

PROLOGUE

A problem with waste

Some time in 1987 I was a freelance writer for the Bangor Daily News in the wilderness state of Maine when there was an ongoing story about a polluted stream in the countryside. Locals had reported the problem over several weeks and the Environmental Protection Agency traced the source of pollution to slurry from a dairy farm. Despite the farmer's assurances that the slurry would be removed, the problem seemed to keep recurring. I went to see for myself.

After driving for many hilly miles up and down almost deserted roads and turning back at least once in the belief that I must have blinked and missed the farm, at last a large open-ended barn and several sheds appeared by the roadside ahead. A black and white cow was sitting inside the barn as I slowly passed by. Pulling off the road a little further on, I walked downhill past a big empty meadow and found the stream emerging from beneath the road on the other side. Although narrow and swiftly flowing it was clouded and grey. The water coursing rick-rackedly down through the uneven and untrampled meadow could hardly be seen as I walked back up towards the farm. It was a very small stream. The EPA is 'very committed.

The black and white cow looked very contented, chewing the cud on clean straw. Other cows could be seen in stalls behind her. Hoppers above them appeared to supply food. A small milking booth occupied an area to one side. Making sure no-one was around, I ventured along by the side of the barn. Ahead, some distance from the road, was a low shed. A score of calves looked out, crowding each other, up to their knees in slurry. At the end of the barn furthest away from the highway was an open space. A miserable group of about half a dozen cows stood together on the concrete floor. It was shocking to see that their hooves had grown abnormally long and the animals were no longer supported by their feet but by their forelegs, looking as if they were on skis. An outdoor telephone bell was ringing and a tractor was approaching. I left quickly. The story was not published.

Across the Atlantic, however, a different story was emerging.....

Beware the risky derivative
Wherein horrendous dangers live;
From Barings B
To BSE
The future's bleak without a div.

BSE - A housewife's rendering

Mad Cow Disease - or Bovine Spongiform Encephalopathy - first made headlines in 1986. In America we were only vaguely aware of black and white cows staggering about on our tv screen before other more pressing news took its place in media priorities. BSE was a British problem, a distant, dead-end story. We returned to England in 1991 and resumed life in Lincolnshire, a county formerly sheep-rich but now more noted for daffodils and cabbages.

Some time after settling into our new home we were surprised by occasional references to Mad Cow Disease and our suspicions were roused. Perhaps the disease was still prevalent among cattle. Having grown used to American ground sirloin in our Shepherd's Pie rather than the frugal British fatty mince it was not difficult to transfer to Brazilian or Argentinian corned beef instead. The argument went that South American cattle roamed the pampas and would therefore be BSE-free, unlike British cattle intensively reared like chickens on broiler farms. Never keen on processed food, we didn't miss meat pies.

To the horror of the public at large it had become apparent that animals had been recycled into food for other animals. The thought of 'cannibal cows' produced a collective shudder. People imagined the docile Daisy gnawing a raw steak sliced from its Aunt Gertrude, jaws luridly dripping. Cud rhymed with blood. This thorough-going reversal of the bovine nature was sufficient in itself to convince many that BSE was 'God's punishment', natural justice for offending the natural order. Modern intensive farming methods were a sinister mystery most people preferred not to confront. With BSE there was no escaping the unpalatable truth.

Sheer curiosity impelled me to begin reading *Farmer's Weekly* and to write to the Ministry of Agriculture, Fisheries and Food. But their answers only provoked further questions.

Thus began a search for the facts about BSE and a worldwide correspondence encompassing Australia, New Zealand, the United States of America, the European Union and several addresses in the United Kingdom. Scientists and bureaucrats have generally been helpful and cooperative, even those at MAFF, whose patience was often sorely tested by my continued insistence on having ambiguities explained and my unscientific grey cells educated at the expense of their time and effort. Long delays in answering were probably due to pressure of work rather than to any conscious desire to discourage questions, but the suspicion persists. Unfortunately a sceptical nature is easily persuaded that suspicions are justified by an apparent reluctance to cooperate. My suspicions continue though the search dwindles.

A morning spent in Spalding Library filled in some early gaps in my limited knowledge. There seemed to be some connection between BSE and Scrapie, a sheep disease. Hungerford's *Diseases of Livestock* (McGraw Hill 1975) illumined several black holes. On my way to Scrapie it was interesting to note that sheep suffered widely from

Salmonella, particularly after long journeys under stress¹. But, unlike Scrapie, Salmonella could be inactivated by formalin. Scrapie was described as having 'numerous strains', its nature quite different from other diseases, and resistant to temperatures as high as 1150C, even after boiling 36 hours. It appeared too small to have nucleic acid and resembled 'Kuru', a human brain disease in New Guinea. Symptoms included severe itching, driving sheep to scrape against hard surfaces, often rubbing off their wool in the process, hence the name 'Scrapie', (also 'Rubbers'), with stumbling and loss of coordination. Moreover the genetic make-up of the host was important; sheep ate foetal membranes, a possible source of infection; incubation was anything from 5 months to 5 years. Control was by slaughter and surveillance of all contacts for several⁴² months.

Notebook at the ready, next was Black's '*Veterinary Dictionary*' 17th edition (Black 1992) with ominous references to human diseases: Kuru, Creutzfeldt-Jakob Disease (CJD), Gerstmann-Straussler-Scheinker syndrome (GSS), Prusiner's article in the *Cornell Veterinarian* 81, 85 and the possibility of prions (preons) as the cause of BSE, a prion being a self-replicating infectious protein thought to be responsible for 'Downer-cow syndrome' in the USA. My notes also included a reference to a question asked by a New Zealand scientist at Massey University, N H Christensen: Could BSE survive the pelleting process and pass through other hosts - chickens, for example? This question was later partly answered in a letter from Dr David Taylor of Edinburgh Institute of Animal Health.

A sinister note in the notebook states ominously: 'MAFF Food Advisory Committee bans information on animal rearing and slaughter.' No wonder I was suspicious.

Letters to the Australian High Commission and New Zealand Ministry of Agriculture produced interesting scientific papers² about the introduction of Scrapie into those countries in sheep imported in the Fifties from Great Britain. The British herds had been certified free of the disease and there were no apparent symptoms³. It was thought that earlier imports, if infected, did not survive the long sea journeys⁴. Improvements in travel methods reduced the time spent en route, thus allowing infected sheep to live long enough to infect native flocks. Such infections were relatively easy to trace and all affected flocks were slaughtered⁵. This was extremely important because America was a major customer and trade with the United States depended - and depends - upon scrapie-free animals.⁶

Extracts from the *New Zealand Journal of Agriculture* (85 pp305-306) published in 1952 describe Scrapie: 'The disease has been known in Britain for about 200 years and at one time was fairly prevalent in a large part of of England.....Both ewes and rams are equally susceptible. The incidence in infected flocks is commonly from 4 to 20 per cent. its spread through a flock is usually slow, but very occasionally may be so extensive that the deaths in a year outnumber the births. Severe outbreaks sometimes occur in which the disease spreads rapidly, especially among the younger sheep....It is usually spread by the lambs being born with the infection, which they may derive from either the

ram or the ewe, even although both parents appear healthy at the time of mating. The infective parent may never show the disease or may do so only after a lapse of several years.

'The disease can also be spread through pastures, although experience shows that farms can safely be restocked very soon after the removal of infected animals. The infection may be introduced into a clean flock by the purchase of apparently healthy ewes or rams, and the ram may spread the infection both to his progeny and to the ewe at mating time.

