
5 Mad Cow Disease and the Elusive Prion

5 Mad Cow Disease and the Elusive Prion

In 1986 a new disease of cattle struck the UK. At first it looked like a fairly rare, rather esoteric disease of more interest to white-coated scientists than to anyone else. But within 5-years the disease had resulted in collapse of the UK's prestigious beef industry, a political furore that resulted in the Brits no longer trusting what they were told about food safety, and the rest of the world looked on with horror as people contracted the disease from their food and later died in an horrific way with no hope of cure. This, of course, was Bovine Spongiform Encephalopathy (BSE), or Mad Cow Disease as the UK press christened it. It changed the face of food safety and diminished public trust in politicians and scientists throughout the world.

A New Disease Emerges

During April 1985 a veterinary surgeon, Colin Whitaker, examined a cow on a farm in Ashford, Kent in the UK. The normally placid animal was behaving oddly and becoming hyperactive and aggressive towards the farmer and found it difficult to control its limbs (ataxia) so it staggered about when it tried to walk. The cow's symptoms closely resembled Staggers, a condition known for many centuries and caused by a lack of magnesium in the diet. Vets normally treat Staggers with magnesium, but in this case the treatment had no effect. In fact the animal got

worse. Mr Whitaker referred the case to the Central Veterinary Laboratory (CVL) in Surrey, UK for the experts to have a look.

At first the CVL scientists and vets thought that the cow might have been poisoned, perhaps with an organophosphorus (OP) pesticide that attacks the nervous system which would explain both the behavioural changes and the animals difficulty in controlling its limbs. A great deal of work ruled out poisoning, and left the scientists with no explanation. They were almost at the point of accepting that they had been beaten by the case, but that it was probably a one-off and would have to be accepted as one of life's unexplainables. Then another case arrived, and another, and another, until, by the end of 1986, nine herds had been affected often with more than one affected animal per herd.

The incidence of the disease increased dramatically until its peak (7,267 herds affected) in 1992, with a steady decline to the latest figure of 18 herds affected at the end of July 2003 (Fig. 5-1).

By 2003 there had been a total of 180,166 cases. Nobody in their worst nightmare would have predicted an epidemic of such devastating proportions to have followed that first ataxic cow in April 1986.

What is BSE?

When the pathologists examined the ataxic cow they found that the brain looked very characteristic under the microscope. It had numerous "holes" that gave it a spongy appearance. This is indicative of a group of diseases called the transmissible spongiform encephalopathies (TSEs):

- *Transmissible* can be transferred from one animal to another
- *Spongiform* because of the spongy microscopical appearance of the brain
- *Encephalopathy* disease of the brain (*cephalos* – Greek for head; *pathos* – Greek for suffering)

