so far (Hornsby, 1997) can be linked to BSE. Just a couple of months ago, researchers in the UK demonstrated, using Western blot analysis, that the pattern produced by new variant CJD is highly distinct (or significantly different) from the other three forms of CJD in humans, yet very similar to the pattern seen in cows with BSE, mice infected with BSE, macaques infested with BSE, cats infected via BSE (Collinge et al., 1996).

Mink TSE Raises Concern

In the proposed rule, FDA outlined three possible sources whereby US cattle could become infected with BSE: i) importation of BSE-infected live animals or animal product, ii) transmission of a TSE from an infected species, and iii) spontaneous occurrence. We agree that the actions taken by USDA in 1989, to bar importation of live ruminants or ruminant products from all countries known to have BSE in native animals, effectively address the first concern (i). The current proposed rule claims to address (ii) and (iii). However, it does not do so adequately.

In the proposed rule, FDA argues that BSE has never been diagnosed in US cattle, and that they do "not have evidence to support the theory that BSE already exists, in an undiagnosed form, in this country." We think that indirect evidence, from research on a TSE in farm-raised mink, suggests that a spongiform encephalopathy could be present in a small number of cows here.

Transmissible mink encephalopathy (TME) is a rare disease. TME has only been seen in farm-raised mink and was first described in the scientific literature in

1966, in a paper that discussed outbreaks of this disease on 8 or 9 mink farms in Wisconsin over a 16 year period (Hartshough and Burger, 1966a,b). The disease was first recognized on a mink farm in 1947. In 1961, another outbreak of the disease appeared on 5 or 6 mink farms, from three adjoining counties. Since all the farms with affected mink used a ready-mix feed ration which came from the same feed plant, the scientists assumed that the feed was the source of the infectious agent (Hartshough and Burger, 1966a).

Two years later, in the summer of 1963, TME appeared again, almost simultaneously, on 2 more mink farms. Suspecting a possible feed-based source of contamination, based on the 1961 outbreak, the scientists went through the two farms' feed records. A striking finding was that from July through October 1962, meat "from beef carcasses unfit for human consumption (so-called "downer" cows)" that came from Farm A were fed to minks on both Farms A and B. As the scientists noted, "Since mink on both farms developed the disease almost simultaneously, we believe this feed component has to be incriminated" (Hartshough and Burger, 1966a: 389). Although the authors could not completely rule out the possibility of sheep parts being part of the suspect meat shipment, this was the first suggestion that "downer" cows may be connected to TME. ("Downer" cow is a term that refers to cattle that are killed because they no longer can stand up or have collapsed or died for any of a number of reasons. There are an estimated 100,000 downer cows in the U.S. each year.)

The next recorded outbreak of TME in the U.S. occurred 23 years later, in 1985, on a mink ranch in Stetsonville, Wisconsin. Dr. Richard Marsh and his colleagues (Marsh et al., 1991) studied this outbreak and produced a number of lines of evidence that linked "downer" cows to TME. First, the mink's diet consisted of 95% "downer" cow and 5% horse meat. The minks received no sheep meat so scrapie can be ruled out as the infectious agent. Dr. Marsh also performed a number of experiments that further supported the notion that cows were the source of the infectious agent. Dr. Marsh injected brains from Stetsonville mink with TME into the brains of 2 Holstein calves. Within 19 months, both calves had developed a fatal spongiform encephalopathy. Furthermore, when brains of these cattle were either injected into mink or simply fed to them, the mink developed TME. The authors concluded that, "These results suggest the presence of a previously unrecognized scrapie-like infection in cattle in the United States" (Marsh et al., 1991: 589).

Some scientists have tried to argue that TME is caused by feeding scrapie-infested sheep to the ranch mink. To test this possibility, Dr. Marsh has tried feeding brains from scrapie-infested sheep to mink. Since there are believed to be at least 20 different strains of scrapie, Dr. Marsh tried feeding brains from a number of different strains of scrapie-infested sheep. In none of these cases was he able to infect mink with TME (Marsh et al., 1991). Indeed, we know of no studies in the scientific literature which demonstrate that mink fed scrapie-infested sheep develop TME.