'The onset of the disease is insidious and the earliest signs may not be noticed. Probably the first thing noticed, as in the New Zealand outbreak, will be skin irritation and loss of wool, frequently in the region of the hindquarters and loins. Apart from possible injury from rubbing or biting, the skin over the bare area looks quite normal. The loss of wool may gradually extend over the whole body. If the skin is rubbed, the animal shows a "scratch reflex" by making nibbling movements of the lips.....The sheep is more excitable and restless than usual, especially if rounded up, and trembling of muscles...is apparent. There may be grinding movements of the teeth. The expression is fixed and staring and the head and neck are carried high and stiffly. Movement is often unsteady and in one form a peculiar trotting gait takes the place of the normal sheep gallop.

'The nervous symptoms gradually become worse over a period, usually of several months. There is gradual loss of condition and the sheep eventually becomes paralysed. In most cases the disease runs a course of from 2 to 5 months and invariably ends fatally. Curative measures are useless.

'Unfortunately it was necessary in the recent outbreak to slaughter all the sheep in the infected stud and also the sheep that had been sold. The total number involved..was about 225. Compensation at full market value was paid for all sheep slaughtered.

'It is hoped that this drastic action will prevent further spread.....Imports from Britain...may possibly not be renewed until a reliable biological test for scrapie is evolved or until it is considered safe to allow further imports.

In *The New Zealand Veterinary Journal* (Vol.1, p29) also in 1952, the same author, A G Brash, refers to the work of Greig in 1940 and 'a large volume of evidence indicating that under natural conditions the disease can be transmitted from either the ram or the ewe to the immediate progeny.' Further work by Greig in 1950 indicated that Scrapie 'can be transmitted through the medium of the pasture.' It was also demonstrated that the agent causing the disease was 'resistant to formalin and storing' and was 'present in transmissible form in sheep fifteen months before they might have been expected to show signs.'

It must be noted that the above information comprised what was known about Scrapie more than 40 years ago. Some of the evidence was based on hearsay and some on scientific research. Scrapie was not considered a danger to human health and no

particular attention was paid to the few facts known. Research into brain diseases was in its infancy and scrapie did not at that time represent a threat to humans.

A later paper by A N Bruere for the New Zealand Veterinary Association in 1985 on *Scrapie in New Zealand - Its history and what it could mean* describes Scrapie as 'an infectious disease of sheep caused by as yet an unidentified agent with unusual characteristics.....known to exist as several strains...in an obscure way affected by the genetic make up of susceptible animals.....certain strains can be transmitted to mice and a variety of other animals.'

Bruere notes the existence of 'many written reports...particularly from Britain' but laments the fact that Scrapie was not a notifiable disease in the UK, thus making estimates of its seriousness almost impossible; moreover it was known from 'the earliest reports' that shepherds sent affected animals to slaughter as soon as they recognised the symptoms. Assuming these animals may have entered the food chain, this fact alone demands further examination in the search for causes of human brain diseases. Bruere refers to Scrapie infecting American flocks of goats and sheep, (reported in a *Veterinary services memo no.557.1APHIS*) Nonetheless he confirms that the USA 'have excluded from human consumption all meat from scrapie farms' based on evidence of transmission of spongiform encephalopathies between species, including higher primates.

The above statement over a decade ago indicates an awareness by American authorities of the possibility of transmission to humans of the Scrapie agent through sheep. British authorities continued to ignore such a possibility: Scrapie was still not notifiable and sheep from infected flocks continued to enter the British food chain.

Bruere makes the point that once Scrapie has become established in a country with a large sheep population 'eradication has not been possible'. He cites the American example where an official eradication programme has operated 'since 1970' (*ANH discussion memorandum 557.1 20/6/70*) but the disease continues. This is at variance with a report from USDA (see below). Successful eradication programmes in New Zealand took place in 1952, 1954, 1976 and 1977 involving the slaughter of thousands of sheep and insistence on strict stock controls: 'tempers were frayed and..the Department of Agriculture did not gain popularity'. Bruere concludes: 'if you wish to avoid scrapie don't import sheep from a country in which the disease is endemic.....although all properties (in Britain) were reported free from Scrapie the assurances of the owners were in hindsight worthless.'

New Zealand relies on a five-year extendable quarantine period, importing sheep from Denmark and Finland, both countries Scrapie-free in 1985, the year before BSE was recognised in Britain.

Bruere ends his paper with quotes from Sir John M'Fadyean (1918) and Sir Stewart Stockman (1913):

1. 'My own study of the disease leads me to think that the only certain method of eradicating it from a large flock is to dispose of the whole flock of sheep young and old and restock with ewes and rams from a sound source.'

2. 'A farmer who has got the disease in his flock should sell his sheep for slaughter. I believe that by adopting a measure of this kind a great deal could be done to control the spread of the disease until science is able to come to the rescue with a practicable method of eradication.'

'We are still waiting for science. In the meantime it would appear that the New Zealand policies on Scrapie past and present have been conservative but correct. If we wish to continue to exclude this disease from entry all the previous experiences and rules must be adhered to and succeeding generations of veterinarians would be wise to always carefully look at the past while they plan for the future.'

A whole decade had passed without much further progress into the study of Scrapie. In Britain, rules were not adhered to and previous experiences were ignored. Everything that was known about Scrapie in 1986 was based on ancient history and a few unfortunate experiences on the other side of the world. Ironically the history of Scrapie began over two centuries ago in continental Europe in Spain and Saxony, when infected Merino sheep were allegedly imported into Britain.⁷ By the 19th century British flocks were badly infected, causing great losses, but eradication was not considered an option. It was not considered a zoonosis, transmissible to humans. However the presumption that Scrapie had mutated in cows to cause BSE should have prompted enough alarm signals to eliminate any complacency about the harmlessness of the disease.

DOES THE AMERICAN EXPERIENCE OF SCRAPIE EXPLAIN OURS?

Although Scrapie affects American sheep, the incidence is relatively low since eradication programmes started in 1952, continuing in 1992 with strong incentives to farmers by way of indemnity payments and certification of Scrapie-free flocks over 5 years. Sheep farming has declined in recent years, thus reducing risk of Scrapie. This information was contained in documents received from the United States Department of Agriculture including '*Qualitative Analysis of BSE Risk Factors in the United States*' 1991, '*Quantitative Analysis (etc)*' 1991, and '*BSE: Implications for the United States*' July 1993. Various letters were also exchanged.

These papers laid out the possibility of BSE developing in America based on the assumption that Scrapie caused the disease. Comparisons of farming methods and food production techniques in both countries are significant. The sheer size of North America suggests lower risk factors, apart from the low cost of grain available for livestock. Although the American dairy industry is even more intensive than in Britain and heavily reliant upon concentrates, these are generally cereal or soya-based.⁸ BSE has not yet been found in America but a transmissible spongiform encephalopathy has occurred in farmed mink. It has been suggested that this may have derived from

rendered material from 'downer' or 'nonambulatory' cows.⁹ The BSE crisis has focused fresh attention on the cause of 'downer cow' syndrome.

