SUSAN ATKINSON

BEARING ALL THEIR FAULTS AWAY

Skapegoats in Agriculture

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BIOPIC

Susan Atkinson originally trained as a teacher and is a married to a farmer. Together they run a 200-acre farm in South Nottinghamshire. The farm has been in her husband's family for 90 years. During this time it has seen many changes. It is now mostly arable and is involved with various environmental schemes.

Over the years Susan became increasingly concerned about issues connected with farming and the environment. This concern prompted her to start writing letters and articles, which have been published in farming and Christian publications as well as local newspapers.

She wrote *The Money Go Round*, a booklet on globalisation and farming, which was published in 2002 by the Agriculture and Theology Project. In 2007 she contributed to a book about the social and cultural impact of foot-and-mouth disease crisis in the UK in 2001, published by Manchester University Press Her chapter is titled "FMD and the abuse of democratic process – a farmer's perspective." Susan is a member of the national committee of Agricultural Christian Fellowship and is a member of the UK Food Group. Amongst other things she is a former trustee of Farmers' World *network* and a volunteer with Farm Crisis Network.

Contents

Foreword

Aaron is to offer the bull for his own sin offering to make atonement for himself and his household. Then he is to take the two goats and present them before the LORD at the entrance to the tent of meeting. He is to cast lots for the two goats—one lot for the LORD and the other for the scapegoat. Aaron shall bring the goat whose lot falls to the LORD and sacrifice it for a sin offering. But the goat chosen by lot as the scapegoat shall be presented alive before the LORD to be used for making atonement by sending it into the wilderness as a scapegoat. (Leviticus 16.6-10 NIV)

In the ancient world people developed all kinds of rituals involving scapegoats to restore broken relationships. In the First Testament we have a record of one such ritual played out between the people of Israel and God.

In our society the practice of scapegoating is different in significant ways. There is no awareness and confession of wider responsibility and general atonement. Rather there is a rush to find the second goat and to heap all blame onto it, leaving nothing else changed.

This is the story of a local CJD cluster – a small group of people afflicted by new variant Creutzfeld-Jakob Disease at the height of the BSE outbreak in cattle. After careful enquiry Susan came to believe that the search for the cause of the outbreak turned into an example of scapegoating.

We will be looking at a small number of farming scapegoats, not in order to take our turn in blame games, but because there are important lessons to learn. The chosen scapegoats are unjustly blamed and suffer severe consequences, whilst the real causes of a problem remain undiscovered, or unacknowledged and therefore unremedied. That, of course, goes a long way to ensure that the problem will recur. Looking ahead, the challenge and complexities of climate change will offer many opportunities for scapegoating – pouring blame on the weak or inarticulate rather than confronting real problems, powerful interests or cherished illusions.

Christopher Jones

CHAPTER 1

The Beginning

How I became involved

In the late 1990s it was realised that, of the then slightly more than one hundred cases of variant Creutzfeld-Jakob disease (vCJD), five had connections with north Leicestershire, particularly in the area around a small village called Queniborough, which soon found itself in the spotlight for all the wrong reasons. An investigation was ordered and carried out by the Leicestershire Health Authority (LHA), but the resultant report came out to a mixed response. As many had expected, it tried to prove the cause was BSE infected beef and pinned the blame on the methods used by local butchers. However, the reasoning was questionable and residents of Queniborough, who knew all the people concerned, rejected the report. They wanted the truth. This could only be done by thorough investigation of the facts, not by putting forward a hypothesis and then looking for the evidence for it. After I had a letter concerning this report published in the *Leicester Mercury*, I was contacted by some of Queniborough's residents and invited to visit them to hear their story. This I did.

...in the spotlight for all the wrong reasonsin the spotlight for all the wrong reasons.

The LHA had published a report of their findings after their investigation, carried out by two doctors, in 2001. The investigation focused on the period 1980-1991, as being that of common exposure. It stated the victims had lived in both the Wreake and Soar valley areas. In fact the investigation covered a very wide geographical area. It looked at farming practice in the area, butchery methods and the lives and diet of the victims. The report based its conclusions on the hypothesis that "there was an association between the cases of variant Creutzfeld-Jakob Disease and the consumption of beef purchased from

butchers where there was a risk of 'cross-contamination' of beef carcass meat with bovine brain."

The essential elements of their hypothesis were that:

- Infective material came from locally reared cattle
- The beef cattle were a by-product of the dairy industry and were fed meat and bone meal from day six onwards giving them a greater lifetime exposure to feedstuff that was potentially contaminated with the BSE agent
- They were predominantly Friesian crossbred cattle which were slow to fatten and therefore slaughtered at close to three years
- The cattle pinpointed were slaughtered in small abattoirs which employ a technique known as pithing without the washing down of the carcass
- The heads were split to remove the brain
- During brain removal, if the meninges (the membrane that covers the brain) are broken, because of a gelatinous consistency, when handled it then has a tendency to be adherent
- Carcasses were wiped with cloths increasing the risk of cross-contamination
- The vCJD victims ate beef from these abattoirs

In other words the report's findings hang on where the vCJD victim meat came from, how it was butchered, what cattle the meat came from and how these cattle were fed.