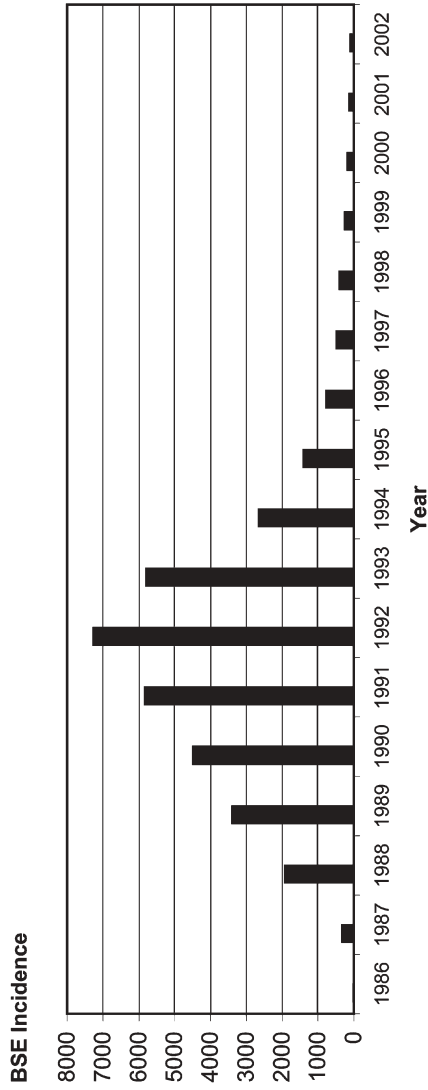


Fig. 5-1. BSE incidence in the UK (data from the Department for Environment, Food and Rural Affairs [DEFRA], UK at <http://www.defra.gov.uk/animalh/bse/bse-statistics/bse/herds.html>)

This was the first time that a TSE had been seen in a cow; it was a brand new disease about which nobody knew anything.

Other Animal TSEs

The TSEs comprise a group of quite unbelievable (their causative agent is truly fantastic, but more of this later...) diseases of many animal species, including humans.

Scrapie

Scrapie of sheep and goats was the first TSE to be discovered and has since become the most studied and reviewed of all the STEs. Until the early part of the 20th century this disease was rarely considered by vets, not because it was a new disease, in fact it was first described in 1732, but because shepherds often tried to conceal it. Scrapie is thought to have started in Spain and is now widespread in Europe, Asia and America but has never been seen in Australia or New Zealand. Trade in live animals spread the disease around the world. New Zealand's and Australia's strict biosecurity laws protected these countries.

Scrapie sheep have been found in 35 counties in England and Wales and it has been suggested that about a third of flocks in the UK are affected.

Scrapie can be transmitted within flocks by infection of lambs at birth or by other members of the flock contacting (perhaps eating) afterbirth tissue (e.g. the placenta).

Transmissible Mink Encephalopathy (TME)

TME was first identified in Wisconsin, USA in 1947 with further outbreaks in the 1960s. The symptoms and progression of the disease are strikingly similar to scrapie. In captivity mink are often fed a diet derived from animal remains, including sheep meat and bone, for this reason it was suspected that TME was due to infection of mink with the scrapie agent.

Chronic Wasting Disease (CWD) of Deer and Elk

Chronic Wasting Disease was first noted in 1967 in captive mule deer in Colorado, USA. It closely resembled scrapie and is known to be transmissible within species. In the period 1981–1995 CWD was confirmed in 49 free-range deer from North Central Colorado (USA), and later in 2002 there was an outbreak in deer in the mid-west of the USA.

Feline Spongiform Encephalopathy (FSE)

FSE was first identified in a five year old Siamese cat at the Bristol Veterinary School, UK. Retrospective examination of tissue sections from cats dating back to 1975 revealed no similar cases and therefore FSE was designated a new disease. By October 1996 75 cases had been reported in the UK. In addition, by 1997, FSE had been confirmed in 2 pumas, 6 cheetahs, 2 tigers and 1 mountain cat in UK zoos, suggesting that this is a disease of the cat family rather than being confined to the domestic cat.

Human TSEs

Creutzfeldt-Jacob Disease (CJD)

In 1920, Hans Creutzfeldt reported a new and unusual neurological disease. The following year Alfons Jacob reported four cases of progressive fatal dementia which he grouped together as examples of “spastic pseudosclerosis” and believed that they resembled the case originally described by Creutzfeldt. The disease now bears both names in recognition of their contribution. The clinical course and features of CJD have been well documented. CJD is a rare disease which is found world-wide at a rate of approximately 1–2 cases per million population per year.

Kuru

Kuru is a condition confined to highland New Guineans in the mountainous interior of Papua New Guinea and is believed to result from ritualistic cannibalism. The brain of deceased relatives was eaten by women and children and the muscle by men. The level of infectivity was greatest in the nervous tissue and this is reflected in the fact that women and children were those mainly affected by Kuru. This cannibalistic practice has disappeared over the last 30 years and by 1985 the disease was no longer seen in anyone under 35 years of age. Our knowledge of Kuru was instrumental in sorting out how BSE spreads.