There are many differences between American and British farming methods, not least the proportion of sheep to cattle. In Britain the sheep population, and therefore Scrapie, has been rising steadily while falling in America; the ratio of sheep to cattle in Britain is 32 times greater than in the United States.¹⁰ Concentrates derived from animal protein included 14% sheep in Britain compared with 0.6% in America.¹¹ British sheep tend to be older and more likely to be infected.¹² Scrapie in Britain was not notifiable until 1993 and is undetectable while incubating.¹³ It is probably endemic in most British flocks, particularly in Suffolks.¹⁴

In Britain it is common for animals to mix at slaughter. American slaughterhouses tend to be species specific and limited in operation to 150 miles radius¹⁵ 97% of American slaughter came from 1,300 federally inspected plants whereas only 50 British abattoirs out of about 200 had fulltime inspectors and were licensed for export.¹⁶ In America sickly animals tend to fall prey to coyotes and other scavengers but in Britain they were traditionally disposed of at the knacker's.¹⁷ The reduction in numbers of knacker's yards led to an increase in casualty and condemned sheep at the rendering plants.¹⁸ Eventually everything ends up at the renderers.

RENDERING - or -What goes around comes around

Rendering means reducing left-over raw material from livestock that is otherwise wasted. 400,000 tonnes per year of protein material was normally produced of which 5% was exported¹⁹.

For many decades raw materials have been rendered down to produce greaves, gelatine and tallow.²⁰ Greaves are further rendered for their protein content as 'meat-and bonemeal' (MBM).²¹ MBM was used as fertiliser as well as for feedstuffs.²² Tallow is mostly fat and is used in a surprising number of ways including cosmetics, soap, glycerine and many other products as well as in animal feed.²³ More recently the huge problem of waste disposal from intensive farming methods was partly solved by processing urea. Even urea contains valuable protein. Broken down into its constituent chemical parts and combined with molasses, this form of protein supplement has formed a regular part of animal diets²⁴ and was advertised for feeding to livestock, recommended in *Farmer's Weekly*²⁵.

Farmer's Weekly magazine was a goldmine of esoteric information on such matters. For instance, its Opinion column May 7 1993 reported a pilot plant in Kansas making fuel from rendered animal fat with a return of 20% net profit before tax. A mix of methanol, fats, oils and methylesters was used. FW Opinion commented that this 'might produce a marginal lift in prices for overfat stock and by-products' then concluded: 'But don't bank on it.' British power stations have been considering cull cattle as fuel more recently.

The subject of waste has become one of this century's most pressing problems. Not so long ago an enormous barge caused uproar when it was towed out from the American coast with the intention of dumping the contents at a safe distance from its origin. The contents were garbage and the barge was prevented from discharging its malodorous cargo at the intended site. Increasing resistance to the dumping of waste materials and the closing of landfill sites threatened to tarnish the bright new millennium before it had begun. Recycling as much as possible promised to be one solution. Water is already chemically recycled in cities. The recycling of food was a logical step. After all, the process had been carried on naturally for eons.

For decades, rendering was done in batches, heating quantities of raw materials to a high temperature long enough to kill known diseases such as Salmonella and produce equal heat throughout the mixture before applying hydrocarbon solvent to finish.²⁶ This tried and true process appeared to be safe without benefit of official standards²⁷. New developments and sophisticated chemistry encouraged greater savings to be made and concomitant greater profits to be garnered in the wake of intensive farming. Such a solution to the problem of waste seemed ideal and allowed for the removal of waste products from 'factory farms' and consequent recycling with the utmost economic efficiency. Poultry farms supplied broiler litter (not only urea but feathers also have protein); nothing was wasted.²⁸

Livestock waste was reduced to a dry, inoffensive pelleted product to be compounded for further use.²⁹ Protein concentrates for livestock varied according to the animals receiving them. Pigs, for example, were given three times as much protein supplement as herbivores like cattle and sheep.³⁰ Cows in calf were fed concentrates until birthing.³¹ Suckler calves received concentrates for faster, leaner growth.³² The problem of waste and consequent pollution appeared to be solved wherever adequate rendering facilities were available.

Consumer demand for leaner meat was satisfied by the new leaner product. Prices were kept down through the supply of cheap and plentiful feedstuffs; British profit margins began to soar to new heights. Imports of expensive soya meal were less important. Thus the circle of consumer satisfaction revolved happily within the larger wheels of commerce. Leading the way to even better profitability, American methods of continuous rendering with lower temperatures and little, if any, solvent, were introduced into Britain³³. The more conservative Scots and French, however, retained the old methods.³⁴

Recent research into the effectiveness of different rendering processes was reviewed in a paper in *The Veterinary Record* of December 9 1995, suggesting that BSE comprised one particular strain of the Scrapie agent peculiarly resistant to inactivation.³⁵ Funded by MAFF, EU and the rendering industry with equipment from renderers Prosper de Mulder of Doncaster, Dr Taylor of Edinburgh and his co-authors concluded that the 'timescale over which....changes in production methods occurred is not consistent with the onset of the effective exposure of the cattle population to BSE'. Rather, they wrote: 'the rapid

decline in the use of solvent extraction as an adjunct to rendering processes which occurred during the late 1970s and early 1980s in Great Britain has been identified as consistent with the estimated onset of the exposure of cattle....'. It should also be noted that the use of superheated steam to remove the solvent may also be significant.

This paper further concluded that 'field strains of scrapie which are more thermostable than BSE may exist'. (Despite such conclusions the American authorities do not plan to reintroduce solvent extraction after 40 years without it, according to a letter from USDA of August 2 1996). It should be noted that during these experiments measures taken to prevent cross-contamination included frequent changes of protective clothing, wellington boots scrubbed with hypochlorite, floors and surfaces covered with disposable polythene sheeting, and equipment scrubbed with detergent, cleaned and sprayed with strong sodium hypochlorite.

Such strict laboratory measures are used in commercial food preparation for humans but do not normally feature in preparing animal feedstuffs, especially as portrayed in the media.

A report on the BBC's *'The World at One'* June 9 1996 portrayed a squalid picture of a rendering plant near Chartham in Kent. Dr Alan Colchester of Guy's Hospital, who diagnosed the first case of CJD associated with BSE, and a further cluster of cases from the same area near Ashford, visited the rendering operation known as Canterbury Mills with a local woman who had complained about it. They described a huge midden caused by liquid effluent oozing from pipes from the rendering plant. Vegetation had died. Dr Colchester described the pipework system as 'Heath-Robinson'. He stressed the resistance of the prion to inactivation, saying it could remain in the soil for years. (Mouse experiments in Edinburgh found some SE survival in their faeces.³⁶)

On television a manager was interviewed and said that the plant could not possibly keep up with the present cull of 1800 a week. Piles of offals and animal remains could be seen in the blazing sunshine of an exceptionally hot and humid day. The rendering plant had been prosecuted in the past by Kent County Council. The Environment Agency confirmed a prosecution took place in 1995. It is said to be one of only nine in the United Kingdom dealing with the BSE cull. A local woman, Mrs Sheila Webb, said she had seen many spillages of effluent from tankers on the road nearby.

A London firm was asked about their operation. Paul Foxcroft of Prosper De Mulder in east London said they had spent thousands on new equipment. He said the renderers 'must stick together' and refused to reply to questions about soak-aways as used by Canterbury Mills. He said his firm withdrew from the official scheme. Member of Parliament, David Hinchcliffe, would ask 2 questions in the Commons that week. MAFF refused to comment on the programme. The Environmental Agency said they were not consulted about rendering plants.

Chalk filters bacteria and viruses but the prion agent is more elusive. Water companies are allegedly insisting on filters ten times as effective. Doubt was expressed about the

temperatures used to incinerate remains. It was difficult to maintain constant high temperatures, especially in the event of power failures. Testing was not simple and could take years. Dr Colchester said it was time to start 'erring on the safe side'; the whole area should be fenced off and inspections should be audited. Since the broadcast there have been demands from the French to increase the cull. Power companies were reported to be considering using beef for fuel.

