Bubonic plague has erupted on a global scale a number of times in the past two millennia, in Europe most dramatically in the Black Death of the fourteenth century. Its impact then was profound for two predominant reasons. It killed between a third and a half of the population and its origins and dissemination were mysteries which persisted for centuries.¹ The impact of bubonic plague was so great as to lead to a second, popular definition of plague — 'an affliction, calamity, evil, a “scourge”'. In other words, a plague is a highly dramatic disaster which combines widespread devastation with mystery as to its origins and spread. This conception of plague remains potent. Folk memories of the great influenza pandemic immediately following the first world war invest it with the title of plague (in Australia at least). More recently, AIDS has reawakened acute anxieties previously stilled by the progress of modern medicine, and has achieved plague status.²

Epizootics can also be plagues. The term ‘rinderpest’, German for ‘cattle plague’, testifies to its devastating impact; the panzootics of rinderpest which swept Europe in the eighteenth century are estimated to have killed 200 million head of cattle. Rinderpest also destroyed millions in southern Africa at the end of the last century, devastating societies in which cattle were the major form of wealth.³ As for its incursion into Great Britain between 1865 and 1867, this was the most dramatic episode in nineteenth-century British agricultural history.⁴ No other single event has had the same impact on public consciousness — until the present epizootic of Mad Cow Disease, of Bovine Spongiform Encephalopathy (BSE). After 130 years, a livestock disease has again become the prime focus of public concern in Britain.

Beyond the scale of the attention they have generated, the two epizootics share a number of features which are characteristic of all plagues, human and animal. The salient features of the saga of mad cow disease are reviewed in the following section, with an emphasis on the similarities between these and the cattle plague, especially the initial responses. There are also significant differences between the two episodes, not least in terms of outcomes. Taken together, these similarities and divergences provide considerable illumination as to why mad cow disease has been such a disaster for all concerned, especially government and the regulatory bodies whose role is to meet the threat.

According to official figures, there were some 160,000 cases of BSE in Britain up to 1996 and a negligible incidence elsewhere. These are low figures by usual plague standards — only half the mortality of the cattle plague in 1865–7 (when the British herd was half its present size of 11,800,000) and insignificant compared to losses from bubonic plague or European rinderpest epizootics. Further, the official peak of the BSE epizootic was in 1992 and 1993, when there were some 35,000 cases annually. This is less than one-half of one per cent of the total British herd and since then the official tally has declined substantially. There were fewer than 15,000 cases in 1995 and the number was expected to fall to some 8000 in 1996.6

But such figures are now irrelevant. No one knows how many animals have had BSE in the past 15 years. There have been consistent claims of underreportage through the plague, while the negligible incidence in Europe has been convincingly queried. Most importantly, the latest estimate is of more than a million cases in Britain, with the peak of the epizootic, 460,000 cases, in 1988.7 Even these figures do not reveal the scale of the disaster represented by BSE, but their variance from official totals serves to emphasize the central reality of its status as a plague. Ten years after BSE was first diagnosed in a dairy cow, mystery and uncertainty continue to surround virtually all aspects of it.

Uncertainty, and therefore controversy, still attend the question of the origin of BSE, its transmission within and between cattle herds, in and outside Britain, and between cattle and other species. Above all, the feature which makes BSE a genuine plague is the probability that it is a zoonosis, that it has crossed the species barrier into humans. While this may not have happened,

---

5 278,943 deaths to rinderpest had been officially notified by 1867, but this was officially acknowledged as a gross understatement; see Parliamentary Papers, 18 (1867–68), Veterinary Department of the Privy Council, Report on the Cattle Plague in Great Britain during the Years 1865, 1866 and 1867, 1868, 218–19.

6 The Economist, 30 April 1996; Ministry of Agriculture, Food and Fisheries (MAFF), Programme to Eradicate BSE in the United Kingdom (London 1996), 6–7.

the weight of evidence points to its implication in a few human deaths already — and the possibility of thousands in the future. This possibility, combined with the horrific nature of the disease and speculation on the extent of dissemination outside Britain, has ensured that BSE has acquired a global rather than merely a British or even European status. Mad Cow Disease has led herdsmen near Samarkand to express sympathy with British tourists over their likely fate; New Zealand butchers display notices emphasizing the disease-free status of their supplies. Modern communication has meant that the economic losses to cattle producers spread faster and more widely than the disease itself.

