Tracking the media and Mad Cow Disease

A look at how science, politics and media coverage influenced policy decisions

By Michelle Badash ©1999

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The Beginning

Often one can pinpoint the roots of historical events or calamities to a single, sometimes seemingly insignificant occurrence. In the case of Mad Cow disease – a disease epidemic that eventually wreaked global havoc in the arenas of science, trade, politics, industry, farming and public health – it all began in the quiet English countryside with a sick cow named Jonquil.

In the spring of 1985, Jonquil, a cow on a prosperous Holstein dairy farm in southeastern England, began exhibiting rather odd behavior. The head stockman noticed that this normally quiet, good-tempered animal began to behave aggressively, was unsteady on her legs, and "seemed to hallucinate."

Examination by a veterinarian yielded no clues, and her symptoms became worse. Fearing a brain tumor, they had no choice but to end her life. Little notice would have been made of this incident, except that six months later, several more cows began exhibiting the identical symptoms. An investigation into other farms in the area found additional cases. Local veterinarians were mystified; in November, 1986, they decided to send a sample of a diseased cattle head to the Central Veterinary Laboratory of England's Ministry of Agriculture, Fisheries, and Food (MAFF) – the British equivalent to the USDA - for examination.

The cattle head ultimately created quite a stir in the MAFF laboratories; the brain was found to be riddled with spongy holes, a characteristic sign of a common disease called scrapie. There was just one problem: scrapie, until now, had only been found in sheep – never cows.

Anatomy of a disease

Scrapie had been documented in England as far back as the mid-1700's. Sheep who developed this disease exhibited such traits as clumsy gait, shivering, seizures, or rubbing (scraping) against posts (hence the name "scrapie"). Scrapie was categorized as part of a family of diseases defined as transmissible spongiform encephalopathies, or TSEs.

TSEs have several characteristics in common: they are infectious, produce no immune response, and are universally fatal. Except for the occurrence in sheep, this type of disease is relatively rare; various forms have appeared naturally in captive animals such as ranch mink, deer herd, zoo animals and cats. Humans, on occasion, have contracted TSE-type diseases; kuru, Gerstmann-Straussler-Scheinker disease, Alpers disease and Creutzfeld-Jakob disease are all examples of rare human diseases that fit the TSE mold. Although the incubation period varies depending on the host, it is generally long, and can range from several months to several years with no telltale symptoms. Cattle for instance, can harbor bovine spongiform encephalopathy (BSE – the TSE version in cows)

for three to eight years; this would later become a significant factor in assessing the food safety of animals killed at an early age, such as veal.

But what makes TSE's so perplexing – and, as yet, nearly impossible to combat - is that they seem to break all rules of biology. Like a burglar who enters a home and disables the alarm system, TSE's don't trigger any detectable antibody response, so there are no blood tests to diagnose symptoms. The only way to confirm diagnosis is through autopsy and examination of the brain for the microscopic spongy holes after death.

Transmission

Unlocking the mysteries of transmission is key to learning how to control the disease. Most diseases are transmitted either by hereditary causes (in which case, the transmission is "vertical" from parent to offspring) or infection such as bacteria or virus (called "horizontal" transmission, by contact from one source to another.) Vertical transmission may take many years, while horizontal transmission may spread quite rapidly. If the disease is caused by an infection, you can control the spread by isolating and killing the infected animals; if the disease is hereditary, you can change the breeding practices. But when BSE surfaced, it did not conform to either of these models, and no one could figure out how to stop its spread.

In addition to its inscrutable transmission pattern, what was a TSE that was usually found in sheep doing in a cow? Although experiments had shown that TSEs could be introduced from one animal to another in a laboratory setting, there was a general belief that the disease could not naturally jump the "species barrier". If this proved false, the implications could be quite alarming. How could you possibly control a disease if you weren't sure how it was spread – and who (or what) it would affect next?

In 1987, MAFF veterinary epidemiologist John Wilesmith began studies to determine what was causing the outbreak of "cow scrapie". The following year,

he pinpointed two culprits: the practice of rendering (grinding up) infected sheep used in bonemeal feed for cows, and the recently instituted shortcut in the rendering solvent/steam treatment process which reduced infective agents. His findings were published in the *Veterinary Record*. [1]

But few journalists, or even physicians, read the *Veterinary Record,* so initially this information was circulated only to those in the agricultural or veterinary communities. As time went on, however, it would become common knowledge to nearly everyone interested in beef in Europe and North America.

Wilesmith's theory gave a plausible explanation as to why scrapie had surfaced in cattle. But the biologic mechanism for transmission was still stumping scientists. And to this day, no researcher is absolutely sure how TSE's are transmitted. But several years before Jonquil's death in 1986, a scientist named Stanley Prusiner was developing a radical theory that would turn the scientific world upside down – and possibly offer one of the few viable explanations for the cause of TSE's.

