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WHAT ARE WE DOING TO PREVENT ENTRY OF POTENTIALLY
CATASTROPHIC FOREIGN ANIMAL DISEASES INTO THE U.S.A.

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What are Foreign Animal Diseases? By definition, a Foreign Animal Disease (FAD) is one which originates in another country and can, or does, enter the USA. An agency of our federal government, the Animal and Plant Health Inspection Service (APHIS) of the U.S. Department of Agriculture (USDA), is charged with responsibility for preventing entry of FADs into our country and is especially vigilant regarding those FADs which would have potentially catastrophic effects on farm-animal populations, the agricultural economy and/or the public (human) health. Activities of APHIS specifically related to FADs include (USDA-APHIS, 2001a): (a) Guarding our borders against foreign agricultural pests and diseases through activities at U.S. ports and overseas in foreign countries. (b) Detecting and monitoring animal and plant diseases in this country. (c) Carrying out emergency operations if foreign pests or diseases get past our border defenses.

Why are we concerned about FAD? Horn (2001) described our concerns about FADs as: (a) These diseases are common overseas. (b) Because these diseases are spread by natural means, constant efforts are needed to exclude their entry into the USA. (c) Our livestock would be unbelievably susceptible to these FADs. (d) An outbreak of an FAD would cripple agricultural trade, would be devastating economically and would have psychologically depressing societal effects. Examples of effects of four FAD outbreaks follow:

- (a) Foot-and-Mouth Disease (FMD), in swine, in Taiwan, in 1997—FMD was diagnosed in one tiny area of the country in March and, in three months, had spread throughout the entire country (Horn, 2001). Four million hogs died or were killed, there was huge societal impact generated by dead/rottening pigs and smoke from hog funeral pyres and Taiwan lost \$7 billion in agricultural and export trade. Some believe that Taiwan may never recapture their export markets for pork (Horn, 2001).
- (b) Classical Swine Fever (CSF), in swine, in the Netherlands, in 1997-1998—CSF was diagnosed in February 1997 and brought under control in May 1998 (Horn, 2001). CSF was ultimately found on 60% of pig farms, resulted in death or slaughter of 11 million hogs, cost the Netherlands \$2.3 billion in agricultural and export-trade losses, and ultimately spread to Italy, Spain and Belgium (Horn, 2001).

- (c) Foot-and-Mouth Disease (FMD), in cattle/sheep/swine, in Great Britain, in 2001—FMD was detected on one pig farm on February 28, had spread throughout England and into France and Ireland two weeks later, and was occurring at the rate of 350 new cases per week in the UK and EU by April 1 (Horn, 2001). As of October 17, 2001, animals that have died or been slaughtered total 600,000 cattle, 150,000 swine and 3,000,000 sheep. Psychological and social impacts have been huge; American Express reported that tourism in the UK has been reduced by 85% since outbreak of the disease (Horn, 2001). Beef (2001) reported that: (a) The UK recorded its 2,000th case of FMD in early September 2001. (b) Government scientists now say the outbreak could last, at least, until January 2002. (c) To date, 3.8 million head of livestock have been destroyed with another 19,000 awaiting euthanasia. For purposes of analogy, Horn (2001) said “Great Britain is the size of Oregon; if an FMD outbreak of the same magnitude as has occurred in the UK were to occur in the USA, probable numbers of dead or slaughtered U.S. livestock would be 4 million cattle, 1 million swine and 1 million sheep.
- (d) Mad Cow Disease (MCD), in a single cow, in Japan, in 2001—An example of results of a single case, not an outbreak, of an FAD provides further support for catastrophic effects on domestic and export demand for beef (National Meat Association, 2001a). Japan announced on September 10, 2001 that a Holstein dairy cow tested positive for MCD in an area near Tokyo. Three weeks later, Junichiro Koizumi (Japan Prime Minister) said: People are saying they don’t want to even eat safe beef products. This is a frightening thing!” Confusion over the disposal of the infected cow, which was ground into meat-and-bone meal (MBM), has deepened consumer mistrust, with some 2,000 schools nationwide clearing beef from their lunch menus. Widespread news coverage of the BSE outbreak has taken a toll on the shares of some restaurant chains, as well as beef prices and sales in Japan, possibly denting the outlook for its Kobe beef exports. Photographs have circulated of workers standing next to literally tons and tons of unused beef product (National Meat Association, 2001a). Domestic prices for beef in Japan have fallen about 60% (Reuters, 2001). And, Clayton (2001) reported that demand for U.S. beef in Japan has declined by 50%.

Were lessons learned from recent FAD outbreaks? Lessons learned from the FMD outbreak in Taiwan (Horn, 2001) were: (a) The source of the virus was probably pigs smuggled in from mainland China. (b) It took several weeks to identify it as FMD (it was first thought to be Vesicular Stomatitis). (c) Officials did not initiate preventive slaughter of at-risk animals soon enough. Lessons learned from the CSF outbreak in the Netherlands (Horn, 2001) were: (a) The source of the virus was food brought from Bosnia by returning soldiers. (b) Officials allowed uncooked garbage to be fed to swine. (c) Officials did not require proper cleaning of trucks between loads of hogs. Lessons learned from the FMD outbreak in Great Britain (Horn, 2001) were: (a) The source of the virus was probably pork smuggled in from China. (b) Officials allowed uncooked garbage to be fed to swine. (c) The virus was present for some weeks before FMD was diagnosed. (d) The measures taken to control spread of the virus were not sufficient. The lesson learned from the BSE incident in Japan (National Meat Association, 2001a) was the extent of the danger associated with

feeding at-risk animal protein (meat-and-bone meal) imported from a country that had an epidemic of MCD. Obviously, traffic across the borders of the USA creates opportunities for inadvertent entry of FAD-infected foods that could trigger potentially catastrophic effects on our food-animal and human populations; Horn (2001) reported that, in 1999, 475 million people, 125 million vehicles and 21.4 million import shipments crossed U.S. borders.