It also rested on more general assumptions about the origin of BSE and its means of spreading, and the relationship between BSE and vCJD. The first was that however the BSE epidemic in cattle arose, the principal means of its spread was the feeding of meat and bone meal from cattle carcases to cattle as a protein supplement. The second assumption, that was perhaps less certainly held, was that vCJD in humans resulted from infection from certain parts (e.g. brain and spinal column) of BSE infected cattle. (I will look first at the particular assumptions of the report).

The following is what I was told by people who had lived in Queniborough throughout the relevant period, who knew the victims and their families and, in some cases, had been interviewed by those compiling the LHA report. In this chapter, the facts that they gave me, and the results of my own research, are set out.

However, before looking at the evidence from Queniborough itself, it is necessary to look at the basic facts about cattle rearing and the history of BSE.

CHAPTER 2

Bovine Normality

Methods of Cattle Rearing

Cattle are mammals and, like all mammals, produce milk in order to feed their young. The ancestors of our domesticated cattle only produced enough milk to rear their calves. However, after many hundreds of years of selective breeding there are now both dairy and beef cattle. The latter are bred to convert feed into muscle (meat) and give only enough milk to rear a single calf: between one to one-and-a-half gallons a day. Dairy cattle give several times more milk each day than a calf would need but are relatively poor at gaining flesh. However, a dairy cow still has to give birth in order to give milk and is then milked for ten months, then dried off and rested for two months before giving birth again.

Most of the dairy calves born each year are not required for breeding purposes, even if female. They are used for beef. The most common practise is for a dairy farmer to breed (usually by artificial insemination) the highest yielding cows to dairy bulls and the rest to bulls from the various beef breeds. The pure dairy-bred male calves were mostly exported to France (until BSE stopped exports) and reared as veal. Until the 1960s, the most popular beef-dairy cross was the Hereford-Friesian, a moderately fast growing animal that produced good meat and, if female, could be reared as a suckler cow and bred to another beef breed (e.g. South Devon) producing yet another very good meat animal, Thanks to the Friesian part of its make up, it's able to produce enough milk to have a second calf adopted onto it. However after the introduction of the Charolais breed from France in the early 1970s (quickly followed by breeds such as Simmental, Limousin or Blonde D'Aquitaine), the Hereford crosses declined in popularity. This is

because these Continental breeds, when bred with dairy cows, produce a faster growing, leaner animal. By the mid 1980s, most dairy cross calves were sired by Continental bulls and Hereford-Friesian crosses were a minority.

All this means that calves intended for beef production grow up in two ways. The offspring of suckler cows, purebred beef or crossbred, stay on their mothers for much of their first year and have no need of high protein cattle food. Towards the end of their life they probably receive some concentrate food. The offspring of dairy cows, pure or crossbred, on the other hand, are rapidly transferred to milk substitute and after a few weeks to a high protein solid concentrate. Either of these could well have contained meat and bone meal. "Could have" because ready-made cattle feed was sold without any list of contents. The third source of beef was from dairy cows whose working life was over. They consume higher protein concentrate both as calves and in adult life.

The English Midlands has always been a beef rearing area as it is relatively cheap to bring calves here from the dairy areas to the west of the country and straw and fodder crops from the arable areas in the east. Calves reared in Leicestershire, by whatever means, need not have been born in that county. In fact, when we were rearing cattle (though our farm is in Notts) all our calves came from Cheshire via Crewe market.

The composition of the concentrate feed also varies. Some farmers buy in some or all of their concentrate in ready mixed forms containing various grains etc while others mix their own rations and include for example barley, sugar beet pulp, soya bean pulp, brewers grains (barley-based waste from the brewing industry), maize and beans. For many years before BSE the government had resisted farmers demands that ready mixed feed had a list of ingredients attached. Instead, the bags just had a label stating how much protein, oil and ash the mixture contained plus a list of vitamins and minerals that had been added. It was impossible to know what the mixtures contained, but, besides

meat and bone meal (MBM), it was legal to include dried poultry manure, hydrolysed chicken feathers and other doubtful substances. Some farmers who mixed their own rations used bone meal, but many herds that did so never had a case of BSE. Once again, this was part of the drive to make food cheaper. Waste products like MBM were cheaper to feed than other proteins and so the resulting meat could be sold at a lower price. At the time the scientists claimed it was perfectly safe to feed MBM to ruminants even though it not only turned herbivores into carnivores but into cannibals. At some point the government legalised a change in the process for sterilising MBM. It is thought that is when MBM could have become an agent in the spread of BSE. It is impossible to know what cattle were fed MBM in any feed (from milk powder onwards).

