

Mad Cows or Mad Scientists?

THE SUPPRESSION OF ALTERNATIVE EXPLANATIONS

By David Crowe

The smoke and flames from funeral pyres for hundreds of thousands of British cows are fading into distant memory, but the fear of this disease affecting livestock or wildlife continues to circulate the globe.

Most people do not realize that there is a non-infectious explanation for Mad Cow disease and other spongiform encephalopathies and chronic wasting diseases. This is due to the reluctance of scientists, health and agriculture bureaucrats and most of the media to question a theory that affects public health once it is active policy.

One man, Mark Purdey, has turned himself from organic dairy farmer into an amateur scientist and globe-trotting epidemiologist to doggedly continue building the major alternative theory.

The infectious theory of Mad Cow disease not only resulted in the possibly unnecessary destruction of hundreds of thousands of cows, but it diverted attention from other causes of health problems facing livestock and wildlife. It created a fear of eating beef (perhaps not entirely misplaced, but for the wrong reasons) and resulted in the circulation of tons of toxic materials from the slaughtered cows into the atmosphere. It also prevented investigations into alternative solutions to the epidemic of disease, even though these might be cheaper, more constructive and far less destructive.

The dominant belief is that Mad Cow disease (also known as Bovine Spongiform Encephalopathy or BSE) and the related diseases Scrapie in Sheep and vCJD (variant Creutzfeldt Jakob Disease) in humans are caused by a prion, a mutant protein. These semi-living beings are thought to be able to withstand temperatures that would kill the hardiest bacteria, viruses and parasites. It is believed that this allowed them to be transmitted from sheep to cows through the rendering of sheep brains into MBM (Meat and Bone Meal) protein supplements for cows.

An apparently unrelated health problem in cows that existed before Mad Cow disease was warble fly infestation. These flies lay their eggs in a cow's skin, causing health problems and reducing the value of cow hides. To combat this, in the early 1980's the British government mandated the use of heavy doses of organophosphate insecticides. These were poured in an oil-based liquid along the spinal column of cows. It was intended that they be systemic, absorbed into the cow's body, as it was believed that this was necessary to provide full and enduring protection from warble flies.

Mark Purdey was one of a handful of farmers who refused to use organophosphates (such as Phosmet) on their cows in 1982. He was concerned that the high doses would damage the health of his cows because the application was so close to the spinal column. He was also concerned about the health of people who drank milk from his cows. In 1984, Purdey won his court fight, and gained the right to use less toxic methods to combat warble fly.

When the first cases of neurological problems were reported in cows in 1985, Purdey felt that his avoidance of these pesticides had been vindicated. However, researchers and the British Government had a different idea, blaming the rapidly emerging disease on the recently postulated prion, based on the detection of protein plaques in the brains of sick cows.

Purdey started to publicly argue his theory that organophosphate pesticides were actually the cause

of neurological problems, attracting some attention, and seriously annoying the British scientific establishment and government who were starting to act as if the infectious theory was fact.

Purdey noted many inconsistencies in the prion theory. Cows were supposedly infected by feeding on supplements containing the brains of sheep with Scrapie, yet Shetland Islanders had been eating potted sheep brains for centuries without similar diseases occurring. He also noted that British byproducts were exported around the world, yet the 170,000 British cases of BSE far outnumbered the total in the rest of the world. Cases of BSE had been found on organic farms with cows brought in from outside, but not on those raised from birth on the organic farms, even though organic farming rules allow restricted amounts of the suspect MBM feeds.

Other ruminants, such as goats and sheep, were not affected by Mad Cow-like diseases in England, even though they were fed MBM supplements. Conversely, several antelopes at the London Zoo and cattle at the Liscombe experimental farm developed BSE, but had never been fed MBM supplements.

When BSE was found in other countries it was in places like Bretagne in northwest France where organophosphate pesticides were first encouraged by the French government. As in the UK, BSE cases first occurred a few years after the pesticide program was initiated. The lower number of cases may be due to the lower doses used, the use of annual treatments (as opposed to twice a year in the UK) and because the program was not mandatory.

As further evidence, the decline in BSE cases in the UK began about the same time the warble fly eradication program ended.

British cases of vCJD in humans also fit the environmental theory. The disease was found in some long-term vegetarians and in humans who had never eaten cow brains. There is no good explanation of why cows could only get BSE from eating sheep brains, but humans could get it from eating only other parts of cows.

Although there was a great deal of panic, there were actually few cases in humans. Purdey noted that about 80% of the 82 cases were in rural areas, even though more than 80% of Britons live in urban areas. One cluster in the Weald district of Kent is in a hops growing area where organophosphate pesticides are used at 100 times average levels for all crops.

Purdey lobbied for government funding to test his research. Eventually, he did get a small amount, and Dr. Stephen Whatley of the University of London was able to show in a test tube that organophosphates were found to produce 3 of the 4 protein transformations required to create the mutant prion protein. A victory, but also a major defeat. The UK BSE inquiry admitted that "the door is not yet closed on the possibility that OPs [organophosphates] played a role in rendering cattle susceptible to BSE infectivity," but the infectious theory was still cast in the primary role because of the inability of Whatley to show all four transformations.