Two further television programmes followed in swift succession, both on the same evening of 17th June 1996. The first, on Independent Television, dwelt heavily on the broken rules and faulty regulations governing several aspects of BSE including preparation of feedstuffs, veterinary procedures, compensation payments and slaughter. A Warwick University epidemiologist condemned the methods used to control the feed compounders and renderers.

The second programme, on BBC, began with the first case to be diagnosed in 1985 at Ashford. Vet Colin Whittaker, described how the affected cow was condemned and sent to the knacker's yard to be subsequently rendered for feedstuff. Several more 'mad' cows were condemned from the same farm and others over the next 18 months from Yorkshire to Cornwall. All were presumably recycled into the food chain. Rendering plants did not keep adequate records.

Record-keeping is notorious with many busy people, not least farmers who resent the time spent on form-filling. Office work is generally alien to the outdoor life on the land. Enforcement of record-keeping is not a pleasant duty. MAFF epidemiologist Dr John Wilesmith refers to the 'high degree and rapid rate of communication' within the national infrastructure of the State Veterinary Services VI Centres and the 'relatively high degree of active communication between animal keepers and their veterinary surgeons'.³⁷ This resulted in the identification of BSE 'at a relatively early stage of the epidemic'. Unfortunately this happy relationship in the chain of communications was betrayed by several weak links and Wilesmith himself admitted to being 'disappointed'³⁸

FEED COMPOUNDERS

As an ordinary member of the public I paid an unannounced visit to a feedstuff producer early in 1993 and asked the manager what he thought about BSE and the feed ban. He replied with a shrug that the ban was 'just to keep the public quiet' and didn't make much difference to the way things were done. The feedstuffs were going to pigs and poultry as they were 'different animals'. The ban seemed to mean little although it was introduced in 1988.

In 1996 I made a second visit, assuming I would not be recognised after so long. Asking if any of them ate beef, one employee said he no longer ate beefburgers. I left it at that.

Unfortunately the ambivalence of the industry towards BSE was reflected in a general lassitude in the enforcement of control measures, no doubt due to the temporary nature of the first restriction on feedstuffs in 1988.³⁹ No-one took it seriously. Although banned

in the United Kingdom, there was no immediate ban on exports and there is no public record of quantities sold abroad. Accusations that contaminated feed was sold to Europe appeared in British newspapers in June 1996 followed by MAFF's explanation that the feed was harmless to pigs and poultry.⁴⁰ It would be interesting (but probably impossible) to know the stocks involved in the export trade. There is considerable confusion about production of MBM after 1988.⁴¹

Attempts to find more information about animal feedstuffs were frustrated by apparent official and unofficial collusion. Asking my Member of Parliament for help in obtaining a list of producers brought the reply that such a list was confidential and the Member could not break a confidence by forwarding it to me. In reply to my complaint that MAFF was being particularly uncooperative the Parliamentary reply was that it would be easier to obtain secrets from the Ministry of Defence than the Ministry of Agriculture! A bland enquiry to a major company about pig food was met with guarded suspicion and questions about my 'farm'. I gave up.

Neither supermarket chains nor the Food Safety Advisory Centre they fund proved any more helpful on the question of feedstuffs; Michael Young of the FSAC did not believe that animals were recycled in feed⁴² and despite recent university degrees his staff seemed equally unaware of how food was produced. Like the public they allegedly serve, they preferred to believe what the producers wanted them to believe, merely repeating bland assurances. Advertisements for dairy products encourage popular belief in cows enjoying country meadows and lush green grass; few want to believe that even sheep and lambs receive pelleted food manufactured by chemists.

A letter to The Times in June 1996 from the Agricultural Counsellor at the French Embassy stated that 'meat meal is used very seldom' in French ruminant feed and the import of MBM for ruminants was 'forbidden' in 1989 and banned in August 1990, which may have accounted for the low incidence of BSE in France. He added that when the disease is identified, the entire herd is slaughtered and burnt. With unGallic imprecision he continues: 'specified bovine material coming from animals born before July 1991 - ie, those that may have been given infected feedstuff imported before August 1989 - these are systematically destroyed'. Lawyers will be rubbing their hands with glee. Is it the animals or the offal that 'are systematically destroyed'? Even without foreign tongues, such linguistic quirks linger in nearly every phrase relating to BSE, as elusive and slippery as the mysterious agent itself.

Even MAFF studies of the feedstuffs proved virtually impossible for various reasons: lack of staff, lack of documentation, an overall lack of concern in the industry. There were no manufacturers' records to check quantities or mixtures.⁴³ Much perseverance was required to find out anything relevant; it was 'a difficult study to design'. Wilesmith confessed '**no other country has such a combination of risk factors**'.⁴⁴ Yet the 1988 ban was assumed to have been 'totally and immediately effective' and the epidemic to be 'virtually extinct' by 2000.⁴⁵

A challenging hypothesis advanced by Dr Narang⁴⁶ suggested that the enzymes added to animal feedstuffs to aid digestibility also broke down a hard protein shell enclosing the Scrapie agent, thus enabling it to cross the species barrier. In common with Professor Lacey and others he expressed grave concern for the vulnerability of all mammalian species and reported that even ostriches were found with SE in Germany in 1991.⁴⁷

The reason given by MAFF for 27,000 affected calves born since 1988 was cross-contamination of feedstuffs and/or the illegal use of feedstuffs banned in 1988.⁴⁸

In a letter to the *British Medical Journal* of 20 January 1996 Professor Lacey pointed out that calves born after the ban (BAB) developed BSE at the same age as those born before 1988 despite evidence that the incubation period is inversely related to the infecting dose; had the calves received minute quantities of infected material due to cross-contamination, they would not be expected to develop the disease so quickly as calves receiving fully-infected feed. By the same token, relatively large amounts of infected material would be necessary to cross the species barrier, from cattle to humans. In Lacey's opinion, the amount of law-breaking necessary to accommodate so much infectivity was unrealistic and he remained convinced that horizontal and vertical transmission were undeniable.

MATERNAL TRANSMISSION: MYTH BECOMES REALITY

The possibility of the maternal transmission of BSE was a nightmare dreaded by many and denied by others. The early assertion that a mad cow was a dead-end host⁴⁹ satisfied official scientific demand for a controllable, predictable disease confined to a few unfortunate beasts. The *Southwood Report* of February 1989 forecast a total of 17000-20000 cases, despite awareness that the disease had been recycled over and over again from 1981 to at least 1988 and based on the assumption that maternal or horizontal transmission did not occur. The actual total so far was over 160,000 and still rising.⁵⁰

Infection of placental material was deemed to be the source of infection in calves where no contaminated feedstuff was concerned.⁵¹ This method of 'maternal transmission' was generally accepted to have occurred in scrapied sheep, enhancing belief that the disease was inherited.⁵²

More than 27,000 calves born after the ban of 1988 (BAB) developed BSE. *Southwood* referred to 300 offspring of affected cows under observation at MAFF's Central Veterinary Laboratory; results from such observation would not be available 'until after 1990'.⁵³ A highly complicated statistical paper on the subject from Dr Wilesmith proved to be unreadable to this non-statistician.⁵⁴ A paper by Drs Ridley and Baker of Cambridge University Department of Environmental Psychology in 1995 referred to '*The Myth of Maternal Transmission*' with a commentary by Dr R Will followed by a letter in *BMJ* 20 January 1996.