CHAPTER 3

Bovine Calamity

The Outbreak of BSE

Early cases of BSE were reported in 1986 and were promptly publicised. Farmers realised immediately that they were looking at a new disease (though there may have been one or two cases earlier than this). However, the government was slow to react. First it insisted there was no problem. Then it said there may be a few dozen cases, then a few hundred. Finally it admitted there was a serious problem. Because of the symptoms, attention was immediately focused on the possibility that scrapie-infected sheep had been turned into MBM with cows infected in turn.

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Scrapie is a disease of sheep that has been known for about 250 years (it was named then but in all probability has been around for much longer). It was believed to have a genetic origin. It has never affected humans and it was common practise to eat sheep's brains, as poor people especially could not afford to throw away any food.

The theory pursued by government and scientists was that feeding scrapie infected feed to cattle had caused the onset of the disease. It was thought that after this BSE infected MBM from cattle enabled the rapid spread of the condition. It was further suggested that material from infected cattle was responsible for these cases of vCJD. The LHA report assumed this theory to be correct, although, as I shall show, there were other theories about the cause of BSE which were also relevant to Queniborough.

BSE Statistics

Since the BSE epidemic started around 180,000 cases[1] of it have been confirmed. However, the epidemic peaked in 1992 at around 37,000 cases (0.3% of the national herd) in that year and has declined rapidly since. In 1986, seven cases were recorded, 413 in 1987, 2185 in 1988 and 6839 in 1989 (DEFRA's figures) It means that there were less than ten thousand cases by the time John Smith retired. This has to be seen in context: there are about 2,000,000 dairy cattle born each year (plus all the beef cattle). The LHA report states that some cattle developed BSE as young as 20 months of age. Data from MAFF given in response to a Parliamentary question on 10.3.97 shows that of all the cases between 1988 and 1996, only two animals developed the disease at less than two years of age. In fact, of all the 165695 cases of BSE included in this data, only 300 (0.18%) were less than three years of age at the onset of the disease (there are no figures available for those under thirty months when the disease started developing). BSE is essentially a disease of older cattle, with the majority of cases being around five years old or older at the onset of the disease. The Meat and Livestock Commission has a very detailed breakdown of all cases of BSE according to breeding which records that of the 177046 cases of BSE confirmed by the beginning of June 2000, 176006 (99.4%) were in female cattle. Cases in Friesian, Holsteins and Holstein x Friesians, (the main breeds of dairy cattle), total 148184 (83.70%). BSE doesn't solely affect female cattle; it is just that very few male cattle live long enough to develop the disease; especially once all beef animals were slaughtered before thirty months of age.

BSE is essentially a disease of older cattle, with the majority of cases being around five years old or older at the onset of the disease.

Age for age, there is no evidence that female cattle are more likely to catch BSE. Of the 505 cases recorded as male (535 don't have any details recorded), 474 (93.86%) are recorded as purebred animals, presumably pedigree breeding bulls. Butchers prefer steers (castrated

1 *Farmers' Weekly* 30th October 2009 estimates 179,000 cases

males) to heifers (females that haven't given birth to a calf) as heifers carry more fat and craft butchers seldom if ever buy heifers. Though the LHA report blames Hereford x Friesians for the Leicestershire cases (though Charolais crosses were used also), only ONE Hereford x Friesian male and ONE Charolais x Friesian had contracted BSE by June 2000 in the whole of the UK, though eight males are recorded as Hereford x and three as Charolais x which means their mothers may have been any breed or cross-breed.

In fact, as most BSE cases occur in dairy cows that eat the highest amounts of concentrate, it was believed that continuous exposure to infected feed made them more at risk. Why did the LHA theory reject the idea that vCJD victims could also be infected by repeated exposure?

Economic Statistics of BSE

Until the ban of the export of beef from the UK on March 25th 1996, beef exports were worth over £500 million per year, plus £61 million from the export of calves. Those exports were finally resumed in 2006. The domestic market for beef was worth £4.5 billion in 1995, slumped to £3 billion in 1996, recovered to £4 billion by 1998 and is now fully recovered, though many farming businesses did not survive the slump[2]. It is calculated that the total costs were over £4 billion and the crisis cost the British taxpayer the equivalent of 3p on the basic rate of income tax. The policy of having cheap food had cost far more, both in human suffering and economically, than had ever been saved by consumers.

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Having related the national statistics, I shall now come to what happened at Queniborough and start by outlining the history as it was told to me, and the claims of the LHA within the context of the assumption made by what I shall call the main theory.

