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Bovine Spongiform Encephalopathy: European Union’s reactions to the crisis in the UK

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According to a study sponsored by the European Association for Animal Production the total cost of the outbreak of Bovine spongiform encephalopathy (BSE) could be up to $130bn by the time the disease has disappeared (Anderson 7). Starting with the first outbreak in the United Kingdom in 1986 to the most recent confirmed cases in August 2006 in Canada the disease has brought major changes to the topic of food safety.

“Mad cows” – BSE in the UK

The first case of BSE appeared in April 1985 on a farm in Sussex but was not identified until 2 years later. The case of a single cow did not justify an in-depth investigation at that point. Between April 1985 and February 1986 nine more cows fell ill with the same symptoms (Schwartz 142). In November 1986 the Central Veterinary Laboratory (CVL) examined three cow brains and diagnosed a “multifocal spongy transformation of the brain” closely related to scrapie, a well-known disease affecting sheep and goats (Pennington 143). British vets were informed about the possibility of the new disease and urged to make declaration when symptoms were detected. But the scientific community was not informed until October 1987 when Gerald Wells from CVL published his paper. He was the first to describe the symptoms and stressed the similarity with scrapie. (Schwartz 142)

The demand for an epidemiological investigation was high after several more cases were reported by veterinarians. The study started in June 1987 and concluded in 1988 with a report stating transmissibility, probable causes and steps taken to put an end to the epidemic (Schwartz 143). As the origin of the disease the report identified Meat and Bone Meal (MBM), a protein-rich nutritional supplement derived rendered carcasses of livestock including sheep (Brown 6). The use of such supplements goes back to the 1920s and scrapie is an old disease, therefore a recent change must have occurred that changed the number of scrapie agents in MBM (Pennington 147). This early hypothesis concluded that since more sheep were processed and the processing plants were using a lower temperature the number of scrapie agents has increased over the last couple years. MBM was therefore to be banned as a food supplement for cattle (Pennington 148-149). The British government enacted a ban on feeding ruminants with feeds containing animal proteins derived from ruminants in July 1988. These measures taken were supposed to lead to the disappearance of BSE in the UK (Schwartz 144).

The hypothesis that BSE is scrapie in cattle was not challenged in the early years of the BSE crisis. It was widely accepted that due to an increase in scrapie agents in MBM the disease had jumped species. The question raised, why this had never happened before since MB is used very widely for year, was ignored (Pennington 154-155). In summer 1988 worries about a possible species jump to humans became loud. A regulation requiring all animals suspected with BSE to be slaughtered was enacted by the British government. Farmers received compensation of 50 percent of the value of the animal, later increased to 100 percent in order to encourage declarations (Schwartz 145). A report examining the possible harms to humans states in February 1989 that the risk of transmission of BSE to humans appears remote and there are no implications for the human health. However the study group also “points out that if their assessment proves incorrect the implications would be serious” (Pennington 160). As a protection measure the British government declared a ban
on all offal, including brain, spinal cord, thymus, tonsils, intestines, and spleen – from the food chain in June 1989 (Schwartz 145).

Both enactments by the government were very leaky in their implementation. In 1988 after the MBM ban about 12,000 new cases appeared in cattle and the number increased even more in 1989. The numbers were a result of feeding existing supplies of MBM not affected by the ban, accidental contamination of feeds, and the continued use of animal proteins by feed mills. The removal of high-risk spinal cord according to the offal ban was not done properly either, especially in the production of mechanically recovered meat for low-grade sausages and burgers. But the risk of contamination was believed to be minimal and no actions were taken. The government confirmed again that eating beef proposed no risk to humans (Pennington 164-165).

After the initial crisis in the UK the disease has spread to several other countries due to imported food supplements and livestock. Especially contaminated feed intended for pigs and poultry is a contributing factor (Brown 7). The countries affected by BSE increased from eight in 1999 to twenty in 2001 (Schwartz 192). According to December 2000 data from the Organization if International Epizootics (Paris) and Ministry of Agriculture, Fisheries, and Food (UK) the total number of reported BSE cases in the UK is 180,376. The numbers in other countries are much smaller – Ireland 499, Portugal 452, Switzerland 363, France 151, Belgium 18 – which shows the magnitude of the crisis in the UK (Brown 8).

From cows to humans – nvCJD

The possible threat of BSE to humans was considered as soon as the disease became known. An article published in the British Medical Journal in June 1988 debated the degree of protection for humans imposed by the species barrier. Experiments with scrapie in the 1960s had shown that the agent could change if passed to a new host. The fear of BSE affecting human health persisted throughout the entire cattle crisis (Schwartz 153-155).

Several early warning signs about a possible species jump to humans were misread. In spring 1990 several domestic cats were diagnosed with spongiform encephalopathy. A short lived panic among the British population emerged due to a possible contamination with BSE agents through food (Schwartz 155). In May 1990 a surveillance unit was established in the UK to monitor CJD hoping to detect any change in the numbers quickly. This surveillance was extended three years later to other European countries, coordinated by the European Union (Brown 8). In March 1993 a British farmer was diagnosed with Creutzfeld-Jakob Disease (CJD). Two following cases raised now further concern. In April 1994 a 15-year old girl showed symptoms of CJD but the diagnosis was never confirmed due to the youth of the patient. In October 1995 two more cases of CJD occurred in a 16-year and an 18-year old. This time the diagnosis was confirmed and led to a closer surveillance of CJD. The alarm bell was finally ringed on April 1996 when ten more cases of CJD with a new profile were identified. A new variant of CJD (nvCJD) was confirmed and linked to the occurrence of BSE (Schwartz 156-158).

