Farm Disease Crises in the United Kingdom: Lessons to Be Learned

10
CHAPTER

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Introduction

Over the last fifteen years, a diverse succession of disease-related crises has befallen farm animal and food industries in the United Kingdom. Some have involved animal health, with little risk to humans. Some have involved human health, with animals acting as a reservoir for infection but little affected themselves. Some, however—including the most alarming—have involved both animal and human health through zoonoses, diseases transmittable from animal to human. All of these crises are linked in the public mind and in many commentaries, and indeed there are issues that many of them share. The most common of these is concern for food safety, but concern for animal welfare is also a recurrent theme. These concerns play out against a backdrop of:

- Prevalent attitudes, including complacency, about issues of biosecurity, from animal health to food safety;
- Relevant legislation, law enforcement, and law breaking;
- The practices of transporting animal feed, animals, and animal products; and
- An emphasis on the economics of animal production to the exclusion of all other considerations.

Few of these crises have been limited to the United Kingdom, but the problems do seem to have been worse and more frequent there.

Salmonella in Eggs

In 1988 a government minister's statement that "most egg production in the United Kingdom is infected by salmonella" received wide publicity (BBC 1999). The statement proved to be true, and it was confirmed that salmonella bacteria from eggs cause food poisoning. Egg sales fell by about half. To some extent this was a "non-story," since salmonella had not recently increased, the number of eggs affected was small, and the health risk for consumers was relatively minor. (Healthy adults are unlikely to suffer more than a stomach upset even from raw, infected eggs, although children and old people may be more seriously affected. Cooking infected eggs kills the bacteria.) However, the scare demonstrated clearly that public expectations of food safety had increased, and understandably so, since an earlier time when occasional food poisoning was routine.

Fifteen years later, the food industry is only beginning to grapple with this heightened expectation. The industry's complacency has been hard to shake off and, although perhaps ill-founded, this could be justified at least partially by the fact that the poultry sector has led other agricultural sectors in health care for its animals. Because of the susceptibility of chickens and other poultry to disease, in the years following World War II the industry developed a positive approach to health control, including farm design, hygiene, preventative vaccination, and general management (Julian 1995). Perhaps the most important element of general management has been the "all in, all out" approach in which houses are emptied completely between one flock's departure and the next's arrival. The ability to clean facilities thoroughly between flocks and to reduce transmission of disease vectors from old to young animals, has

made a paramount contribution to poultry health. It also underscores the irony of the 1988 scare's relationship to poultry products rather than to others more vulnerable. (The approach is only now, many years later, being adopted by other agricultural sectors such as pig farming.)

Despite all this, the industry has not eliminated salmonella in eggs, in part, perhaps, because there are many different types of bacteria involved. The consequences for poultry are variable (Curtis 1990); mortality ranges from 1 to 75 percent (Sainsbury 2000) but is usually low. By contrast, "a few areas of the world, notably Scandinavia, have been able to virtually rid themselves of salmonella infections in animals" (113).

The furor in the United Kingdom provoked an extensive response from the government, including three orders introduced in 1989. The Zoonoses Order required registration of flocks, regular testing for salmonella, and stringent measures if it was detected. The Processed Animal Protein Order and the Importation of Processed Animal Protein Order required salmonella testing of such protein, intended for feeding animals—although it is notable that these orders did not identify the sort of problem that was even then exacerbating Bovine Spongiform Encephalopathy, or BSE (see the next section). The promise of such measures, and their implementation, reassured the public. Egg sales slowly recovered. However, salmonella in eggs still has not been eliminated in the United Kingdom and in most of the European Union (EU). Sainsbury (2000, 116) suggests that even if it were, this status would be difficult to maintain. He comments as follows about the lack of effective government policy to reduce salmonellosis and other zoonoses:

> Our poultry . . . will always be vulnerable to the introduction and re-infection from other sources, such as wildlife, over which we have no control, the whole environment around them and above all from man himself . . . . Also, animal products, including poultry, enter the United Kingdom from European Union sources in an almost unrestricted way, and several EU countries have no salmonella control programmes. Poultry products are currently entering the United Kingdom from other parts of the world where salmonella control programmes are absent. Thus, with people and animal products constantly putting our birds at risk, the United Kingdom government's policy is, at the least, worrying.