Which FADs concern us most? U.S. beef producers are most concerned about Bovine Spongiform Encephalopathy (BSE) and Foot-and-Mouth Disease (FMD).

What are BSE, CJD and nvCJD? The “B” in BSE means bovine (i.e., cattle), “S” stands for spongiform (i.e., sponge-like appearance under a microscope) and “E” is for encephalopathy (i.e., brain illness); thus, BSE is a disease causing a sponge-like appearance of the cattle brain (Smith, 2001a). BSE is manifested as “craziness” of cattle, hence the name Mad Cow Disease (MCD). BSE was first observed in the United Kingdom in 1972 and diagnosed/described as a cattle disease in 1986; the incidence peaked at 36,680 cases in 1992 and since has declined to 1,311 cases in 2000 and 459 cases through September 2001 (DEFRA, 2001). Creutzfeldt-Jakob Disease (CJD) is named for the two physicians who first diagnosed/described it (in the 1920s) in human patients (National Cattlemen’s Beef Association, 2001b). CJD is a progressive, degenerative, spongiform and fatal encephalopathy that occurs worldwide at a rate of about one case per million persons (U.S. Meat Export Federation, 2001a), usually in persons over the age of 55 years (Franco, 2001). nvCJD (new variant Creutzfeldt-Jakob Disease) was first diagnosed/described in 1996 and is also a progressive, degenerative, spongiform and fatal encephalopathy (Brown *et al.*, 2001). nvCJD is different, though, from classical CJD because it has largely, but not exclusively, affected much younger people; the age of the first ten known victims of nvCJD ranged from 16 to 39 years suggesting that nvCJD has a much shorter incubation time than classical CJD (Will *et al.*, 1996).

BSE, CJD and nvCJD belong (along with scrapie in sheep, chronic wasting disease in deer and elk, and kuru in humans) to a group of diseases called (Acheson, 2001) Transmissible Spongiform Encephalopathies (TSEs). Since BSE was first diagnosed/described, numerous possible causes for the anomaly have been suggested (Smith 2001b; Doyle, 2001): (a) Sheep scrapie jumping species because of changes in meat byproduct rendering practices (U.S. Meat Export Federation, 2001a). (b) An unconventional virus (Food Processing, 2001). (c) A “virino,” which is described as an incomplete virus composed of naked nucleic acid protected by host proteins (Food Processing, 2001). (d) Copper/manganese irregularities in the animal’s diet (Purdey, 2001). (e) High-dose treatment of cattle with Phosmet, an organophosphate used to treat cattle for ox warbles (National Meat Association, 2001b; Render, 2001). (f) “Molecular mimicry” by the bacteria *Acinetobacter* found in “winter feeds” for cattle (Ebringer *et al.*, 1997). (g) Occurrence of a rogue-protein, or abnormal, prion in cattle feed. Food Processing (2001) describes a “prion” as an abnormal partially proteinase K-resistant protein devoid of nucleic acid, capable of causing a cell to produce more abnormal protein. Acheson (2001) says the causative agent was termed a “slow virus” in the 1950s. Stanley Prusiner, in 1982, coined the term “prion” for this protein; he was awarded the Nobel Prize In Medicine in 1997 for his work (Franco, 2001). Prions occur normally in the brain, spinal cord and certain nervous tissues of all

animals; normal prions may have a role in copper transport, nerve conduction, cell signaling and antioxidant reactions (Doyle, 2001). It is the occurrence of aberrant forms of prions (the so-called “rogue-protein prion” or “BSE prion”) that causes the problem. Pathogenic prions pervert normal prions by changing their structure and causing them to aggregate and interfere with brain function (Doyle, 2001).

The “aberrant” or “abnormal” or “rogue-protein” BSE prion is now—almost universally—considered to be the causative agent for BSE and nvCJD (Collinge *et al.*, 1996; Bruce *et al.*, 1997; Hill *et al.*, 1997). BSE is the most recently identified disease caused by unique infectious proteins known as “prions” (Doyle, 2001). How rogue-protein prions originated and why animals became susceptible to them is not known. Perhaps, the original abnormal prion was from a sheep with scrapie, or was mutated in a bovine animal (the “spontaneous rogue-protein prion generation” theory), or as has been recently suggested, was introduced into England by importation of Nyala or Kudu antelope from Africa (Wall Street Journal, 2001). And, maybe, mineral imbalances or bad bacteria or improperly heated meat-and-bone meal in the feed, or inappropriate use of organophosphate pesticides compromised the immune system of some cattle, allowing the rogue-protein prion to gain a foothold—starting the spiral of BSE infections that were—almost certainly—spread from bovine to bovine (and, as it turns out, from cattle to meat, cats and people) by the feeding of ground-up cattle cadavers to living cattle (Smith, 2001b). Horn *et al.* (2001) have concluded that changes in rendering processes, use of meat-and-bone meal in starter calf diets and a relatively high proportion of sheep in rendered material probably combined to initiate the BSE epidemic in the UK.