The Times of June 30 1996 reported that ⁴⁷ calves with BSE had been born in MAFF's herd of 630 cattle. This herd consisted of 315 calves born of affected dams in 1989 and 315 from apparently unaffected animals. The results of this experiment were expected to be published in 1997. Presumably these animals were the same as those mentioned above in *Southwood*, where the date given for results was 'after 1990'. There is quite a big difference between 'after 1990' and 1997, enough time to produce at least two generations of cattle. In the week ending August 3 1996 an official announcement was made that maternal transmission had occurred.

SO WHAT CAUSED BSE?

The official version was that Bovine Spongiform Encephalopathy was a variant of the Scrapie agent.⁵⁵ An unofficial but well-argued alternative came from a British farmer, Mark Purdey. After studying for a decade Purdey became convinced that BSE was caused by Organo-Phosphates used to prevent warble-fly in cattle. Also used in sheep dips, OPs are known to affect the nervous system, having developed from nerve gas used in war. In a very convincing television programme on 12th June 1996 in Channel 4's *Despatches*, Purdey explained his ideas supported by lawyer/farmer Sir Richard Body MP, well-known Tory rebel and independent thinker. Body's books against the Common Market were a well-established source of irritation to the Establishment and he was not afraid of being judiciously controversial.

In order to secure a sound basis for his theory Purdey studied biochemistry while running his farm and gained several adherents including Dr Richard North. He reasoned that in England OPs are used at a far greater strength than abroad; after being poured over the backs of cattle the chemical enters the bloodstream and affects the brain, thus causing the symptoms recognised in BSE. By the use of maps Purdey demonstrated the areas of the country where OPs have been used regularly and these were almost identical to the areas most affected by BSE. Several farmers suffered the effects of using the chemical in sheep dips and were trying to claim compensation from a government that insisted on their use in the first place. One farmer even committed murder while allegedly under the influence of OPs.

Epidemiologist Dr John Wilesmith of the Central Veterinary Laboratory presented his comments, repeating the official view that Scrapie must be the cause. This view was expressed in the *February 1989 Report of the Working Party on BSE* (Southwood Report) set up by the Department of Health and MAFF. OPs were not considered a sufficiently common factor and were vastly outweighed by the evidence for Scrapie. This embraced several aspects (including the interesting statement (4.2.2) that genetic studies revealed 'some familial relationships in a small number of herds'): the predominance of affected dairy cattle rather than beef herds due to the larger proportions of protein concentrates given to dairy calves; the occurrence of transmissible spongiform encephalopathy in farmed mink attributed to scrapie-infected feedstuff and similar occurrences of TME in exotic deer soon after receiving commercial concentrates; every case of BSE so far had received animal protein whereas the small

number of herds (under 3%) receiving none were so far BSE-free; the ages of the affected cattle; the inclusion of more sheep and also more sheep heads in rendering; the inclusion of more sick sheep due to a reduction in knackers' yards; the lower temperatures and/or less time in rendering; the decrease in rendering solvents. A later paper notes: 'The difficulty of inactivating the unconventional agents which cause transmissible degenerative encephalopathies such as scrapie is well recognised (Taylor 1991a)'.

Further evidence for the same hypothesis appeared in Wilesmith's contribution to a definitive book: *Methods in Molecular Medicine: Prion Diseases*, ed. Baker & Ridley 1996. BSE was found in Guernsey after cattle feed containing MBM was imported from the mainland; meanwhile Jersey was BSE-free, having refused such imports. A second observation was the surprising speed with which the exotic deer had succumbed after receiving MBM when the price of vegetable proteins like soya had increased. The onset of the disease was found to coincide most closely with the reduction in use of hydrocarbon solvents in the rendering process in order to yield more tallow while making calf rations 'at low-cost formulation'. BSE was recycled several times into the food chain before a possible link was perceived and control measures imposed.

Intriguingly, research into an equally baffling disease, AIDS, had facilitated the science of detecting dates of probable exposure.⁵⁶ It was determined that 1981-1982 was the period of initial exposure to BSE by calves and the average incubation was about 4-5 years. Monitoring the disease in the field was hindered by lack of staff although Wilesmith expressed the highest opinion of the cooperation between vets and farmers. Unfortunately the hard work and effort contributed by such goodwill were severely undermined by the inability to enforce controls at every stage.

The initial difficulty in obtaining sufficient brain material for study was outlined by Wilesmith as one of the trickier problems encountered in the early stages of the BSE epidemic.⁵⁷ Despite the extremely congenial relations between vets and livestock keepers, it was a logistical nightmare trying to salvage the vital parts of cattle condemned for slaughter and adequately fixing the brains for laboratory study. While there were no plans to study the brains of cull-cows being slaughtered under current EU policies⁵⁸ it was reported that cows in control studies could be used⁵⁹; it was possible such a survey might reveal significant data about pre-clinical conditions. There appeared to be no plan to keep infected cattle alive to discover any further effects as the disease progressed.

Support for the Scrapie hypothesis appeared in a letter to *The Lancet* of 26th May 1990 confirming that experiments in Texas had successfully transmitted the disease from sheep to cattle.⁶⁰ The significance of information relating to an animal disease finding publication in a leading journal concerned with human health should not be wasted on anyone despite protests that neither Scrapie nor BSE was considered to be a zoonosis, one of the main factors behind official complacency. Further endorsement of the Scrapie hypothesis came from Dr Tyrrell, author of the 1989 *Interim Report* and

statement from the Medical Research Council (MRC) in July 1990 that 'the disease behaves..in cattle like scrapie does in sheep' and 'that the agent will be present in spleen and similar tissues, and in brain'.

However, the MRC also made the point that 'changes, sometimes permanent, can occur as a result of passage through a different species and the agent...may behave differently from scrapie'. Despite this the MRC concluded that 'beef can be eaten safely'.

Voices dissenting from the official view were heard, though muted. Brief soundbites were given to Drs Helen Grant, Stephen Dealler, Gareth Wynn-Roberts of Wellcome and Professor Richard Lacey of Leeds University. A former government scientist, Lacey roundly condemned official handling of BSE and produced a book and papers⁶¹ voicing his opinion that the disease was carried in many other organs than those specified under the ban. Lacey also cooperated in making a television drama on the subject. This dour 'gamekeeper turned poacher' continued to make forceful comments highly critical of his erstwhile employers and could be considered among the most prominent and most gloomy prophets of doom.

In his '*Short Report: BSE - The Disputed Claims*', Lacey questioned six official statements and painted a very grim picture for the future, estimating a possible maximum half-million cases of CJD or a minimum 5000 by the year 2015AD. The cost of slaughtering and restocking the national herd would be £30 billion. Contrary to official opinion, Lacey believed Scrapie might be irrelevant to the discussion and that BSE might have existed before new rendering methods encouraged its proliferation. He also believed gelatin from bovine bone marrow to be extremely hazardous, 'the greatest threat to humans'. He viewed BSE as more deadly than Anthrax. After years of rejection his efforts were being taken more seriously by puzzled politicians and Lacey went to Westminster in July 1996 to present his case. The following week the BBC reported that gelatin was now considered suspect.