It is reassuring that outbreaks of TSEs on mink farms have been rare, and that none have been documented in the last ten years. However, one unsettling thing about Dr. Marsh's work on TSE is that the two calves infected with diseased mink

brain did not act aggressively or exhibit any of the other behavioral symptoms typical of "mad cow" disease; rather, they just became a little stiff and sluggish and ultimately collapsed, much as a "downer" cow would. Furthermore, histopathological examination of the calves brain did not turn up spongy holes in the places of the brain where "mad cow" disease usually attacks. In fact, the brain lesions were variable and did not resemble those seen in British cattle with "mad cow" disease. However, these cattle died of a spongiform encephalopathy (Marsh et al., 1991). These data suggest that TSEs could occur in cattle without being noticed or properly identified as such.

FDA Proposed Action Inadequate

Given this information on how TSEs work, FDA's proposal to ban only the use of ruminant (sheep, cattle, goats, deer, elk) and mink protein in ruminant feed, does not adequately protect human health and is not scientifically defensible. Since all food animals have prion genes, it can reasonably be assumed that mutations resulting in a TSE could occur in all food animals. Any such mutation would most likely remain undetected due to the fact that most food animals, except for dairy, are slaughtered before they reach an age where the disease would manifest itself.

In addition, experimental efforts to induce TSEs show that all mammals tested, including pigs, are capable of becoming infected with one of the TSEs. (By contrast, efforts to experimentally induce BSE in chickens have not resulted in infection).

FDA's proposed rule would permit feeding cattle and sheep rendered protein from pigs and poultry and vice versa. It would also permit pig and poultry to consume rendered pig and poultry protein. Thus the possibility will remain that these animals could conceivably get a TSE from eating infected animals. The largest risk would probably come from eating rendered protein from the same species, i.e. pigs eating rendered pig protein, as there is no species barrier; the refeeding process could just serve to silently amplify the infection until the levels are high enough for the TSE to become apparent. However, since cattle and sheep will still be allowed to eat rendered swine and poultry, and vice versa, there also remains a possibility that a TSE originating in cattle could infect pigs, and pig protein could reinfect cattle, and thus the entire food supply could be jeopardized.

Consumers Union therefore urges FDA to immediately adopt a ban on feeding rendered *mammal* protein to ALL food animals. The latter ban would mean that only poultry and fish protein could be used in animal feed. Given the current state of scientific knowledge, and the lack of some basic information about the infectious agent, this is the most prudent course.

We further urge FDA to conduct additional research on whether TSEs can occur in poultry, and whether any such TSEs can cross species barriers into mammals, pursuant to deciding whether poultry should be included in the ban.

We note that the UK government has already enacted a ban on feeding of any mammalian material to farm animals. The UK's Spongiform Encephalopathy Advisory Committee (SEAC) made the conservative proposals that *no* meat and bone meal from any mammals should be used as feed for farm animals or even used as fertilizer. As the SEAC statement on March 24, 1996 said, "The Committee confirmed its previous advice that the use of mammalian meat and bone meal should be prohibited in any circumstance where there might be a risk of the material being fed to or ingested by ruminants. In particular, it must not be incorporated into any feed for any farm animals, including fish or horses, or into fertilizer to be used on land to which ruminants have access" (SEAC, 1996: 2). Four days after the SEAC announcement, on March 28, legislation (BSE (Amendment) Order 1996) enacting this ban was passed.

The FDA currently assumes that feeding animal protein to pigs and chickens is free of risk because they have no evidence of a TSE occurring in those species. However, the fact that there has not been an outbreak of a TSE in pigs or poultry in the UK, or the US, is not a guarantee that these animals have not been infected. TSEs have a long latency period and both chickens and pigs are routinely slaughtered at a young age. For example, on large commercial chicken farms, the time between egg hatching and slaughter of the adult bird is 3 months. With hogs, most are sent to the slaughterhouse before they are one year old, usually when they are about 5 months old. Thus, even if pigs and chickens were infected via contaminated feed, they are slaughtered at too young an age to exhibit any symptoms.

Dr. Paul Brown, a renowned expert on CJD (and Medical Director of the U.S. Public Health Service) stated last year that "It must also be emphasized that the link to cattle products is itself only a presumption; how ironic, for example, if 11 million British cattle should be slaughtered in a preemptive strike to eliminate the risk of zoonotic Creutzfeldt-Jakob disease, only to belatedly find that the true villains were pigs or chickens which were also fed contaminated nutritional supplements but were brought to market at such a young age that the disease had not had time to become manifest" (Brown, 1996: ??).