The obscurity which attends the origins and early dissemination of plagues is inevitably a contributing factor to their ultimate status. It helped ensure that the Black Death was but the first (if also the worst) of a series of periodic outbreaks which afflicted Western Europe until the late seventeenth century and menaced it for another 200 years. The drama of the initial impact of rinderpest in Britain in 1865 was due as much to its novelty as to the high level of deaths it caused. Despite the fact that the disease was endemic in Europe, the general British ignorance about it ensured its rapid early spread. The history of both AIDS and BSE provides abundant testimony that the advance of science has not ended the mystery and uncertainty which attend the advent of new diseases. In the case of BSE, the first official diagnosis of a cow with spongiform encephalopathy came in 1986, while there was later confirmation of the same symptoms on a Kent farm a year earlier. However, livestock producers thought that these were neither the first nor the only cases, a view since confirmed. As John Wilesmith points out, the early cases diagnosed were too spread-out to have originated from a single source. BSE was widely disseminated by 1986 and, given a normal incubation period of between four and five years, its origins lie early in the 1980s if not before.


9 The Economist, 26 October 1996, 18 January 1997; pers. comm. and see, for example, Australian, 18 January 1997, for the harm to cattle interests in BSE-free regions. However, dramatic falls in meat consumption in Japan and Germany may be as much a result of outbreaks of food poisoning due to E coli bacteria. See the Guardian Weekly, 4 August 1996 and Private Eye, 9 August 1996 for the deaths of seven children among thousands affected in each case. Also Private Eye, 11 January 1997 for the British E coli outbreak, the death rate from which is greater than that from the new CJD variant in humans.


There was thus no possibility of an early containment of the threat. In fact, given that, as in the case of bubonic plague, rinderpest or Aids, BSE was 'something completely unknown and quite unexpected', there were problems in establishing what the nature of the threat was. Full diagnosis was and is impossible outside of a post-mortem; some 15 per cent of animals slaughtered on suspicion of having BSE in fact had other ailments. Finally, and of continuing critical importance, the long and variable incubation period makes for difficulties in establishing incidence in the face of the different life-span of stock in dairy, suckler and beef herds.

In these circumstances, the State Veterinary Service (SVS) of the Ministry of Agriculture, Fisheries and Food (MAFF) had made impressive gains in knowledge of BSE by 1988, the date when the disease first became notifiable. In what has been described as ‘an excellent piece of epidemiological detective work’, BSE was pronounced an encephalopathy, probably originating from scrapie, the encephalopathy found in sheep, with the mode of transmission the feeding of contaminated meat and bone meal concentrates (MBMs) to cattle. Although the hypothesis on the scrapie origins of BSE has since been convincingly challenged, that on the mode of transmission has been generally vindicated. BSE is spread overwhelmingly by feeding cattle on the remains of other cattle; it remains the case that ‘action on animal feed is . . . the most effective way of eradicating BSE’. This basic premise underlay policies which have (eventually — after 1993) led to a steep decline in recorded cases of BSE.

Despite its novelty, if BSE had proved a disease strictly confined to cattle then it would not have gained the status of a plague. It will not be eradicated easily; despite the general success of the ban on feeding MBMs, there are still puzzling features to its incidence and spread among cattle. The extent to which vertical or lateral transmission occur has been vigorously disputed; inheritance became a vexed question when cases of BSE in calves born after the feed ban first appeared. Although a number of studies found no evidence for maternal

---

13 J.W. Wilesmith in Veterinary Record, 27 August 1996.
14 The claimed test of the virologist, Harash Nerang, has never been successfully replicated; The Guardian, 23 March 1996. Other tests proposed offer partial but not fully-confirmed diagnoses; The Times, 25 February 1997.
15 MAFF, Programme to Eradicate BSE, op. cit., 3.
18 MAFF, Programme to Eradicate BSE, op. cit., 6.
transmission, this is now conceded as a possibility (if at low levels). As for horizontal transmission, the SVS argues that this would be inconsistent with the high number of isolated cases of BSE, but other European countries act on the assumption that it can happen.19

An even greater puzzle is the limited incidence of BSE outside Britain. Apart from Switzerland, there have been very few cases reported in European countries, even those which imported large quantities of contaminated MBMs and live cattle from Britain in the 1980s when, as is now realized, BSE incidence was at its peak.20 These imports have been a growing source of strain between Britain and its European Union partners (especially when the French discovered that they had been supplied MBMs banned in Britain21), but the real concern has centred on the export of British beef (among other cattle produce), reflecting concern over the possible transmission to humans. In this respect, increases in knowledge have played a seemingly perverse role. Not much was known about spongiform encephalopathies (SEs) before the onset of BSE. Much more is known now, but this has accentuated rather than assuaged the uncertainties which promote fear and tension.