The Prion Theory

One of the most basic tenets of scientific theory is that bacteria, viruses and every living organism contain some form of nucleic acid (either deoxyribonucleic acid – DNA- or ribonucleic acid – RNA) which carries coded genetic information. It is this information that is the "blueprint" for disease replication.

In 1982, Stanley Prusiner, a neurologist at the University of California School of Medicine in San Francisco, developed an astonishing theory: that a plain protein, which in and of itself is considered harmless, could mutate and become infectious. Prusiner dubbed these abnormal proteins "prions" – a word he coined by combining the term "**pro**teinaceous **in**fectious particles." He believed that TSEs were caused when a normal corkscrew-shaped cell protein (PrPC) entered brain cells, and then folded into a flat shape and converted to the

infectious prion form of the protein (PrPSC). As these infectious prions continue to multiply, they clog infected brain cells, causing them to misfire, eventually die, and release into the tissue. Gradually, whole clusters of brain cells die, which causes the characteristic "holes" in the brain – as well as the strange behavior as the organism is dying.

The most controversial element of this theory is that these prions contain no nucleic acid – an idea that Prusiner acknowledged was "…a "heresy" against the principle that genetic information invariably flows from nucleic acids to proteins, a principle so entrenched that it has been called the central dogma of molecular biology."[2] In other words, a protein without nucleic acid to "mastermind" transmission and replication is like suggesting that a body can function without a brain.

As might be expected, Prusiner's theory created an uproar amongst scientists. His many critics scoffed at the notion that disease could be transmitted in a way that was so contrary to the most basic tenets of biology. But over the course of a decade, his theory gained credibility as other scientists were able to replicate his findings, eventually identifying 20 different mutations in the prion protein that could be linked to inherited spongiform diseases. Although there are still questions that the prion theory does not fully explain, and other theories that have been formulated, the prion theory is, to date, the most widely accepted theory on TSE transmission. After years of ridicule, criticism and even ostracization from his fellow scientists, Prusiner garnered the ultimate retribution by winning the Nobel Prize in 1997 for the prion theory.

Aside from the radical concept of prions, further research into TSEs also uncovered one other startling fact that made TSEs unique - and quite unnerving - from other known diseases: it can be transmitted through **both** inherited and infective modes.

Politics and trade make for difficult decisions

After John Wilesmith's findings in 1988 implicating meal and the rendering process as the probable cause of BSE, the obvious choice was to change these practices to prevent further spread of the disease. But this decision had severe financial implications. Rendering solved two problems: it provided a cheap source of animal food supplements, and also solved a handy waste disposal problem. According to industry representatives, England alone could be saddled with a $600 million bill to change rendering practices. This in turn would cause beef prices to soar, and eventually cripple England's lucrative beef export industry. As noted in the book, *Mad Cow U.S.A.*,

> "In the absence of clear knowledge, political and economic factors became the determining factors shaping the government's policies. At stake was a market for beef and veal worth $3.1 billion in U.S. dollars. England employed 40,000 dairy farmers and 70,000 beef farmers, plus another 8,000 truckers engaged in the transportation of cattle to the country's 200 livestock markets…"[3]

Clearly, the UK government was in a tight spot: if they insisted on drastic changes, they could cause irreparable damage to some of their biggest industries. If they did nothing, they ran the risk of perpetuating a public health epidemic.

Scientists were able to offer little in the way of solid evidence – but the few studies that did exist were unnerving. "Experiments had shown that passage from one species to another could alter the subsequent host range of the scrapie agent. In the process of jumping from sheep to cattle, therefore, the disease might become infectious to humans."[4]

Faced with this difficult decision, the government appointed a committee, headed by Sir Richard Southwood, a well-known professor of zoology, to study the problem. Curiously, not one specialist in the field of transmissible spongiform encephalopathy was invited to join the committee.

Two weeks after the Southwood committee's first meeting, they issued the recommendation that infected cows were to be destroyed; farmers were to be

compensated at 50% of the animals' worth, subject to a ceiling. The government also imposed a ban on feeding ruminant offal (internal animal organs) to cows. The following month, milk from cows suspected of infection was also ordered to be destroyed.

In retrospect, these measures were not adequate, and came too late to stop the spread of the disease, which had started nearly three years ago with Jonquil's death. Furthermore, critics note that the offer to compensate farmers only 50% of market value was, in effect, a deterrent to reporting the true extent of the disease.