We may never know the how or the why of what started this spiral, but this much, we now believe, we know (Smith, 2001a): (a) Prions are present normally as relatively straight molecules (with several α -helical structures) in the brain, spinal cord, cornea, pituitary gland and nerve ganglia of cattle and people. (b) Rogue-protein prions are malformed, misfolded proteins that occur as bent, curved or widened molecules (with more β -sheet structures) that gain entry into the body through the tonsils or Peyer’s patches of the small intestine and are transported to the brain via the lymphatic system. (c) When rogue-protein prions enter the brain, these malformed prions convert normal prions into malformed prions. Smith (2001b) said “Imagine, if you will, a ballroom filled with people who are waltzing. A person enters the room, doing the twist. Every time the twister bumps into a waltzer, the waltzer starts doing the twist...and converting other waltzers to twisters.) Because the malformed prions eventually aggregate (clump together) they collect in areas of the brain, kill the surrounding tissue and form holes.” Evidence supporting the rogue-protein prion theory of BSE disease transmission among cattle and that BSE can be transmitted to humans (via consumption of beef or offal from BSE-infected cattle) causing nvCJD, but not classical CJD, is highly documented in the scientific literature (Scott *et al.*, 1999). These findings argue unequivocally that BSE and nvCJD are caused by the same strain of rogue-protein prion (Almond and Pattison, 1997).

How are BSE and nvCJD transmitted? It is believed (Brown, 2001) that at some point in time—probably prior to 1970—cattle were given feed contaminated with rogue-protein prions; that feed most likely was, or contained, meat and bone meal (MBM) that was

made from cadavers or other remains of sheep with scrapie, cattle with mutated prions or African antelope that had, or had died from, a TSE (Smith, 2001b). In nature, prion diseases are known to be transmitted orally and mutant genes may cause disease or an increased susceptibility to abnormal prions (Doyle, 2001). The incubation time (time required for malformed prions to convert enough normal prions to malformed prions to cause the neurological and physical symptoms of BSE) was such (perhaps 8 to 15 years) that by the time enough “mad” cows were observed to establish a trend (in the early 1970s), the transmission cycle (feeding remains of a few cows that died of BSE to other cattle and thus infecting very many of them with BSE), the problem was out of control (Smith, 2001a). The spiral continued upward as newly infected cows were slaughtered, and brains and spinal cords were put back into animal feed; that feed was given to more cows, spreading the disease (Brown, 2001). In the USA, a test developed by Schmidt (2001) is used to test beef trimmings and beef products for presence of a unique protein (Glial Fibrillary Acidic Protein) that occurs almost exclusively in central nervous tissue, as a means of assuring that spinal cord and ganglia (that would carry BSE prions if US cattle have BSE, and we don’t yet know it) are not present.

The disease moved from cattle to humans when infected meat was cut-up, ground or processed (USA Today, 2001a); the vehicle could have been ground meat or sausage (which could have contained brains, spinal cords or the ileum portion of the small intestine) or it could have come from solid-muscle cuts contaminated by splitting the carcass (spreading spinal cord tissue in the process) or contaminated by butchers or meat cutters using sloppy techniques (e.g., some meat cutters in the UK purchase whole carcasses with the intact head attached, open the skull to extract and merchandise the brain—for breakfast food—and then proceed to fabricate the carcass into steaks, roasts and mince) without first cleaning their tools, cutting boards or equipment (New Scientist, 2000, 2001b). Pathogenic prions are extremely resistant to heat and disinfectants, and the high concentration of pathogenic prions in central nervous system tissue makes it important to prevent these tissues from entering the food chain (Doyle, 2001). The difference in spread between BSE and nvCJD may be due to the fact that, in humans, recycling of infected tissue has not occurred, and thus the epidemic will evolve much more slowly than in cattle (Brown *et al.*, 2001). Estimated infectivity of bovine tissue containing BSE prions (FDA, 2001) is as follows: (a) One gram of nervous tissue contains 10 million infective doses of the BSE prion. (b) One gram of any of the spleen, lymph node or colon contains less than 25,000 infective doses of the BSE prion. (c) One gram of any of the pancreas, liver or lung contains less than 100 infectious doses of BSE prions. (d) One gram of any of muscle, bone or heart contains less than one-tenth of one infectious dose of BSE prions (FDA, 2001).

BSE popped up in herds all over England in 1988; by the year 2001, 180,500 cattle had been diagnosed with BSE in the UK, and other cattle with BSE had been discovered in 15 other European countries (Office International des Epizooties, 2001). In Great Britain, 106 probable and proven cases of nvCJD had been diagnosed through August 2001 (UK Department of Health, 2001) and 3 deaths due to nvCJD have occurred in other EU countries (Smith, 2001a).

Fox (2001) said “The only surefire way now to test for BSE is to check an animal’s brain after it has been killed. There is still no treatment for prion diseases, including BSE and nvCJD, but a reliable and sensitive diagnostic would permit the testing not only of cattle, but also of human blood products and tissues before they are used in medical procedures.” At least six companies—Paradigm Genetics, Prion Developmental Labs, Boehringer Ingelheim, Genescan Europe AG, IDEXX Laboratories, Caprion Pharmaceuticals—are working to develop tests to detect BSE and/or nvCJD while the diseases are incubating—before physical symptoms occur (Hollingsworth, 2001; Barto, 2001; DeFrancesco, 2001; Giese, 2001).

New Scientist (2001a) recently reported that: (a) Mad Cow Disease scandals just don’t stop. In a week when Great Britain recorded 6 new cases of nvCJD—the biggest real monthly increase so far—it has become clear that it most certainly inflicted the curse on the rest of the world. (b) Great Britain continued to export animal feed made from ground-up remains of infected cattle long after it knew that the pellets spread BSE to other cattle; the rest of the EU was still exporting it in January 2001. (c) Great Britain insists that the feed was meant only for pigs and chickens but you can bet this message didn’t reach farmers in places like Southeast Asia. Even restricting the feed to pigs and chickens may not be safe because these animals end-up in cattle feed and could pass the infection on. (d) Great Britain exported meat-and-bone meal (containing brains until 1991) from 1980 through 1996, to more than 80 countries; the rest of the EU more than trebled MBM exports during the 1980s to non-EU countries (New Scientist, 2001a). USA Today (2001b) reported that the first indication that MCD may have spread to Eastern Europe was reported on June 6, 2001 by agriculture officials in the Czech Republic; tests have not yet confirmed the disease which is suspected in a 6-year-old animal in Jihlava but the European Commission said in April that the Czech Republic is at risk of BSE because it has imported live cattle and MBM from EU countries. No MBM was exported to the U.S. between 1988 and 2001 (National Cattlemen’s Beef Association, 2001b); it is believed that no MBM was exported to either Canada or Mexico during that same time period, but we are not certain that is correct (Smith, 2001b).