2 Janie Axelrad p.64

CHAPTER 4

The Local Story

The Queniborough Victims

The first victim was Pam Beyless, who died in 1998 aged 24. Pam had lived at Glenfield, but her mother had been brought up in Queniborough. Pam's maternal grandparents moved to Syston about 1974 after their son was killed in a car crash, but they kept buying their meat from Bramleys' the Queniborough butchers. About three times a year they bought bulk orders of meat for the freezer which were divided between them and Pam's parents. Pam left home when about 17 and moved to Southampton. After splitting up with her boyfriend, she went to live in Basingstoke. She became ill and returned to Glenfield when she was 22 and was nursed there until she died. A friend that she knew in Basingstoke has also died of vCJD and, in fact, three people connected with the Basingstoke area have died of vCJD. Basingstoke has a large number of electronics firms in the area and they use large quantities of manganese, which as we shall see, might be significant. Strangely, these facts have not been reported elsewhere.

Basingstoke has a large number of electronics firms in the area and they use large quantities of manganese, which as we shall see, might be significant.

The second victim was Stacy Robinson, who was born in Queniborough in November 1979, and moved to Thurmaston when aged 13. Her mother was only ever seen once in Bramleys' shop and otherwise shopped at supermarkets. Stacy had a child when 16 and shortly afterwards became ill. She died in 1988, aged 18. The third victim was Glen Day, who was the oldest, dying in 1998 aged 34. He was a van driver and consequently travelled around a large area. The fourth victim was Chris Reeves, who was born and lived at Rearsby, where his father worked for a farmer. The family bought meat from the

local butcher and Chris worked on a farm in Queniborough after leaving school, where he was a good stockman. Chris died in September 2000, aged 24. Nothing is known about the fifth victim apart from the fact he was a 19-year-old male whose family didn't buy meat from either the Queniborough or Rearsbybutchers. Considering that the group is a very small one, it is surprising that a theory was put forward that excludes 20% of the sample.

The Butchers at Queniborough

The Queniborough butchers were Ralph and Ian Bramley. Ralph had owned the shop since 1948 and his son had joined him in the business when old enough. Until 1975 they had sourced their meat from a local farmer-grazier who had farmed between Barkby and Queniborough. This farmer bought in Welsh Black and Hereford calves and reared them. . The Herefords were purebred animals, with the distinctive white head and deep red-brown coats (which farmers call 'red' and the general public 'brown'). At least 99% of Hereford x Friesians have black coats. This farmer reared his cattle quite traditionally and they may have been older than 30 months at slaughter. They were slaughtered by the farmer's cousin, a licensed slaughter man, with his own premises at Thurmaston. The meat was brought to the Queniborough shop as whole quarters and cut up there. After the farmer died in 1975, his cousin bought cattle for the Bramleys at Leicester market and then slaughtered the animals for them. Contrary to Dr. Monk's claim that the Bramleys butchered four or five beasts a week, they actually only bought one a week, though extra stewing meat, joints and occasionally whole sides were bought from a meat wholesaler if required. Many small butchers only sell one side of beef in a week and only a butcher with three or four shops would sell fourbeasts in a week. The Bramleys only had the one shop. The cattle bought from Leicester market were Hereford x Friesian, with some Charolais x Friesian also. The animals

Many small butchers only sell one side of beef in a week and only a butcher with three or four shops would sell fourbeasts in a week.

purchased by the Bramleys would have been between 18 months and two and a half years of age, depending on breed (the Charolais are killed at a younger age than a Hereford x Friesian of the same weight), rearing system and whether they had been treated with the growth hormones that were then legally used in this country.

John Smith, the Rearsby butcher, bought his cattle from Melton Market or from local farmers. Again, through the 1970's, they were mostly Hereford x Friesian but by the time he retired in 1989, they were mostly Continental crosses (it is uncertain whether he bought other breeds besides Charolais crosses, quite possibly he did). He had a larger business and bought two cattle a week on average, but by the time he retired he was only buying one animal a week. Mr Smith killed his own animals in the slaughterhouse adjoining his shop. There was a slaughterhouse adjoining the Queniborough shop, but that has not been used since 1948 and cattle have not been slaughtered in it since 1945.

It is important to realise that quality retail butchers were very unlikely to purchase purebred Friesian cattle.

The Slaughtering Process

The slaughtering and butchery procedures have been described to me by Chris Hollis and John Gamble of Syston, who are experienced in both the practises of large abattoirs and small butchers. The process is as follows.

The animal is shot with a captive bolt (a bolt which retracts back into the gun) at a precise point on a 'cross' between the ears and eyes. As the animal may still kick in reflex action, it is then pithed. A pithing cane is pushed into the wound, through the base of the brain into the spinal column) to prevent this from happening. These rods are 3-4 feet long and made of stainless steel, though there are also disposable plastic ones. Pithing rods must be sterilised after each animal. In a large slaughterhouse one man would be assigned to pith all the animals, merely wiping off brain tissue with his fingers and then putting the rod in the steriliser before moving on to the next animal. Contrary to Dr Monk's claim, pithing rods are used in abattoirs and slaughterhouses,

large and small, all over the country and it is still legal to use them. Farmers' Weekly reported (5-1-2001) that half of the 70 members of the British Meat Federation were still using them. A ban on their use was proposed as a precautionary measure but that was postponed to allow members to adapt premises to the alternative measure. However, there was concern about this proposed ban because of the risk of slaughter men being injured by animals kicking.