NEURO-DEGENERATIVE DISEASES

Neuro-degenerative diseases were largely a mystery until the invention of the electron microscope and molecular biology. The discovery of spongy areas of the brain after death confirmed a similarity between some human neurological diseases and animal spongiform encephalopathies (SEs)⁶². However, scrapied sheep brains were not always identical and sometimes lacked the vacuolar appearance generally accepted as typical of the disease.⁶³ It appeared that there were several strains of Scrapie. In humans there appeared to be a link between several degenerative disorders: Alzheimer's Disease, Creutzfeldt-Jakob Disease (CJD), Amyloid Lateral Sclerosis, Gerstmann-Straussler-Scheinker Syndrome (GSS), Kuru, for example. The most definitive study has been that of Kuru in Papua New Guinea.⁶⁴

A deadly degenerative disease, Kuru was found in people who had eaten the brains of the dead, often as a sign of respect and affection. Significantly the bodies were often allowed to decompose before eating and the addition of maggots was considered an

enhancement. The longevity of the agent responsible for the disease was therefore well attested.⁶⁵ It survived long after the death of its host. This fact alone is worthy of particular note. The cessation of this traditional ceremony virtually ended the incidence of Kuru but for a few cases thought to have incubated over decades.⁶⁶

Alzheimer's Disease and CJD have become the most familiar human degenerative diseases. Alzheimer's, formerly known as Senile Dementia, is commonly associated with older people while CJD was - and still is - rare, occurring spasmodically throughout the world, mostly in older people; clusters have been observed in Libyan Jews and a possible genetic link suspected.⁶⁷ There has never been any direct evidence of links with sheep and these diseases seem to occur equally in populations regardless of diet.⁶⁸ Scientists admit that much more work is needed. Stress appears to be a common factor, especially relating to females with Alzheimer's.⁶⁹

Like Scrapie, none of these diseases could be detected before clinical signs appeared.⁷⁰ Human incubation periods measured decades. There were no tests. Unfortunately a large group of people were put at risk of CJD by the use of infected growth hormone in tissue taken after death from human subjects without clinical signs of disease.⁷¹ These hormones were used to stimulate growth in under-sized people with disastrous results. Responsible administrations have since replaced pituitary extracts with synthetic hormones.⁷² The extreme infectivity of the agent responsible for the disease was a matter for record, believed to have been removed in very small elements of neural tissue accompanying the removal of the pituitary gland, itself non-infectious.⁷³

Equally damaging 'friendly fire' cases of iatrogenic CJD resulted from an infected cornea transplant in 1974⁷⁴ and electrode implants in 1977 despite sterilisation in 70% alcohol and formaldehyde⁷⁵. As far back as 1965 a resistance to formalin had been noted by Pattison.⁷⁶

The only examination possible was *post mortem*, after death. The electron microscope had revealed structural changes in the brain tissue common to most spongiform encephalopies: vacuolation and fibrils associated with Scrapie in sheep.⁷⁷ The brains had become spongy. The mysterious agent responsible could not be readily identified but appeared to be a proteinaceous particle without nucleic acid.⁷⁸ Its method of replication was therefore highly mysterious.⁷⁹ After years of research Professor Stanley Prusiner of the University of California at San Francisco coined the term 'prion' (preon) in 1982.⁸⁰ A co-authored book on *Prion Diseases* (1991) is a standard work. Professor Prusiner kindly supplied his latest Review from *Neurology*, November 1993, a somewhat technical paper but extremely informative. His conclusions were inescapable: SEs appeared to be both infectious and genetic.⁸¹

Other scientists argue against the prion hypothesis, suggesting that the agent is 'an unconventional virus'.⁸² Dr Harash Narang chose the term 'nemavirus' based on his own research in association with Dr Gajdusek⁸³, an eminent leader in the field. Dr Narang

argues the case for greater care in the handling of BSE and has postulated a method of identifying the disease in the urine of live animals.⁸⁴ (The Dutch have since claimed success with tonsil tests from scrapied sheep.)⁸⁵ Dr Narang's conclusions were published in a 22-page two-part *Minireview* in 1996, updating his work published in 1987. He believes at least 29% of affected cattle entered the food chain and also refers to a conclusive link with DNA.⁸⁶

The transmission of SEs to pigs was successful only after massive injections directly into the brain⁸⁷ while both pigs and poultry survived in good health for six years while receiving deliberately infected feedstuffs under MAFF control.⁸⁸ Up to the time of writing there had been no tests to discover whether or not SEs survived in the gut of pigs or poultry but tests with mice showed survival in faeces.⁸⁹ Tests in Edinburgh found possible infectivity through the eyes,⁹⁰ making it imperative that all workers in the field should be adequately protected.

The itching and skin irritation distinctive of Scrapie clearly affect other parts of the body than the brain. A sheep's loss of wool from rubbing obviously does not occur in cattle, therefore skin irritation is not so obvious. The possibility of the spread of the disease from the nervous system to other tissues has been discussed elsewhere. The fact is that the only species to die naturally from spongiform encephalopathy while under observation is man. The progress of CJD has been charted in many instances and is associated with pain in the feet.⁹¹ Aggression and excitability are common SE factors in most species, especially under stress, associated with loss of coordination, loss of balance and mental confusion.⁹² Some tests have shown magnetic resonance imaging may be instrumental in diagnosing CJD in live subjects.⁹³

At the outset of the BSE epidemic there were loud denials that humans might be at risk although the *Southwood Report* indicated special surveillance for increased cases of CJD in the future⁹⁴. From 1990 such cases were referred to the Neuropathology Laboratory at Edinburgh University Western General Hospital. All death certificates involving CJD are referred there.⁹⁵ The purpose of the surveillance was to identify any changes in the pattern of CJD. According to a paper in *The Lancet*, April 6 1996, ten such cases had been identified. The victims were unusually young with evidence of the same variant never seen before in humans. Experiments with Macaque monkeys proved conclusive.⁹⁶

CONTROLS, COMPENSATION AND CASES **from MAFF News Releases and Reports**

Existing laws required cattle to be tagged and their movements recorded, such records to be kept at least 3 years;⁹⁷ the meaning of the word 'meat' was defined in 1984 to include muscle, head meat, tail meat, diaphragm, heart, liver, kidney, pancreas and thymus while banning most offals from 'uncooked meat products'.⁹⁸ This meant that brain, spinal cord, spleen, oesophagus, stomach, small and large intestines, rectum, lungs, testicles, udder and feet could be used in cooked products.

The Minister for Agriculture, the Rt Hon John Macgregor MP, on advice from the Southwood Committee (formed April 1988), introduced the following legislation:-

June 1988 the first statutory order relating to BSE making it notifiable; and a temporary ban on animal protein in ruminant food until 31 December 1988.

Cases so far: **511**

July 1988: Compulsory slaughter of affected cattle and compensation at 50% market value

Cases: **637**

December 1988 Milk from suspects destroyed except for own calves. Feed ban extended.

May 1989 14 measures listed by a government 'fully committed to protecting human and animal health' including recognition of BSE as a transmissible spongiform encephalopathy. Various research projects underway. Makers of human baby foods advised to 'avoid the use of ruminant offal and thymus' (not statutory). Labelling foods containing brain or spleen* considered unnecessary. (see * MRC 24/7/90)

June 1989 Specified bovine offals (SBO) from all cattle over 6 mths banned from food.