Encephalopathies are a very special type of disease. They present scientists with fascinating puzzles, many of which show no immediate sign of being unravelled. The causative agent, for example, is unknown. By 1993, it had come to be generally accepted that protein particles, containing no DNA or RNA, 'that have some of the characteristics of a slow virus', were intimately involved in such diseases. These are now referred to as prions, but whether they are cause or effect is still uncertain — the latest research findings suggest the latter. Stanley Prusiner's work may have been a major scientific breakthrough but, even given later research on how prions become malignant,22 this has no immediate relevance to the control of BSE or other SEs.

Observation and experiment have mainly served to demonstrate the variability of the transmissibility of SEs within and between different species. Beyond the 20 strains of scrapie, there are at least three affecting humans.


20 B.E.C. Schreuder and O.C. Straub, 'BSE: A European Problem', Veterinary Record, 138 (1996), 575; Dealler, op. cit.. There are strong suspicions of evasion and under-reportage while sporadic cases of BSE, the origins of which have been hard to establish, have since appeared in Germany and the Netherlands to general consternation: Farmers' Weekly, 7, 14 and 28 February 1997.

21 Le Monde, 13 June 1996.

Only BSE has appeared in British cattle, although American veterinarians have speculated on the incidence of a mild form of SE in their stock. The vertical (maternal) and lateral transmissibility of some strains of scrapie is known, while about 15 per cent of cases in humans ‘are thought to result from inherited mutations in the gene implicated in Creutzfeldt Jakob disease’ (CJD). It was already known before BSE appeared that some SEs, including scrapie (but with no known case of transmission to humans), could cross the species barrier.\textsuperscript{23} Enhancing knowledge on this score provided a rich research agenda but no common pattern has emerged.

As for transmission to humans, attempts of some ingenuity have been made to circumvent the unacceptability of direct experimentation. Ominously, intracerebral injection of BSE into macaques produced brain lesions similar to those in the human victims of a variant of CJD. John Collinge began his experiments on mice with introduced human genetic material in 1995 and, early in 1996, these had still not succumbed to BSE.\textsuperscript{24} However, any possible heartening effect on public opinion was swamped by the dramatic announcements of the most significant addition to knowledge on transmissibility since the first official diagnosis of BSE itself.

In March 1996, a team of investigators ascribed the deaths of eight young people to ‘a new variant of Creutzfeldt Jakob Disease in the UK’.\textsuperscript{25} The eight exhibited clinical symptoms which differed from CJD; they were more akin to kuru (an encephalopathy found in Papua New Guinea) or, it has since become clear, to BSE.\textsuperscript{26} Confronted with this evidence, the Spongiform Encephalopathy Advisory Council (SEAC) advised the Ministry of Health that, although there was no direct evidence, ‘the most likely explanation, in the absence of any credible alternative, was that the cases were linked to exposure to BSE’ before MBMs were banned in 1989. The health minister duly informed parliament of SEAC’s findings.

The response to the ministerial statement confirmed the coming of age of BSE as a plague. The public sense of unease which had been simmering — and flaring regularly — for several years now erupted in hysteria. Beef sales fell precipitously, and not only in Britain. Cattle production and marketing were thrown into chaos; the already delicate relationship between Britain and its partners in the European Union was brought under further strain when a total

\begin{thebibliography}{99}
\bibitem{collinge} \textit{Veterinary Record}, 6 January 1996. Nor did they later: \textit{Daily Telegraph}, 12 October 1996; \textit{Nature}, 6 June and 25 October 1996. See Collinge’s interview on the difficulties but not the impossibility of crossing the species barrier. For a suggested extensive research programme utilizing chimpanzees (as being genetically more akin to humans), see \textit{Nature}, 15 August 1996.
\bibitem{collinge2} Collinge, \textit{op. cit.}, 685–90; \textit{Veterinary Record}, 30 March 1996.
\end{thebibliography}
ban on the export of British cattle and beef was instituted. Finally, inexorably, a search already in place for culprits, for those to blame for the disaster, was reinvigorated.