By contrast, Pennington (BBC 1999)—professor of bacteriology at the University of Aberdeen and well known due to the *E. coli* inquiry (see page 153)—suggests that salmonella should not still be a problem:

> I don't know why we have a problem with salmonella still, probably because we have not worked hard enough to try to get rid of it. We shouldn't have a problem with salmonella. We know how to sort it out. Other people have sorted it out, we should have had it sorted out long ago.

In 2001 the EU Commission announced that it would target salmonella poisoning as a number one priority in a food safety crackdown, bringing in new controls affecting producers of breeding poultry, laying hens, broilers, turkeys, and pigs over the next eight years (Meade 2001).

In the United States, about 40,000 cases of salmonellosis are reported annually (more probably go unreported), and about 1,000 deaths (Marler Clark, L.L.P., P.S. 2001). Evidence that a significant number of these cases derives from animal products is circumstantial, but convincing, given the extent of infection in poultry and other units (Altekruse, Cohen, and Swerdlow 1997):

> The doubling of salmonellosis incidence in the last two decades has accompanied modern food industries' centralized production and large-scale distribution. . . . The trend toward larger markets and consolidation of industry has exacerbated the *Salmonella enteritidis* problem in another way. Changes in egg production have adversely affected infection control in poultry flocks. In 1945, a typical hen house contained 500 birds. By 1995, many houses contained 100,000 hens, and multiple houses were often linked by common machinery, resulting in large flocks with common risk profiles. Large-scale distribution of shell eggs from infected flocks has caused outbreaks in which contaminated eggs were distributed in many states over a period of months.

Bovine Spongiform Encephalopathy

As its name indicates, BSE—a disease discovered in 1986—causes the brains of cattle to become spongy. The resulting behavior, such as staggering, has given rise to BSE's common name, "mad cow disease." After 1986 the incidence of BSE increased gradually and then rapidly, peaking in 1992 at more than 3,000 cases per month in the United Kingdom. Early on, there was concern that BSE might be transmittable to humans, and in March 1996 it was confirmed that such is probably the case. Eating infected animal material is the likely cause of a new variant of Creutzfeldt-Jakob disease (vCJD), which has similar physical symptoms in humans to those of BSE in cattle. As of 2000 about 115 people had died from vCJD, mostly in the United Kingdom, according to an official inquiry set up in 1997 and chaired by Lord Phillips (Phillips, Bridgeman, and Ferguson-Smith 2000).

BSE must have become established in U.K. cattle in the 1970s. Its origin is not known, but a major possibility is that it arose from a similar disease in sheep called scrapie (Horn et al. 2001). Other sources suggested include a mutation in a single cow, zoo antelopes (Phillips, Bridgeman, and Ferguson-Smith 2000), and U.S.

cattle (H.W. Reid, Moredun Research Institute, personal communication, June 27, 2001). The latter idea stems from the fact that U.S. mink have had outbreaks of a similar disease since the 1940s, of which beef is a likely source (Phillips, Bridgeman, and Ferguson-Smith 2000).

It quickly became clear that the disease was spread by feeding cattle meat and bone meal (MBM) from animal carcasses. The biological agent that causes scrapie may have changed to make it infective in cattle. Alternatively or in addition, new management practices introduced in the 1970s and 1980s probably increased infectivity; these included changes in how MBM was produced, and increased feeding of MBM to young calves (Horn et al. 2001).

In 1988 the Ministry of Agriculture, Fisheries, and Food (MAFF) introduced a ban on feeding ruminant protein to ruminants. This was followed in 1990 by a ban on using material from cattle tissue most likely to be infective (brain, spinal cord, and intestines) for human food, and another in 1991 on using such material for feeding pigs and poultry. Unfortunately, implementation of these bans was made less effective by two mistaken assumptions. First, it was thought that the infection was coming directly from sheep. In fact, whatever its original source, the infection now was being spread by MBM from infected cattle being fed to other cattle. This had a self-amplifying effect that was not understood for several more years: as more animals were infected, so the rate of infection accelerated. The assumption that scrapie was responsible also inappropriately lessened the urgency of measures to protect human health, as scrapie is not transmittable to humans. Second, it was thought that infection was possible only if a large amount of infective material was eaten, whereas it proved that as little as one gram was needed. Perhaps because of this assumption, farmers and feed suppliers were relaxed about continuing, illegally, to use existing stocks of MBM for their cattle and to export MBM around the world. There also was contamination of cattle feed from that prepared for pigs and poultry (Phillips, Bridgeman, and Ferguson-Smith 2000).

