

Copper-Bottomed Answers to Mad Cow Disease? There May Be a Simple Explanation for BSE

by George Monbiot, *The Guardian Weekly*

The most interesting aspect of France's BSE scandal is that it makes no sense at all. Britain stopped exporting contaminated cattle feed to Europe in 1991 (though it continued sending it to the third world until 1996). In most other European Union countries cases have already peaked and declined, as expected. But in France the number of infected animals has doubled in the past year. It is impossible to see how this pattern could result from the export of British bone meal.

The transmission of BSE has never been satisfactorily explained by the prevailing theory. The consumption of meat and bone meal from infected cows has doubtless played an important role. Yet this alone fails to account for the huge numbers of cattle in Britain that continued to become infected after most contaminated feed had been removed from the food chain. The latest research on the human form of the disease, Creutzfeldt-Jacob Disease (CJD) published last November, failed to find any link with the consumption of infected beef.

You might imagine that when its theory isn't working, a government would wish to test the alternatives. But the British government has so far sought only to attack a hypothesis that does appear to fit the facts. Since 1988 a Somerset farmer, Mark Purdey, has been arguing that scientists have overlooked the root causes of BSE. Self-taught and self-financed, he has studied the brain's complex biochemical pathways, and this year published a groundbreaking paper in a respected medical journal. His reward is to have been reviled, misrepresented and physically attacked. Prions, the brain proteins whose alteration seems to be responsible for BSE, are designed to protect the brain from the oxidising properties of chemicals activated by dangerous agents such as ultraviolet light, Purdey argues. When, he suggests,

the prion proteins are exposed to too little copper and too much manganese, the manganese takes the place of the copper that the prion normally binds to. The protein becomes distorted and loses its function.

BSE arose in British herds in the 80s, Purdey asserts, because the Ministry of Agriculture started forcing all cattle farmers to treat their animals with an organophosphate pesticide called phosmet, at far higher doses than are used elsewhere in the world. The pesticide had to be poured along the line of the spinal cord. Phosmet, Purdey has shown, captures copper. At the same time cattle feed was being supplemented with chicken manure, from birds dosed with manganese to increase their egg yield. The prion proteins in the cows' brains were both deprived of copper and dosed with manganese. In France the use of phosmet first became mandatory in Brittany. Twenty of France's initial 28 cases of BSE emerged there. BSE's subsequent spread, Purdey maintains, mirrors the use of the pesticide.

Poisoning by similar means may explain the distribution of the human form of the disease. Of the two main clusters of CJD in Britain one, in Kent, is in the middle of a fruit- and hop-growing area where huge quantities of organophosphates and manganese-based fungicides are used. The other is in Queniborough in Leicestershire, whose dyeworks (until they caught fire a few years ago, spraying chemicals over the village) used to dump some of their residues into the sewerage system, Purdey alleges. The sewage was spread over the fields. Dyeworks use shedloads of manganese.

Purdey has tested his theory on BSE and CJD clusters in Iceland, the United States, Slovakia and Sardinia. He found that people and animals had been exposed to deficiencies of copper and surfeits of manganese. Most of the clusters, intriguingly, are in

mountainous areas, where levels of ultraviolet light are high.

But the most compelling evidence in support of his hypothesis comes from a paper published by a team of biochemists at Cambridge University this year. They found that when copper was substituted by manganese in prion proteins, the prions adopted precisely the distinguishing features that identify the infective agent in BSE.

If Purdey is right, he deserves a Nobel Prize for medicine. Instead he has been shot at, his phone lines have been cut, and his house has been burned down. The Ministry of Agriculture, which for 50 years has had a dangerously close relationship with the agrochemical industry, has repeatedly sought to discredit him. Suddenly, however, its tone has changed, and it has now promised to start funding his research. The families of the French victims of CJD are threatening to sue the British government, and it desperately needs an alternative transmission theory.

With funding on its way, and new evidence accumulating every month, a self-educated dairy farmer may be about to overturn the entire body of scientific research on the biggest public health scandal of modern times.