It is noteworthy that no slaughter man, abattoir worker or butcher has contracted vCJD, yet they would inevitably breathe in any such particles due to their proximity to the animal.

Dr Monk claimed that microscopic particles of brain tissue, together with the BSE agent, being sprayed by aerosol effect over meat is what caused contamination in the Queniborough cases. At this stage the animal's hide is still on it, so any particles would be on that, rather than on the underlying meat. It is noteworthy that no slaughter man, abattoir worker or butcher has contracted vCJD, yet they would inevitably breathe in any such particles due to their proximity to the animal.

After being shot with the captive bolt, the animal's throat is then cut. Small butchers do this while the animal is on the floor but the larger slaughterhouses first hoist the beast with a chain around a hind leg. It is then allowed to bleed for 3-4 minutes before the feet are removed, at hocks and knees, and then the head is cut off and put to one side, with the brains still inside it. Again, contrary to Dr. Monk's theory, it is dealt with later, after the carcase has been prepared for hanging, and not as the first item. No butcher is going to leave several hundred pounds worth of meat lying on the floor in order to deal with a head that is worth pennies, especially as the long-term future of his business depends on the meat getting to his shop in peak condition. In the mid-1970's a sheep's head was worth about 15p and cattle brains about 20p each. A special knife called a sticking knife (6-8 inches long and very sharp) is used to cut the animal's throat and remove the head. All knives used are put aside as their particular purpose is completed and sterilised before being used again.

The next job is to skin the animal. For this, a flaying knife is used (four to six inches long with a curved blade). The skinning starts at the back legs of the animal, which is hoisted gradually as the skin is removed, so the hide falls away from the carcase. Any splashes would be 'inside' the folding skin. At the end of the process, the skin is pushed aside out of the way.

Then another set of knives is used for the evisceration process (removal of internal organs). The sternum (brisket) is split and the internal organs removed in a set order and the liver, spleen, heart and lungs hung up. Unwanted offal is disposed of, usually being put into a wheelbarrow for the time being. After that the tail is taken off and the carcass split, with a saw at the pelvis, to expose the spinal column. A chopper splits apart the two halves. There is far less risk from a small butcher doing this with a chopper than in the big slaughter houses, where a splitting band saw is used that sprays minute fragments everywhere. This was pointed out on Radio 4's 'Food' programme, as was the point that a small butcher then removes the spinal cord very carefully as it deteriorates faster than the rest of the meat and would spoil the carcass if any remained. This is particularly important to a small butcher who will hang the meat for up to three weeks before selling it (a supermarket does this for a much shorter period). On the Continent, the common practice is for the spinal cord to be rolled up neatly and sold as a delicacy, but vCJD is still rare with only four cases in France by 2001.

The two sides of meat were then wiped with a cloth. The small butchers dislike using too much water for fear of spoiling the meat and also because they do not have enough space for spraying a carcass. The cloths were muslin and boiled after each day's use and were kept in buckets of hot water. The big slaughterhouses use power hoses for washing the sides and again there is more potential risk with this process as blood skin particles and faeces are blown everywhere by it. The two sides are then left to cool for 24 hours before being put into the chiller. If the meat is put into the chiller too soon the meat

contracts and becomes tougher. After cooling the carcase is quartered and put into the chiller for hanging and then taken to the shop for cutting up into joints etc (with yet more knives being used in the shop).

Once the sides are put aside to cool the head is then dealt with. The tongue is taken out for salting before being cooked and pressed. After that the cheeks are taken off. These are not put into mince as they are tough. They have a different consistency and are often used as dog meat. Then the rest of the head meat is trimmed off and used in mince. If the brains were required, a small butcher would split the head with a saw and chopper, cutting through the bone at strategic points so that the skull would prise open and the brains lifted out as a whole with the meninges intact. Brains have to be poached whole when cooked, so if a butcher split a brain, it is rendered unsaleable (again a contradiction to Dr Monk's claims). Also, splitting a head is not difficult. It was often done by a lad (preparing the head was a good way of developing their skills) and was a routine job for a skilled butcher, as the Bramleys and Mr Smith were.

Further Facts about Queniborough

Though the LHS report makes much of the fact that old people ate brains, while it is true that some Queniborough inhabitants who are now old ate brains when younger, their eating habits have altered along with the rest of society and the Bramleys' customers. Only one or two liked brains and they were only supplied when these customers requested them, which was two or three times a year. Rosemary Smith, the daughter of Jack Sharpe (the village butcher who preceded Ralph Bramley) who worked in the Bramleys' shop and delivered meat locally for them, stated that brains were only brought into the shop at these times. Sheep's brains were the more popular as they have a sweeter taste, but the sale of them was prohibited in 1978 due to fears of scrapie transmission when the first cases of BSE emerged.