The result of the mistaken assumptions was that the disease became much more widespread than it otherwise might have. As of 2002 there had been more than 180,000 cases of BSE in the United Kingdom. In the rest of Europe, more than 3,000 cases had been reported; presumably these started from MBM from the United Kingdom, but recycling of infected MBM also is likely to have been a problem within those European countries. There also had been a small number of cases in non-European countries; these involved imported animals.

One reason it was difficult to understand—and therefore control—BSE more quickly is that the disease has a long incubation period; it takes four to six years before infected cattle show symptoms. Most have been slaughtered before then. For a long time, it was mistakenly believed that animals not showing symptoms could not infect others. Indeed, confidence that beef was safe to eat meant that for some time even animals slaughtered because they were showing symptoms were used for human food. The incubation period in humans may be ten or more years.

With increased knowledge of BSE, including its probable transmission to humans, a complete ban was imposed in the United Kingdom in March 1996 on use of all mammalian MBM in farm animal feed. Even this ban could be described as conservative, and indeed there is a lack of clarity on exactly what has been banned. Phillips, Bridgeman, and Ferguson-Smith (2000) get it wrong in their summary (vol. 1, 66):

> [Previous measures] were replaced after 20 March 1996 by the radical step of banning the incorporation of all animal protein in animal feed.

Phillips, Bridgeman, and Ferguson-Smith are suggesting that, despite all that had happened up to that point, the ban still seemed radical. That certainly is not true now, especially since the ban includes not "all animal protein" but only mammalian MBM. They make this clear elsewhere, but this still leaves room for confusion. It remains legal, for example, to feed poultry protein to animals, including poultry. Even as of 2002 a complete ban on intra-species recycling was only being considered, not pressed.

Whether or not the ban was radical when it was introduced, MAFF resisted it for a long time. It seems bizarre now, but this resistance came in the face of proposals from feed manufacturers, as represented by the U.K. Agricultural Supply Trade Association (UKASTA) (Phillips, Bridgeman, and Ferguson-Smith 2000):

> MAFF was concerned not to do anything that would lead UKASTA members to cease using animal protein as an ingredient of feed for non-ruminant animals. UKASTA, for its part, was anxious that its members should be able to continue to do this without incurring risk of prosecution should it result, on occasion, in cross-contamination of ruminant feed. UKASTA was to threaten repeatedly that it might have to advise its members to cease using animal protein, while MAFF officials sought to allay UKASTA's anxieties. (vol. 1, 63)

No cases of BSE had been diagnosed in the United States as of mid-2003. Importation of ruminants and ruminant by-products from countries with BSE have been banned for some years, and in 1997 the U.S. Food and Drug Administration banned the use of mammalian carcasses in the production of feed for ruminants. A study commissioned by the U.S. Department of Agriculture concluded that risk of BSE in the United States is low (Harvard Center for Risk Analysis 2001), but some commentators argue that there is much too little surveillance, especially as compliance with feed rules is known to be weak (Newman 2001).

At the height of the BSE epidemic there were at least four serious public concerns in addition to the obvious

worries about possible effects on human health and the farming industry. First was a view that MAFF was divided in its loyalties on the BSE issue and could not be trusted to defend consumers' interests as well as those of farmers. Second, there was a perception that the government had concealed the truth about the risk to humans. Phillips, Bridgeman, and Ferguson-Smith (2000) reject both of these charges. Nevertheless, they acknowledge (vol. 1, xviii) that "confidence in government pronouncements about risk was a further casualty of BSE." One consequence of this (in combination with other crises, particularly the outbreak of *E. coli* discussed next) was the establishment in 2000 of an independent Food Standards Agency. Another was redistribution of MAFF's responsibilities after the 2001 general election and its replacement with a Department of Environment, Food, and Rural Affairs.

The third public concern had to do with the effects of the disease on the cattle themselves. There has been surprisingly little discussion of the actual impact of BSE on cattle welfare. However, Phillips, Bridgeman, and Ferguson-Smith (2000) make it clear that vCJD is very unpleasant for humans (Table 1), and it may be presumed that BSE in cows has at least some similar mental as well as physical effects. Furthermore, media coverage suggested that concern for the animals involved was not limited to considerations of suffering. It also expressed that it is wrong for animals to have a disease, especially one seen as avoidable, irrespective of its mental effects, and that killing of animals (including healthy herd mates of cows with BSE) is of serious ethical concern. The concern about killing may be based in part on the fact that the animals are not being used for meat or other purposes. It also is possible that the concern simply came to the fore because the killing was brought to public attention. These ideas will be discussed again below, in the context of foot and mouth disease.