The Southwood Committee met again in November; their report was not released for another four months. Their explanation of the cause of BSE focused on the commercial feed rations; they expressed optimism that the disease would eventually disappear. The report acknowledged that at this point, it was impossible to be certain of the magnitude of risk, but " …the risk of transmission of BSE to humans appears remote." It was the first of many government assurances that there was "no need to worry".

At the time of the report, the number of cattle found to have BSE was approaching nearly 1000, with over 1000 more infected.

During the late 1980's, minimal media attention was given to Mad Cow disease; it was considered primarily an agricultural problem, with little notice from the general public. But as the early '90's wore on, and thousands of cattle continued to die with little sign of abatement, the stirrings of anxiety began to take hold. The British press was prolific, racking up nearly 400 news reports on this issue, in contrast to the U.S. press, which languidly published a total of 20 newspaper reports until 1993. Most articles focused upon the effects of BSE on society. "Mad Cow Disease kills 10,000 cattle in Britain; government sees little risk", claimed the *L.A. Times* in April, 1990. The *Washington Post* followed with "Hysteria over Mad Cow Disease Sweeping Britain," in May, and the *Wall Street Journal* ran articles examining the international trade implications of unsafe beef.

But in 1993, a mysterious incidence of a very rare disease began to strike inexplicably in a very unlikely population; by the time the implications of this disease were realized, the media worldwide exploded in a frenzy.

Creutzfeld-Jakob disease: the spark that ignited hysteria

In the early 1920's, German researchers identified one of the rare transmissible spongiform encephalopathy disease that affected humans - about one person per million worldwide. Named Creutzfeldt-Jakob disease after the researchers who identified it, it was symptomatically similar to another TSE called kuru. Researchers believed that kuru (which was found in only one tribe in New Guinea) developed as a result of cannibalistic eating rituals that involved eating and handling brain matter. In contrast, CJD cases were noted in ethnic populations worldwide, including Libyan Jewish immigrants to Israel, Czechoslovakia, and Northern African immigrants to France, and appeared to be from genetic causes. As with other TSEs, CJD was invariably fatal, and those who contracted it showed signs of dementia and loss of motor function. Although it affected men and women equally, it was found only in older people, with the average age of onset around 60.

CJD is classified three ways, depending on the method of transmission:

Sporadic - appears due to no known cause. This is by far the most common, and accounts for 85% of all cases.

Hereditary – family history or genetic predisposition. About 5-10% of people with CJD account for this type.

Acquired CJD – Transmitted through brain or nervous system tissue. Although this accounts for less than 1% of the worldwide cases, this is the type that was linked to BSE, and received the most media attention.

With the exception of cases which developed for iatrogenic reasons (ie, caused by medical surgeries such as corneal transplant and cadaver-derived

growth hormone injections) the rate of CJD has remained fairly consistent throughout the years.

But in 1993, this rare disease began to surface in England. Fifteen year old Vicky Rimmer was the first young person to be diagnosed with Creutzfeldt-Jakob disease; within three years, nine more people under the age of 42 were also confirmed to have contracted the disease. The young age of these new cases startled physicians – it was a dramatic deviation from the age parameters of previous CJD cases. The British media began reporting disturbing stories of young people suddenly suffering from dementia and dying – not unlike the cows in previous years.

Given that both BSE and CJD were from the same spongiform family, scientists began to question whether there might be a connection between the two diseases. Clues to a potential link came when the CJD Surveillance Unit autopsied the first few teens who had fallen victim to CJD. Dr. James Ironside, a neuropathologist with extensive experience in CJD, found that the manifestation of CJD differed significantly from previous cases: it had struck at the base of the brain instead of the cerebral cortex (much like BSE) , the disease had lingered longer than traditional CJD cases, and the patients had all exhibited different types of behavioral dementia than those usually associated with CJD. Neurologically, heavy concentrations of amyloid plaques were found that were much larger than previous CJD cases.

Because these symptoms differed from traditional CJD, scientists felt that these cases represented a new strain of the disease; eventually, it came to be known as new-variant CJD (nv-CJD). And in 1996, a study published in *Nature* was the first direct scientific evidence to date that this nv-CJD did indeed seem to be linked to BSE.

Science vs. politics

"In uncertain areas of science, it is a fool who professes perfect vision…"

(Tim Lang [5])

As the Mad Cow crisis continued to unfold over a decade, the question of what scientists knew – or didn't know – about BSE and CJD became a virtual commodity utilized by various factions to promote their own agendas. Government officials frequently sought to reassure the public by saying that there was "no scientific evidence" that BSE could cause CJD. Those in the beef-related industries also touted the lack of scientific certainty on the BSE issue, and instead blamed the media for provoking "hysteria." Frank Burnham, a spokesperson for the rendering industry, exemplifies this position when he commented, " ... the current BSE scare is due largely to sensationalized stories in the British press – based on speculation not fact – linking BSE in cattle to CJD in humans. There is in fact, no scientific fact directly linking CJD to BSE or BSE to sheep scrapie." [6]

These statements all assumed a singular premise: that the determination of action should be predicated on proven scientific findings. However, scientific research methods are based upon building a body of knowledge that is constantly evolving. At what point are decisions made regarding public health and policy?