The old people of Rearsby were buying at the village shop, as they wanted a quality product above all. About 75% of the populations of

Rearsby and Queniborough are middle class, but the vCJD victims are all of working class backgrounds.

After Ralph Bramley handed the business over to his son and retired (about the end of 1980), Ian worked to build up the business. He supplied meat to pubs and hotels etc within a ten-mile radius of Queniborough, buying in extra portions of beef from wholesalers in order to do so. It was then fashionable to have large eight and twelveoz steaks and Ian bought in loin sections of beef to cut up for these. It is also believed that he bought in about three heads each week for processing and these were split and the brains sent to one outlet, but the purpose is unknown. It is possibly this factor that has caused confusion about how many cattle Ian sold each week. Ian tragically died in June 1982 (three years before the first case of BSE was diagnosed) and a caretaker butcher kept the shop open to supply the village trade until it was sold. At the time of Ian's death, Stacy Robinson was only about thirty months old.

Claims that their techniques were particularly likely to contaminate the saleable parts of the carcase with infective material, is at least questionable.

Thus we can see that the victims did not all eat beef regularly from these butchers or exclusively from them. Also, claims that their techniques were particularly likely to contaminate the saleable parts of the carcase with infective material, is at least questionable. There is also the fact that none of the supposedly infected cattle would have been above 36 months of age. The thirty-month rule that was imposed due to BSE was formed because nobody believed that animals under thirty months of age could be infective even if incubating the disease.

CHAPTER FIVE

Every Morsel

What about MRM?

If the local vCJD cases were related to beef consumption there is an alternative to consider. Since the discovery of vCJD, there has been the possibility that cheap meat was a factor in the spread of the disease, as the victims tend to be not only young but low income. There is also a slightly higher incidence of the disease in the north of the country, which eats more cheap cuts of meat. However, this has caused confusion for many as to what is meant by cheap meat. Some people think mince carries more risk than steak. In meat animals, the tenderest meat comes from the parts that have had the least movement i.e. the back and rump. The roasting and grilling cuts come mostly from the back quarters and the stewing cuts from the forequarters, with mince being made from the flanks and other trimmings. In spite of the often-repeated statement "only one mouthful of infected steak is enough to contract CJD", no trace of BSE has been found in steak (or milk for that matter) and there is no evidence that mince from a prime young beef animal is any more risk than steak from it [3].

For the decade since 1996 no animal over thirty months of age was allowed to enter the food chain. As most BSE cases were five years old or more when they developed the symptoms this rule was to ensure that no animal that was not displaying any symptoms but which was incubating the disease could be incorporated in the food chain. Before then, thousands of old cows were killed for human consumption every year. The best, biggest and fattest animals were exported to France while the others were turned into canned and frozen stews, pies and beefburgers. No craft butcher would ever buy old cows, as their meat

3 David R. Brown, University of Cambridge, 2001

is tough and totally unsuitable for roasting and grilling. Not only beef was incorporated into canned stews etc before this ban. For a decade, beginning in 1975 until 1986 when the Meat Products Regulations came into force, cooked products contained 'meat' that was in fact such things as feet, testicles, rectum, udder, brains, spinal cord and spleen (brain, spinal cord and spleen are the specified bovine offal removed from the food chain in 1989). (The Meat Machine, 1989).

Even more draconian measures were taken by food manufacturers to extract as much meat (and profit) from the carcasses as possible. Mechanically recovered meat (MRM) is a product obtained by recovering residual raw meat from bones that cannot be removed by manual boning processes. The process is carried out under high pressure and the result is a slurry that contains other tissues besides meat. Animal heads were put into this process. Between 1985 - 1995 (when MRM manufacture was stopped) two companies had up to 90% of the market for MRM. David Walker, then Chief Trading Standards Officer for Shropshire has estimated that as much as 25% of MRM could have contained spinal cord. MRM was added to products such as sausages, burgers, pies and baby foods and it has been estimated that a 100g pie or burger could have contained as much as 280mg of spinal cord. Some MRM was manufactured on the continent but there it was only used in pet food. Pam Beyless' father worked for Tesco at one time and took out of date pies home to put in the freezer. Glen Day, Stacy Robinson and Chris Reeves all went to Wreake Valley School, though at different times. This school was supplied with meat for several years by a Syston firm that supplied meat and meat products to schools, nursing homes and old people's homes throughout Leicestershire. This firm bought in burgers from Derbyshire that were so cheap that, though their precise manufacture is unknown, it is almost certain they contained MRM. Stacy Robinson enjoyed going to burger places and Glen Day would have stopped for breaks at various places while on his van driving

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rounds. Pam Beyless ate burgers and donor kebabs and left home while still a teenager and so would have had to watch her budget. It is unknown precisely what she ate during these years. In none of the four known cases was the village butcher the only supplier of meat to the victim. While the LHA theory tries to lay the blame on a single contaminated beef animal bought by Ian Bramley sometime between 1980 and 1982, it is unclear that infection is caused by a 'one off' occasion. In fact, as most BSE cases occur in dairy cows that eat the highest amounts of concentrate, it was believed that continuous exposure to infected feed made them more at risk. Why did the LHA theory reject the idea that repeated exposure could increase the chances of contracting vCJD?