Gerstmann-Strausser-Scheinker Syndrome (GSSS)

In 1928 Josef Gerstmann reported “An interesting case of hereditary familial disease of the central nervous system”. After his patient’s death in 1932, Gerstmann joined with Ernst Strausser and Isaac Scheinker to publish a detailed case report. GSSS is a rare disease which occurs at an order of magnitude lower rate than CJD. It only occurs in families and is closely related to CJD, it is genetically controlled and occurs in families by inheritance of a group of genes.

The scientists investigating BSE soon realised that it was a TSE and were able to call upon the vast amount of knowledge of the other TSEs to unravel this incredible new disease. In the five or so years following the discovery of BSE an unprecedented research effort resulted in a good understanding of the disease, its causative agent, and its means of transmission. It is a pity that this truly wonderful research was lost amidst the political argument that raged about the disease and its possible effects on consumers ... but more if this later.

What Causes BSE?

Stanley Prusiner had been working on Scrapie at the University of California, USA for many years. He, and others, had tried to identify the cause of scrapie.

- Was it a bacterium? No, bacteria can't survive 100°C (since then bacteria living around volcanic vents deep in the ocean that thrive at temperatures above 100°C have been discovered) – boiling scrapie extracts did not significantly reduce their infectivity.
- Was it a virus? This was a bit more difficult to decide, but most scientists thought not because scrapie extracts treated with UV or γ -radiation were still infectious and viruses are killed by these high energy rays.

So what causes scrapie? Professor Prusiner discovered a truly amazing causative agent. He called it a proteinaceous infectious particle or *Prion*. It is a protein. It is not alive. But it behaves just like any other infectious agent in that it replicates itself in the infected animal's body. Many scientists simply did not believe this apparently far-fetched hypothesis.

However the prion has stood the test of time and incredible scientific scrutiny, and now it is accepted as the causative agent of the TSEs. Prusiner was awarded the Nobel Prize for Medicine in 1997 for his remarkable discovery – his tenacity paid off.

What is the BSE Prion and How Does it Cause BSE?

Prions are medium sized proteins (molecular weight = 33–35,000 daltons), they are found in most, if not all, cells and are thought to play a role in communication and recognition between cells. They are called cellular prions (or PrP^{C} in scientific jargon – PrP stands for *prion protein*, and C stands for *cellular*). There are damaged forms of prions which resemble very closely PrP^{C} , but are different enough not to function properly – indeed they are highly dangerous. These are the so-called scrapie prions

(PrP^{SC}), but in fact are the TSE prions – they are called scrapie prions because they were identified from scrapie sheep.

So, what is the difference between PrP^C and PrP^{SC}? The simple answer is, very little. They have the same amino acid building blocks in their protein structures, they have the same molecular weight, but they have one very important difference. The shape of their protein make-up is different. Proteins are made up of long strings of amino acids that are folded to make complex structures. The folding (i.e. conformation) of PrP^C and PrP^{SC} is different. This apparently small difference in shape makes an enormous difference to their biological activity – this story is almost incredible, I never cease to marvel at the ingenuity of the prion as a disease causing agent.

Protein biochemists have given names to the different types of protein molecule folding. PrP^C has a lot of α -helix, PrP^{SC} has more β -pleated sheet. α -Helix proteins look like spiral staircases (well they would if it were possible to magnify them enough), β -pleated sheet proteins look like stacks of plates or folded sheets (Fig. 5-2).

Now comes the amazing bit. If a molecule of PrP^C comes into contact with a molecule of PrP^{SC} the PrP^C is pulled into the same molecular shape as the PrP^{SC}. This is called an induced conformational change because one molecule has induced a change in shape (conformation) of another. This, of course, has significant implications because it means that dangerous PrP^{SC} can be created from safe PrP^C. The implications become even more worrying when the PrP^{SC} is in the brain next to PrP^C molecules doing their important job helping cells to communicate with each other. The multiplication of PrP^{SC} in this way is devastating to the brain's function. It stops cell to cell information flow and causes very serious brain malfunction (Fig. 5-3, see also p. 111).