The fourth and most fundamental concern was that the whole process of forcing herbivores to eat animal protein, of making cows into cannibals, was an unnatural practice. This may be partly a concern for the cows, partly a feeling that treating animals unnaturally is wrong in itself, and partly an opinion that it should have been obvious that such a practice would lead to disaster. Phillips, Bridgeman, and Ferguson-Smith (2000) reject this last point:

> The practice of feeding MBM to animals in the United Kingdom dates back at least to 1926. . . . It is a practice which has also been followed in many other countries. It was recognised that it was important that the rendering process should inactivate conventional pathogens. Experience had not suggested that the practice involved any other risks. In these circumstances we can understand why no one foresaw that the practice of feeding ruminant protein to ruminants might give rise to a disaster such as the BSE epidemic. (vol. 1, 20)

If producers were going to give cattle supplementary protein to boost their productivity, then perhaps it was not unreasonable for them to use animal protein that was readily available and had the right mix of nutrients. However, the practice now can be seen to be part of a general approach to animal agriculture, common particularly in the second half of the twentieth century, that pushed for increased production at decreased cost with scant regard for the animals concerned. It now is well recognized that dairy cows are under huge metabolic stress to maintain their greatly increased milk production, with many effects on welfare such as negative energy balance and lameness (Webster 1994). In developed countries most people do not need milk to be as cheap as it currently is, so that less economy-oriented dairy systems—those that do not use protein supplementation—could be adopted.

The most important question now is not whether the practice of feeding cattle protein to cattle was culpable, but how to adapt agriculture to reduce the chance of similar disasters in future—disasters that are perhaps intrinsically unforeseeable. An important part of the answer must be to reduce the emphasis on cheap production and to take into account the evolutionary history and biology of the animals involved—in other words, to treat the animals more naturally.

Table 1
Symptoms of vCJD in Humans

An early age of onset or death (average 27.6 years, range 18–41 years).
A prolonged duration of illness (average 13.1 months, range 7.5–24 months).
A predominantly psychiatric presentation including anxiety, depression, withdrawal, and progressive behavioral changes.
First evidence of neurological involvement in four patients was dysaesthesiae (unpleasant abnormal sensations) in the limbs and/or face.
Development of a cerebellar syndrome, with problems with gait and limb muscle coordination after a period of weeks or months.
Development of forgetfulness and memory disturbance, often late in the clinical course, which progressed to severe cognitive impairment and a state of akinetic mutism (paralysis and inability to speak) in the majority of cases.
Development of muscle twitching or spasms in the majority of patients (myoclonus), preceded by purposeless involuntary movements in some (chorea), with EEG appearances typical of sporadic CJD absent.

Source: Phillips, Bridgeman, and Ferguson-Smith 2000, 8:2

Escherischia coli O157

Many strains of the bacterium *Escherischia coli* (*E. coli*) live harmlessly in the guts of humans and animals. One of the exceptions is *E. coli* O157, a virulent, toxin-producing strain first identified as causing human illness in 1982. Infection is frequent; for example, there are an estimated 73,000 cases of infection and 61 associated deaths in the United States each year (Centers for Disease Control and Prevention 2001). The main reservoir is in cattle and sheep, for whom it causes no illness. The main route of human infection probably is contamination of meat by animal feces. Heating of meat kills *E. coli*, but only if it is thorough. People also can be infected directly by live animals and each other—for example, in nursing homes where hygiene is poor.

An outbreak of *E. coli* food poisoning in central Scotland in 1996 affected about 500 people, 18 of whom died (Pennington Group 1997; most of this account depends on this report, supplemented by Pennington 1999). This was the world's second highest number of deaths from such an outbreak.

Events moved with impressive speed (Table 2). A likely outbreak was identified on Friday, November 22, 1996, with fifteen confirmed or suspected cases. By that evening it was known that at least eight had eaten food from John Barr's butcher shop in Wishaw (although that did not prove it was the source) and health officials visited the premises. On Saturday, November 23, an outbreak-control team was formed, chaired by a local health board consultant. On Wednesday, November 27, Barr's closed. On Thursday an inquiry was announced in Parliament, chaired by Professor Hugh Pennington of Aberdeen University.