A striking characteristic of the historical reaction to plagues has been the pervasive belief that they are a form of retribution exacted for the sins or failings of society and that these sins or failings can be expiated through the punishment of offenders. The logic seems clear enough: 'blaming has always been a means to make mysterious and devastating disease comprehensible and therefore possibly controllable'. What is variable is the choice of culprits or scapegoats.

Society as a whole may be to blame, as the Archbishop of Canterbury presumably felt when he prescribed a 'National Day of humiliation' as the appropriate response to the cattle plague in 1865. Social deviants have always been popular as scapegoats: the Jews in the Black Death, homosexuals and drug-users in the Aids epidemic. Conversely, as the state took on the responsibility for the execution of policies designed to contain or eradicate plagues, it and its agencies became a logical target in the case of perceived failure. During the British cattle plague, the inaction of the Liberal government became the focus of increasingly fierce criticism as losses to rinderpest mounted. The criticism was unwarranted; the administration had attempted to institute measures against disease in the previous year only to be rebuffed by producer interests concerned about the disruption of trade. That producers and traders were also the main agents responsible for the rapid dissemination of rinderpest did not stop them venting their fears and frustrations.

The government then was accused of neglecting the interests of producers; in the BSE saga the charge has been reversed. From as early as 1990, it has been contended and widely accepted that the Conservative government, and especially MAFF, have employed suppresio veri and suggestio falsi in order to downplay the scale of the BSE epizootic and the risk of transmission to humans in order to protect the interest of cattle producers. 'BSE might be a slight risk to human health (but) it is a far greater risk to MAFF's credibility.' By 1992, as concern over transmission to humans grew, 'Old Muckspreader'

---

28 MAFF, Animal Health: A Centenary, 1865–1965 (London 1965), 134. Canon Girdlestone argued that the plague was retribution on farmers for the treatment of their labourers: Orwin and Whetham, op. cit., 222.
in *Private Eye* thought that ‘MAFF mandarins seem more concerned to protect animals than people.’ The pursuit took on a new stridency after the March panic. Two British historians found the origins of MAFF behaviour in its history: it had always ‘minimised the human health risk (of livestock disease) for fear of panic about one of the farming industry’s most important products’. Scares like BSE ‘will continue until the administrative ethos of the Ministry of Agriculture, which frankly seems to have changed very little over the last 100 years, can be fundamentally readjusted to be more responsive to consumer needs’.

If the specious nature of public statements was their only fault, then such accusations might not need to be taken too seriously. Governments and their agencies have an evident and natural interest in minimizing public alarm in conditions of uncertainty, but this does not necessarily correspond to deliberate misinformation. As noted earlier, the initial investigations into BSE were highly successful, regulations were progressively strengthened as more became known about the disease and MAFF’s principal control measure worked. Unfortunately, however, MAFF’s record on containing the possible spread of the disease to humans, what really mattered as far as national and international opinion was concerned, was grossly deficient.

In 1993, in the course of a periodic bout of concern over BSE, an editorial in *Nature* accepted that ‘the raw materials of cattle feed are now tightly controlled, while there are rigorous inspections of meat sent for sale’. It was wrong. MAFF failed to gain or enforce compliance with its regulations in virtually every area of concern. With compensation payments often inadequate, notification of BSE has been haphazard and the export certification system (given a substantial price premium for cattle exports) has been rife with evasion. The situation in feedmills and abattoirs, on the basis of evidence from MAFF itself, was even worse. An audit in 1994 could not account for half of the specified bovine offal (SBO) from slaughtered cattle. The amounts involved were massive and the Assistant Chief Veterinary Officer conceded that most would have gone into animal feed. According to John Wilesmith and Danny Matthews, cross-contamination from this source probably accounted for half of the BSE cases in stock born after the original feed ban. As for abattoirs and slaughterhouses, surveillance by the SVS up to March 1995 found that nearly half were not complying fully with regulations, with 34 per cent still imperfect two months later. Claims for the perfect

---


35 For various cases, see Dealler, op. cit.; *Private Eye*, 13 August 1993, 16 December 1994; *The Sunday Times*, 24 March 1996.