Are Prions Alive?

When scientists first saw the PrP^{SC} replication process they thought that the prion was dividing and growing like a virus. ——— This is not the case. The prion is simply a chemical that is able

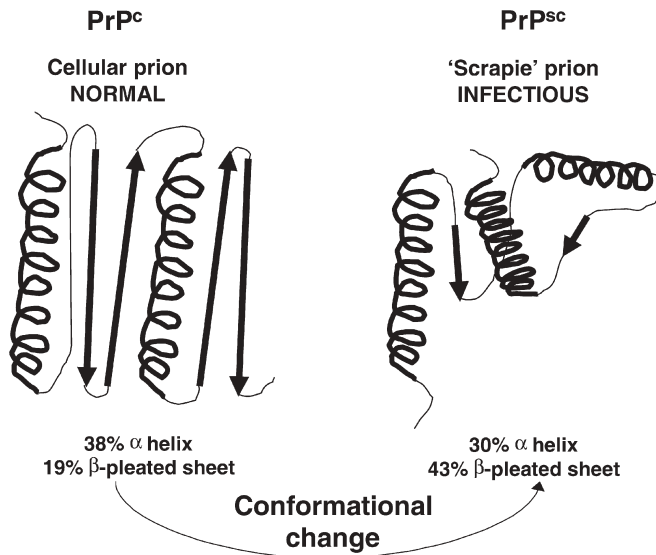


Fig. 5-2. The differences between the “normal” and BSE prions and the shape change that leads to BSE – the curly line represents α -helix protein; the arrows represent β -pleated sheet protein

to reproduce its form by changing other molecules into the same shape as itself. It is a very devious poison.

What Happens if you Eat Beef Infected with BSE Prion (PrP^{Sc})?

In the early days of our understanding of TSEs it was thought that nothing would happen. After all Scrapie was first described in 1730s, many people have eaten infected lamb since then, but nobody had contracted Scrapie. But humans get CJD, could this be the human form of Scrapie? Studies showed that Scrapie and CJD are not connected – CJD is caused by a spontaneous change (mutation) in the gene that codes for human PrP^C. The mutation causes the synthesis of a CJD prion rather like PrP^{Sc} that has just the same devastating effect.

The UK government took this information to heart and were unable to move with changing scientific ideas. Partly be-

cause they did not want to believe the evidence that was beginning to appear, and partly because the scientists advising them were uncertain – this was cutting edge science in a field that was almost unbelievable, and the implications would be devastating to the UK's farm economy.

It was becoming clear that the BSE prion was different to the Scrapie prion. It behaved differently. Might it jump species? Might human consumers contract it if they ate meat from a BSE cow? These were real scientific questions, based on uncertainty, but they had no answers. A great deal of very expensive research was necessary to answer them. But there was no time – answers were needed NOW!

The press got hold of the possibility that people might catch BSE – a furore of unprecedented proportions broke out. The Brit's were scared. Would they catch this terrible disease? Would everyone who had eaten beef since the onset of BSE die a terrible death? There was confusion and fear.

Then came the worst possible news. Researchers had shown that a new form of CJD, which they called new variant CJD (*nvCJD*) was caused by eating BSE-infected meat. In the UK at this time, there was that terrible feeling of a lull before an enormous storm. Scientists and medics alike were shocked, silent, even frightened. What were the implications? Would our worst fears be realised?

nvCJD

nvCJD or *vCJD* as it is now usually called, first appeared in England in 1995/96. The cases were very like CJD, but unlike CJD they occurred in young people – CJD is a disease of later life, not usually occurring before 50 years old. The peculiar CJD cases were in people in their teens and 20s. The first case was considered a curiosity. Then came another, and another – just like BSE in cattle. By 1996 there were 5 early onset CJD cases. They had strikingly similar symptoms.