Prior to the first cases of nv-CJD in 1993-5, scientists had published a small number of studies on the biological mechanisms of transmission. Although findings from these studies were not definitive, the evidence was mounting that transmission of BSE should be carefully monitored. Several studies used animal models to confirm that TSEs could indeed "jump the species barrier"; successful experiments involving innoculation and transmission of BSE to mice and pigs, as well as cattle to cattle, were published before 1990.

The early 1990's marked a time of increasing concern within the scientific community regarding the British government's lack of swift response to the BSE

epidemic. In fact, some scientists charged that funding of BSE-related research was actually being discouraged by the UK government in order to minimize economic injury to Britain's beef trade industries. By taking a slow and cautious approach to the eradication of known risk factors, and maintaining assurances that British beef posed no threat to the consumer, some scientists feared that BSE could eventually explode into an unmanageable epidemic. Richard Lacey, of Leeds University, was one of the first well-known scientists to suggest that meat products should be avoided; he also recommended the destruction of cattle herds which harbored even one infected animal. This resulted in a dramatic *Sunday Times* news story entitled, "Leading Food Scientist Calls for Slaughter of Six Million Cows." [7] The story sparked continuing controversy about agricultural practices as well as food safety; two weeks later, overall beef consumption was down 25% in the UK. In June of 1990, one month after the appearance of the *Times* article, Lacey testified in a hearing on BSE before the Parliamentary Agriculture Committee. This revealing exchange is a good example of the widening philosophical differences between government and science:

> **Alan Amos, member of Parliament:** "…it does seem to me, and I am not an expert in these matters at all, that you seem to be expecting the Government to give a degree of scientific certainty which no scientist could or would wish to give, yet in your submission it seems a cry vague when you talk about 'may' 'could be' 'possibly'. On the one hand you are demanding scientific certainty, yet in your submission you accept that certainty is not available and it is not possible."
>
> **Lacey:** "The point about the difference between science and philosophy is not that I am demanding proof. I am not. I am saying it is impossible to generate scientific proof because it will take too long. The possibility exists that there is very substantial risk to man, and I am saying that we cannot wait to generate scientific proof…I cannot believe that a scientist will say: In order to find out how big the problem is, we are going to see how many people die. I cannot accept that." [8]

Lacey, who at one time had been appointed a member of a scientific panel which advised the government, became the target of government ire and scathing rebukes for being "sensationalist and irresponsible."

Stephen Dealler, a microbiologist at Burnley General Hospital, was another scientist concerned about the epidemic potential of BSE. In 1995, he published studies which examined the threat of BSE to public health, as well as statistical projections of potential transmission and when infectivity was likely to occur. His research findings were alarming: he noted that cases of BSE were severely under-reported (a mere 40% of all cases in 1993); he also predicted that 1,800,000 cattle incubating BSE would be eaten before the year 2001 even if no cases are born after 1991. Dealler's report also warned that "the risk to humans in Europe from BSE is unacceptably high, but cannot be stated precisely at this time." [9]

In November, 1995, the esteemed *British Medical Journal* (BMJ) devoted an issue to scientific discussion of BSE and its potential link to CJD. Researchers weighed in at all levels of the spectrum, but many noted that the available information was insufficient to draw sound scientific conclusions. However, there was disagreement about how to proceed. Should a lack of evidence that BSE might potentially cause CJD result in more aggressive preventive action, or instead, a conservative "wait and see" approach?

Sheila Gore, an epidemiologist with the UK government's Medical Research Council who also published in the same BMJ issue, was less uncertain. In an article entitled, "More than happenstance: CJD in farmers and young adults", she demonstrated statistically that the then-current incidence of two teenagers and four farmers contracting CJD within four years was so low that it could not have happened by chance; she estimated that the statistical probability was "less than one in 10,000."[10] The work was highly publicized, and added fuel to the growing concern about BSE and CJD.

By the end of 1996, there were 14 confirmed cases of CJD in England, and one in France. After the first few teenage victims of CJD were autopsied, the CJD Surveillance Unit combed carefully through questionnaires detailing the history and lifestyle factors of the cases, looking for a common thread to explain the mysterious onset of CJD. Only one factor cropped up repeatedly: all ate meat. In February, 1996, when autopsies of eight people who died from CJD were found to have identical brain patterns, Dr. Ironside determined that the government's Spongiform Encephalopathy Advisory Committee (SEAC) had to be informed – and action would need to be taken.