36 *Farmers’ Weekly*, 5 April and 19 August 1996; MAFF, *Bovine Spongiform Encephalopathy*
safety of British beef were nonsense. The gap between regulation and actual practice was glaring.

This is hardly surprising. The fundamental basis of public and animal health programmes has always been the principle of isolation. This is simple enough to state but hard to realize: measures such as quarantine and *cordons sanitaires* have always encountered the constant of human frailty. There are strong incentives which make for the evasion of regulation; in the case of animal health these are primarily economic, involving trade as well as farm production. The importance of compensation payments to ensure compliance with regulation, for example, has been acknowledged since the time of the first efforts to control rinderpest. Unfortunately, cattle are not a homogenous good; market values fluctuate, often wildly, over time. Inadequate payments may mean poor compliance but, conversely, compensation can be over-generous, leading to perverse effects and raising the question of who should bear the cost — issues which worried W.E. Gladstone and John Stuart Mill during the cattle plague.37

Even when compliance is high, regulation requires effective monitoring. When compliance is poor, policing is difficult and puts pressure on limited public resources. In 1865, the early regulations against rinderpest were flouted with impunity. Even when stockowners had become convinced of the necessity of stamping out rinderpest, compliance remained far from perfect, ensuring that the epizootic was not eradicated until 1867. In fact, throughout its history, a major problem for the SVS — the descendant of the Veterinary Department hastily established in 1865 — has been finding the resources necessary to ensure effective compliance.38 During the BSE saga, this problem has been exacerbated by growing fiscal stringency in the public sector (a trend reinforced by ideologies reminiscent of the laissez-faire dominant at the time of the cattle plague), and the need to respond to heightened concerns over food safety generally. The inception of a new centralized Meat Hygiene Service (MHS) in 1995 was a response to such concerns but, in the short run at least, the change was counter-productive. A year after its birth, an MHS operations director conceded that, given the attendant structural problems, the proper monitoring of meatworks remained virtually impossible.39

Human frailty has thus been only too evident in the BSE saga. As knowledge

---

37 Dunlop and Williams, op. cit., 218; *Hansard*, CLXXXI (14 and 16 February 1866), 488–92, 609–17. See also *The Times*, 23 January 1866, for Gladstone’s proposals for a stockowners’ fund to pay compensation.


39 *Veterinary Record*, 29 June 1995; *Farmers’ Weekly*, 5 April 1996. Responses to the same imperatives have led to the restructuring of meat inspection services throughout Western Europe, North America and Australasia.
of regulatory inadequacies has grown, it has reinforced scepticism and suspicion of government and government agencies to the point where they have come to be considered the cause of the problem rather than merely an exacerbating factor. Nevertheless, and despite their demonstrated inadequacies, it remains the case that BSE was prevalent before any regulations could realistically have been put in place. While some containment policies could have been mounted earlier and more effectively, this was hardly warranted by the existing state of knowledge and would have been at the expense of other programmes — at a time when resources were inadequate to meet the continued spread of sheep scab and various crises over salmonella.\textsuperscript{40} Even then, subsequent policy defects were of relatively marginal importance in the light of the quantities of potentially contaminated cattle produce which would have entered human food supplies before 1988. The extent to which this point has been overshadowed by the continuing diatribes against government and government agencies points to a further theme which has accentuated the status of BSE as a modern plague.

Mad cow disease is not just frightening and mysterious but possibly a symptom of a deeper malaise. It is one of a range of new diseases, including Aids, the Eboli virus and others, which are suspected of being a result of the impact of modern material \textquoteleft progress\textquoteright on the natural world.\textsuperscript{41} BSE has a special status among these in part because of its mystery, in part because of the greater directness of its connection to this \textquoteleft progress\textquoteright — and a particularly repugnant form at that. When the Prince of Wales asserted that \textquoteleft BSE was just one example of how society, and farmers, had been \textquoteleft paying twice\textquoteright for cheap food\textquoteright, he was echoing a widely-held view that intensive agriculture is practised at the expense of the well-being of the animal, plant and soil resources involved. For Alexander Cockburn it came

\textldots as a sour irony that it\textquotesingle s taken the very rare mad cow disease\ldots to raise the alarm about the consequences of intensive meat and milk production. After all, over the past 150 years such production has destroyed much of the world\textquotesingle s ecological balance and impoverished or otherwise ruined millions of lives.\textsuperscript{42}