Thus within the context of the meat and bone meal theory about the spread and cause of BSE the LHA Report conclusions are hard to accept. Beyond that it perhaps needs to be recognised that the main theory has weaknesses and that there may have been several contributory causes.

CHAPTER SIX

What if?

Some other considerations

THE ALTERNATIVE THEORY

Mark Purdey, a dairy farmer from Somerset, who unfortunately died in 2006, had always been convinced that the pesticide used to eradicate warble fly from this country in the early 1980's was to blame for the onset of BSE. The pesticide used was an organophosphate compound called phosmet and the Ministry of Agriculture vets ordered it to be poured along the spinal cords of cattle at twice the dosage rate used in the rest of Europe. Purdey suggested that prions (whose function in the brain, he describes as being to protect it from the oxidising properties of chemicals activated by ultraviolet light and other such dangerous agents) are adversely affected if exposed to too little copper and too much manganese. Then the manganese takes the place of the copper the prion protein usually binds to, causing it to become distorted and lose its function.

Purdey's work has indicated that phosmet captures copper. Also, at the same time the warble eradication programme was being carried out, some cattle were being fed animal feed supplemented with chicken manure and that in turn came from poultry dosed with manganese to try to increase the shell quality of the eggs (though the birds absorbed little of the manganese and excreted the bulk of it). The combination of the manganese affected feed and the phosmet treatment created the conditions for BSE. As well as this, it is standard practise for farmers to have mineral licks available at all times so the animals get all the trace elements, but some cattle lick extensively at them and so get high doses. There is a strong correlation between the number of times an area was forced to treat for warble fly and the number of subsequent cases of BSE, with the most cases being in the

south and west. Also, some cattle that were never fed MBM but were treated for warble fly developed BSE.

Purdey also travelled the world and discovered that wherever there is a 'hotspot'of scrapie or CJD, there are high background levels of manganese (as in the Iceland case). He did soil and water tests at Queniborough that show that this area is high in manganese. However, the government always refused to investigate Purdey's theory or give him grants to do so (his work was entirely funded by private contributions). In comparison, MAFF's figures show that it budgeted for £32 million to investigate the infected feed theory between 1997-2002. This is not surprising as, if it were proved that organophosphate was the cause or a factor in BSE, it would open the floodgates to those with other concerns about these compounds. There are hundreds of people convinced OP based sheep dips have ruined their health and such compounds have been linked to Gulf War syndrome. Farmer's wives have miscarried after coming into contact with OP's. There are concerns over OP pesticides on fruit and vegetables (and health problems reported in farm workers who have used them for spraying crops) and from their being used in household uses such as pet flea sprays and head lice treatments for children.

THE INVOLVEMENT OF MANGANESE?

Also, why did the LHA report rely solely on the British Geological Survey soil maps to determine whether the area around Queniborough had high or low levels of manganese? Mark Purdey had done soil and water samples in the area that show very high levels of manganese in places. He had visited an area of the Tatra Mountains in Slovakia, which is the CJD 'capital'of the world. There the incidence of CJD is one in a thousand, rather than one in a million as elsewhere. That area of the Tatra has proved to be high in manganese, but the levels recorded at Queniborough are up to twice the levels found in the Slovakian case. Surely the fact that tests taken in the area show high levels of manganese when they should be low should be investigated? It is even

more surprising considering that Dr DR Brown gave a talk in Leicester (6.11.2000) entitled "Life after BSE" in which he referred to a study that shows that CJD patients have a ten-fold increase in the levels of manganese in their brains [4], though it needs further investigation to determine whether this is a cause or consequence of the disease (though Mark Purdey's work does indicate that it is connected to the development of the disease). Dr Brown believed that CJD is a post-industrialisation disease and that is why it is more prevalent in the north of the country, where most of our heavy industry was based. Certainly in the Slovakian example there is a large steel works that is responsible for heavy pollution of the area. Steelworks use large amounts of manganese, which is dispersed as dust in the resultant fumes in the Tatra. Queniborough, though classed as rural, has some industry among which there was once a dye-works. That had a major fire at the end of the 1970s (possibly early 1980s – the date is uncertain though Rosemary Smith remembered the villagers being told to keep inside with their doors and windows shut at the time). Dye-works use vast quantities of manganese and the fire showered the village with chemicals.

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THE INVOLVEMENT OF ACINETOBACTER?