Barr's shop was indeed the source. He had supplied contaminated food to many private customers and several institutions and groups. These included the three clusters worst affected. Eight people who died had been at a church lunch in Wishaw on Sunday, November 17; six who died were at a nursing home in Bonnybridge (all whom were aged 69 or older); and a number of non-fatal cases had followed a birthday party on Saturday, November 23. Some cases resulted from sales Barr made from the back door of the shop after it was shut on November 27.

The main problem was that Barr and his staff did not keep raw and cooked meat properly separated. This was exacerbated by a general lack of proper hygiene in the handling and preparation of food. When contaminated raw meat came in, the contamination spread to cooked meat, which customers did not heat enough to make safe. Thus the contamination got progressively worse rather than being eliminated. In January 1998 Barr's company was fined for breaching food hygiene regulations.

Because failure to follow regulations contributed to this outbreak, the Pennington Group (1997) emphasized the need to educate people on the importance of such regulations, and to improve enforcement of compliance. But it also stressed the importance of events all along the way from cattle to table—on the farm, during animal transport, at the slaughterhouse, during meat transport, in premises processing and selling food, and in the home. The report recommended new regulations, better education, and a general change in attitude to improve hygiene. Involvement of farms, slaughterhouses, and food distributors will be mentioned here.

Farmers have a responsibility to send animals to slaughter in clean condition. This is affected by a number of factors, such as whether they are given clean, dry bedding and whether they are crowded in holding yards (which increases the likelihood that they will soil each other (C.B. Tucker, University of British Columbia, personal communication, June 30, 1999). The Pennington Report also criticizes a practice of "feeding up" cows before slaughter to increase live weight and hence the price obtained; feeding up increases the chance of intestines bursting during removal, and hence contaminating carcasses. By contrast, in instances where the same company both owns and slaughters animals, it is common for food to be withheld before slaughter.

Slaughterhouses also must avoid slaughtering dirty cattle, and must

Table 2
Events in the 1996 Outbreak of *E. coli* Food Poisoning in Central Scotland

Sunday, November 17	Wishaw Parish Church lunch
Friday, November 22	Likely outbreak identified Barr's butcher shop visited by health officials
Saturday, November 23	Outbreak Control Team formed Birthday party at Cascade Public House, Wishaw
Wednesday, November 27	Barr's closed
Thursday, November 28	Expert Group set up under Professor Pennington
Thursday, December 5	Fatal Accident Inquiry announced
Tuesday, December 31	Pennington Group submitted interim report
March 1997	Report commissioned on setting up a Food Standards Agency
April 1997	Pennington Group submitted final report
2000	Food Standards Agency established

improve various practices to safeguard hygiene. The Pennington Group comments that:

> There clearly has to be a cultural change amongst slaughterhouse operators and their staff.... Notwithstanding commercial considerations and the implications of, for example, piece rates of payment for workers [in which they get paid for work done rather than time worked], the speed of the production process within abattoirs needs to be controlled so as to permit the achievement of adequate food safety standards. (1997, 19)

The report includes this statement concerning food distributors:

> The distribution chain of meat and meat products from Barr's was diverse and complex and it took some days for the details on that to be unravelled from a painstaking investigation of the company's records. That caused delays in relation to the identification, publicly, of some of the outlets involved or potentially involved in the outbreak. Some 85 outlets...were eventually identified as being supplied by the company, making the task of outbreak management and control extremely difficult. (5)

So, while response to the Barr's outbreak was rapid, it could have been more rapid—and prevented many cases—if it had not been for this complexity of food distribution.

Around the time of this outbreak, other food safety problems also were publicized, including the danger of Listeria in unpasteurized cheeses. As a response to the accumulating list of such problems, the United Kingdom's Labor Party commissioned a report in March 1997 on the possibility of setting up a food standards agency. Labor won the general election that May, formed the new government, and, shortly thereafter, accepted the report's proposals. The Food Standards Agency was established in 2000.

Classical Swine Fever

Classical swine fever (CSF), or hog cholera, is one of the most important virus diseases of pigs. It is a fast-spreading disease, limited to pigs, with high mortality. Outbreaks are intermittent in Europe. There was a major outbreak in the Netherlands in 1997, for example, and many, smaller outbreaks in Germany (where it is endemic in wild boar) from 1998 to 2000. North America is free of CSF.