The first case was in a dairy farmer, then came a teenager, then a 28 year old woman. Their symptoms were loss of

memory, confusion, mood changes, difficulty walking, loss of coordination, dementia, and death. A terrible scenario, horribly reminiscent of BSE.

All of the early onset CJD (as it was then called) patients died within 14 months of the onset of symptoms (this is different to CJD where the period to death is only 4 months) and were found to have identical brain microscopical appearance at post mortem examination. They looked strikingly similar to BSE.

Since the first cases in 1995, there has been a meteoric rise in cases, until a peak occurred in 2000. Then followed the long awaited decline (Fig. 5-4).

Cases of ν CJD have now been recorded in other countries. Most have been traced back to consumption of UK beef.

How Does the Prion Get to the Brain?

If meat contaminated with the BSE prion is eaten the prions pass through the stomach unscathed (they can withstand stomach enzymes that break down proteins), are absorbed across the intestine – like other food components, and find their way to the spinal cord (possibly via the lymphatic system). When they reach the spinal cord they slowly move up towards the brain. They are not alive remember, so they don't propel themselves in any way. They simply diffuse like any other chemical. This process is slow. It takes many years (this is the “incubation period” of the disease); in humans this probably takes about 10 years (the first case of ν CJD was seen in 1995, and the first case of BSE was in 1986, so the first human exposures were also in 1986; i.e. 9 years from exposure to onset).

When the prion gets to the brain it meets brain PrPC and converts it to PrPSC and there is no going back. ν CJD and death are inevitable. It is likely that the concentration of BSE prion in the consumed meat is a very important determinant of ν CJD risk. Nobody knows how much BSE prion is needed to result in ν CJD, but it is thought that only large doses are certain to cause the disease. This probably explains why the incidence of ν CJD was so low compared to the theoretical exposure of consumers

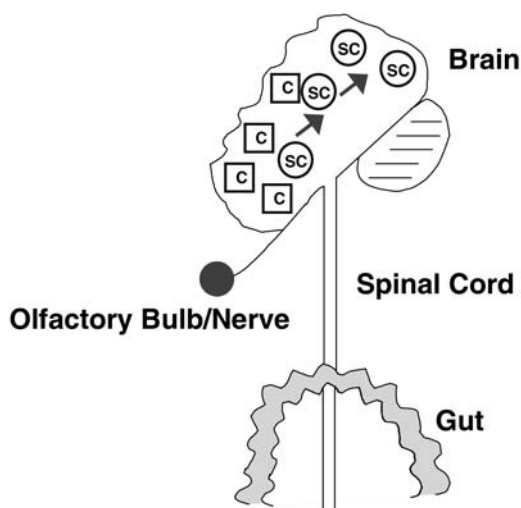


Fig. 5-3. The BSE prion replicates in the brain by changing the shape of normal prions that it comes into contact with

to BSE beef between 1986 and 1989 when the Offals Ban was introduced.

What Did the UK Government Do to Minimise BSE Risk to the Consumer?

Let's back track a little. The first case of BSE was confirmed in 1986, but it was not until 1996 that the first case of vCJD was confirmed and the link between the two diseases confirmed. So, before 1996 there was no evidence that BSE could affect human consumers. I point this out because it is very easy with hindsight to criticise the action of the UK government in the way that they minimised the risk that they knew nothing about.

As research on BSE unravelled, what causes the disease and how it was transmitted, it became possible to introduce risk management strategies to minimise human exposure. The first knee jerk reaction was to ban eating beef. This was a ridiculous suggestion in a nation where roast beef and Yorkshire pudding is the national dish (the French even affectionately call – I think!