The turning point: March 20, 1996

In early March, the CJD Surveillance Unit presented its findings to the SEAC, a committee that had been established in 1990 and had, until then, recommended little modification to existing BSE management policy. By mid-March, two more nv-CJD deaths had occurred, and it seemed clear that an announcement regarding a potential link between BSE and CJD would need to be made to the public.

On March 20, 1996, Health Secretary Stephen Dorrell, made a formal statement* to the House of Commons, and acknowledged that the SEAC had "agreed on new advice about the implications for animal and human health of the latest scientific evidence…[the work of the CJD Surveillance Unit] has led the committee to conclude that the unit has identified a previously unrecognised and consistent disease pattern." Dorrell outlined recommendations from the SEAC regarding risk reduction measures and future research, and tried to assure the public that they would be kept apprised of ongoing scientific findings. Agriculture Minister Douglass Hogg made a statement following Dorrell, and ended by saying, "I recognise that there will be public concern, but the Government's Chief Medical Officer advises us that there is no scientific

evidence that BSE can be transmitted to man by beef…In view of what I have announced, we believe that British beef can be eaten with confidence."

To this day, few statements made by the British government can match the reaction of the shock waves sent around the world by the joint statement made March 20 by Dorrell and Hogg. After nearly a decade of assurances about the safety of British beef, the government was forced to concede that there was a potential risk to public health.

**See Appendix A for full transcript of Dorrell/Hogg BSE Statement*

The media and the message

News of Mad Cow disease had begun as a trickle in the 1980's, and grew into a media tsunami when the March 20,1996 announcement about a potential BSE/CJD link was made. In the United States, fewer than twenty articles per year had been published in the print media on Mad Cow disease between 1985 and February 1996; between March and December of 1996, approximately 250 articles were published in the US, and over 800 in the British press.

But a review of the Mad Cow media coverage reveals some startling differences in comparison to other health crises.

Where was the science?

In general, scientific publications are the springboard for health and medical news coverage. This model – scientific research followed by news - is the standard way that most medical information is disseminated. Perhaps one of the most significant factors of the Mad Cow story was that news reports were **seldom** driven by scientific findings. In a review of over 500 American news items in magazines, five top newspapers*, wire service reports, radio and television transcripts from January 1990 to March 1998, only 46 stories (9%) focused on the medical aspects of Mad Cow disease. (*Newspapers were: *New York Times, Washington Post, Wall Street Journal, LA Times, Chicago Tribune*.)

At first, this conclusion is surprising, because Mad Cow disease is a public health issue, and as such, would seem to fall into the standard scientific reporting model outlined above. But Mad Cow disease is unusual as a health phenomenon; unlike other medical issues, much of the scientific research was conducted as a result of the widening epidemic, in an effort to understand an existing pattern. Thus, the amount of existing medical research in this area was relatively meager. When placed in this context, it is more understandable that the media coverage instead used angles other than science as the basis for its news stories.

However, this is not the first time, nor the last, that the media will encounter a vacuum of information on a health topic that suddenly explodes onto the horizon. Other recent medical mysteries include the development of AIDS and transmission of the Ebola virus.

If the news reports were not discussing the scientific aspects of Mad Cow disease, what were they reporting on? Since the topic of Mad Cow disease spanned so many fields, and evolved over more than a decade, there were plenty of angles from which to choose. Stories dealing with agricultural aspects (e.g., "Mad Cow Disease Threatens the Farming Life" (*New York Times*) , "Britain acts to aid farms facing ruin…" (*Chicago Tribune*) etc.) accounted for 87 stories (17%). Other areas of frequent reporting were actual events related to Mad Cow Disease ("British Government Announces Slaughter of Cattle", CNN News) with 61 stories (11%), and social, and political/trade issues (7% and 9% respectively).

In effect, much of the news that Americans received about Mad Cow disease was rather piecemeal, and tended to focus on elemental features of the story.

Differences in coverage: US vs. UK

Media success is largely based on capturing and maintaining the interest of its audience – usually by reporting on news that is local, and of personal

relevance to the audience. Obviously, the spectre of Mad Cow disease and subsequent cases of CJD became increasingly important to the British; in America, where the incidence of these problems was quite low or nonexistent, the tone and frequency of press coverage differed dramatically. The British press began reporting on Mad Cow disease in the 1980's; articles about this issue did not appear in the American press until 1990.