A CSF outbreak in southeast England started on August 8, 2000. On August 14 movement restrictions were imposed, which, over the next four months, would affect 264 farms suspected of disease and 907 more in the infected areas. CSF was confirmed on sixteen farms; 41,000 pigs were slaughtered on those and neighboring farms as a direct result of the outbreak and 34,000 more as "dangerous contacts."

On December 30 (Anonymous 2001) the outbreak was confirmed to be over, and movement restrictions were lifted. While the outbreak was confined and eliminated relatively rapidly, it had a severe financial effect on a pig industry that already was in difficulties; many pig farmers left the business. It is also noteworthy that the outbreak was at its height when the Phillips report on BSE was published in October 2000.

The most likely source of disease is thought to have been an infected pork product, illegally imported, perhaps dropped on the farm by a member of the public or a wild animal (Gibbens et al. 2000). Initial detection was slow, perhaps in part because CSF's symptoms are similar to those of other, prevalent diseases. The disease probably was present in June, so that movements of pigs to other farms already had occurred before CSF was identified (Sharpe et al. 2001). Subsequently there was some spread between neighboring farms, but no evidence of irresponsible movements. By contrast, one of the ways in which disease spread in the Netherlands in 1997 was via trucks moving between farms (Elbers et al. 1999). Another major problem in the Netherlands was the concentration of its industry, enabling the virus to spread readily from house to house and farm to farm.

In many ways, control of the U.K. outbreak was a success story, and one that must have influenced decisions on handling foot and mouth disease, which followed hot on its heels. However, it renewed concerns about vigilance and effective surveillance for diseases, and about general attitudes regarding the importance of disease control. (The United Kingdom's state veterinary service has been reduced in size, and the number of veterinarians working in large animal practices has been declining (Anonymous 2000).) It also renewed concerns about the killing of animals, many of whom were found to have been healthy. There is an effective vaccine for CSF, but EU and U.K. policy is not to use it on animals who have the disease, who are suspected of having it, or who might become infected. This is because vaccinated animals cannot be distinguished from infected animals, so vaccination hinders eradication. An EU directive adopted in 2001 continued this policy but placed increased emphasis on development of "marker vaccines" that would allow vaccinated and infected pigs to be distinguished.

Foot and Mouth Disease

Foot and mouth disease (FMD) appeared in northern England in February 2001 and rapidly became an epidemic. It broke out on a pig farm and spread to neighboring sheep farms. Sheep from this farm were moved around the country before the disease was diagnosed. Three days after the diagnosis, the U.K. minister for agriculture imposed a complete ban on animal movements. By then, however, the disease had been established for several weeks; the Department of Environment, Food, and

Rural Affairs (DEFRA)—which replaced MAFF in June 2001—estimated that there had probably been over 2 million movements of sheep in that period. Many of these movements were not recorded, as they should have been. Furthermore, farmers continued to move animals illegally during the crisis (Lashmar 2001). Some of these movements were for the animals' sake—for example, because they were in fields with insufficient food—and the government soon issued guidance and help for such cases. However, some movements must have been for commercial reasons. Some animals were moved abroad both before and after the ban, and the disease spread to the Netherlands, France, and Ireland. It reached Ireland by an illegal movement of animals across the border from Northern Ireland.

This was the first outbreak in the United Kingdom since 1967, apart from a small outbreak on the Isle of Wight, off the south coast, which rapidly was controlled. The latter demonstrated, though, just how infective the disease is, as it was caused by the virus blowing across from France. DEFRA attributed the decades-long period without infection, in a world with widespread FMD, to tightened control of imports from countries with the disease after 1967 and improved hygiene and animal health standards. However, illegal importation of meat is common, with more than 200 consignments intercepted on their way into the United Kingdom every month and unknown numbers missed. Some of these go to restaurants, and waste food from a restaurant was found at the pig farm in question. Use of waste food for pigs has been common, and although it is supposed to be heated at 100°C for an hour—which would kill the FMD virus—this often does not happen (Lashmar 2001). (Feeding of waste food to pigs now is banned.) While exactly what happened on this farm has yet to be established, it seems that the farmer failed to notice symptoms of FMD, although his pigs were infected for several weeks before officials traced the source of the outbreak to his farm.

So far, Australia, New Zealand, and the United States have managed to keep FMD out, presumably by more rigorous import controls than are achieved in Europe.