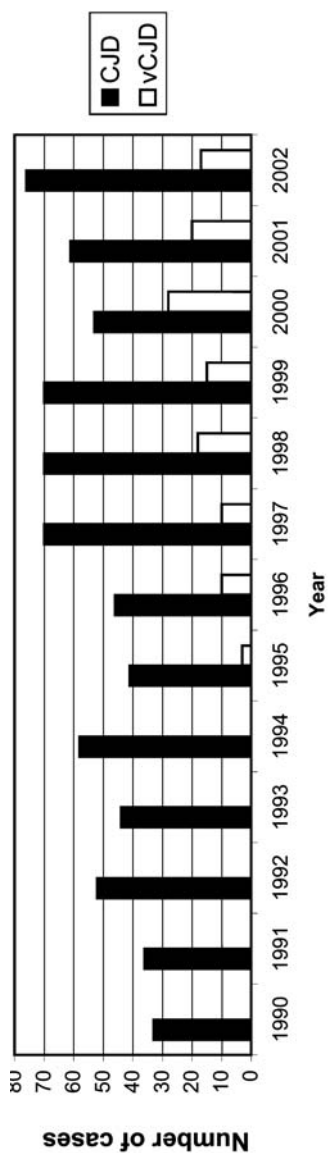


Fig. 5-4. The incidence of CJD and vCJD in the UK showing the long awaited decline that began in 2001 (data from the CJD Surveillance Unit, Edinburgh, UK. See <http://www/cjd.ed.ac.uk/figures.htm> for up to date figures)

– the Brits “rostbifs”). But it was a starting point for the risk managers to grapple with. Research had shown that the BSE prion is at highest concentration in the CNS – so the first risk management approach was to ban eating brain and spinal cord in 1989 (the Offals Ban). You might think that not many people eat the CNS, but in fact most of us did. Brain and spinal cord were included in the mixtures of meat used in making sausages, meat pies, beef burgers, etc. Since the Offals Ban was not introduced until 1986, the Brits (and visitors to the UK) had been exposed to BSE meat for 3 years.

As we learnt more about the BSE prion and where it occurs in the cow’s body, other tissues were included in the Offals Ban (e.g. parts of the intestine). Milk was shown to be safe and other tissues (e.g. muscle) were very low risk.

The Offals Ban was a good risk management strategy that worked well. We did not know this at the time, but looking at the vCJD statistics show us that exposure between 1986 and 1989 probably resulted in the 136 human deaths that have occurred so far (up to September 2003 – there will be more, but not many). The Offals Ban very significantly reduced human exposure to the BSE prion which explains the onset of vCJD decline in 2001. If risk management had not been introduced we would have had a vCJD epidemic of frightening proportions. The sadness, of course, is that anyone had to die, but due to the incredible science that was conducted over a ridiculously short period of time, I am able to report than hundreds rather than 10s of thousands of people died. We should congratulate the scientists, not ridicule them.

Since 1996, there has been a steady accumulation of evidence, often tragic, that supports the vCJD from BSE beef theory. Perhaps the most heart-wrenching was a cluster of vCJD cases that appeared in the Queniborough, Leicestershire, UK in between 1998 and 2000. Five people died of vCJD which is an incidence very much higher than would be expected for such a small village (population 1,800) on the basis of the national statistics (for this population even a single case of vCJD is a very remote possibility). It turned out that a local butcher used traditional butchery methods that might have led to cross contamination of meat from a BSE cow. The butcher’s knives were

suspected; perhaps an infected brain was cut, then the same knife used to cut some meat.

Does Cooking Destroy the BSE Prion?

The BSE prion is an incredibly resilient protein. As discussed above, it can withstand enzymes that destroy most proteins, it is acid and base stable, and is not destroyed by normal cooking temperatures. At 138°C it begins to lose its activity. This temperature might not sound very high, but most meat only reaches 60–70°C in its centre during cooking. If you made sure that your roast beef was kept at 138°C for long enough to destroy the prion, you would end up with a solid charred mass. Safe (perhaps) but inedible! Therefore however hot you cook your food the BSE prion will survive.

