The Pesticide Link to Mad Cow Disease

The disease hits Canada. Is the U.S. next?

by Mark Purdey

Editor's Note: On May 20, 2003, Canadian agriculture officials reported that a cow slaughtered in Alberta in January tested positive for Bovine Spongiform Encephalopathy (BSE), commonly known as mad cow disease. It is the country's first case since 1993. Fearing the disease could spread to the U.S., the U.S. Department of Agriculture has a temporary ban on Canadian beef. Mad cow disease was first reported in the United Kingdom in 1986, peaking in 1993 with almost 1,000 new cases per week. In 1996, variant Creutzfeldt-Jakob Disease (vCJD) was detected in humans and has been linked to eating contaminated beef. Both are fatal brain diseases with unusually long incubation periods, often lasting years. According to the Centers for Disease Control and Prevention, a total of 125 human cases have been reported in the world: 117 from the United Kingdom, six from France, and one each from Ireland and Italy. No human cases have been reported in association with the recent Canadian case.

While officials have tied BSE to an infectious microorganism, a British organic farmer, Mark Purdey, has linked the disease to organophosphate pesticide exposure. Years ago, Mr. Purdey resisted his government's order to spray his cattle with organophosphates for warble fly and went to court for a judicial review. He won his case, was exempted from using the spray, and has gone on to conduct research on the disease. No cows born in his herd developed BSE. He has contributed numerous articles on the subject of BSE to scientific journals. He farms in Somerset, UK. The following article appeared in Wise Traditions in Food, Farming and the Healing Arts, *the quarterly magazine of the Weston A. Price Foundation, Spring 2000, which can be found on their website www.westonaprice.org.*

As the first snowstorm of winter hit the isolated hill where I farm, I pitched out the last forkfuls of hay to my cattle before nightfall. Much like the whirlwinds of snow surging all around me, my brain was turning over and over the catalogue of injustices that successive governments had levied onto the farming community over BSE. I felt paralysed and powerless in the encroaching snowstorm.

My confidence to carry on was battered to pieces by the recent ban on beef-on-the-bone. The announcement—based on the whims of a mere handful of government "experts"—renders my hard graft over the last twenty years in farming into pathetic insignificance. But how can there be any true "experts" from academia when the most basic facets of the Bovine Spongiform Encephalitis (BSE) disease process remain a total mystery? One would have thought that all of those farmers and independent vets living and working in the front line with BSE cattle would have been the first to be consulted. But strangely, their observations have been completely ignored by officialdom.

Cows frequently partake in the bizarre habit of eating their colleagues' afterbirths after calving, and I was particularly intrigued to watch my own home-reared, BSE-free cows positively relishing the delicacies of afterbirth tissues derived from a group of pedigree cows that I purchased into my farm in 1989. As the majority of these imported cows went on to develop BSE, it is interesting that BSE has not surfaced in my home-reared cows, despite their overzealous exposure to the allegedly "infectious" blood and lymph found in the afterbirths of the BSE cows. Other farmers sharing the same experience report the same outcome.

Another anecdote hails from the farming community of Shetland, where the island folk are free of Creutzfeld-Jakob Disease (the human form of BSE), despite their ancient custom of eating "potted sheep's brain." Interestingly, the equivalent of BSE in sheep, called scrapie, has been rife in the sheep flock on Shetland for centuries.

The anecdotes are ever-flowing, and all point to a hypothesis based upon some environmental causal factor that falls a long way short of the current government's nightmare infectious "ingestion" scenario. If the spongiform agent is as infectious as the authorities would have us believe, why has chronic wasting disease (the BSE equivalent in deer) remained uniquely confined to a small cluster zone in the Rocky Mountains for thirty years now, without spreading across to the neighboring deer herds roaming the rest of the Rockies? Why has no spongiform developed in the various predators of those affected deer?