As with CSF, during the 2001 epidemic, the policy of the United Kingdom and the rest of the EU was to slaughter animals with FMD, those who might have it, and those who risked spreading it. The main reason was commercial: a country with infected or vaccinated animals may not export animals or meat to countries free of the disease. At the peak of the epidemic, about fifty farms where the disease was present were identified daily. After about two months, the number had dropped to fewer than ten per day. The outbreak had a long "tail," with three or four farms being infected per day before the disease finally was eliminated late in the year. More than 1,900 farms were affected directly, and 7,000 neighboring farms also were cleared of animals. About 4 million animals were slaughtered.

The slaughter policy was hugely controversial. This was largely a result of the issue's high profile, as television broadcasts showed thousands of farm animals being killed, many of them healthy, and their carcasses burned. Individual stories of pet animals and prime breeding herds received considerable publicity. As with BSE, even though the farm animals would eventually have been killed anyway, the fact that they were being prematurely and very visibly killed was morally repugnant to many people. The outrage probably was exacerbated by the fact that, for many, the killing was seen not to have a useful purpose—such as meat production—but to be done for defense of a meat export trade that they regarded as unnecessary. Indeed, losses to the tourism industry, caused by people being unable to move around in the countryside, heavily outweighed the value of the meat exports. Furthermore, suggestions that the disease is not very severe if left untreated gave rise to discussion of whether it should be allowed to continue rather than eradicated by draconian measures. (In fact, symptoms of the disease vary in severity; it sometimes causes considerable suffering and in particular causes major problems in breeding animals, such as abortion and loss of milk production, and mortality in the young.) On balance, it seems appropriate to eradicate an eradicable disease. However, even though FMD was eventually eradicated, it might break out again sooner or later. If that were to happen, the U.K. government says it would employ vaccination rather than extermination as its strategy in dealing with the disease. (Countries in which FMD is endemic use regular vaccination and, as is the case for CSF, development of marker vaccines has been proposed.)

To re-emphasize the commercial basis of the policy not to vaccinate, the decision to end vaccination in the EU in 1991 was taken on the basis that a major outbreak every ten years would be less costly than annual vaccination (Nettleton and Reid 2001).

There were reasons other than the slaughter policy for controversy. It was apparent that the United Kingdom was ill-prepared for the crisis. The early slaughter and carcass disposal were relatively slow, delaying containment of the epidemic. In addition, the methods used for handling and slaughtering animals evidently were not as humane as they might have been. Accusations also were made against farmers. Some were seen to be profiteering by pushing claims for compensation higher than reasonable, with the direct cost of FMD reaching £1 billion by August. There were suspicions that some deliberately spread the infection to their own animals to claim compensation above market values (Hetherington and Lomax 2001). On the other hand, many farmers were hurt both emotionally and financially by the crisis; a number even committed suicide.

Apart from inquiries into the epidemic and its handling, the FMD cri-

sis finally precipitated wide-ranging discussion of the future of farm animal production in the United Kingdom. One result was establishment by the government of a Policy Commission on the Future of Farming and Food, which produced a report (2002) emphasizing the importance of sustainability.

Is the United Kingdom Exceptional?

One obvious question that arises from this succession of crises is whether the United Kingdom is doing something different from other countries, something culpable. It is true that agriculture is more industrialized in the United Kingdom than in many other European countries, with larger farms and a smaller proportion of the population involved. It also is true that the drive for greater and cheaper food production after World War II was stronger in the United Kingdom than elsewhere, arguably because the United Kingdom is an island nation. It is possible that these factors led to widespread laxity in food handling, which magnified the disease crises. However, it also is apparent that the United Kingdom is not the only country with such problems. Reprehensible actions occur elsewhere; for example, in 1999 it became known that human and animal feces were being incorporated illegally into feed for farm animals in France (Meade 1999). This practice was both repellent and as risky as those that gave rise to BSE. While the United Kingdom has been hit particularly hard by farm animal diseases in recent years, this must at least partly have been bad luck. The lessons to be learned are relevant to all countries.

Biosecurity

When international travellers enter the United States or New Zealand they are asked whether they are bringing in food and whether they have visited farms or plan to do so. While this is done with varying stringency, it is strikingly different from the lax approach used in other countries. Indeed, New Zealand is the only country with a minister for agriculture and biosecurity, and its Biosecurity Authority produces a regular magazine intended for the general public as well as specialists. By contrast, although DEFRA launched a biosecurity campaign in the United Kingdom in June 2001, it was aimed solely at farmers.

