For the past several years, the U.S. Food and Drug Administration (FDA) has been considering ways to prevent an epidemic of "mad cow disease" in the U.S. In Britain, where the disease has killed 170,000 cows and at least 24 people since 1985, the beef industry has been crippled and confidence in government has plummeted because no one took adequate measures to control the disease. Additional human deaths are now expected in Britain because millions of people ate contaminated beef for a decade before authorities acknowledged that mad cow disease could endanger public health. (See REHW #606, #607.) Could such a thing happen in the U.S.?

When animals are slaughtered for human food, at least half of the carcasses hide, hooves, entrails, and so forth cannot be sold for human food and must be sent to a "rendering" plant where it is ground up, boiled down, dried into the consistency of brown sugar and sold as feed for cows, pigs, chickens, and pets. Cows --vegetarians by nature -- can become infected by mad cow disease when they are forced to eat parts of other infected animals.

June 5, 1997. FDA issued a rule making it illegal for rendered animal parts from ruminants or mink to be fed to ruminants. Ruminants are animals that chew their cud --cattle, sheep, goats, deer and elk, among others. Mink are included in the FDA's ban because they can get a disease similar to mad cow disease.

A small group of scientists, led by Michael K. Hansen of Consumers Union, argues that the FDA ban does not go far enough to protect public health, and that FDA's rule is "not scientifically defensible."[1] Hansen wants a ban on all animal feed containing anything derived from rendered mammals. Consumers Union publishes CONSUMER REPORTS magazine.

FDA says its ban is adequate because no cows in the U.S. have ever been confirmed with mad cow disease, nor is there evidence that any humans in the U.S. have been affected. However, last week we reviewed indirect evidence indicating that some cows in the U.S. may already have mad cow disease and that some people in the U.S. may already have a human version of the disease. In Britain, mad cow disease is thought to have infected some people with a variant of an age-old, but very rare, disease called Creutzfeld-Jakob disease, of CJD for short. In this country, there is some evidence that CJD may not be as rare as was once thought because some cases of CJD may have been misdiagnosed as Alzheimer's disease. (See REHW #607.)

Mad cow disease is one of a family of diseases called transmissible spongiform encephalopathies, or TSEs for short. In sheep, the disease is called scrapie; in deer and elk it is called chronic wasting syndrome. In cows, it is called BSE [bovine spongiform encephalopathy] and in mink it is TME [transmissible mink encephalopathy].

TSE diseases all have similar characteristics: they attack the central nervous system, causing disintegration of the brain; they have a long incubation period --months or years (even decades) can pass between the initial infection and the time when symptoms appear; TSEs are invariably fatal; and they are transmitted by eating animals or animal parts, especially brains and spinal cords.

TSEs are now thought to be caused by a protein called a prion (pronounced PREE-on). Prions are normal proteins, present in all mammals and some non-mammalian species such as salmon and ostriches. According to the prion theory of disease, some prions can fold abnormally and then they can kill nerve cells. Furthermore, according to the theory, abnormal prions can cause normal prions to fold abnormally, thus causing a chain reaction leading eventually to disease and death.[2]

Prions are remarkably hardy. They are not destroyed by the digestive system of humans or other animals. And they are very heat resistant. A scientific committee of the European Union says that heating prions to 271 degrees Fahrenheit (133 Celsius) under three atmospheres of pressure will deactivate most, but not necessarily all, of them. Prions also resist destruction by ultraviolet light and by radiation, and they are not destroyed by prolonged immersion in formalin, a potent disinfectant made from formaldehyde and alcohol.[3] Prions are hard to stop.

Under FDA's rule, ruminants can be fed to pigs and pigs can be fed to ruminants. Under the rule, even ruminants that are known to be infected with a TSE can be fed to pigs. FDA allows this because, the agency says, no "naturally occurring" TSE has ever been confirmed in pigs. However, Dr. Hansen notes that British researchers have managed to infect pigs with a TSE by exposing them to high doses of contaminated brains from cattle.[4] This does not answer the question whether pigs can be infected through their normal diet, but it indisputably establishes that pigs, like many other species, are susceptible to TSEs.