The possible relationship to BSE was raised since the first cases only occurred in the UK five to ten years after the period of highest level of food contamination. Experiments conducted showed that the nvCJD agent behaves like the BSE agent which confirmed and proofed the link to BSE. The human disease resulted from the consumption of meat and other related products from BSE-contaminated cattle. Early cases occurring in 1993 and 1995 were identified as unrelated to BSE (Schwartz 158-160).

In order to minimize the risk for NiCad transmission through blood and organ donations, several European governments, including the UK, Switzerland, France, and Germany have implemented policies to exclude blood and organs from people who lived in or visited the UK. But concerns extend beyond that to the safe use of medical and surgical instruments. Paul Brown, Senior
Research Scientist in the Laboratory of Central Nervous System Studies at the National Institute of Health, Bethesda, Maryland, US, suggests the use of disposable instruments and the implementation of a standard sterilization protocol (Brown 11-12).

By the end of 2000 a total of 86 people had died from nvCJD – 82 in the UK, three in France and one in Ireland. In 2001 fifteen more death in the UK raised the total to 101 (Schwartz 194). Predictions about the future of nvCJD are hard to make since it is not clear how people became infected. Predictions based in different incubation times at the end of 2001 talk about a maximum number of 540 to 2600 casualties (Pennington 186). The fact that the incubation period of nvCJD is still unknown contributes much to the uncertainty about the extent of the nvCJD outbreak (Brown 11). The disease is currently untreatable and uniformly fatal. It is also unclear why nvCJD affects predominantly young people (Smith 123, 127).

Early Reactions by the European Union

According to Peter Smith, the BSE epidemic in other European countries was seeded either by cattle exported from the UK that was infected with BSE or by the export of contaminated MBM. Fig. 3, taken from Smith’s article, shows that the peaks of the epidemics occurred at different times in different countries resulting from the impact of the different times that restrictions were put on feed in different European countries and the strictness with which they were enforced (Smith 126).

![Fig. 3 Distribution of cases of bovine spongiform encephalopathy (BSE) in various European countries](image)

As a reaction to the initial appearance of BSE, in July 1989 the European Union (EU) enacted an export ban on all cattle born before July 18, 1988, the date of the UK ruminant feed ban, and on the offspring of confirmed and suspected animals. It was later amended to an export limitation to only calves under six months old. On April 1, 1990 BSE was officially acknowledged by the European Commission as a threat to animal health (Vincent 501-502).

After several European states declared import bans on British beef in May 1990, the EU was pressured to enact an EU-wide ban limiting the export of bone-in beef only from holdings without confirmed BSE cases for 6 years. In 1992 restrictions were placed on the export of bovine embryos. During the peak of the BSE crisis in the UK from 1992 to 1993 no EU regulations concerning BSE
were proposed or enacted. On June 27, 1994 an EU decision prohibited the feeding of mammalian MBM to ruminants, while the export ban were extended and a new cattle identification was required (Vincent 502-503).

After the growing concerns about species jump were confirmed and the first cases of New variant Creutzfeldt-Jakob Disease (nvCJD) were identified, the public mistrust in British meat and other related products increased dramatically in the UK and other European countries. The beef prices declined rapidly. On March 22, 1996 France declared an embargo on the import of beef and live cattle from the UK. On March 26 the European Union reacted on emergency measured by enacting a total ban on all British cattle and products derived from them (Schwartz 158).

The European Food Safety Agency

Dealing with the initial stage of BSE, from 1990 to 1994, has been extremely challenging to the administrative structure of the EU. In many cases this structure failed to deal with it adequately. The failure to acquire timely scientific information on the development of the crisis and to oversee the actions taken by the EU member states, most notably those of the UK, both contributed to the ineffective measures taken (Brown 516-517).

After a number of reports and inquiries about the failure to react, the European Commission took a series of measures to try and rectify the serious problems within administrative structure and obtain accurate scientific data. But this first response to the crisis was just a readjustment of the existing system. But it laid the foundation for the next stage of administrative redevelopment (Brown 509-512).

Following a process of dialogue and debate, the European Commission embarked on a wholesale redevelopment of the administrative landscape in the area of food safety. The result of these changes was the foundation of the European Food Safety Agency (EFSA) in 2002. Although its formal powers are limited, the new agency “is the keystone of European Union (EU) risk assessment regarding food and feed safety” (European Food Safety Authority).

According to the Council Regulation No178/2002, which legally established the EFSA, the agency should be an independent scientific source of advice, information and risk communication in the areas of food and feed safety. While EFSA advises on possible risk related to food safety, the responsibility for risk management lies with the EU institutions. It is the responsibility of the EU institutions to propose and adopt legislation as well as regulatory and control measures based on EFSA’s advice (Council Regulation No178/2002).

Conclusion

The full impact of the BSE epidemic on human health might be impossible to assess. It is likely that millions have been exposed or contaminated but due to the long incubation time predictions are hard to make. As of January 2003, 139 nvCJD cases have been ascertained. BSE itself is no longer a significant public health problem and unlikely to become so in the future (Smith 128-129). The measures that were put in place to control BSE in the UK and elsewhere resulted in a year-to-year decrease of 25-45% in the annual number of cases. The epidemic is under control (Smith 127).