Obviously, other countries should adopt policies on biosecurity similar to those of New Zealand. Given that classical swine fever and FMD probably were introduced into the United Kingdom by illegal imports, the fact that the United States and New Zealand have remained free of FMD must partly be luck. However, stringent regulations and stringent enforcement of those regulations must reduce the chance of disease transmission.

The United States and New Zealand are not blame-free: they are guilty of double standards in restricting imports while aggressively exporting agricultural products. So long as these export policies continue, the two countries risk exporting any disease that does get into their animals or animal products in the future. They also reinforce the tendency to regard such exportation as routine, acceptable, and inevitable. In fact, on the contrary, it is evident that international movement of animal feed, animals, and food from animals is dangerous, largely unnecessary and damaging to animal welfare and the environment (Lucas 2001). Ways must be found of reducing such movement.

Similarly, movement of animal feed, animals, and food from animals within countries must be reduced. A major factor in the foot and mouth epidemic was the enormous scale of sheep movements. Animals often are driven very long distances to slaughterhouses, for instance, frequently passing by nearer slaughterhouses on the way. For biosecurity and animal welfare, animals should be slaughtered at facilities as close as possible to the farm where they are produced; yet the number of slaughterhouses in the United Kingdom has declined considerably over recent years. Local food production and consumption clearly are desirable for animal health—and many other reasons (Valen 2001). Traceability—the principle that it should be possible to track any product "from farm to fork" or vice versa—also is gaining importance, with obvious relevance to animal health and food safety.

Biosecurity frequently has been regarded with complacency. It is imperative that vigilance become the norm, with systems in place in the food industry that lead to containment or prevention of disease.

In the United States, such vigilance has been discussed much more since the events of September 2001 raised the possibility that disease outbreaks might be introduced purposefully. It is striking that little of that discussion has addressed the fundamental structure of the U.S. agricultural industry, despite the prominent role such structure was seen to have had in causing the crises in the United Kingdom—and the fact that the agricultural industry in the United States is much more concentrated and more intensive than that of the United Kingdom, and that there is much more movement in the United States of animal feed, animals, and food from animals. The future is difficult to predict, but it does seem extremely likely that if there is a serious out-

break of a disease such as FMD in the United States—whether accidental or deliberate—it could rapidly become very serious, indeed. These issues would then be given the attention they deserve. What seems surprising, especially as salmonellosis and *E. coli* infection already are widespread, is that such attention isn't already forthcoming. It would obviously be better to address these issues properly before such an outbreak—reducing the chance of it occurring—than after.

Economics of Animal Production

Biosecurity will not come cheap. However, it is increasingly apparent that pressure for cheap food has incurred many external costs, that "cheap food at any price" is not a sustainable policy, and that cost-cutting elsewhere (such as in veterinary surveillance) also has been damaging. As the Pennington Group (1997) emphasized in relation to *E. coli*, disease control includes events on the farm, during animal transport, at the slaughterhouse, during transport of meat, in premises processing and selling food, and in the home—and supply of animal feed should be added to the beginning of that sequence. Improvement of disease control at all those stages will require expenditure. How is it to be paid for? One mechanism might be a levy on food from animals, to be spent on improving animal health and welfare. As the cost of animal products in a meal usually accounts for only a small proportion of its selling price, most consumers would hardly notice such a change.

Cheap food production has not generally increased profits of farmers, because profits constantly are pared away by price competition. Income for many farmers is low and unreliable, which must affect their attitude to and limit their spending on animal health and welfare. A decent, reliable income for farmers—not huge wealth, but a reasonable living—must be part of a sustainable future for farming.

On the farm, relevant issues include:

- Group size: Maintaining smaller groups of animals restricts disease transmission.
- Housing conditions: Giving animals sufficient space and clean, dry conditions, including bedding, increases their health and cleanliness. Hygiene is not increased by barren conditions.
- Feeding methods: These are critical and have many effects on health and welfare. Consideration must be given to the biology of the animals involved, that is, to treating the animals as naturally as possible.
- Concentration of animals: Large, closely spaced units increase disease transmission. Small, well-spaced houses and farms should be favored.

Consideration also should be given to licensing farms or farmers. Most farmers are not criminal or irresponsible, but it should be possible to exclude the minority who are.

Minor increases in expenditure on food and on other aspects of biosecurity related to food production could produce major improvements in farm animal health and welfare. Mechanisms should be explored to achieve these changes.

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