Purdey was not about to give up. He felt that there must be a co-factor that he had missed. To find it he went on a tour of places in the world where spongiform encephalopathies had existed in animals or humans for some time, collecting samples of soil and feed. In these places, where organophosphates had little or no use, he found extremely high Manganese levels and low Copper, Selenium, Zinc and Iron. He did not find this in geographically similar areas where no illness was found. The causes of this mineral imbalance varied, including acid rain, volcanic emissions, lead-free gasoline production, emissions from steel, glass, ceramic, dye and munitions manufacturing and the take-off zones of major airports.

BSE-like diseases were found in Colorado among deer and elk in an area of the front ranges where overpopulation often forced starving animals to graze on pine needles. These showed very high levels of Manganese, perhaps due to acid rain from upwind smelters. In Iceland, Purdey found Scrapie associated with similar high Manganese/low Copper soil conditions. In Slovakia the two clusters of CJD are close to ferromanganese factories and glassworks (heavy users of Manganese). These cases may well be related to the almost eradicated occupational disease known as "Manganese Madness" which occurred among miners exposed to poorly ventilated working conditions. Its symptoms and brain pathology are similar to spongiform encephalopathies.

Purdey was not just randomly testing for mineral abnormalities. Copper is a constituent of the normal prion protein, and Manganese could be a replacement when Copper is deficient, or when Manganese is present at high levels, such as in many mineral supplements for cattle. It is at this point that Organophosphates re-enter the theory. They can remove copper from the body, leaving the door open for Manganese (or other similar metals) to replace it in the prion protein. This results in a non-functional conformation of the molecule, particularly when Manganese is from the 2+ form to the oxidative 3+ and 4+ forms.

Recently, Purdey traveled to Groote Eylandt, an island north-east of Australia where 25% of the world's Manganese is currently produced. He wrote a long [detailed account](#) of his journey on his web site.

About one in thirty people in the largely aboriginal Agurugu village, where the fine mine dust regularly settles most heavily, have Groote Syndrome, a progressive neurological disease.

Researchers supported by the mining company hypothesize a genetic defect introduced by Portuguese sailors 300 years ago, even though this theory does not explain why some white mine workers also have this syndrome, nor does it explain the emergence of this syndrome since open pit mining began in the 1960s.

Purdey's theory was now multi-factorial. Organophosphates were a major factor, but the copper/manganese imbalance could be exacerbated by animal feeds or mineral supplements.

Similar situations could occur where the soil is low in the antioxidant metals and high in Manganese.

After extending the theory, David Brown, a researcher at Cambridge University performed experiments that incorporated high Manganese and low Copper conditions and was able to reproduce all four protein changes in vitro, thus providing full laboratory confirmation that Purdey's theory is at least plausible.

At the height of the Mad Cow frenzy, the British government invited Purdey to make a detailed proposal for research funding. Predictably, after sitting on the proposal for more than a year, they rejected it, and then funded two of its reviewers for some of the studies suggested by it. A cynic might conclude that they had asked for a grant proposal solely to have Purdey reveal his arguments and thoughts in full detail, so that they could then fund some 'reliable' researchers to debunk them, without giving Purdey resources that might strengthen his arguments.

Interest in Purdey's ideas is still growing in a grass roots fashion, although slowly, and usually beneath the radar of major media outlets. Purdey has a small grant from the US Fats and Protein Research Foundation, supervised by Dr. Larry Berger of the Animal Science Lab in Urbana, Illinois. Purdey recently gave 14 lectures in Japan, some Slovakian researchers are studying the influence of Manganese and Copper on familial and sporadic cases of CJD. Some British universities are also

quietly investigating in this area.

Purdey is attempting to obtain brain samples from Grootte Eylandt to test for manganese and copper levels, and has persuaded one local GP there to see whether a chelating drug that removes Manganese will have beneficial effects.

Purdey is now investigating whether ultra-violet light is an additional factor in the development of SE diseases, perhaps in concert with a haze of terpenes from the pine trees that often grow at these elevations. He hypothesizes that the eyes could act as a trigger, because of their concentration of nerves exposed to light.

Purdey and other researchers have turned up many potential factors that could stimulate the development of spongiform encephalopathies and chronic wasting diseases. If some or all components of this theory prove to be valid, the solutions to these devastating diseases could be incredibly simple. It may also open new avenues of research into mental illness.

Supplementation of cattle feeds with minute amounts of copper and regulation of the manganese levels could work near miracles, at minimal cost. Chelation could be used to reduce the levels found in people or animals suffering from these illnesses. Yet, it is likely that governments and the scientific establishment will continue to concentrate their efforts almost exclusively on infectious agents and genetic defects, suppressing anybody brave enough to argue against them on this or other health issues.

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Further Reading:

[The Inquiry into BSE and variant CJD in the United Kingdom](#)

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[Purdey M. The Purdey Environmental Home Page](#)

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