Dr. Clarence Gibbs, Acting Chief of the Laboratory of Central Nervous Systems Studies at the National Institute of Health, made similar points at the recent Congressional hearing: "Pigs held for eight years after intercerebral inoculation with BSE develop a spongiform encephalopathy. . . meat and other tissue of the pig might harbor low-level infectivity at the time of slaughter at an early age. . . . We also do not know whether the agent replicates in chicken or other poultry. BSE-contaminated bone meal was fed to chickens. Poultry would be expected to shed massive quantities of the infectious amyloid in their feces. Chicken manure is widely used as fertilizer on vegetable crops. This means that vegetarians might be at risk. *It is best to admit our ignorance rather than to imply that we have information about this*" italics added (Gibbs, 1997).

Finally, the UK banned the feeding of ruminant protein to cattle in 1988, yet some 28,000 cows born after the ban went into effect have developed BSE (Stekel et al., 1996). One possibility is that non-ruminant protein (from pigs or chickens)

infected the cows. However, a more likely explanation is that since ruminant protein was still permitted in pig feed, there could have been cross contamination, or the use of pig feed on dairy farms. Either way, this step (i.e. banning use of ruminant protein in ruminant feed) clearly failed to stop BSE in the UK.

There is a strong possibility that a spongiform encephalopathy has occurred in US cattle, albeit at very low incidence, or may occur in food animals in the future due to a spontaneous mutation. There is also a strong possibility that FDA may find it very difficult to enforce a ruminant (plus mink)-to-ruminant ban. It currently has no staff allocated to this kind of enforcement effort. Unless a renderer or feed mill is supplying material for use in drugs, the FDA does not routinely inspect these facilities. A 1989 voluntary ban on rendering sheep for use in animal feed was widely ignored, as a 1992 FDA special survey indicated (62 FR 564). It is imperative that FDA learn from the mistakes in the UK, which show that a ruminant-to-ruminant ban does not stop the spread of TSE disease.

Part of the FDA's rationale for why we are safe in the US is that USDA has an extensive monitoring procedure and has looked at over 5,342 brains of cattle as of January 23, 1997 and has not detected BSE (Detwiler, 1997). This surveillance, while a positive step, is not sufficient to protect public health. First, the vets were trained with slides and materials from British cattle with BSE, so that they are looking for the characteristics of the British strain of BSE. Both Dr. Marsh's work on TME along with the work of the USDA and ARS on transmission of US scrapie to cattle suggest that a US strain of a TSE in cattle probably would exhibit different characteristics than British BSE. Thus, the U.S. vets may not be looking for the right brain characteristics. Second, the majority of brains were only examined visually for visible signs of an TSE. Only a few hundred brains have been tested using immunohistochemistry for presence of abnormal prion protein. Third, only a couple hundred "downer" cow brains have been looked at so far (Detwiler, 1997).

Fourth, animals may have the disease and even can be infectious for quite a while before any visible symptoms or tests could detect the disease. Dr. Clarence Gibbs made a similar point at the recent Congressional hearings: "The assay for PrP [prions] is not sufficiently sensitive to detect it before infectious titers—that is, levels in the brain—reach many thousands of infectious doses per gram. In the mid-1960s, we demonstrated with our French and English collaborators that during the early incubation of the TSEs, when the virus titers in the brain was very low, there were already marked functional changes, even though no pathology was yet detectable . . . Thus, it is clear that early replication to only low level infectivity titer, *far below that necessary to detect PrP biochemically or immunologically, can already lead to disease*" italics added (Gibbs, 1997: 4). Thus, the USDA's surveillance systems cannot be relied on to pick up infected animals.

In addition, Dr. Marsh's work with TME suggests that, if a TSE is present in US cattle, it could cause behavioral symptoms different from those in the UK, symptoms that are similar to "downer cow" syndrome. Since there are some 100,000 "downer cows" in the US, a bovine TSE in the US could be difficult to distinguish from other causes of "downer cows" and thus even more difficult to detect (Marsh, 1992). (The UK had a relatively easy time of initially detecting BSE as the behavior

of the cows was so different than that caused by any other known bovine disease in the UK.)

The potential difficulty of detecting a TSE in cattle in the US only strengthens the need for the FDA to take at least the immediate preventive step of banning all use of mammalian protein in food animal feeds.

Gelatin and Blood Meal

Consumers Union opposes the exemption of gelatin and blood meal from the FDA's proposed ban since some scientific evidence suggests that these materials may be infective. Blood from CJD victims has been shown to induce TSE in animals (Manuelidis et al., 1985); this is not surprising as the prion protein is found on the surface of white blood cells (the "buffy coat" fraction). Furthermore, SEAC has recommended that blood meal not be used as either a feed ingredient or a fertilizer (SEAC, 1996).

