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Reaction to the emergence of BSE in the UK: what was done and what perhaps might have been done better

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While it was correctly identified early in 1988 that the spread of BSE was due to the practice of feeding cattle with meat and bone meal (MBM) made from animal carcasses, the sensible control measures adopted to stop this practice were not implemented as quickly as might have been possible. The ruminant feed ban was introduced in July 1988, but stocks of contaminated feed continued to be distributed until at least October 1988. As a result, more animals became infected and the epidemic was unnecessarily fuelled. Affected animals were excluded from the human food chain by the compulsory slaughter and destruction measures, but it was not possible to exclude carcasses of animals infected but not yet showing clinical disease. The numbers of these preclinical cases could not be determined and were underestimated in the assessment of risk. In 1988-89 the exclusion of MBM from all animal feed was regarded as a disproportionate measure, and one which would have led to major problems in waste disposal and economic difficulties for the rendering industry. Consequently, MBM continued to be used and exported in commercial feed for pigs and poultry. It was not appreciated until 1991, that cross-contamination at the feed mills could cause further spread of the epidemic, and over 44 000 new cases of BSE were the result. The introduction of the animal SBO ban in 1990 was a measure designed to limit the spread of BSE to pigs and poultry; however, it was largely disregarded. Cross-contamination of cattle feed may have been more important in the spread of BSE abroad, than the export of MBM for pigs and poultry.

The measures taken to halt the BSE epidemic and to protect against the possibility that BSE might transmit to man were sensible measures [1]. However, it was believed that the risk to humans was remote and that the measures to protect human health were ultra-precautionary. This belief was based on the assumption that BSE originated from sheep affected with scrapie, and that as scrapie had never transmitted to humans in 250 years, the risk of BSE transmitting to humans was equally unlikely. Thus the public were repeatedly reassured that it was safe to eat beef, and those charged with implementing the controls were not unduly concerned by an occasional breach of the regulations.

The public were not advised of the increasing uncertainties of the scrapie origin hypothesis from 1988 onwards. The fact that BSE was exclusively a British disease, was explained on the basis that rendering procedures in the U.K. had led to failure of inactivation of the scrapie agent. However, similar rendering systems were used abroad without leading to BSE. It is now known that no rendering procedure has been designed which is capable of completely inactivating TSE's. In 1990 cats, which were resistant to scrapie

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infection, became infected with BSE. In fact the species range of BSE proved to be much wider than with scrapie. Cows infected with scrapie contracted a disease different from BSE. These and other facts about BSE which increasingly threw doubt on the theory that BSE would behave like scrapie were not concealed, but the public were not informed that the risk assessment had changed. The control measures continued to be relied on to protect the public from risk. When variant CJD was announced in March 1996 the public felt betrayed.

With hindsight, it is possible to speculate what might have been done differently which would have limited the spread of BSE. MBM might have been recalled. Herds in which BSE had been detected might have been destroyed. SBO controls might have been more rigorously implemented. Mechanically recovered meat might have been banned in 1989. Had annual surveillance been more efficient the epidemic might have been recognised earlier.

It now seems likely that BSE originated as a novel disease first in Southern England in the 1970's if not earlier. Thus, the agent may have entered the human food chain long before BSE was recognised. It is inevitable that a small number of people were exposed to BSE at first, then in increasing numbers by the 1980's. It seems from animal experiments that a number of genetic loci are involved in susceptibility and resistance to TSE's. As genetic and environmental factors conferring susceptibility remain unknown, as routes of infection are unclear, as incubation periods are uncertain it is not possible to predict with any certainty the numbers of people likely to succumb to vCJD. The management of the disease in the future depends first on the urgent development of a test capable of detecting the agent in patients incubating the disease, and secondly on therapies that can arrest the disease at this stage or prolong the incubation period beyond the natural lifespan of individual carriers.

References

[1] The BSE Inquiry, www.bseinquiry.gov.uk.