

# *Deadly Feasts: A valuable examination of the “Mad Cow” epidemic*

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This book should be read by all those concerned over the emergence of new diseases such as Bovine Spongiform Encephalopathy (BSE or Mad Cow Disease) and the new form of its human equivalent, Creutzfeldt-Jakob Disease, that has emerged, variant CJD (vCJD). It traces the efforts by scientists, spanning many decades, to unlock the secrets of this group of disease agents.

The author, Richard Rhodes, is not new to writing on controversial scientific subjects. His book *The Making of the Atomic Bomb* won the Pulitzer Prize in 1988.

The class of diseases to which BSE and vCJD belong are called TSEs—Transmissible Spongiform Encephalopathies (infectious spongy brain diseases). They are feared more than other, better known, diseases with bigger death tolls, since TSEs are largely an unknown quantity. They are unique in being able to destroy the body over years, gradually, from within, without producing any immune response. As well, they are uniquely resistant to the usual methods of prevention and cure. Even boiling and disinfectants have little effect upon the infective agent, let alone antibiotics; and the real nature of the agent itself has so far eluded scientists.

The book examines the heated debate amongst scientists about the nature of TSEs. Some scientists claim that the infective agent is a prion (a structure made entirely from protein), others say it is a virus, and others something else again. Rhodes makes no effort to hide his dislike of the views and methods of Stanley Prusiner, the originator of the term prion. While this tends to get in the way of an objective treatment of the contending theories, the prion theory has not been proven and more research is needed before any of the rival theories can be discounted. None of the present theories can give clear-cut answers to all the questions posed by the peculiar properties exhibited by TSEs.

*Deadly Feasts* is a good introduction to the field. It begins in New Guinea in 1950. At that time, the Fore tribe had a custom of eating the dead bodies of tribal elders. The tribe had been suffering from a decimating illness which they called “kuru” (their word for shivering, referring to one of the symptoms of the disease). At its height it caused the deaths of between 5 and 10 percent of the population in the worst hit settlements, which was more than half the total number of deaths in a five-year period. Scientists noted the similarity with Creutzfeldt-Jakob Disease, CJD.

The book then follows the story of CJD between 1913-21 to the present day, from its discovery by Dr. Creutzfeldt, an assistant to Alois Alzheimer, the neurologist who discovered Alzheimer’s

disease, and Dr. Jakob in Germany. The discoverers of CJD described its symptoms and pathology in great detail, but overlooked the importance of one feature: the presence of tiny holes in the brain. The holes give the brain a sponge-like appearance under the microscope, which is common to all TSEs (hence the word spongiform in the name.)

Having related the scientific work on kuru and CJD in humans, Rhodes describes the research on scrapie in sheep, a disease that showed similarities with the human conditions, but which, unlike kuru and CJD, was known to be transmissible. This posed the question as to whether kuru and CJD were also transmissible. Scientists gave chimpanzees food infected with kuru. After many months, one chimp, Georgette, became ill. This was proof that kuru could pass through the food chain, and that it had spread through ritual cannibalism. In 1966 another chimp was inoculated with brain tissue from a human victim of CJD. By 1968 scientists reported in the American journal *Science* that they had successfully transmitted CJD to a chimpanzee.

The book recounts the tragedy of those who died from a CJD-infected human growth hormone made from brain matter and other transplants. “Only fortuitously was it not more like two hundred thousand,” said, Carlton Gajdusek, the scientist whose work is the subject of many chapters of this book.

Rhodes explains the research done into the nature of the infective agent. All infections studied previously had been carried by living organisms. Viruses, previously the smallest known disease carriers, have no means of ingesting food, and rely on the cells of other animals for their survival. But they do have their own genetic material, which is the means by which their characteristics are stored and passed on.

The infective agent in TSEs is a thousand times smaller than the smallest known virus. More than that, it is almost impossible to kill. As Rhodes says in his preface: “Assault with pressurised, superheated steam in the autoclaves that hospitals use to sterilise instruments for surgery barely slows it. It remains deadly after hours of intense bombardment with hard radiation, months of soaking in formaldehyde, years of burial, decades of freezing. It survives even the fiery furnace of a seven-hundred-degree oven.” Any genetic material able to withstand such conditions has to be very different from that which has been found in other living things.

When a saturated solution is gradually cooled until crystals are on the point of forming, a single crystal dropped in leads to “an

amazing cascade of crystal precipitation.” Rhodes compares this process to what may happen when healthy proteins in a cell are exposed to a seed of misshapen protein from a TSE. They may begin to change to the misshapen form, and having done so then cause others to do likewise in a chain reaction, to form the telltale strings of insoluble proteins found in TSE-infected cells.