There is another matter that should be investigated as it bears on the research of Professor Alan Ebringer, who believes that a sewage-born bacteria called acinetobacter is the cause of vCJD. Mike Winterton (a Queniborough farmer) had fields at Wanlip that were spread with sewage sludge from the nearby sewage works every year for several years. It was stopped in 1985 as heavy metals were building up in the soil (manganese is a heavy metal). Other fields in the area have been regularly spread with sewage sludge. One such farm is crossed by a footpath, which leads to Wreake Valley School, and which was used by Stacy Robinson, Glen Day and Chris Reeves (the latter visited friends in

4 Quoting Bost M. *et al* poster presented at "Metals and Brain Conference" Padua, Italy 24th-27th September 2000

Queniborough after school). This field had been tested by Mark Purdey and showed high levels of manganese. The sewage sludge used to be spread by a spinner, which caused a fine mist of sewage that used to spread to the houses in the village. Then it was spread behind a tractor using an umbilical cord and now is spread by direct injection. The firm spreading the sludge used to be based at a nearby farm and, for cost reasons, tried to spread as much sludge as possible within five to six miles of the Wanlip sewage farm. Leicester was, of course, famous for its textile firms, which obviously used high quantities of dye and were connected to the sewers. (The great textile areas of Yorkshire and Lancashire are in the north.) Prof Ebringer has done research work that has found cattle with BSE carry high levels of antibodies to acinetobacter (Guardian 13.10.2001).

LINKS WITH OTHER DISEASES

The LHA report appears to have treated the vCJD cluster in isolation, without looking to see if Queniborough has high levels of any other diseases. Unfortunately, it appears this could be the case. In 2001 Rosemary Smith and Bob Partland were aware of six cases of Multiple Sclerosis in the village and feared that others may be developing the symptoms. Both Mark Purdey and Prof Ebringer have connected MS and vCJD due to the findings of their research. Multiple Sclerosis affects one in 700 of the population and, with a population of 2,279 (Guardian Weekend 22.9.2001), there should only have been three or four cases in the village. Also, someone who lived in the house next to Stacy Robinson's former home died of Parkinson's disease. The neighbour on the other side had Multiple Sclerosis, with the house next to that having a resident with severe Alzheimer's disease (Purdey has linked Alzheimers to vCJD and others have linked it to heavy metals). What are the odds for such a combination of dreadful diseases appearing in four adjacent houses? Besides this, a 51 year old man died of sporadic CJD in Queniborough in 1989. He lived on the same street as did Glen Day. Again, what are the odds for such an occurrence?

CONCLUSIONS

It seems unlikely that the vCJD cluster around Queniborough can be laid at the door of the local small butchers.

1. Whilst some of the cattle they used may have been fed meat and bone meal as calves, they were too young to have been prime suspects as sources of infection.
2. The argument that the practices and procedures of small abattoirs made them more likely sources of infective material is highly questionable.
3. The eating habits of the vCJD victims as a group were not particularly linked with these butchers.

In respect of all three of these points there are more likely explanations.

1. In terms of cattle used, older cows are a more likely source of infective material. Most of these went into the manufacturing sector.
2. It is in this sector that MRM (mechanically recovered meat) was used.
3. There is some evidence of the victims' consumption of beef in manufactured products.

As if that were not enough, this cluster appears to offer grounds for stepping outside the theory of transmission from BSE infected cattle to vCJD victims to consider possible roles for manganese or Acinetobacter, or links with other diseases, remembering that any phenomenon may have more than one cause.

The LHA report claims that vCJD victims were 15 times more likely to have purchased beef from a butcher who removed the animal's brain. On what statistic did they conclude that small butchers were more likely to purchase BSE animals in the first place, especially as supermarkets and food manufacturers purchase far greater numbers of cattle? Or is it, as was pointed out on the Food Programme, that if the report had blamed the major supermarkets they would have sued?

It is all too obvious that a small business has very limited resources and so cannot fight any accusations against it when to do so would involve a great deal of time and cost. The problem is exacerbated when the events one is being blamed for happened in the past and, while a theory is all that is necessary to cast the slur, concrete evidence is really all that can clear one's name. The whole BSE saga is one of small people being ignored or vilified while the roles of big business have been protected from any official blame.

The LHA theory was trumpeted as a breakthrough when first published but all it haachieved is to tarnish the reputations of three well-respected men, two already dead and one then in poor health so none can defend themselves. It is more of a lost opportunity and appears likely to remain so unless real attempts are made to discover the truth. It is also surprising that the investigation was left to the LHA alone, which presumably is as cash-strapped as any other local health authority. This is the largest cluster of a disease that has affected people across the country. Why weren't all the theories tested thoroughly?

By the time the LHA report was published, it was apparent that vCJD was thankfully not going to be the mass killer once predicted and the numbers of BSE cases in cattle was tailing off. The general public was eating more beef and prices for the producers were staggering towards once again making a profit. However, if the tide had not turned, this report, by finding explanations in the wrong place would have done nothing to counter vCJD or to point the way forward for public health or farming. On the contrary, it would have deflected attention and effort into unhelpful directions.