What measures are being taken to minimize risk that BSE will enter the U.S.? According to American Meat Institute (2001a), National Cattlemen’s Beef Association (2001b) and Lazar (2001): (1) The U.S. has not imported beef from the U.K. since 1985; the U.S. has not imported ruminant animals or at-risk ruminant products from countries with confirmed cases of BSE since 1989. (2) More than 60 veterinary diagnostic laboratories throughout the U.S. participate in a BSE surveillance program with the National Veterinary Services Laboratory (Ames, IA). (3) In 1997, a Food and Drug Administration (FDA) regulation banned the use of at-risk, mammalian-derived animal protein byproducts in cattle feed (Meat Processing, 1997) to ensure that, if the rogue-protein prion causing BSE ever entered the U.S., it would be prevented from spreading through cattle feed. (4) In 1997, the USDA banned imports of all live ruminants and certain ruminant products from European countries until BSE is more fully understood. (5) In 1998, the USDA asked the School of Public Health, Harvard University to analyze and evaluate the USDA’s BSE prevention measures. (6) In 2000, APHIS prohibited all imports of rendered animal protein products, regardless of species, from Europe. (7) In 2000, the USDA issued a Declaration of Extraordinary Emergency, obtained some sheep from three flocks imported from Europe,

ethanized these sheep and examined—diagnostically—their brains. (None had BSE.) (8) In 2000, APHIS prohibited all imports of rendered animal protein products from Europe, regardless of species. (9) In 2001, USDA seized the remaining sheep imported from Europe, euthanized them and examined their brains but none had BSE (National Cattlemen’s Beef Association, 2001b).

Salvage (2001) reported that BSE has not been detected in the U.S. and the USDA says we have a “Triple Firewall System” in place to keep it out: (a) Firewall #1—A series of import bans dating back to 1985 ensures that no live cattle or at-risk cattle products are imported from any European country. (b) Firewall #2—FDA banned at-risk animal protein in cattle feed in 1997. (Feeding animal protein to cattle doesn’t cause BSE unless the animal protein includes BSE prions, but the ban would keep the disease from spreading should it ever occur in the U.S.) (c) Firewall #3—USDA has had an active surveillance program for the past ten years that has found no sign of the disease in U.S. cattle herds (Salvage, 2001).

What is FMD? The most contagious disease known to mankind is Foot and Mouth Disease (FMD). Caused by each of seven types of a specific virus in the *Picornia* group, FMD occurs in cloven-hooved animals (cattle, sheep, swine, goats and many game animals) on all continents except North America, Australia and Antarctica and is persistent in about two-thirds of the countries of the world (Torres, 2001). Animals affected with the FMD virus demonstrate profuse salivation and slobbering, have difficulty eating and drinking, and prefer to lie down rather than standing or walking (U.S. Meat Export Federation, 2001b). Vesicles (blisters) form in the mouth and nostrils and on the tongue, on the udder and teats of female livestock, and on the soft tissues between the toes and above the hooves (Crews, 2001). The blisters burst, leaving eroded and pitted areas of exposed flesh that are apparently very sensitive and sore, hence the reluctance of affected animals to eat, drink, stand, walk or—in the case of dairy animals—to submit to hand or machine milking (Ensminger, 1996). The disease is fatal to about 2% of affected adult animals and about 20% of young livestock but often debilitates those farm animals that survive, greatly reducing meat, wool, mohair and/or milk production even after recovery from the disease (National Cattlemen, 2001). People are very mildly susceptible and very rarely infected by the FMD virus (Crews, 2001); in the recent outbreak of FMD in livestock of the United Kingdom and European Union, at least 21 persons were suspected of having contracted the disease but, as of June 2001, virology testing eliminated FMD virus as the cause of their physical symptoms (Associated Press, London, 2001).

FMD is so contagious that essentially 100% of cloven-hooved animals exposed to the virus become infected (National Cattlemen, 2001). The incubation period for FMD is from 3 to 14 days and the disease has a time-course of about 2 to 3 weeks. Transmission (spread) of the disease is: (a) Animal—FMD virus can be spread from animal-to-animal (among and between cloven-hooved species) by direct contact or from air exhaled from the lungs of infected animals, especially swine (Torres, 2001). (b) Feed And Water—grain, hay, silage, pasture and mixed feeds as well as water can spread the FMD virus (National Cattlemen, 2001). (c) Apparel—shoes, boots and clothing can carry and thus spread the FMD virus; the FMD virus can survive in clothing for up to 46 days at room temperature (Ugstad, 2001). (d) Facilities, Equipment and Vehicles—Barns, sheds, pens, chutes and farming/ranching