As for gelatin, there is no definitive evidence that the infectious agent is not present. While the European Commission did lift an embargo on gelatin from the UK in June, 1996, the decision was quite controversial. As pointed out in a recent draft report on BSE prepared for the European Parliament, "Between 11 and 15 April 1996, the Scientific Committee on Cosmetology, the Scientific Committee for Food and the European Medicine Evaluation Agency all expressed their opposition to ending the embargo on gelatin" (Ortega, 1996). Thus, the bulk of the scientific reports looked at by the EC recommended against lifting the gelatin embargo as definitive evidence that the infectious agent is not present in gelatin was not available.

Finally, we note that current regulations ban importation of ruminant gelatin and blood meal from countries with documented BSE. If the FDA believes foreign gelatin and blood meal can carry the BSE infective agent, then it should assume the same is true of US gelatin and blood meal.

Milk

Consumers Union does not oppose the exclusion of milk from the feeding ban. Even though colostrum from a woman with CJD was found to be infective in laboratory animals, her child did not develop CJD (Greger, 1994). Milk is not known to be infective in other animals with TSEs. Scrapie is not considered to be readily transmissible by milk (Detwiler, 1992). With the cases of TME, even though thousands of adult mink died of TSE, none of the offspring that were suckling did (Hartsbough and Burger, 1966b).

Solid Waste Issues and the Continued Use of Animal Protein

Finally, as for what to do with rendered animal protein, Consumers Union would support continued use of rendered protein in pet food, coupled with clear labeling of the animal source of the protein and with public education efforts by FDA to inform pet owners of the possibility of risks.

We realize that the action we recommend—banning all use of rendered mammal protein in feeds—would create a new solid waste problem. FDA evaluated the impact of a mammal to ruminant ban and found "no significant impact." Since only about 10% of all rendered animal protein is fed to ruminants, while some 20% is fed to pigs, we realize that our proposed mammal to food animal ban would have significantly greater impact. We feel that the rendering industry has served a very useful purpose in the past, given that over half of the weight of food animals is considered a waste product. So that the rendering industry can continue to function we recommend that FDA encourage research on different uses to which the rendered animal products could be put. We recommend that FDA look into the situation in the UK, where much research is being done along these lines. The UK is strongly considering using the waste as fuel to generate electricity, and, the tallow and fat from rendered cattle is being used as a fuel to run some of the rendering facilities. (William Hueston, pers. comm.)

Conclusions

We urge FDA not to take half measures. The UK failed to act early and decisively out of fear of economic impact. The UK now faces estimates of a minimum of 100 human deaths due to eating infected meat, and plausibly as many as 80,000 deaths (Cousens and Smith, 1997). Some have put the maximum possible death toll higher, approaching over 50% of the population (Lacey, 1996). Given the long latency period of the disease, if there ever were a TSE problem in the U.S., by the time we had good scientific evidence (as the UK now has) the problem would be impossible to fix. We would face a public health disaster about which we would be able to do nothing. We urge FDA to act decisively now to prevent any possibility of a UK scenario here. We agree with the FDA's stated goal of developing regulations that will erect a firewall to prevent TSEs in food animals. Given that data show that all mammals including pigs are susceptible to TSEs and that TSEs are notoriously resistant to deactivation, this means that FDA should ban the use of all mammalian protein in food animals' feed, as in the UK.

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News  from Simon & Schuster

1230 Avenue of the Americas, New York, NY 10020
Contact: Kerri Kennedy, Publicist 212/698-7537

Deadly Feasts

Tracking the Secrets of a Terrifying New Plague

"Explosive...[an] important, gripping book. . . . In cinematic fashion, Rhodes creates a complex, colorful and sometimes gory medical documentary."

—*Publishers Weekly* (starred review)

"Gripping. . . A powerful and alarming book. . . Rhodes weaves careful research and powerful stories into a chilling narrative that often reads more like science fiction."

—*Kirkus Reviews* (starred review)

Deadly Feasts: Tracking the Secrets of a Terrifying New Plague (Simon & Schuster; March 20, 1997; \$24.00) by Richard Rhodes, Pulitzer Prize-winning author of *The Making of the Atomic Bomb* and *Dark Sun: The Making of the Hydrogen Bomb*, tracks the threatening emergence of a new group of bizarre, deadly brain diseases known as transmissible spongiform encephalopathies (TSEs) that kill 100 percent of the animals and humans they infect. The best-known is mad cow disease (bovine spongiform encephalopathy, BSE), which has infected hundreds of thousands of cattle in Great Britain and Europe and has now begun killing humans who have eaten infected beef.