The most striking difference between American and British coverage of the BSE and CJD issues was – perhaps predictably – the sense of immediacy or distance to the situation conveyed by news reports. Obviously, Britons had a sense of personal involvement in the BSE issue; many felt personally at risk as the crisis continued to escalate. However, upon review of U.S. media reports about this issue, one is struck by how often the news story was framed in the context of "it can't happen here." This premise was reinforced by an emphasis on stereotypes of English as "beef-eaters". Some sample headlines include:

- "Hold the kidney pie; Mad Cow disease panics UK beefeaters" (*Newsweek*)
- "British officials can't calm Mad Cow disease fears" (*Washington Post*)
- "British Beef crisis: A menu for despair" (*LA Times*)

A recent media analysis of American media coverage of Mad Cow disease noted that the *New York Times* actually ran an article on April 7, 1996 that highlighted the redundancy of U.S. headlines which evoked English stereotypes. At least seven major U.S. newspapers ran stories entitled in some form, "Mad Cows and Englishmen…" The author commented:

> "More than a simple coincidence, these headlines reflect a trend in U.S. newspaper coverage linking beef consumption, mad cow disease and England. Many of the articles … focused on the history of beef in England, and pointed out the connection between British identity and beef consumption." [11]

Media coverage of mad cow disease, in retrospect, was a mix of politics, agriculture, economics, trade issues, lifestyle changes, social fear – and not the

least, scientific mystification and public health concern. Given the complexity of the topic, did Mad Cow play "better" in certain mediums rather than others? Perhaps so; the mediums that provided the most coverage were television and the (print) wire services, at 14% each. Radio and magazines had by far the least amount of coverage, with only 3% and 7% respectively.

And if this eclectic stew of ingredients wasn't enough, the colorful Oprah trial landed in 1998 to add yet another element: freedom of speech issues.

Prevalence of Oprah trial

In May of 1996, Mad Cow disease once again grabbed headlines – this time as the focus of a lawsuit brought by Texas cattlemen against Oprah Winfrey, the TV talk show host. The cattlemen were protesting one program which focused on the dangers of beef – particularly Mad Cow disease. When Oprah, one of the most popular and influential media personalities in the U.S., declared on the show that she would "never eat another hamburger", cattlemen claimed that beef prices subsequently plummeted, resulting in an alleged loss of billions in revenue.

The trial ended in victory for Winfrey, and for a few weeks highlighted some interesting freedom of speech issues. Ironically, the issue of mad cow disease, the ostensible lightning rod for the whole show, became merely incidental.

Yet it was the Oprah trial that received the most Mad Cow-related press coverage; a total of 93 news stories out of 534 (18%) focused upon the Oprah trial.

This fact is surprising – yet also understandable. The trial was concrete, dramatic, relatively short, offered a high degree of celebrity and human interest, and was "spun" by Oprah's lawyers to center around one of the cherished American rights, freedom of speech. In contrast, other news regarding mad cow disease was sketchy and mysterious. In addition, Americans felt that they "didn't have to worry about it" because they had been assured by their government that

it "couldn't happen here." Thus, interest from the American media was predictably less than in Europe, where it posed not only public health issues, but significantly affected trade and politics as well.

Antecedents to the Mad Cow crisis: a pattern of government risk mismanagement

In retrospect, many communication experts now suggest that the Mad Cow story developed in the media through convergence of a lack of factual scientific information and a pattern of government obfuscation and denial. Ultimately, this created an information vacuum that the media ended up filling on its own. But the attitudes of the public also played an important role; when assessing the role of the media, it is also important to understand the pre-existing perceptions of the public. And in the case of Mad Cow disease and CJD, the British public had reason to be suspicious: unfortunately, Mad Cow was just the latest in a string of food-related crises experienced by Britons.

In 1988, when BSE was first starting to attract attention from MAFF scientists, the Ministry of Agriculture had their hands full trying to handle an epidemic of salmonella-tainted eggs. When a junior health minister publicly acknowledged that 1,000 people had been poisoned by salmonella that year, she was promptly fired. Ironically, part of the salmonella outbreak was attributed to the farming practice of using rendered chicken remains as feed to chickens – a foreshadowing of BSE feed practices. The salmonella scare resulted in a 50% drop in egg sales.

The following year brought a fresh problem: listeria. Richard Lacey, who was to gain notoriety later in the BSE crisis, went public with evidence of listeria in frozen microwave dinners. Although the British government initially tried to downplay Lacey's findings, they admitted to discovering "disturbing" listeria levels in frozen dinners a few months later.

In both the salmonella and listeria cases, the British government attempted to allay public fears about food safety by initially denying or minimizing risk. This had the reverse effect on the public; eventually, government assurances were perceived as suspect, and the public grew highly mistrustful. A poll taken in July, 1989 by the Consumers Association found that a startling three out of four people agreed with the statement "The government has failed to protect consumers from unsafe food". [12] Sue Dibb, the co-director of the independent watchdog Food Commission, commented that " nobody is trusting what is coming out of the government any more…every time a minister gets up and says beef is safe, there is absolutely no danger, there is absolutely no risk, a whole lot more people stop buying beef because they don't trust the government." [13] It was in this tense atmosphere that the BSE crisis erupted.