November 1989 SBO ban widened under new Minister John Gummer to include brain, spinal cord, thymus, spleen, tonsils and intestines but calves under 6 months exempt, described as 'a further addition to the very tough measures...earlier this year'

January 1990 following a Paris meeting of the Office International Epizooties (OIE) MAFF's Chief Veterinary Officer Keith Meldrum welcomed endorsement of measures taken, repeating that 'there is no evidence whatsoever of a risk to human health', citing Scrapie. There was no reason to extend the protein feed ban to pigs and poultry.

Total cases 9000 so far

9/1/90 New funding allocated for research: £2.2M 1990/91 and 1991/92, £1.7M 1992/93

February 1990 Changes to compensation to encourage detection of BSE; total £5M.

May 1990 'BRITISH BEEF IS SAFE, GUMMER

15 May 1990 News Release with letter from Gummer to Lady Wilcox, Chairman of Consumer Council: 'There is no reason to believe BSE will be any different from scrapie...'

15 May 1990 Information from 'MAFF Food Safety Directorate' Questions and Answers: Since 1986 over £2M spent and further £12M allocated over next 3 years for research. All results to be available to the public.

17 May 1990 Tyrrell Committee said there was no scientific basis for official advice against breeding from BSE cows and farmers should make their own decisions, even if infection can pass down.

12 July 1990 Tyrell said: Given the similarities between BSE and Scrapie, experience in operating measures to control Scrapie is clearly relevant in the case of BSE.

24 September 1990 New legislation to begin October 15: all calves and dams to be recorded

September 1990 SBO banned from all feedstuffs and exports but other ruminant protein still allowed. This followed experimental infection of a pig via inoculation.

1990 Total: 14000+

October 1990 new record-keeping requirements for farmers; must be kept 10 years.

21 November 1990 New laws to enforce removal of cattle brains in slaughterhouses and an inquiry into animal feed to be set up; independent scientific advice to be considered.

November 1991 law on movement of SBO protein or exports. Compensation £15.5M

Letter of July 14 1992 stated 600 per week confirmed cases 'not out of line' with forecast (the Southwood report forecast 300-400 a month) but that recycling was significant and the ban on ruminant feed 'will not be reflecteduntil the end of the year' with 'eventual disappearance' by the end of the decade, enclosing News Release on research into SEs.

June 6 1992 News Release on Parliamentary Question from John Whittingdale to Minister Gummer who replied that 'all studies justifying a high priority have been started' and that the Committee were 'content with the progress of implementing the earlier recommendations' and 'believes all necessary safeguards' in place to minimise further spread of SEs and 'to prevent any risk of transmission to humans'.

June 1992 Report stated: 'no change in characteristics of CJD'. Cases peaked: 1000 per week.

November 10 1992 Stats showed 700 per week confirmed. Compensation so far £35M

1993 total BSE confirmed cases since 1986 exceeded 27000 and compensation total £66M

November 24 1992 reply to mine of 12th on seasonal variations. 'As usual with BSE, nothing is simple' enclosing Report of EC Standing Veterinary Committee evidence of continued use of infected feed despite ban; of 500 affected calves born since ban 2 did not have feed but risk of maternal transmission was impossible; their dams were untraceable (see 24/9/90 above); there was no evidence of horizontal or vertical

transmission and this 'will not significantly affect the progress of the epidemic or prevent successful eradication'.

January 1 1993 Scrapie now notifiable to meet EU requirements and have assessment made. Research into diagnostic tests 'underway'; no indications of human transmission; controls necessary only 'on animal health grounds'. No changes in feed regulations. Enclosed a statement from the Chief Medical Officer, Dr K. Calman, on the death of a farmer from CJD on March 11 1993; he was 'confident that all necessary action has been taken; beef was safe.

January 19 1993 Safe for ruminant protein to be fed to poultry and non-ruminants. Renderers to meet 'Operational Standards'.

April 6 1993 letter from Sir Richard Body MP that BSE could be 'a major catastrophe'.

April 16 1993 letter from KC Taylor, Assistant Chief Veterinary Officer, Tolworth stating that pigs and poultry were no problem; pigs had 'far more animal protein'.

May 19 1993 letter clarifying terms used in regulations on feedstuffs: ruminants, animals, mammals, poultry: 'every species of bird' (includes ostriches). There was confusion over what kind of animal could be fed to what. Ruminants could not receive feedstuff made from ruminants but could still receive food made from non-ruminants (see note above).

May 21 1993 letter from Dr D Matthews, Senior Veterinary Officer, on Bovine Brain Disorder (BBD); occurred in Scotland beef cattle (80%) and all incinerated.

August 6 1993 BBD still occurring sporadically, presumably a different disease but little is known except its violence.

September 24 1993 Graphs of statistics in reply to my complaint that figures appeared to be 'classified' and unobtainable; explanation that the information was 'not readily available in tabular form'. This was very curious as the figures had been available in tabular form before.

Following further 'reluctance' to answer my enquiries, I sent a letter to several provincial newspapers, published anonymously, and the Cabinet Office suggesting that all was not as it should be. This produced a prompt reply from one T E D Eddy dated October 20 1993 assuring me that I was wrong to be suspicious and that beef was safe because no infected tissue could enter the food chain.

November 1 1993 J F Howard explained that from February 1992 suspected cases born after the ban were not to be killed until at least 7 days after a second visit 'to reassess ..progress' and many returned to normal; 91% proved negative in 1991; 26% in 1993. Manipulation of the figures denied.

November 24 1993 letter stating that date of birth and clinical onset of disease were 'recorded centrally' as 'the only dates of relevance'.

Explanation for delay in reply was Computer Change.

January 27 1994 Compensation changes February 14 1990, April 1 1994. Total £92,622,031

April 1995 Meat Hygiene Service created to supervise control measures.

July 8 1996 letter on regulations for knackers' yards: 'regularly visited by State Veterinary Service' under the Bovine Offal (Prohibition) Regulations 1989, Slaughterhouses Act 1974, Meat Regulations 1982.

22 July 1996 Media announcement that sheep offals are banned on advice from EU.

WHY THE CONTROLS DIDN'T WORK

The secrecy surrounding rendering operations and feed compounding seemed to infect those entrusted with their regulation. A reluctance, or inability, to impart information was a common feature in communications with local authorities. Letters to the County Health & Safety Executive produced replies referring enquiries to other departments in other areas; enquiries to Environmental Health Officers brought telephone calls from Trading Standards. Who was responsible? Changes in the system had transferred responsibility to other people with different titles. The person who was responsible in 1993 was no longer held accountable in 1996.

Formerly the bailliwick of local councils, supervision of control measures and the measures themselves had mutated into a bewildering mish-mash destined to confuse all concerned.

Tussles between central and local government abounded in at least one report. Lincolnshire Trading Standards Officer Peter Heafield described laws as 'poorly drafted', blaming central government for its 'failure to take account of local authority view'. A week of 'round-the-clock' inspections recommended to the State Veterinary Service was dropped due to lack of funds while banned materials were found eight times at a local rendering plant in the same period in 1995 and continued to be found as late as April 1996. Enforcement of 'unclear' and near impossible instructions continued to vex authorities charged with carrying them out. The regulatory structure looked distinctly vacuolar and subject to many variants at a time when MAFF announced that only one gramme of infected material could infect a victim⁹⁹. Laws drafted in Whitehall offices proved worse than useless down on the farm.

Government Ministers also changed. After Gummer came Waldegrave, Mrs Shephard and Douglas Hogg - but the assurances remained the same: British Beef is Safe.

