

# Is U.S. Beef Safe for Consumers to Eat?

Lynn Knipe, Ph.D.
Extension Specialist, Processed Meats
The Ohio State University

William Shulaw, D.V.M., M.S. Extension Veterinarian, Cattle and Sheep Ohio State University Extension

In May of 2003, the Canadian government announced that an Alberta Cow had been diagnosed with bovine spongiform encephalopathy (BSE) or in layman's terms, Mad Cow Disease. This is the first case of BSE found in an animal of North American origin. Since then, there have been many questions about how safe our beef products are in the United States. This fact sheet was developed to help you answer your customer, employee, and local media questions, regarding BSE.

#### **TSEs**

There are a number of diseases that belong to the group called transmissible spongiform encephalopathies (TSEs). They are found in humans and animals and include BSE; chronic wasting disease (CWD), found in deer and elk in North America; scrapie, found in sheep and goats in most countries of the world; feline spongiform encephalopathy (FSE), found in cats in the UK; and the human diseases: Creutzfeldt-Jacob Disease (CJD), Gerstmann-Straussler-Scheinker Syndrome, kuru, and fatal familial insomnia (FFI).

TSEs cause death of nerve cells resulting in a deterioration of the brain. Microscopically the loss of these cells gives the appearance of holes in the brain tissue, resembling a sponge—giving rise to the term "spongiform" encephalopathy. Muscle dysfunction results, causing cattle to become uncoordinated, or stagger, when attempting to walk. Some cattle will become belligerent or aggressive—hence the name "Mad Cow Disease." The incubation period (time from original infection to showing symptoms of the disease) for BSE is typically 2 to 4 years. There is no treatment for these diseases, and they all progress to death within a few weeks to months.

BSE, like all TSE diseases, is believed to be caused by the conversion of a normal cellular protein called the "prion protein" to an abnormal form. It is believed that this abnormal form causes the death of brain cells. Animals and people have prions naturally existing in their bodies, but TSEs develop when they become infected with the abnormal form which then corrupts the

normal prion proteins. Although the origin of BSE in the UK is unknown, one theory is that cattle were infected by the scrapie agent, after consuming meat and bone meal from scrapie-infected sheep. Another theory is that a spontaneous mutation of the prion protein gene in a cow created the first case of the disease. Other theories have been proposed, and it is likely that we will never know the cause for sure. However, scientists believe that BSE was spread by feed sources containing BSE-contaminated meat and bone meal.

#### CJD

How to better name the TSEs to eliminate some of the confusion is currently being debated. Historically, scientists described four types of Creutzfeldt-Jacob Disease found in humans—iatrogenic CJD, familial CJD, sporadic CJD, and variant CJD (vCJD). All are potentially infectious. Familial CJD occurs in some families and is believed to be due to genetic mutations that favor the development of the disease. Sporadic CJD seems to just appear, at the rate of about 1 case per million of population, but the exact cause is not completely known. Iatrogenic CJD occurs when contaminated surgical instruments (usually brain or eye surgery), or organ transplants, introduce the disease from a CJD victim to a healthy person. Most scientists believe that vCJD is caused by the BSE agent, most likely acquired by the consumption of BSE-contaminated foods. About 135 persons are believed to have died of vCJD thus far. The vast majority of these cases have been in the United Kingdom where, at its peak, over 1,000 cases of BSE were diagnosed weekly.

#### Relative Risk to U.S. Beef Consumers

Although a BSE-infected cow has been found in North America recently, to this point, there have not been any BSE-infected cattle found in the United States. Current evidence suggests that the risk to beef consumers in the United States is low. Both the United States and Canada have taken a number of measures to

prevent the transmission of BSE both into and within our North American countries.

In 2001, the results of an extensive, three-year study of the risk of BSE in the United States were published. George Gray, deputy director for the Harvard Center for Risk Analysis, reported that "The risk that mad cow disease could happen in the U.S.A. is low, and the risk that it could spread as it did in Europe is lower still." A direct quote from the Executive Summary: "Our analysis finds that the U.S. is highly resistant to any introduction of BSE or a similar disease. BSE is extremely unlikely to become established in the U.S."

## **Animal and Product Handling**

The United States was one of the earliest countries to ban live ruminants (cattle, sheep, goats, etc.) and most edible ruminant products from the United Kingdom when the BSE outbreak was recognized. This occurred in 1989 before human health concerns had surfaced. Subsequently, the United States has moved to ban these products from all of Europe and Japan as evidence became available to indicate that BSE occurred in these countries.

In spite of what occurred in the UK and other western European countries, there are major differences in animal and meat product handling in England versus the United States. With the relatively higher grain costs in western Europe, compared to the United States, cattle raisers depended much more on the nutritional value of animal by-products, such as meat and bone meal, than on soy-protein sources which are abundant in the United States. Although the exact source of the BSE agent is still in question, most experts agree that the spread of the disease was by the feeding of contaminated ruminant by-products. Feeding of ruminant by-products to other ruminants was prohibited in the United States in 1997, and feed mills are inspected by FDA for compliance. In the United States the numbers of sheep entering the slaughter chain relative to the volume of cattle, pigs, and other animals is also quite small relative to the proportion of that seen in the UK when BSE began. If indeed the presence of scrapie in the meat and bone meal being fed to cattle was the source of the first BSE cases, the situation here was, and is, very different from that of the UK.

In countries in which meat raw materials are relatively more expensive than in the United States, food processors have depended more on low-cost materials, such as mechanically-separated tissue (MST), which is obtained from the soft tissue clinging to bones. This process typically involves salvaging this tissue (meat, connective tissue, as well as bone marrow from round bones) by pressing it through a sieve, which separates the MST from the bone particles. Neurological tissue, such as spinal cords (if not properly removed) could easily contaminate the neck and back bones used to produce MST.