Mad Cow Disease not only threatened strongly-entrenched cultural eating habits – it endangered British agricultural and food companies that relied on the billions of dollars generated by the export of beef to international markets. Because of the economic magnitude of these industries, the decisions about how to handle this crisis became strongly political.

The UK government, struggling to avert widespread collapse of the agricultural industry and salvage trade relationships, maintained a stoic message throughout the evolving crisis. In nearly all cases, the government asserted that:

- BSE could be controlled
- scientific evidence did not warrant drastic measures
- the disease posed no threat to humans

As news of Mad Cow disease grew, the UK government consistently attempted to assure the public that the threat to humans was minimal, and that any suggestion to the contrary was simply "scare tactics." Prime Minister John Major, declared that the safety of beef was "not in question." David Maclean, Britain's Food Minister, noted in a *New York Times* article, "the cow disease does

not pose a health risk to humans. We are not in the business of subjecting our people to unsafe food." [14] Agriculture Minister Douglas Hogg proclaimed "I am an enthusiastic eater of British beef", and declared beef to be "wholly safe". One of the most zealous and memorable attempts to calm the public's fear came when Agriculture Minister John Gummer initiated a public relations photo session with his own four year old daughter, Cordelia. The initial idea was for Cordelia and her father to be shown eating tasty hamburgers; however, with the prerogative unique to young children, Cordelia refused to eat the hamburger when the cameras started rolling. The final photos show Cordelia with a half eaten hamburger strategically held by her father; when the BSE/CJD link was confirmed, these photos would later come back to haunt government officials.

The problem with these tactics, however well-intentioned they might have been, was that they denied both real and perceived risk. In the face of uncertainty, government officials usually opted to make statements of certainty that sometimes conflicted with the facts in hopes of decreasing public concern. This pattern was so entrenched that a 1990 editorial in *Nature* warned government officials of rudimentary lessons in risk management by admonishing, "Never say there is no risk. Instead, say there is always risk. The problem really is to calculate what it is. Never say the risk is negligible unless you are sure that your listeners share your own philosophy of life." [15]

Tim Lang, an outspoken critic of government risk management, and the Director of the Centre for Food Policy at Thames Valley University, concurs. He noted in his paper to the House of Commons joint Agriculture and Health Committees' hearings into BSE in 1997:

> "From the very beginning of the BSE saga…some ambivalence was built into the nature of information on the subject – certainty alongside uncertainty. Industrial and government interests have tended to favour certainty, while consumers to a greater extent asked the "what if" questions more overtly…Public confidence is a key factor in crises, yet the Government has seriously misjudged and mishandled it, exacerbating

> consumer volatility and engendering entirely understandable behavior which went against Government's stated intentions."

And finally, he warns, "Treat people like children, and they behave accordingly, quickly sensing victimhood." [16]

On March 20, 1996, when the government was forced to acknowledge that the increasing incidence of CJD might be plausibly linked to Mad Cow Disease, public support and trust in government information was virtually eradicated.

Summary

The Mad Cow phenomenon was tremendously complex; it posed a very real threat to the stability of a wide range of industries, the European economy, but most of all, to human health. Because of the bizarre trajectory of the disease, the future remains uncertain. Although the incidence of BSE has dramatically diminished since changes were made in feeding and rendering practices, no one knows for sure whether another CJD epidemic will recur, due to the lengthy incubation period.

Undoubtedly, there will be instances in the future when a new crisis – with little medical foundation – will strike. What has this experience taught us?

Although each individual case will be different, and require special tactics, the ultimate goal will be to generate trust and facilitate an open, ongoing dialogue amongst all parties involved. Douglas Powell and William Leiss, experts in risk communication, note the following:

> "The public carries a much broader notion of risk, one incorporating, among other things, accountability, economics, values and trust." They also note that "the public expects experts to understand that it is harm to particular individuals that concerns them above all; that some ways of falling ill and dying are more feared than others; that, in view of the massive scientific databases…there should be more certainty in expert judgements, as opposed to the familiar refrain, "more research is needed"; that lifestyle choices (with their attendant risks) voluntarily made are legitimate and may not be questioned…"

"The risk information vacuum arises where, over a long period of time, those who are conducting the evolving scientific research and assessments for high-profile risks make no special effort to communicate the results being obtained regularly and effectively to the public. Instead, partial scientific information dribbles out here and there and is interpreted in apparently conflicting ways, mixed with people's fears. ..failure to implement good risk communication practices give rise to a risk information vacuum; and …this failing can have grave and expensive consequences for those who are regarded as being responsible for protecting the public's interest." [17]

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Appendix A

Transcript of BSE Statement to British House of Commons, March 20, 1996
Stephen Dorrell:

"I would like to make a statement about the latest advice which the Government has received from the Spongiform Encephalopathy Advisory Committee.