It is interesting that if you look at CJD incidence statistics (from before we knew about vCJD) you will see that medics and paramedics have a greater incidence than the general population. This is because neurosurgeons sometimes cut themselves accidentally with their surgical instruments. If they have operated on a CJD-infected patient, who has not yet begun to show the symptoms of the disease, they might infect themselves with the CJD prion. The normal sterilising procedures used in hospitals use pressurised steam at 121°C (autoclaving) is not hot enough to deactivate the prion. When this was realised, new procedures were introduced to sterilize surgical instruments used in CNS surgery – they now have to be dry heated in an oven at 140°C to be sure to destroy infectious prions.

Why Was the First Case of BSE in 1986?

This is a key question. What happened, or changed, in farming practice to cause the disease. Or was it just a fluke?

Prions are passed from animal to animal orally (including humans, remember Kuru). If a non prion-infected animal

The incidence of CJD in selected* occupations in the UK between 1990 and 1995. This shows that medics and paramedics are more likely to die of CJD than non-prion exposed groups. The data are from the annual report of the CJD Surveillance Unit, Edinburgh, UK (1995)

Occupation	CJD Incidence/million
Managers	0.3
Secretaries/clerical workers	0.5
Shop workers	1.7
Medics/paramedics	5.7
Teachers	0.7

* Some data have been omitted because there is not sufficient space in this section to discuss in detail the higher incidences in some other professions.

eats a prion-infected animal it is likely to contract the prion disease (e.g. BSE). But cows don't eat cows ... or do they? It is true that cows are not carnivorous by nature, but farming developments have forced them to eat each other by using meat and bone meal (MBM) as a component of cattle feed. This began a long time ago, and was a way of putting back valuable elements (e.g. calcium) into farm animals' food chains.

When farm animals are slaughtered, their meat is removed from the carcass, bones, plus some attached meat are left. This is treated to remove the fat – the product is tallow used to fry food in. The remaining material is dried and ground up to make MBM. This used to be added to animal feed to prevent waste and to get rid of a waste product that otherwise would be of very little use. This is the ultimate in recycling.

Until the early 1980s in the UK, the tallow was extracted from the animal remains after the meat had been recovered using solvents. The solvents were then evaporated off to leave the fat which was sold to fish and chip shops and the like. The remainder was dried and ground to make MBM. In the 1980s the process for recovering tallow was changed. Instead of solvents, heat was used. The carcass minus its meat was passed through an oven and the tallow melted off. This removed the need for solvents and allowed the process to be streamlined.

It is thought that the solvents inactivated the BSE prion, but the heating process did not. So, the change in process allowed the BSE prion to survive and infect cattle via the MBM in their feed.

This still does not explain fully where BSE came from. Some very elegant work at the Central Veterinary Laboratory in Weighbridge, UK came up with the answer.

The Origins of BSE

The first few cases of BSE were all associated with a single animal feed producer in the south of England. It was thought that scrapie sheep MBM had been used to make cattle feed and that the scrapie prion had jumped the species barrier and infected cows. The cows then formed a new batch of MBM, which was fed to more cows so amplifying the disease. This mechanism of infection would very quickly result in a widespread epidemic. The Scrapie hypothesis was accepted for quite some time, but it did not quite ring true. Scrapie is a common disease and so why did only one feed producer transmit the prion? This simply did not make sense. A better, and now widely accepted, explanation is that a prion mutant cow – i.e. a cow making defective prion (PrP^{SC}) was produced randomly due to a spontaneous and ‘natural’ change in the gene (mutation) that codes for the prion protein, this cow was made into MBM and incorporated into cattle feed. This is a far better explanation of the point source of infection. So the fact that BSE occurred in the UK was just damn bad luck. The mutation could have occurred anywhere. Indeed such mutations might happen from time to time, which might explain the few BSE cases around the world that cannot be traced back to UK feed or UK breeding stock. So countries that say they are BSE free should beware – nature might well prove them wrong!

Can BSE Be Transmitted from Cow to Cow?