It matters not that many forms of traditional farming can be destructive or that cattle have long been fed on various materials including the remains of other cattle.\textsuperscript{43} Knowledge of the probable cause of BSE touched a deep nerve in

\textsuperscript{40} Dealler, op. cit.; Veterinary Record, 4 and 18 November 1995.
\textsuperscript{41} Garrett, op. cit., passim; Dennis Pirages, \textquoteleft Microsecurity: Disease Organisms and Human Well-Being\textquoteright, Washington Quarterly, CCCLXXIX (1995) 7–12; Arno Karlen, Plagues and Progress (London 1996). But see also Sergio Budiansky, \textquoteleft Plague Fictions\textquoteright, New Scientist, 2 December 1995 and Raymond Tallis, \textquoteleft Apocalypse Now?\textquoteright, Times Literary Supplement, April 1996.
\textsuperscript{42} Daily Telegraph, 28 September 1996; Alexander Cockburn, \textquoteleft Who\textquotesingle s Mad Now?,\textquoteright New Statesman, 29 March 1996; see also Cockburn, \textquoteleft A Short Meat-Orientated History of the World: From Eden to the Marrole\textquoteright, New Left Review, 215 (1996), 16–42.
\textsuperscript{43} Paul Cohen, \textquoteleft Old England\textquotesingle s Bitter Harvest\textquoteright, New Scientist, 149 (1995), 15; Ford, op. cit., 79–91.
society: it came ‘as a shock to many — particularly city-dwellers — that some of the animals we eat are fed other animals’. Mad cow disease showed what happened ‘when humans tamper too much with the food supply’ and has become a symbol of all that is wrong in modern life. As John Grey put it, ‘a society in which the feeding of cow-remains to other cows can pass almost unnoticed is one in which the very idea of nature is all but dead’. The theme of retribution again looms large. Just as the Black Death embodied divine anger at society’s deviations from the way of the Lord, so BSE is a punishment for the sins of modern society, for its wilful disruption and destruction of nature.

Another group of culprits can be identified through this approach. The roots of the problem may lie in the capitalist system or, more simply, individual and collective human selfishness, but the means to wreck nature have been provided by science-based technology. And while scientists like Richard Dawkins might consciously accept their modern status as priests, the BSE saga provides a reminder of the impact of the Black Death on the medieval Church. As Grey argues: ‘People turn to science for what religion can no longer supply — a sense of certainty.’ But this certainty ‘is, of course, an illusion’, as BSE has so amply demonstrated. Mad cow disease is just one of the ‘vast unsupervised experiments, whose risks we can know, if at all, only retrospectively’. The moral is, of course, in the words of a respondent to Grey, ‘to resist the scientific onslaught on nature’.

The scientific community itself has responded variously to the fact that ‘BSE has damaged the credibility of science as well as of politicians’. Naturally enough, it has joined in the onslaught on government: ‘Through greed, expedience, and ignorance of science we have liberated the ancient cannibals’ plague [an allusion to the causes of kuru] in Western society.’ ‘The BSE episode has been shamefully mismanaged’, and the columns of the Veterinary Record and of Nature have been full of complaints about government under-funding and a selective control of information which threatens to erode the ‘public guarantee of reliable, disinterested knowledge’, which scientists have always sought to maintain.

At the same time, scientists have also identified the root cause of the specific problem of BSE: ‘Presently, science simply does not know the truth.’ The irony of the coincidence of the March 1996 panic with National Science Week, dedicated as this was to bridging the gap between scientists and laity, was not lost on participants. It was evident that the public still did not

---

46 Ford, op. cit., 13, 193 and 199.
appreciate that there is not a ‘universally-applicable answer to every problem’. British veterinarians, especially, arguing that ‘science must reassert itself’ in response to the BSE crisis, have been mortified at the institution of policies, such as the cull of older cattle and the European export ban on British beef, for which they could see no scientific justification. In imposing the ban, the European Union’s Standing Veterinary Committee, ‘the highest point of decision-making for veterinary science’, the members of which were supposed to be ‘scientists not political puppets’, had connived in ‘the needless destruction of animals’.