vehicles (both for humans and livestock) can spread FMD virus (National Cattlemen, 2001). (e) Raw or Improperly Cooked Garbage Containing Infected Meat or Animal Products—this is the most likely potential source of FMD occurrence in the USA (U.S. Meat Export Federation, 2001b); the virus can survive up to one year in such products. (f) Semen or Biologics Containing the FMD Virus—animals inseminated or given medicines containing the virus would get FMD (U.S. Meat Export Federation, 2001b). (g) Bio-Terrorism—FMD virus is in the germ-warfare arsenal of many countries and could be used by bio-terrorists to disrupt the economy of the U.S. (Torres, 2001). (h) Airborne—with appropriate wind (direction and speed), the virus can travel 60 km over land and 220 km over sea (Ugstad, 2001). (i) Aerosol—aerosols created by the jostling of milk in a vented transport tanker can spread the virus to farms and ranches adjacent to the road being traveled (Ugstad, 2001). (j) Human Nasal Passages—FMD virus can survive, and thus be disseminated, in human nasal passages for at least 28 hours (Veterinary Services, USDA-APHIS, 2001) and perhaps as long as 5 days (Crews, 2001). (k) Milk—FMD virus can survive milk pasteurization performed at 72°C for 15 seconds (Ugstad, 2001). (l) Meat—the FMD virus can be in bone marrow and lymph nodes of chilled and frozen meat. FMD is not present in the USA largely because neither live cloven-hooved animals nor their fresh, chilled or frozen meats can be legally imported into the USA from any country (or region of certain otherwise FMD-free countries) in which it has been determined that FMD exists (Ensminger, 1996).

What steps are being taken to keep the U.S. free of FMD? The U.S. has been free of FMD since 1929 (Crews, 2001), when the last of 9 outbreaks here was eradicated, because of the superb monitoring and surveillance system of Animal and Plant Health Inspection Services (APHIS) of USDA. The last two FMD outbreaks in the U.S., in 1924 and 1929 (U.S. Meat Export Federation, 2001b), were both caused by feeding raw or unrecooked garbage from steamships to swine and were quickly contained; the February 2001 outbreak of FMD in the UK is believed to have been caused by feeding raw or unrecooked garbage from illegally smuggled meat served in Chinese-style restaurants to swine in northern England (National Meat Association, 2001c). In that outbreak, one case on one pig farm was detected on February 28; FMD had spread throughout England and into France and Ireland two weeks later and was occurring at the rate of 350 new cases per week in the UK and EU by April 1, 2001. The pivotal importance of swine in development of an FMD outbreak cannot be overemphasized. Although the virus replicates in the blisters of infected animals and erupts—as infected particles—when blisters burst, swine infected with FMD shed the virus in exhaled air—as infected aerosols—before they exhibit clinical signs of the disease. Torres (2001) says there is enough FMD virus in one breath of exhaled air from an FMD-infected pig to generate 10,000 infectious doses of the disease. FMD is so feared in the U.S. that our Congress passed legislation in the 1950s making it illegal to possess live virus anywhere (except at the USDA Plum Island Animal Disease Center which houses the North American FMD Vaccine Bank) in our country (Torres, 2001).

USDA monitors diseases among U.S. cattle herds and takes aggressive action to prevent animal diseases (including FMD) from entering the U.S. whenever an outbreak occurs in another country (Veterinary Services, USDA-APHIS, 2001; American Meat Institute, 2001b). Examples of actions that have been taken since the February 2001 FMD outbreak in England, Ireland and the UK include (National Cattlemen's Beef Association,

2001c; Walloch, 2001; Crews, 2001; National Meat Association, 2001d): (a) USDA issued an interim rule in February 2001 prohibiting or restricting the importation into the U.S. of live swine and ruminants and of any fresh swine or ruminant meat (chilled or frozen) or animal products from Great Britain or Northern Ireland. (b) In March 2001, USDA expanded the earlier ban to temporarily prohibit the importation of animals and animal products from the entire EU due to concerns about FMD there. (c) USDA dispatched teams of experts to England and to Argentine in March 2001 in response to FMD outbreaks (Argentina announced an FMD outbreak there in March 2001); these experts help to stem the tide in affected countries and guard against introduction of the disease into the USA. (d) In March 2001, heightened alerts were enacted at ports of entry and airports to ensure that passengers, luggage and cargo are checked; this included deploying additional inspectors and dog teams (to detect meat and other food in parcels, luggage, baggage and cargo) at airports to check incoming flights and passengers. (e) In April 2001, USDA authorized an additional \$32 million to hire 350 additional staff members to work at critical ports and airports. (f) In April 2001, USDA initiated an aggressive public education campaign that included additional signage in airports, public service announcements, a Web site and other means to inform the public about this important issue and steps they can take to prevent the virus from entering the U.S. (g) In May 2001, President George W. Bush signed the Mad Cow and Related Disease Prevention Act of 2001 (S. 700) which established an interagency task force with oversight by Secretary of Agriculture Ann Veneman, to prevent the introduction of foreign animal disease—particularly BSE and FMD—into the U.S.

Ugstad (2001) described USDA measures against entry/spread of FMD as consisting of: (I) General Prevention—Control of importation of livestock, zoo animals, and animal products from countries which have FMD. (II) Response To The Outbreak In The UK In 2001—(a) USDA sent 40 people to the UK to help contain the disease. (b) USDA heightened alerts at airports and points of entry. (c) USDA restricted imports of semen and cheese processed after February 19, 2001. (d) USDA heightened coordination with state agriculture and veterinary science officials. (e) USDA coordinated efforts to prevent entry of FMD into the U.S., Mexico and Canada (the “North America FMD Prevention Plan”). (III) Response Plan—(a) State and national health officials have a response plan that integrates government and industry action and involvement. (b) There will be extremely strict animal-movement controls. (c) There will be depopulation with indemnification (payment to farmers/ranchers for euthanized animals). (d) There will be vaccination to limit the scope of the outbreak. (e) USDA will begin immediately to optimize recovery of lost international trade opportunities (Ugstad, 2001).