There are only two ways that the BSE prion can be transmitted from animal to animal:

-
- Horizontally – by a cow eating BSE-infected MBM
 - Vertically – at birth a calf can be infected from its BSE infected mother

It is not possible for one animal, or a person for that matter, to catch a TSE from another animal or person in any other way. In people, *v*CJD is transmitted from cow to human horizontally, whereas Kuru is transmitted from person to person horizontally. There is no known vertical transmission.

The Politics of BSE

The risk of getting *v*CJD from BSE-infected beef is very low indeed. There have been 136 *v*CJD deaths in the UK out of a population of about 60 million. Not all of the population eat beef, and so some could never have been exposed to the BSE prion. If we assume that 40 million Brits eat beef (in reality it is likely to be more), if they were all exposed in the same year (which of course they were not), the risk of “catching” BSE is 1 in 294,000. This is small, but the real risk is much smaller because exposure was over at least 4 years (i.e. the period before the Offals Ban). Despite the relatively low risk, consumers around the world shunned UK beef and beef products. The UK beef industry collapsed.

Politicians were desperate to support this important industry and so hawked the “there’s no risk to the consumer” story. This was stupid. They were doomed to be proved wrong. There is no such thing as no risk. It would have been better to explain the issues to the public, but they were immensely complex, and the science was unravelling as the news stories broke. The result was a series of knee jerk reactions, contradictory stories, changed advice – all of which undermined the public’s confidence in both politicians and scientists. It will take many years to heal this wound.

A look at some of the newspaper headlines published around the world throughout the Mad Cow Disease saga says it all.

Beef brains banned in food

The Times, 14 June 1989

Mad cow disease ‘now in decline’

The Independent, 23 September 1993

Human ‘Mad Cow’ deaths at new high

The Daily Telegraph, 6 October 1995

Death toll from brain disease increases

The Times, 8 October 1995

Top scientist adds to BSE warnings

The Independent, 4 December 1995

Why we should all give up beef

The Independent, 7 December 1995

Food giant may sue BBC in beef scare

The Independent, 7 December 1995

Lax regulations to blame for BSE, says new study

The Independent 12 December 1995

Expert warns of epidemic

Evening Post [Lancashire, UK]. 21 March 1996

Latest BSE scare puts farming in crisis

Farmers Guardian, 22 March 1996

Beef industry faces ruin as bans spread

The Times, 23 March 1996

Mafia linked to sale of herd infected with BSE

The Times, 27 March 1996

Birds Eye stop making burgers

The Times, 27 March 1996

Food firms try to cut all sources of British beef

The Times, 27 March 1996

Businessman, 42, latest suspected victim of CJD

The Times, 3 April 1996

Cattle slaughter may be doubled to 30,000 a week

The Times, April 12 1996

Laboratory mice carry secret of human BSE risk

The Times, 25 October 1996

We won't swallow any more lies about food

The Independent, 31 January 1997

CJD kills five around village

The Sun, February 10 2000

Transfusions can spread BSE

The Times, 15 September 2000

Madness in the blood

The Sunday Times, 24 September 2000

Mad-cow fears prompt EU meat ban

The Press [Christchurch, New Zealand] 8 January 2001

Warning of second wave of vCJD

New Zealand Herald, 16 May 2001

BSE sold a great many newspapers, destroyed thousands of farmers' livelihoods, lives seriously impacted on the global beef industry, has killed over a hundred innocent consumers, ended the careers of a handful of politicians, but led to some of the most wonderful science of our time. This was truly a disaster, but it was a triumph of discovery too.

STANLEY B. PRUSINER

1997 Nobel Laureate in Medicine – *For his discovery of Prions – a new biological principle in infection.*

<http://www.springer.com/978-3-540-21286-7>

Is it Safe to Eat?

Enjoy Eating and Minimize Food Risks

Shaw, I.

2005, XI, 251 p. 55 illus., Hardcover

ISBN: 978-3-540-21286-7