The House will be aware that this committee, which is chaired by Professor John Pattison, was established in 1990 to bring together leading experts in neurology, epidemiology and microbiology to provide scientitically based advice on the implications for animal and human health of different forms of spongiform encephalopathy.

The committee provides independent advice to Government. Its members are not Government scientists, they are leading practitioners in their field and the purpose of the committee is to provide advice not simply to Government but to the whole community on the scientific questions which arise in its field.

The Government has always made it clear that it is our policy to base our decisions on the scientific advice provided by the advisory committee. The committee has today agreed on new advice about the implications for animal and human health of the latest scientific evidence.

The committee has considered the work being done by the Government Surveillance Unit in Edinburgh which specializes in Creutzfeldt-Jakob Disease This work, which relates to the 10 cases of CJD which have been identified in people aged under 42, has led the committee to conclude that the unit has identified a previously unrecognized and consistent disease pattern.

A review of patients' medical histories, genetic analysis and consideration of other possible causes have failed to explain these cases adequately There remains no scientific proof that BSE can be transmitted to man by beef but the committee has concluded that the most likely explanation at present is that these cases are linked to exposure to BSE before the introduction of the specified bovine offal ban in 1989

Against the background of this new finding the committee has today agreed to

the series of recommendations which the Government is making public this afternoon. The committee's recommendations fall into two parts:

Firstly, they recommend a series of measures to further reduce the risk to human and animal health associated with BSE Agriculture Minister Douglas Hogg will be making a statement about those measures which fall within his department's responsibilities immediately after questions on this statement have been concluded.

In addition the committee recommended that there should be urgent consideration of what further research is needed in this area and that the Health and Safety Executive and the Advisory Committee on Dangerous Pathogens should urgently review their advice. The Government intends to accept all the recommendations of the Advisory Committee in full, they will be put into effect as soon as possible.

The second group of recommendations from the committee offers advice about food safety on the assumption that the further measures recommended by the committee are implemented. On that basis the committee has concluded that the risk from eating beef is now likely to be extremely small and there is no need for it to revise its advice on the safety of milk.

The Chief Medical Officer will be writing today to all doctors to ensure that the latest scientific evidence is drawn to their attention. In the statement by the Chief Medical Officer which we have placed in the Vote Office, Sir Kenneth Calman poses to himself the question whether he will continue to eat beef I quote his answer: “I will do so as part of a varied and balanced diet. The new measures and effective enforcement of existing measures will continue to ensure that the likely risk of developing CJD is extremely small.”

A particular question has arisen about the possibility that children are more at nsk of contracting CJD There is at present no evidence for age sensitivity and the scientific evidence for the risks of developing CJD in those eating meat in childhood has not changed as a result of these new findings.

However parents will be concerned about implications for their children and I have asked the Advisory Committee to provide specific advice on this issue following its next meeting.

Any further measures that the committee recommend will be given the most

urgent consideration. As the Government has repeatedly made clear, new scientific evidence will be communicated to the public as soon as it becomes available."

(When Dorrell had finished speaking, Douglas Hogg stepped forward to make his statement:)

The House will wish to know the action I propose to take to ensure the risk to the public is minimized.

The additional recommendations just made by the Spongiform Encephalopathy Advisory Committee that most immediately affect agriculture departments are that carcasses from cattle aged over 30 months must be deboned in specially licensed plants supervised by the Meat Hygiene Service and the trimmings kept out of any food chain, and that the use of mammalian meat and bonemeal in feed for all farm animals be banned.

The committee goes on to state that if these and their other recommendations are camed out the risk from eating beef is now likely to be extremely small.

The Govemment has accepted these recommendations and I will put them into effect as soon as possible. Any further measures that SEAC may recommend will be given the most urgent consideration.

Also, and with immediate effect. I have instructed that existing controls in slaughterhouses and other meat plants and in feed mills should be even more vigorously enforced.

I do not believe that this information should damage consumer confidence and thus the beef market. But I should say that support mechanisms exist in the Common Agricultural Policy and the Government will monitor the situation closely. I will naturally report developments to the House.

I recognize that there will be public concern, but the Government's Chief Medical Officer advises us that there is no scientific evidence that BSE can be transmitted to man by beef. Indeed he has stated that he will continue to eat beef as part of a varied and balanced diet as indeed shall I. In view of what I have announced, we believe that British beef can be eaten with confidence."