What are the general prospects of BSE and/or FMD leaping from one continent to another and occurring in cattle (BSE) or cattle/sheep/swine (FMD) in the United States? Smith (2001c,d) said “Having recently spoken to two of the most respected experts on the subject (Dr. Will Hueston, University of Minnesota, about BSE; Dr. Alfonso Torres, USDA-APHIS, about FMD): (a) I do not believe we will have outbreaks of either disease within the next 12 months, and perhaps never, and (b) If either disease does occur in the U.S. during the next year, or ever, I believe the response to the incident by those in industry and government will be so rapid that it will be of minimal consequence to the livestock industry but of

variable consequence to the meat and food industries—depending on how the media reports the incident and how consumers react to such reports.”

What have been the consequences of outbreaks of BSE and FMD in the European Union? Smith (2001d) and U.S. Meat Export Federation (2001c) reported that EU Agriculture Commissioner Franz Fischler reported that beef consumption in the EU is down 25% (comparing October 2000 vs. March 2001 data) and that EU export performance is down to 10,000 to 15,000 tonnes per week; he said “Coping with the BSE crisis or FMD separately would already be an enormous challenge for the European agriculture sector. Now they are striking at the same time. And, the silver lining on the horizon of the beef market might turn out to be an illusion if consumers turn their back on meat altogether.” At the time of Fischler’s report, in June 2001, changes in EU beef consumption by country ranged from highs of -60% (Germany), -42% (Italy), -35% (Spain) and -30% (Greece and Luxembourg), to lows of 0% (Denmark, Netherlands, Ireland, Finland and Sweden) and +3% (United Kingdom) with the latter (+3% in the UK) suggesting no negative effect of the February 2001 outbreak of FMD in the UK on beef consumption (U.S. Meat Export Federation, 2001c; Smith, 2001d).

In May 2001, Great Britain’s Meat and Livestock Commission (Smith, 2001a,c) reported that “New research tells us that consumer confidence in red meat has been rocked by the graphic visual images of FMD represented by the media, particularly funeral pyres and the tipping of dead animals. More than 1 million families throughout Great Britain may have given up red meat because of health fears prompted by the foot-and-mouth disease epidemic; meat sales have fallen by as much as 15% and could worsen, weakening Britain’s red meat market. The sight of burning funeral pyres in the countryside revived fears over food safety that had begun with revelations about BSE being passed on to humans. Among some consumers, this has served to rekindle concerns about meat and farming practices, suppressed since the BSE outbreak of 1996 and its aftermath; sales and household purchase data indicate that some 1,000,000 households may have stopped purchasing beef, pork and lamb in the short term.”

If BSE and/or FMD were to breach our defenses, how damaging could this be to the U.S. meat supply and what would be the impact on consumer buying decisions? Smith (2001c) reported that “We do not believe we have BSE in the USA and we have not had a human death attributable to nvCJD. The federal government has erected three firewalls to preclude entry, and prevent spread—if it does occur—of BSE: (1) The U.S. bans cattle and beef products from countries where BSE has been detected. (2) The U.S. bans the feeding of certain animal products back to animals. (3) The U.S. has a surveillance program that includes examination of brain tissue from suspected animals.” Chandler (2001) reported that Dr. George Gray (School of Public Health, Harvard University) said “Mad Cow Disease is not likely to occur here. And, even if it does occur, it is virtually impossible to imagine how we could have a UK-like epidemic. So long as brain, spinal cord and ileum (which carry the rogue-protein prions of BSE) are carefully avoided, only small incremental changes in government policy and regulations will be needed.” Gray (2001) said the final report of their risk assessment would be released in the next few months.

Smith (2001d) said “A U.S. outbreak of FMD would cause loss of productivity and closing of U.S. borders to export of meat and byproducts, especially edible offal, and would lead to great economic loss. Outbreaks of FMD in the UK, EU and other countries have heightened awareness and concern of U.S. consumers but the actual effect, should an outbreak occur in the USA, would be economic—decreasing profitability of farmers/ranchers and packers and, perhaps, increasing meat prices to consumers.”

Would an actual outbreak of either BSE or FMD—or even concern about such outbreak—have the eventual impact of promoting greater use of food safety programs throughout the meat supply chain? Hueston (2001) has said that “Healthy livestock is the basis for a safe food supply.” Drovers Journal (2001) reported that the case is made that U.S. beef consumption could drop by 50% and major export markets (U.S. beef export values, including variety meats, totaled more than \$4 billion in 2000) could suddenly close if either BSE or FMD were to occur in this country. Because these diseases have wreaked such havoc in other parts of the world, it is imperative that we do everything possible to prevent entry into the U.S. But, if they do, what will be the most valuable arrow in our quiver to minimize spread of the disease? It is “traceability” (Smith, 2001a).

Smith (2001a) reported that: (a) Traceability, or trace-back, in this context refers to the ability to identify the source of meat from farm-to-fork and/or from fork, back to the farm on which the animal source originated. (b) It is now possible to follow cattle, through harvest, to the carcass form by using retinal scanning, plastic/metal tags and trolley-tracking. (c) It is more difficult to follow carcasses through fabrication (into primal/subprimal cuts and trimmings), distribution and retail-cut preparation in foodservice and supermarket operations. (d) Meat at retail can though—as is being done in Ireland, New Zealand and Australia—be traced back to carcasses of origin by use of DNA-fingerprinting technology (Smith, 2001a).

The Scientific Steering Committee (SSC) of the European Commission (2001) has recently developed (May, 2001) a “Rapid Response” program of action, in the event that BSE occurs in the sheep population of the EU; included in that program are: (1) An EU-wide culling program. (2) A long list of BSE-risk parts which should be removed from carcasses. (3) Culling of all suspect animals, their offspring, all traceable relatives and all animals with TSE susceptibility in the affected and contact flocks. “Traceability of individual animals is an essential part of the plan, hopefully also enabling identification of the parents; certification of flocks as TSE-free would then be possible” (Fleischwirtschaft International, 2001).