The U.S. Food and Drug Administration announced in early January 1997 that it would soon ban the feeding of processed ruminant animal wastes to cattle — believed to be the cause of the British contagion — tacitly acknowledging what TSE experts have warned for years: that a form of BSE infects US cattle and could spread to infect Americans who eat beef. The US rendering industry estimates that incinerating or burying the millions of tons of perishable animal wastes which it formerly processed and sold for recycling into food animals will cost it \$1.6 billion a year.

Besides its immediate focus on this developing national and international story, *Deadly Feasts* narrates for the first time in a book for general readers the remarkable history of the discovery of the TSEs, of other, little-known outbreaks and of the pathbreaking scientific research that led to present understanding of the TSEs' unusual biology. In brief:

Richard Rhodes

• In animals and humans, the TSEs kill by damaging the brain. Victims lose control of their muscles, stumble, stagger, lose the ability to walk, have seizures, go mute, become demented, lose the ability to swallow and die of pneumonia or starvation, usually within a year. On autopsy, their brains turn up riddled with microscopic sponge-like holes (hence "spongiform encephalopathy"). While the diseases incubate, sometimes for years, the victims show no symptoms. There is no way to trace TSE infection prior to the appearance of symptoms, the agent is almost unkillable, there is no treatment and no cure.

• The first human TSE epidemic, of a disease called kuru, occurred among a tribe of cannibals, the Fore (for-ae) people of the Papua New Guinea highlands. An American physician, Dr. D. Carleton Gajdusek (guy-do-sheck) studied the kuru epidemic extensively beginning in 1957 and won a Nobel Prize in Medicine for proving that the disease was transmissible (it had been spread by the Fore practice of eating their deceased relatives). Five or six Fore still die of kuru annually, having incubated the disease for more than forty years since cannibalism was banned. Richard Rhodes traveled to Papua New Guinea in the course of researching *Deadly Feasts* and located and interviewed one of these rare kuru patients, a 45-year-old grandmother.

• Gajdusek and his team at the US National Institutes of Health subsequently demonstrated that TSE agents were hard to kill and that a little-known but widespread lethal human brain disease, Creutzfeldt-Jakob disease (CJD), was a TSE. (The best-known US victim was the choreographer George Balanchine, who died of CJD in 1983.) In the mid-1970s, they warned that CJD might be spread through surgery and dentistry.

• A patient who received a corneal transplant at Columbia Physicians and Surgeons Hospital in New York City died of CJD contamination in 1973. Two Swiss patients infected through brain surgery died in 1976. These cases affected surgical practices throughout the world, much as AIDS has done.

• The worst outbreak of TSE prior to the British epidemic occurred beginning in 1985 among young people who had been treated as children with human growth hormone extracted from donor pituitary glands, a few of which proved to have been infected with CJD. Further infection was limited by banning natural human growth hormone, which was replaced by a genetically-engineered synthetic. But nearly 100 young people have died so far in the US, Great Britain, France, New Zealand and elsewhere from human growth hormone contamination. According to Gajdusek, "It was only luck that saved thousands of people when we contaminated...human growth hormone...and injected [it] into our children in an instance of high-tech necrocannibalism.... Only fortuitously was it not more like 200,000...deaths."

• The British epidemic began among dairy cattle in 1985. By 1996, 170,000 cattle had died of a new form of TSE known as bovine spongiform encephalopathy (BSE) — mad cow disease. British authorities identified the practice of recycling animal rendering wastes into protein supplement fed to cattle as the source of the infection. Bent on saving their cattle industry, they assumed without evidence that the disease being transmitted through contaminated meat-and-bone meal was an old natural TSE of sheep called scrapie which is widespread in Great Britain. Since humans have eaten scrapie-infected lamb and mutton for hundreds of years, the sheep disease is evidently not transmissible to humans. In 1989, the British government banned the cannibalistic feeding of cattle and sheep rendering wastes to cattle and sheep — the same kind of ban the FDA is promulgating in the US.