Hansen offers evidence that some pigs in the U.S. may be infected with a TSE.[5] In 1979, Dr. Masuo Doi, a U.S. Department of Agriculture (USDA) hog inspector, began noticing pigs with central nervous system (CNS) disorders arriving at a swine slaughterhouse, the Tobin Packing Plant, in Albany, New York. Because there was no single source of the animals, and because the Tobin plant did not routinely deal in diseased animals, Dr. Doi suspected that the symptoms he was observing might be present in pigs nationwide.[6] During a 16-month period, Dr. Doi observed CNS symptoms in 106 pigs, taking careful notes and retaining tissue samples, including brains. Researchers examined the brains of the 106 pigs and found telltale "spongiform damage" --holes in the brain tissue --in only one of the 106. They did find other brain damage that occurs in TSE diseases --so-called "glial changes" in brain cells --in 40% of the animals. Dr. Doi, and Dr. Langeheinreich, the pathologist who examined the brain tissues, both say they believe they were dealing with a single disease in all the pigs. Dr. Clarence Gibbs, the leading expert on TSEs at the National Institute of Health, has said he believes all the pigs had the same disease, based on behavioral abnormalities evident in motion pictures taken while the pigs were alive.

There are 83 million pigs slaughtered in the U.S. each year.[6] They are killed at an average age of only 5 months --long before symptoms of a TSE would ordinarily become apparent.[1] Therefore, if pigs were infected with a TSE, they still might end up in food products for humans and in animal feed.

Even if a pig had the behavioral symptoms of a TSE disease, it might not be noticed by USDA inspectors. To see the symptoms of such disorders, one must observe an animal in motion. The way they walk, turn corners, and hold their tails and heads can all be important clues to their condition. Most pigs are so jammed into pens with other pigs that they have no room to move. If the animals are not in motion, symptoms of TSEs (or other CNS disorders) can go unnoticed. At present, USDA observes only 5% to 10% of pigs while they are in motion.[6] Thus USDA's inspection program seems inadequate to detect symptoms of TSE diseases in pigs. And, as we have noted, even if a pig were identified with a TSE, FDA's rule would allow its infected carcass to be fed to all non-ruminant animals, including pets, chickens, fish, and pigs.

Do humans who eat pork and other pig products have high rates of CJD, the human TSE associated with mad cow disease in Britain? There have been two epidemiological studies on this point.[7,8] Both were suggestive, though not definitive. The first study, in 1973, examined 38 patients with Creutzfeld-Jakob disease. The control group consisted of the nearest relatives of the CJD patients, often their spouses. These controls then selected a friend of the patient of the same age and sex to act as a second control.

The study revealed that this group of people had an unusual diet. More than one-third of the CJD patients ate brains "and the great
majority of patients had a specific preference for hog brains," the authors wrote. [7] One-third of the control group also ate brains, but not necessarily hog brains. Obviously the control group, composed of close relatives and close friends, shared dietary habits with the patients, reducing the power of the study to discern differences between the two groups. [7]

The second study, in 1985, compared 26 patients with Creutzfeld-Jakob disease with 18 of its family members and 22 other people selected from a hospital population. [8] Compared to the control group, the CJD patients had an unusually high consumption of roast pork, ham, hot dogs, pork chops, smoked pork, and scrapple. Scrapple is made by adding cornmeal to the liquid derived by boiling pig bones and meat (usually from the head, feet and internal organs). Compared to controls, CJD patients also had an excess consumption of roast lamb, rare meats [meaning not thoroughly cooked], and raw oysters and clams.

Could TSE-infected meat enter human food chain from other sources besides pigs? The state of Colorado requires deer hunters to turn in the heads of any deer they kill. In 1996, 6% of the deer in northeastern Colorado were found to have a TSE. In 1997, 4% of the deer there had a TSE. [9] Diseased deer in Colorado are usually incinerated or buried in a landfill (where the prions remain infective for an unknown period). However, if any diseased roadkill deer were sent to a rendering plant, they could become animal feed for pigs and chickens.

It is not known at this time whether chickens can become infected by TSE diseases. However, even if it turns out that chickens cannot get a TSE disease themselves, they still might carry such a disease if it were in their feed. As we have noted, the FDA rule allows chickens to be fed rendered animal protein even if it is known to be infected with TSE diseases. Dr. Clarence Gibbs, Acting Chief of the Laboratory of Central Nervous System Studies at the National Institutes of Health, testified before Congress January 29, 1997, saying that bone meal derived from infected rendered animals has been fed to chickens. "Poultry would be expected to shed massive quantities of the infectious amyloid [prion protein] in their feces. Chicken manure is widely used as fertilizer on vegetable crops. This means that vegetarians might be at risk," Dr. Gibbs testified. [1]

[To be continued, but not next week.]

--Peter Montague (National Writers Union, UAW Local 1981/AFL-CIO)

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