What Are Government Agencies Doing To Help Prevent Entry/Spread of FADs? Principles of FAD Management involve Preparedness, Prevention, Discovery and Response and were characterized by Torres (2001) as follows: (I) Preparedness—(a) Characterization of FADs worldwide, (b) Foreign intelligence and activities, (c) Education and training, and (d) Emergency management systems. (II) Prevention—(a) Strict regulations on importations of animals and animal products, (b) Control of foreign-origin garbage, (c) Control of domestic feeding of garbage (33 states allow such feeding but federal and state regulators make sure it is cooked prior to feeding), (d) Restrictions on feeding animal byproducts, (e) Maintain a strong animal health infrastructure, (f) Active surveillance and monitoring, and (g) Prompt diagnosis (there are FAD diagnosticians located in all states and territories, and

there are federal diagnostic laboratories). (III) Discovery—(a) Producer (via awareness and previous education) “observes it,” (b) Private veterinarian “suspects it,” (c) State or federal veterinarian “investigates it,” (d) State or federal veterinarian accurately/promptly “reports it” (“red-flags it”), and (e) National Emergency Management Response is activated (and, hopefully, “stops it”). (IV) Response—(a) Quarantine; stop movement of animals, (b) Disinfect vehicles and personnel, (c) Slaughter all affected animals, cohorts and progeny, (d) Destruction of affected carcasses, (e) If appropriate, vaccination, and (f) Surveillance and tracking (Torres, 2001).

USDA-APHIS (2001b) has developed a document entitled “National Emergency Response to a Highly Contagious Animal Disease” which provides guidance for a response to a highly contagious animal disease; this document includes (a) Concept of Operations, (b) Movement Control Guidelines, and (c) Foot-and-Mouth Disease Operational Guidelines. The Concept of Operations preamble states “The goal of an emergency response plan is to detect, control and eradicate a highly contagious disease as quickly as possible to return the U.S. to free status. A presumptive positive case will generate immediate, appropriate local and national measures to eliminate the crisis and minimize the consequences. A confirmed positive case will generate additional measures on a regional, national and international scale” (USDA-APHIS, 2001b). Movement Control Guidelines (USDA-APHIS, 2001b) characterize the “Infected Zone” (at least 6 miles beyond the presumptive or confirmed infected premises” and the “Surveillance or Movement Control Zone” (this zone surrounds the infected zone; its exact boundary will be established to assure containment of the outbreak). Foot-and-Mouth Disease Operational Guidelines provide specifics for Depopulation and Disposal, Cleaning and Disinfection, Estimated Personnel Requirements, Animal Welfare, Equipment, Indemnity and Appraisal, Milk and Milk Products, Meat, Zoologic Parks, Germplasm Centers and Disinfectants for FMD—Field Use (USDA-APHIS, 2001b).

Beef Business Bulletin (2001) reported that NCBA President Lynn Cornwell and Secretary Joaquin Ponce de Leon of the Confederacion Nacional Ganadera of Mexico signed an agreement in April 2001 expressing the intent of their organizations to keep North American free of FMD and other FADs; in September 2001, as an outgrowth of the U.S/Mexico agreement, cattlemen’s organizations from North and Central American countries (Belize, Costa Rica, Guatemala, Honduras, Panama, Canada, Mexico, USA) pledged to work together to ensure appropriate measures to prevent FAD are in place and enforced.

What Can U.S. Beef Producers Do To Help Prevent Entry/Spread Of BSE?
Producers must comply with the Food and Drug Administration (FDA) requirement that anyone feeding ruminant animals must save copies of invoices and labeling of all feed they receive that contains animal protein (National Cattlemen’s Beef Association, 2001a); feed that does not have an invoice or label from the manufacturer or distributor does not comply with the law, and the feed cannot be fed to cattle. National Cattlemen’s Beef Association (2001a) urges that: (a) Producers have written documentation from their feed suppliers that the premixes, supplements and complete feeds they buy are free of prohibited materials. (b) Cattle feeders/producers should consider buying feeds exclusively from feed mills that do not

handle prohibited materials. While this is not a part of FDA regulations, NCBA believes this is a reasonable step to reduce the risk of prohibited materials being incorporated in premixes, supplements and complete feeds destined for cattle. Wren (2001) quoted Dr. Linda Detwiler of USDA-APHIS as responding to the question “What can the U.S. meat protein industry do to help prevent BSE from occurring?” with the answer “Compliance. Any person or company that imports products should strictly comply with the regulations for importation. Domestically, producers should comply with feed ban regulations, as they are critical. If we can learn nothing else from the situation in Europe, it is that compliance with live animal and feed ban regulations is essential.

What Can U.S. Beef Producers Do To Help Prevent Entry/Spread Of FMD? California Department of Food and Agriculture (2001) says “Visitors from FMD-affected countries may unknowingly introduce the virus into the U.S., and meat and dairy products from affected countries may harbor the virus.” Beef Today (2001) says in order to protect your operation and the U.S. cattle industry from FMD, follow the guidelines listed below: (1) Learn all you can, and provide diagnostic training to employees to facilitate early detection. (2) Minimize exposure by asking visitors and employees to register, declare animals at home and exposed to, change clothes/footwear and wash hands thoroughly, disinfect via foot-bath or wear booties, and consider restricting animals/equipment entering and leaving the premise. (3) Discourage contact with livestock by international visitors and do not allow meat or animal products from FMD-infected countries to enter your facility. (4) Park all arriving vehicles in a restricted area; have vehicles available for on-premise transport. (5) Receive incoming cattle only during daylight hours, make sure health papers match the cattle and record truck license numbers. (6) Maintain vigilance on livestock and susceptible wildlife (Beef Today, 2001).