• By then, however, BSE was widespread among British cattle. If animal protein was the material transmitting BSE, then the cattle would be protected from further infection. But the ban did not protect British citizens, who were still eating animal protein and fat in the form of beef. Early in 1996, British scientists indicted BSE — eating beef, that is — as the cause of 10 deaths among young people from a new

form of CJD. As of early 1997, the number of such confirmed deaths in Britain and France has increased to 17, and confirmation of further deaths is expected. How many human deaths will ultimately result is anyone's guess, but in January 1997, British government scientific authorities projected a range from "a few hundred cases or less" to "many thousands."

- Not all TSE researchers agree that animal protein is the carrier of BSE infection. Gajdusek, among others, suspects animal fat — tallow — a much more serious problem because disease agents are undetectable in tallow and tallow is an ingredient in widespread use in human food, cosmetics and pharmaceuticals. There is no known test for TSE agents in tallow.

- As early as 1947, outbreaks of a virulent TSE among mink fed meat from diseased cattle on mink ranches in Wisconsin led some researchers to suspect the existence of a native TSE of cattle in the US. Further outbreaks occurred sporadically down through the decades in Wisconsin and elsewhere, always associated with feeding mink diseased cattle, a common practice. Based on these outbreaks, mink TSE researchers estimate that one in a million US cattle may die of a native cattle TSE (more than enough to cause widespread contamination when the disease agent is recycled from cattle through cattle). Although the FDA's public focus has been the British outbreak, it is this danger that the FDA ban on recycling cattle and sheep remains really addresses.

- The US meat industry produces millions of tons of animal wastes annually. Incinerating or burying these wastes turns a profitable byproduct into a serious disposal problem and a major expense. The British ban on recycled wastes has been marked by widespread cheating. If US government meat inspection is incapable of preventing contamination of meat and eggs with salmonella and *E. coli*, both of which can be easily identified under a microscope (unlike the TSEs), why should Americans trust their government to police animal waste recycling when nearly \$2 billion dollars in lost profits and new expenses are at stake?

- Beyond direct contamination of the human food supply, another potential area of human risk explored in *Deadly Feasts* is the new biotechnology of xenotransplantation: transplanting organs and tissues from specially-bred transgenic animals into humans. Pig-heart transplantation is projected to begin before the end of 1997, to meet a demand that extends to some 50,000 heart patients annually within the US alone. Xenotransplantation industry pioneers expect to transplant pig lungs, kidneys and other organs as well if heart transplantation proves successful — including, significantly, brain tissue. Pigs have been shown to be susceptible to TSE infection. The TSEs raise major issues of risk whenever the species barrier is breached.

- The TSE agent has not been finally characterized after more than forty years of intensive research. *Deadly Feasts* describes the course of that research, a fascinating scientific detective story. No DNA other than the victim's has yet been found associated with a TSE; Gajdusek and other leading researchers believe the TSEs are not viruses at all but rather a new kind of disease agent, an infectious protein (sometimes called a prion) that causes disease by serving as a template that misshapes a normal protein which the victim's brain manufactures. The misshapen form then proliferates, blocking normal brain function and causing lethal damage. TSE infection incubates stealthily and without symptoms for years, one reason the agent is so dangerous.

No book yet published for general audiences in Great Britain, Europe or the United States reports in such depth on these issues. *Deadly Feasts* will prompt national debate on issues of vital importance to millions of Americans.

About the author: Richard Rhodes is the author of 15 books including *The Making of the Atomic Bomb*, which won the 1988 Pulitzer Prize in Nonfiction and the National Book Award. His *Dark Sun: The Making of the Hydrogen Bomb* was one of the three finalists for the 1996 Pulitzer Prize in History. He lives in rural Connecticut.

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By Richard Rhodes

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OBSERVATION OF CNS SIGNS

IN HOGS AT EST. 893

TOBIN PACKING CO., INC.

BY: Dr. M. Doi - SVMO - IIC - Est. 893

Dr. K. Langheinrich, Pathologist-in-Charge, Eastern Laboratory

Dr. F. Rellosa, Staff Assistant - NERO

(Presented by Dr. Langheinrich at the MPI National Pathology
Meeting in Seattle, Washington on July 20, 1979)

This film was developed by Dr. Doi at Est. 893. It showed two (2) cases of hogs exhibiting TIC - a spasmodic muscular contraction, usually in the face or extremities, which is a typical CNS symptom. A longer 8MM version of this film show 13 hogs with CNS symptoms, 7 of which were confirmed by the laboratory to have CNS lesions histologically.