The cattle cull and the export ban were gestures designed to appease public anxieties in the wake of the March panic and in themselves were a typical response to the traumas induced by a plague. These gestures have been costly for government and farmers, and the cause of widespread disruption to livestock production and of exacerbated tensions within the European Union. Scientists, however, might find some comfort in an equally typical response. One consequence of the cattle plague was the granting of the most substantial public funds for medical research in the nineteenth century, while the furore over BSE has led to a major allocation for research into encephalopathies. The comfort might be lessened by the extent to which the allocation came at the expense of other research, but the response also points to another potentially positive outcome.

The animus against science and science-based technology evident during the BSE saga is ultimately sterile. A return to a pre-scientific society is unrealistic: apart from the unattractive nature of the ‘sickness culture’ then an integral feature of society, the associated costs are not worth contemplating. Economic development is a treadmill which the human race can get off only at an unacceptable price. Whether scientific advance is a process which can be consciously controlled is another question, but it is evident that the mysteries and uncertainties of mad cow disease can ultimately only be resolved by scientific investigation and research.

The cattle plague of 1865–7 was eradicated in a campaign which provided an outstanding validation of the principles of isolation and ‘stamping-out’ in checking an infectious disease. It was the more impressive because, coming immediately before the advent of ‘germ theory’, the control programme faced a host of major constraints: the regulatory machinery necessary was put together

48 New Scientist, 30 March 1996.
50 See, for example, Farmers’ Weekly, 14, 21 and 28 February 1997.
from scratch, its personnel from the incipient veterinary profession were incompetent and ignorant of rinderpest (and of cattle diseases generally),\textsuperscript{54} while compliance from stockowners and traders was far from perfect. These factors helped to prolong the period before rinderpest was finally eradicated in 1867 but, given the appropriateness of the measures adopted, otherwise proved incidental.

Ultimately, the control programme was successful because the ignorance which marked the onset of rinderpest had been overcome. This ignorance was a product of British insularity, of a refusal to listen to those few veterinarians who knew something of rinderpest, how it spread and how it could be contained. The cattle plague raged while the lessons of European (and even previous British\textsuperscript{55}) experience were ignored. British doctors experimented with cures; British stockowners refused to comply with measures constraining cattle movement. However, as losses soared, the futility of such responses led to recognition of the necessity of the drastic measures embodied in a stamping-out programme.\textsuperscript{56}

The cattle plague and the nature of its eradication had major positive consequences for public human as well as animal health in Britain. The inappropriateness of a laissez-faire ideology in the face of epidemic disease was forcibly demonstrated. Public action against disease was in the interests of consumers and producers alike and a permanent regulatory apparatus was established, although still in the face of a hostile British Treasury. Despite such difficulties and an initially narrow perspective on the role of science, the historical record of the veterinary authority which became the SVS in eradicating and controlling epidemic animal disease and in improving food hygiene has been impressive.\textsuperscript{57}

The contrast to the contemporary position of the SVS is striking. The policies it has mounted against BSE are founded on the same principles that were successful in the past and have proved reasonably effective in the present. The staff of the SVS are well-trained professionals able to draw on a wealth of knowledge from domestic and international sources. Yet, never has a policy of disease-containment been faced with the level of opprobrium lavished on the British government, MAFF and the SVS alike, in the case of BSE. The crucial difference between the two episodes lies in the fact that uncertainty and mystery were only temporary in the case of the cattle plague while they have been continuing features of the BSE saga. They are also likely to persist and it


may even be, as with rinderpest in Western Europe, that the disease will be effectively contained before it is fully understood.

BSE is the first livestock disease known to have originated in Britain. Mystery and uncertainty militated against effective action before the disease became widespread and have been significant constraints ever since. More effective preventative action could have been taken by the SVS after 1988 and the first notification of a new disease. However, the resources necessary to this end were hardly warranted in the context of the information available and would have been used at the expense of other desirable ends. Unfortunately, prescience is a scarce commodity in or outside of plagues. It has been absent in the saga of mad cow disease and the results can hardly come as a surprise to anyone familiar with the history of past plagues.

John Fisher

is a Senior Lecturer in the Economics Department of the University of Newcastle, New South Wales, Australia. He has published numerous articles on agricultural and veterinary history in Britain and Australia, and recently collaborated with Professor J.A. Perkins of the University of New South Wales to examine the implications of mad cow disease for the European Union.