Over the last 10 to 15 years, newer mechanical processes for removing residual meat from bones have been developed. These are called Advanced Meat Recovery Systems (AMR or AMRS) and do not involve crushing bones. These systems are more efficient than the older systems, are safer for workers, and result in a product that can be labeled as "meat." With this label, the product cannot contain spinal cord or other neurological tissue. In June of 2002, the USDA/FSIS proposed revisions of existing

directives regarding AMR systems. The rule was finalized in December of 2002, and it requires processors of beef who use these systems to take routine samples to verify that spinal cord tissue is not present in the product. USDA has indicated that it expects additional rulemaking activity in this area this year to further ensure that nervous tissue is not present in meat products derived from this process.

There are several other theories about how humans who have developed variant CJD in the UK might have been exposed besides direct consumption of beef. It has been proposed that accidental or intentional adulteration of meat products or other foods may have exposed people in the UK. Several instances in Europe in recent months of the finding of spinal tissue in products that shouldn't have had it suggest that there may have been multiple avenues of exposure. Contaminated instruments used in tonsillectomies (young people) are also proposed to have been a means of spread. Small children may have been exposed by consumption of pureed baby food, which has been shown to previously contain large quantities of MST. None of these hypotheses have been proven to be involved in human exposures, but are compatible with the accepted incubation period for variant CJD of approximately 15 to 20 years.

# **USDA Testing of Suspect Animals**

The safety of the U.S. beef supply is further protected by the USDA targeted surveillance program. In this program animals most likely to harbor BSE, if it is present, are examined after death. These include animals with suspected neurological disease found on-farm and presented to veterinarians in diagnostic laboratories; rabies suspects that are found not to have rabies in our public health laboratories; cattle condemned at slaughter for neurological disease; and down or fallen stock. The USDA has been looking for BSE in the United States for more than 12 years and is currently testing these high-risk cattle at a rate that is 41 times the standard testing rate, as established by the Office of International des Epizooties (OIE). In 2002, nearly 20,000 cattle in these categories were tested in the United States, with no BSEpositive cattle found. Although it is possible that this program will identify a case, as did Canada's surveillance program, the number tested so far gives us a high confidence that the disease is not present or is present at an extremely low level. This data supports the 2001 Harvard Risk Assessment.

# **Communicating Risk**

In the past 15 years or more, the topics of hazard analysis, risk assessment, and risk management have received much discussion. Risk *communication* to a public that is bombarded by sensationalistic media reports and which has come to expect "zero risk" or "zero tolerance" policies from its government is also much discussed, but very difficult to perform. There is no denying that finding a BSE case in the Unites States as has happened in Canada would cause tremendous political and economic upheaval. But it may be helpful to try to put the risk of BSE in our food supply in some perspective given the evidence we now have.

At the peak of the BSE outbreak in the UK in the early 1990s, and before the human health risks were recognized, about 1,000 cows were being diagnosed with BSE *weekly*. Certainly some

animals that were infected, but not yet obviously ill, entered the human food chain prior to and during this time, and large numbers of people must surely have been exposed to some level of the BSE agent. Although the personal tragedy associated with any case of vCJD cannot and should not be minimized, to date only about 135 cases of vCJD have been recognized; the numbers of cases diagnosed yearly seems to be coming down; and the estimated number of potential cases has been lowered from hundreds of thousands to perhaps a few hundred or a few thousand. Certainly the safeguards in place in the Unites States and Canada will not allow anything like the UK experience to be repeated.

It is important to keep potential health risks in perspective. For comparison, as of June 3, 2003, 770 deaths from SARS have been reported to the WHO—a disease just recognized this year; a May 5, 2003, article in the online version of *USA Today* reports that *annually* about 70 people are killed and 300 injured in lightning strikes in the United States (http://www.usatoday.com/weather/resources/basics/wlightning.htm); each year in the United States about 90 to 100 people die of bee stings (http://ohioline.osu.edu/hyg-fact/2000/2076.html); and in an article in the journal, *The Physician and Sportsmedicine* (Vol. 29 - No.7 – July, 2001, http://www.physsportsmed.com/issues/2001/07 \_\_01/mueller.htm) researchers reported that during the years of

1987-1996 there were more than 29,000 injuries in Little League Baseball players aged 5 to 12 years. About 25 percent of these were considered serious, and 13 players died.

Certainly it is appropriate to strive for "zero risk" in our food supply, but we must realize that "zero" is often unattainable. The available evidence suggests that the risk to the *consumer* of BSE in our American beef supply is very minimal. As we identify new potential risks, we will move to implement additional safeguards—as we have been doing.

## **Internet Links:**

### **USDA**:

www.fsis.usda.gov/oa/topics/bse.htm#3 http://www.usda.gov/news/releases/2003/01/0012.htm http://www.aphis.usda.gov/lpa/issues/bse/bseoverview.html

#### Canadian Food Inspection Agency:

http://www.inspection.gc.ca/english/anima/heasan/disemala/bseesb/bseesbindexe.shtml

## ProMED (SARS):

http://www.promedmail.org/pls/askus/f?p=2400:1202: 424240::NO::F2400\_P1202\_CHECK\_DISPLAY,F2400\_ P1202\_PUB\_MAIL\_ID:X,21768

Visit Ohio State University Extension's WWW site "Ohioline" at: http://ohioline.osu.edu

All educational programs conducted by Ohio State University Extension are available to clientele on a nondiscriminatory basis without regard to race, color, creed, religion, sexual orientation, national origin, gender, age, disability or Vietnam-era veteran status.

Keith L. Smith, Associate Vice President for Agricultural Administration and Director, OSU Extension