National Cattlemen’s Beef Association (2001a,c) in answer to the question “What can beef producers in the U.S. do to help prevent FMD?” said: (a) Know who is on your farms/ranches/property at all times. If people from other countries where confirmed cases of FMD have been found are scheduled to visit your property, make sure they wear freshly cleaned clothing and footwear. Make sure people wash their clothes and footwear before traveling to another farm/ranch/property. (b) As always, farmers should watch for excessive salivating, lameness, and other signs of FMD in their herd and immediately report any unusual or suspicious signs of disease to their veterinarian, state or federal animal disease control officials, or their country agricultural agent. (c) Food waste used as feedstuffs is required to be fully cooked before feeding to livestock (National Cattlemen’s Beef Association, 2001a).

Are we at risk to agricultural bioterrorism? Peter Chalk, a policy analyst with RAND Corporation, in remarks at the World Food Prize Annual Symposium (Reuters, 2001a), said: (a) More must be done to protect the U.S. agriculture industry and food supply from attacks by extremists. (b) We have a highly critical sector that is vulnerable, and it’s not that difficult to exploit that vulnerability. (c) An attack on the nation’s food supply could have a devastating impact on the economy and damage consumer confidence. (d) The introduction of an FAD into a cattle feedlot (e.g., BSE) or large hog farm (e.g., FMD) would set off a tidal wave of effects. (e) The concentration of many animals that makes U.S. livestock facilities so efficient is also a security liability. (f) USDA would not be able to quickly and effectively

contain an outbreak because they don't have the ability to institute local preparedness programs (Reuters, 2001a).

According to Associated Press (2001), the U.S. Congress is concerned that the nation's food supply could be the target despite assurances by USDA Secretary Ann Veneman to American consumers and farmers that "the government is prepared to respond quickly should the nation's food supply be the target of an attack." FDA wants to hire 410 new food inspectors, lab specialists and other personnel to check fruits, vegetables and other products and to buy additional equipment to detect pathogens; USDA has put veterinarians on alert, and wants more guards to protect its labs around the country that work with food pathogens (Associated Press, 2001).

Which agents might be used for agricultural bioterrorism? Biological agents that could be used for bioterrorism (Kvasnicka and Thain, 2001; CDC, 2001; New York City Department of Health, 2001) include: (a) BSE prion—not likely because it takes too long; cause:effect is not readily apparent. (b) FMD virus—USDA Secretary Ann Veneman said (Associated Press, 2001) "My biggest concern is that terrorists would contaminate a feedlot with FMD virus." FMD doesn't affect human health but wreaks havoc on the livestock industry and agriculture economy. (c) Clostridium botulinum toxin—placed in a food or water supply could kill millions; it doesn't spread person-to-person. (d) Bacillus anthracis spore—Anthrax spreads via cutaneous contact, inhalation or ingestion; it doesn't spread person-to-person. Kvasnicka and Thain (2001) reported that 110 pounds of Anthrax spores, properly aerosolized and released at altitude over a city of 5 million people could kill up to 100,000 people but it would be "very technologically demanding" to prepare spore membrane coats to minimize particle-charge and to prepare ideal particle-size to properly aerosolize the spores. (e) Yersinia pestis cell—Bubonic Plague is spread largely by fleas but also by cutaneous contact and inhalation (sneeze/cough). (f) Variola virus—Smallpox is spread by saliva via contact with an ill person. Brasher (2001) said "After attacks from the air and the mail, government officials worry the nation's food supply could be next. The government considers potential targets to be fruits and vegetables that people eat raw and cattle that could be infected with fast-spreading FMD. The only known terrorist attack on U.S. food occurred in the 1980s, when a cult in Oregon contaminated salad bars with *Salmonella*. So, fresh produce may be the food most vulnerable to attack because it's often eaten raw, is subject to little inspection and *Salmonella* (obtained from supermarket chickens) or *E. coli* O157:H7 (commonly found in cattle manure) are readily available" (Brasher, 2001).

USDA (2001) announced that President George W. Bush proposed, on October 19, 2001, the allocation of \$45.2 million to USDA—to enhance other actions taken throughout other federal departments to increase homeland security—as a part of a \$20 billion submission to Congress for emergency funding to strengthen essential programs and services related to biosecurity issues. A variety of actions have already been taken by the government to protect America's livestock—and the food supply (USDA, 2001); these include: (a) Award of \$2 million in grants to 32 states to bolster emergency animal disease prevention, preparedness, response and recovery systems. (b) Funding to develop the regulatory

infrastructure for the Presidential Initiative To Counter Bioterrorism. (c) A Centers for Disease Control and Prevention, four-step preparedness plan to counter bioterrorism.

What should cattle producers do about agricultural bioterrorism? Reuters (2001b) reported that Texas Agriculture Commissioner Susan Combs held an emergency meeting of farm and ranch producers and said “An assault on our food supply through either biological weapons or the introduction of a foreign animal or crop disease or pest would be a highly destructive force that could be used against agriculture and America.” At that meeting, Texas State Veterinarian Linda Logan said: (a) We’re urging producers to keep a closer watch on their animals. (b) The cattle industry could be severely disrupted if diseases such as Brucellosis or Mad Cow Disease were to break out. (c) A reaction, including quarantine, would be led by a Texas rapid response force of state and federal agencies. Max Coats (Texas Animal Health Commission) added “The key is prompt identification and rapid diagnosis, so a prompt response can be mounted to contain any possible outbreaks” (Reuters, 2001b).

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