Since January, a number of hogs in this establishment have been found, in antemortem, to show what appears to be CNS. Sets of tissue samples were sent to the laboratory for examination. Various tests were done which includes histological study (E-H stain), fluorescence antibody technique, virus neutralization and viral and bacteriological isolation. Differential diagnosis was also done to exclude vitamin B deficiency, post vaccination reaction, chlorinated hydrocarbon, arthritis, and transport stress.

Of the more than 50 cases observed, almost all showed CNS lesions which range from congestion and focal hemorrhages to generalized meningo-encephalitis.

The Veterinary Services has been alerted on this. The North Central Region will do their own sampling to verify the observation in the plants and the findings of the laboratory.

2739
11-5-79
Rush

UNITED STATES DEPARTMENT OF AGRICULTURE
FOOD SAFETY AND QUALITY SERVICE
MEAT AND POULTRY INSPECTION PROGRAM

October 29, 1979
Case #316071

Pathology Group
USDA, FSQ3, STS, Eastern Lab.
Russell Research Center
Athens, Ga. 30604

Gentlemen:

On Oct. 19, 1979, a barrow, weighing approximately 200 pounds, was retained on AM inspection because of suspected CNS disease. The animal was slaughtered on Oct. 23. The carcass is being retained pending laboratory findings. (ret. tag # MPD-272321)

The clinical signs were characterized by excitable temperament and repeated lifting of right pelvic limb in standing position and on ambulation (tic). Rectal temperatures were 103.5°F, 102.8°F and 101.6°F during four days of retention.

PM examination showed a greatly enlarged cystic rightiliac lymphnode containing numerous small abscesses.

Specimens

10% formalin - #1 bottle - lung, liver, spleen, kidney, heart, lymphnodes
 * #2 bottle - brain and spinal cord.

* frozen specimen - collected aseptically in sterile plastic bags.
 - brain and iliac lymphnode

Slides - 2 blood smears

* Serum - 1 test tube

Sincerely,

M. Doi

MD/ndm

M. Doi, STMC

* being kept at Est. freezer pending lab request

LAB ACC. NO. & DATE REC'D

USDA FSQS

SERIAL NO.

LABORATORY REPORT

(Read instructions on reverse before completing)

316074

1. PROJECT NAME OR CASE NO.

CASE NO.

2. RELATED SERIAL NOS. Use item 15 if needed

3. RETAIN TAG NO.

11-5-79

316074

316075

19PD-272321

4. REASON FOR SAMPLING

☒ 1 INSPECTOR INITIATED☐ 2 PROGRAM INITIATED

5. NAME AND ADDRESS OF PRODUCER, HERD OR FLOCK OWNER

LOUISIANA, IND.

6. STATE CIR-
CUIT CODE

7. ESTABLISHMENT NO.

ZIP CODE

573613

8. PRODUCT OR SPECIES

SWINE

CODE

570

9. APPROX. AGE

MOS.

YRS.

6

10. SEX

1 M

2 F

3 N

☒☐☐

11. DATES

A. COLLECTED

MO.

DAY

YR.

10

2

5

7

9

10

2

9

7

9

7

9

B. MAILED

MO.

DAY

YR.

10

2

9

7

9

7

12. IMPORT PRODUCT

A. COUNTRY NAME

CODE

B. FOREIGN EST. NO.

C. CUSTOMS ENTRY NO.

D. MP FORM 410 NO.

13. REQUEST FOR:

☒ PATHOLOGY☐ DIAGNOSTIC
MICROBIOLOGY☐ FOOD MICROBIOLOGY

RESIDUE (Enter residue code & request if known -

See reverse)

CODE

☐ FOOD POISONING☐ SPECIES IDENTIFICATION

14. SPECIMEN

☐ 01 FAT☐ 02 LIVER☐ 03 MUSCLE☐ 06 OTHER (Specify)☐ 04 KIDNEY☐ 05 INJECT SITE

15. RELATED INFORMATION (Including Imports, Ante-Mortem and Post-Mortem remarks)

See attached sheet

16. NAME OF INSPECTOR (Type or print)

MATZUER, NIO

PHONE NO. (Include Area Code)

