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BSE - A statement by the Royal Society

Many people have, rightly, looked to the scientific community for guidance about bovine spongiform encephalopathy and its possible connection with Creutzfeldt-Jakob Disease (CJD). The subject has been of concern to the Royal Society for some time. For example, in 1990 the Society, jointly with the Association of British Science Writers, organised an authoritative briefing for science journalists on the scientific and research aspects of BSE; and in 1993 the Society ran a two-day scientific conference with leading experts from around the world to discuss prion diseases (of which BSE is an example). The Proceedings were published in 1994.

Spongiform encephalopathies (SEs) are found in various forms in a variety of animals. The human form is CJD. The sheep form is scrapie, which is a common and long-standing sheep disease and in which the agent is known not to infect humans. Research on SEs has been undertaken in the UK for several decades, with groups of world class renown now based at several centres, including the BBSRC/MRC Neuropathogenesis Unit at Edinburgh and the Prion Diseases Unit at St Mary's Hospital, London.

The infectious agents of SEs are called prions, and are very unusual in several respects. Their only known component is protein, whereas all other infectious agents contain nucleic acid. They are highly resistant to being inactivated by chemicals, irradiation or heat (which normally inactivate proteins). Moreover, they cannot be grown in a laboratory (which means that they can be studied only by means of animal experiments taking many months). Research on SEs is therefore difficult and time-consuming. Nevertheless, considerable progress has been achieved, by the efforts of research teams in Europe and North America, in defining the molecular nature of the causative agents of SEs and in identifying genetic factors that influence susceptibility to SEs and the epidemiology of the diseases.

BSE in cattle was first seen in 1986. Its appearance appears to stem from changes during the early to mid 1980s in the preparation of protein supplements for cattle feed from rendered sheep and cattle carcasses (including sheep with scrapie), and in the practice of rendering. Previously the carcasses had been processed to produce tallow, bone meal and animal feed, by the use of solvents to remove the tallow and high temperatures to remove the organic solvents. It appears now that this destroyed the infectious agent (the prion) from scrapie. However, driven by economic factors (fall in the price of tallow, increase in solvent and energy costs) and by safety factors (exposure of workers to solvents), the process was changed to avoid solvents and the high temperatures. The scrapie prion survived this new process and subsequently infected cattle, creating an apparently new cattle disease (BSE).

BSE has been found to infect other species fed on infected material, despite the barriers against prions from one species infecting another. It must therefore be possible that BSE could infect humans. Ten cases of an unusual form of CJD in

subjects of no known risk factor have recently been confirmed in the UK, and have rightly given rise to concern that they could be attributed to BSE that has crossed the species barrier.

The risk of exposure of humans to BSE is believed to have been at its height in the late 1980s, before the problem was recognised and before regulations were introduced in 1988 and 1989 to remove infectious material from the animal and human food chains. The risk of infectious material occurring in the human food chain is now believed to be much less, and should be reduced even further by rigorous implementation of regulations to remove infectious material from both animal and human food chains. In the 1950s, an epidemic of kuru in humans (a spongiform encephalopathy associated with the eating and smearing of brains of dead kinsmen in Papua New Guinea) was brought to an end by abolishing the practice.

It is a matter of urgency to carry out further research into how these causative agents operate and how they move from cattle to humans. A particular need is to develop a test for the presence of prions that is as sensitive as bioassay but does not require an animal experiment taking at least eight months.

Other high priority objectives of further research are to establish ways of treating the disease or preventing its development in individuals thought to have been exposed to BSE. We are already aware of several possible approaches, including reducing the extent of expression of the prion-related protein in the host (high levels of expression lead to increased risk of developing the disease), and making agents that prevent the aggregation of the prion-related protein. Further approaches could emerge from the continuing basic research in the general area of prion diseases. Such basic research should therefore be supported fully and urgently.

The present state of detailed scientific knowledge about SEs does not allow us to estimate with any real confidence the degree of risk that the BSE epidemic poses to human health. In due course, perhaps over the next year or so, scientists will be able to give more precise advice. In the meantime, it is important that the scientific understanding we do have of this complex disease and its epidemiology should continue to underpin public policy in dealing with the disease.

In view of the importance, here as elsewhere, of the interplay between scientific understanding and public perception of risk in determining public policy, the Royal Society is now exploring with the Economic and Social Research Council and other Councils the scope for taking forward its work on risk perception and management to the public benefit.

The BSE outbreak illustrates the need to maintain scientific research in areas not of apparently immediate relevance. Much of our understanding (incomplete as it is) of prion diseases such as scrapie, CJD and, now, BSE has come from the work of the Neuropathogenesis Unit of the Institute of Animal Health (IAH) supported by both the Medical Research Council and the Biotechnology and Biological Sciences Research Council over many years, through times when CJD occurred in only a handful of individuals and scrapie-infected sheep were harmless to humans. The IAH and other public sector research establishments are currently being reviewed by the Government for possible privatisation. We doubt whether the private sector could have been expected to support work for which it could see no immediate need or short-term outcome.