Asking for information as a possible investor with two of the country's biggest agrobusinesses was similarly hedged with obfuscation and rebuttals. Dalgety denied involvement with rendering plants and Harrison & Crosfield reacted waspishly to my enquiries about possible compensation claims by suggesting my investment would be better made elsewhere. Nobody wanted to be associated with 'the scene of the crime'. A glance at the televised footage of the Kent rendering operation and anecdotal experience confirmed the impression that such places were at the bottom of the heap in matters of social desirability and public concern.

Anyone who has suffered the stench of rotting flesh will confirm that no-one in their right mind would want to work in such a place. Employees in the less modernised plants are unlikely to have good education or high communication skills. Like working in a fish factory, the stink clings to skin and clothes. Most of us would flinch from such employment. But someone has to do it. Left to their own devices for decades with minimum public interest or official intervention, can we blame them if they were not entirely conscientious about a few extra rules, especially if their pay was below average and their conditions appalling? After all, few in the food industry or government acknowledged a risk to human health. Most of the controls applied to 'animal health'.

Severe impediments to the swift study and assessment of the epidemic were the lack of records and an initial lack of planning. From November 1986 until June 1988 the disease gathered speed unrecorded, unchecked and unrecognised. There was no contingency plan to cope with the unforeseen emergency. Introduction of a temporary ban on feedstuffs was not taken seriously by an industry lacking regulation and control. Allowed to operate almost entirely according to its own standards and with only a mere Code of Practice to govern the late prohibition of possible 'comminglement' of diseased and healthy tissues,¹⁰⁰ the meat processing industry was 'an accident waiting to happen'.

Inability to enforce legal requirements, the soft-peddalling of 'tough new measures' as in the non-statutory 'advice' merely to 'avoid' the use of offals in human baby food, indicate a serious weakness at the top of the chain of command. Nobody was sacked or demoted for incompetence. Despite repeated assurances that 'Beef is Safe' it had become increasingly obvious that doubts abounded; 16 cases of new variant CJD attested it. The raw fact was that nobody knew. Unlike Germany the law did not protect the British public from such risks.

If Mad Cow Disease was indeed the bovine equivalent of Scrapie, commonsense dictated that affected herds should have been slaughtered in the same way that affected flocks of sheep were culled to eradicate Scrapie in the Antipodes. Instead we had muddle and more muddle while officials tried to escape the unpleasant and unenvied tasks incumbent upon them as guardians of the public welfare and paid employees of the State.

Moreover, if scientists believed BSE to be a mutant form of Scrapie, the very fact of its crossing the species barrier should have been a warning in itself. If the elusive prion could cross the species barrier, no animal could be safe, including humans. Mad cats,

mice, deer, pigs, apes, macaques, ostriches and many others bear witness. Once established, Scrapie is ineradicable. Only time will tell if BSE follows a similar path. If it passes through mice, it may also pass through pigs and poultry. As Sir Richard Body put it: BSE could be the horror story of all at the turn of the century.

The farming industry regarded BSE as a mere hiccup - but a hiccup causing paralysis and heart failure in the industry as a whole in Britain and abroad. Like the official scientists, farmers believed the disease was no threat to humans. Incidence of disease was kept quiet for various reasons, insufficient compensation payments, the associated depreciation in land prices, a general reluctance by officials to risk the anger experienced by their New Zealand colleagues during the Scrapie-eradication decades before. Cushioned by Common Agricultural Policies and British farm subsidies since World War Two, farmers were a class apart, secure in the unique privileges granted by a grateful nation. The farming community all but collapsed under a tidal wave of official indifference and callous disregard familiar to thousands of victims of pit closures and the demise of British manufacturing industry, all victims of the elusive but deadly agent called Greed.

CONCLUSIONS

As a housewife and mother who never gave anyone food poisoning in over 40 years' domestic cooking, I am horrified by the ambivalence of a government that placed economic considerations above concern for public health. Never in all the years when money was sometimes short and it was tempting to take a risk with dubious leftovers or specially cheap offers from food shops, never did I consider it worth while saving money at the expense of my family's health. Nothing 'iffy' was allowed to enter my family's food chain. Why did the Government play Russian Roulette with the nation's health?

The intensification of the methods of food production has condensed the number and variety of producers to a few major operations where the fiercely competitive nature of the profit motive has combined to produce an atmosphere of cavalier disregard for the welfare of the many in the interests of the few. Such is governmental concern for its own ideological relationships with large profit-making conglomerates that minds were closed to any other propositions than those which served to bolster the continuing success of the British food industry. The 'Blitz on Bureaucracy' hailed in Farmer's Weekly of June 25 1993, promoted by the Deregulation Unit of the Department of Trade and Industry to further praise in the July 7 issue could only increase disasters from which the farming industry may never recover.

I can only express outrage at such reckless and unjustifiable complacency 'in the name of the people', echoing Wilesmith's damning remark that 'no other country has such a combination of risk factors'.

The Government's insistence that 'science' should provide the sole basis for action on BSE ignored the one, and only one, proven scientific fact about the disease: Science did not know what it was. The scientists admitted their ignorance and the government failed to safeguard the public from that ignorance. .After a decade of officially-induced

inertia Bruere's words remain valid: 'We are still waiting for science'. Evidence suggests that BSE does indeed mimic Scrapie, a good enough reason to apply the same safeguards and to heed the warnings of Scrapie. Wishful thinking is not enough.

The lack of legal enforcement, the cutbacks in scientific and veterinary services, the miserly allowances for urgent research when fortunes are made simply by being in the right place at the right time, and the absence of respect for the rights of the ordinary consumer smack of cynicism beyond belief in a so-called 'civilised' country. The financial aspects alone are daunting, equalled by the sheer enormity of the problem of disposing of thousands of carcasses in an inadequate system already grossly overburdened. Farmers, butchers, auctioneers and many others sink in a morass of conflicting interests. Some markets have closed forever. While confusion reigns, opportunities for double-dealing and fraudulent scheming replicate in the nervous tissue of the body politic and economic, an infection that may be as ineradicable as Scrapie.

Failing to grasp the nettle with decisive action at the beginning of the BSE crisis left the government isolated in a mire of its own making. Any straws offering hopes of escape were clutched at as an election drew closer and tax cuts were threatened by the awesome consequences of inaction. Europe's demand for a greater cull was answered by mathematical modelling to suggest a smaller one. If patriotism is 'the last refuge of a scoundrel', statistical analysis must be the first. Mathematical models are the playthings of economists and even Anatole Kaletsky of The Times admitted that economists cannot predict the future (13/9/96). There was no mathematical model of future human casualties from eating 700,000 mad cows.

While uncertainty remained the only certainty, Sir David Naish of the National Farmers' Union confidently proclaimed British Beef 'the safest in the world' (BBC Countryfile 16/9/96).

From the blueberried highlands and rocky coasts of the State of Maine to the misty mountains and lochs of Scotland, the problems engendered by intensive farming methods and the disposal of waste faced, in the words of Peter Heafield, 'the likely collapse of the system', an environmental and human catastrophe beyond solution while Mammon holds the purse-strings.

As of 16 July 2000, 75 people have contracted nvCJD. A recent cluster of 5 cases including 4 deaths at Queniborough in Leicestershire is now under investigation.

26 Sept 2000 BBC R4 Today misreported 1000 cases per month at peak (per week); 30 cases per month now reported; 84 CJD cases in humans, 79 dead.

July 2000 revision from September 1996, originally submitted to official BSE Enquiry Mrs Joan Woolard, 35 Eastgate, Fleet Hargate, Lincs PE12 8NA.

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