511845441390

17. RESIDUE ONLY - IN COMPLIANCE

18. FOR LABORATORY USE ONLY

Microscopic examination of the barrow tissues revealed a encephalopathy and diffuse gliosis characterized by vacuolated neurons, loss of neurons and gliosis in a confined region (nucleus) of the brain stem (anterior ventral midbrain). Only an empty sometimes divided vacuole was present instead of the normal morphology of a nerve cell. Occasionally a shriveled neuron was seen. According to Jubb and Kennedy in the text Pathology of Domestic Animals, vol. 2, Second edition, p. 413, "The degeneration of neurons, the reactivity of the glia ... are the classical hallmarks of viral infection of the central nervous system and imply a sequence of viral cytopathogenicity and reaction to cellular degeneration." Scrapie of sheep, and encephalopathy of mink, according to the literature, all produce focal vacuolation of the neurons similar to the kind as described for this pig. I was unable to locate any lead as to the cause of this interesting phenomenon in other species including swine. Examination of the lymph node identified by you as the right iliac revealed multiple cystic partially hemorrhagic abscesses. Special stains failed to reveal any etiologic agent. The liver was congested. The WBC of one of the blood smears (the other was of poor quality) revealed a neutrophilia with 72 neutrophils, 4 bands, 20 lymphocytes and 4 monocytes.

Notified Insp. Matzuer @ 1530, 11/8/79

19. DIAGNOSIS 1. Encephalopathy and diffuse gliosis of undetermined etiology; 2. Abscesses in lymph node; 3. Hepatic congestion; 4. Neutrophilia

20. LABORATORY
CODE

113013

21. CONDITION ON RECEIPT

Satisfactory

22. ANALYSIS DATES

A. STARTED (MO., DAY, YR.)

B. COMPLETED (MO., DAY, YR.)

11/9/79

11/6/79

23. NO. T

24. NO. S

25. SIGNATURE OF ANALYST

INITIALS

26. REVIEWED BY

113013

KALANGHEINRICH, DVM, MS, Diplomate ACVP, Pathologist-in-Charge

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United States
Department of
Agriculture

Agricultural
Research
Service

Midwest Area
National Animal
Disease Center

P.O. Box 70
Ames, Iowa
50010

September 6, 1996

Dr. Patrick McCaskey
USDA - FSIS
Russell Research Center
950 College Station Rd.
Athens, GA 30605

Dear Dr. McCaskey:

Enclosed are the 4 pig slides I stained for prion protein, the H&E slide I didn't stain, and a positive control slide from a scrapie sheep.

In the pig sections you will see a small particulate type of staining that is confined to neurons and as I indicated on the phone, I would interpret that as normal PrP. It is a marked contrast to the massive amount of extraneuronal staining seen in the scrapie section. There is quite a bit of artifactual staining in the largest pig section, which looks like it is due to a sectioning problem. All in all, I thought the slides came through the process in pretty good shape.

Sincerely,

Janice M. Miller, D.V.M., Ph.D.
Veterinary Medical Officer

cc: Dr. Al Jenny

SEP 11 1996

13 November 1996

TO: Patrick c. McCaskey
U.S.D.A./F.S.I.S./Eastern Lab
950 College Station Bld., Suite 205
Athens, GA 30605

FROM: W.J. Hadlow
908 So. Third St.
Hamilton, MT 59840

Herewith are my findings in the microscopic section of pig brain (B2709 Path-
BI SCI-ESQS-USDA) you sent for my opinion.

I concluded that the faintly stained specimen is a sagittal section through the caudal medulla oblongata and upper spinal cord. The black dot on the slide partly covers what I have identified as the area postrema. Ventral to it is the parasympathetic (dorsal motor) nucleus of the vagus nerve. Subjacent to it is the hypoglossal nucleus.

About twelve (12) neurons in the parasympathetic nucleus have unilocular optically empty vacuoles in the perikaryon. This is the site where such vacuolated neurons have been seen in swine (as well as in cats and sheep) as an incidental finding. So I do not think such cells have any significance in this pig. All other neurons are intact, and spongiform change in the neuropil is not apparent.

I am impressed, though, with what seems to be an increase in the number of astrocytes in the section. Some astrocytes are in clusters, some are enlarged and vesicular. Where they are most numerous, a few rod cells (activated microglia) are seen. These findings suggest some perturbation of the nervous tissue. Although such a glial response occurs in the transmissible spongiform encephalopathies, I do not know its significance in this case without examining other parts of the brain for changes characteristic of these diseases.

Thus, from looking at this one (1) section of brain, I cannot conclude that the pig was affected with a scrapie-like spongiform encephalopathy.

W.J. Hadlow
W.J. Hadlow, D.V.M.

NOV 15 1996