In addition to this insight, the third part of the book explains the experience with BSE and vCJD in Britain. Rhodes starts with the first official identification of BSE in April 1985 by Dr Colin Whitaker, who worked as a vet in Kent. BSE quickly appeared in farms across the country. Virtually the only common source from which the disease could have spread was food. Since the disease struck dairy cattle far more often than beef cattle, it was likely to be food given mainly to the former. This made the “protein supplement” rendered from meat and bone meal (and used to boost milk production) the most likely candidate. “But,” Rhodes notes, “meat and bone meal had been fed to cattle in Britain and throughout the world for decades without transmitting BSE. Had something changed?”

“Something” had indeed changed. The use of meat and bone meal had increased from 1 percent to 12 percent of the cows’ diet due to the rising cost of alternative protein sources. Temperatures at which the rendering took place had been lowered and the fat content in the product, which protects microorganisms from heat, had been increased. These were all cost-cutting measures motivated by a desire to maximise profits with no regard for safety. Rhodes provides a good description of these changes prior to the onset of BSE, though exactly how they led to the epidemic will remain a puzzle until a proper research program is carried out.

Rhodes makes a critique of the missed opportunities and unwarranted assumptions of the British authorities following the outbreak of BSE. The Conservative government and its advisers based all their decisions on the assumption that BSE was scrapie, picked up “through infected sheep incorporated into meat and bone meal by inadequate processing.” While measures were taken in 1988 to reduce the spread of BSE in cattle—though even these were not properly policed—no action was carried out to protect the human population.

Rhodes quotes from Professor Richard Lacey’s book, *Mad Cow Disease*, to drive the point home:

“No wonder the UK government was delighted. The risk of BSE to man was ‘remote,’ it would die out spontaneously all on its own once the feed source had been cut off. No real action was needed. Farming could carry on much as before. There was to be no slaughter of herds, and no curtailment of cattle movements or breeding. Beef was safe.”

This approach flew in the face of the long experience with TSEs, and how they can pass from species to species. Alan Dickinson had been researching scrapie since the mid-1960s. It was his team in Edinburgh which discovered that there are more than 20 different strains of the disease. Rhodes tells us that the “government’s position made Alan Dickinson furious. As long ago as 1976, well before the BSE outbreak, Dickinson had written that ‘we should not assume at this stage of our knowledge, that scrapie agents are never transmissible to man from infected meat, particularly as we know that some types of cooking would not

inactivate the infectivity.’”

Dickinson is scathing about the British government’s disregard of science: “The way you train the high-flying administrators, the mandarins, can be summed up in one sentence. It is to train people to be at ease with their consciences when they take decisions about things they do not understand... What is a ‘recommendation’? Ah, it’s a thing that’s written in their final report, where they prenegotiated away whatever wouldn’t be acceptable.”

This same approach was taken to the export of the suspect feed: “I badgered our chief veterinary officer,” a British government veterinarian confessed, “saying that having identified a ‘poisoned food’ it was immoral to export it. But I was firmly put in my place, and told that it was up to the importing countries to put in place all the guarantees needed.”

Rhodes explains that the low figures for BSE and vCJD in other European countries should not be taken at face value. Since the response to cases of BSE is often to slaughter the entire herd, depriving the farmer of his livelihood, this discourages farmers from reporting them. Instead, the BSE-infected cows are often buried on the farm.

An interview with Professor Lacey gives a glimpse of the scale of what may be posed in the worst-case scenario. If the average incubation period for CJD turns out to be “about 25 years, maybe 30 years, then the peak human epidemic will come around the year 2015. If the current numbers of variant CJD cases increase by 50 percent per year compound, as they well might, that would take it to about 200,000 cases a year by then.”

Rhodes’ afterword calls for the feeding of animal protein to other farm animals to be banned completely. He warns that similar conditions to those that gave rise to BSE are present in the USA and other countries, and presents evidence that there is already an American form of BSE that is occurring sporadically: “The United States and Canadian governments have taken steps to limit risk, but their actions represent utilitarian compromises between public health and the rendering industry’s profit margins ... anything less than the most effective possible barrier against the insidious spread of a stealthy, untreatable, incurable and invariably lethal disease is gambling with our lives.”

This book shows that working people cannot rely on a sense of public duty or social responsibility in ministers, civil servants or businessmen to protect public health. This demands a well-informed response by the millions who are being put at risk. From this standpoint, *Deadly Feasts* should be circulated as widely as possible.



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