From the very beginning of the crisis, the farming community has been the unfortunate victim of the whole BSE campaign. Yet, ironically, the same presiding authorities who are responsible for foisting off the burden of BSE are, no doubt, totally oblivious to the fact that more farmers have committed suicide as a result of official BSE blunderings than people have died of new variant Creutzfeld-Jakob disease (nvCJD).

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Government research flawed

A body of government experts was quick to take exclusive control of BSE research, and very rapidly the cause of the disease was attributed to the feeding of scrapie-diseased sheep brains to cattle. In other words, scrapie was said to jump from sheep to cattle by virtue of some sort of infectious agent. And it naturally followed that this same assumption of disease cause was extrapolated into the human CJD context—the presumed "microorganism" had now jumped from cows into humans. But this was no more than unproven hypothesis, and it still remains that way today.

Not surprisingly, only a handful of folk had insight into the unsavory world of the meat and bone meal (MBM) rendering business. But for anyone who had scratched the mere surface of the global distribution of British MBM products, it became strikingly obvious that the very mainstay of the official hypothesis was radically flawed. For instance, during the 1980s thousands of tons of this very same incriminated MBM was exported to cattle farms in BSE-free countries such as the Middle East, Malta and South Africa. Officials have always brushed this challenge aside, arguing that the cattle in these countries did not receive sufficiently large doses of scrapie to contract BSE. But this contradicts their current official explanation for the 30,000-plus cases of BSE that have developed in cattle born after the 1988-ban on MBM, where government scientists conveniently claim that leakage of micro amounts of MBM (destined for pig and poultry feed) into the cattle rations, caused the 30,000 cases.

Furthermore, USA and Scandinavian rendering systems duplicated exactly the same prerequisites that were supposed to kick off BSE in Britain—scrapie affected brains being milled into feed—yet their livestock remained BSE-free.

Nor were we told of the numerous unsuccessful attempts by U.S. scientists to induce BSE in cattle that had been experimentally fed or injected with massive amounts of scrapie brain material. Apparently, the cattle either just "got fat" or went down with a sickness more akin to motor neurons disease than BSE.

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The media loved the theory because they could drum up a viral holocaust-horror scoop. The vegetarian and green lobbies found themselves landed with a powerful propaganda weapon on their plate— turning cows into cannibals. And the UK scientific establishment could go on drawing generous grant funding for their viral witch-hunt without the embarrassment of having to account for years of barking up the wrong tree. And then the government could foist the blame of BSE onto a naturally occurring agent for which no significant vested interest or official body could be held accountable.

Whilst the maligned renderers and feed merchants got the full brunt of blame for BSE, it surprises me that neither were held accountable for the financial damages of the crisis. Instead, they all received generous compensation payments to the tune of millions.

Almost on a weekly basis we are now finding ourselves listening to the same experts regurgitating the same stereotype claims of how BSE has now jumped from cattle into humans. On Channel 4 Dispatches, despite no reported cases of BSE in the British sheep flock, it was assumed that sheep must be affected with BSE because they had eaten meat and bone meal. We are now warned of the danger of eating sheep. Professor Blakemore summed up the program by saying that we should all eat chicken and avoid beef and mutton. But as poultry received their fair share of meat and bone meal as well, should we not be cutting chicken out of our diet too, according the dictates of the official theory?

Questioning the conventional hypothesis

These spokespeople would do better to start questioning the entire foundation of their hypothesis, rather than squeezing the last drop of "infected" blood out of the sinking stone. What is more, the conventional consensus on BSE is ignoring that well-recognized academic yardstick, Koch's postulates, which is employed for assessing the cause of disease. The first postulate dictates that a theory begins to carry weight once the hypothetical causal agent can be identified in every victim of the disease in question. The conventional hypothesis on scrapie/BSE/CJD certainly fails to fulfil this basic postulate on several counts. In this respect it is particularly interesting that spongiform disease has been experimentally induced in animals after receiving injections of brain tissue derived from people who have died of Alzheimer's and Parkinson's Disease. Why is nobody freaking out about Alzheimer's disease?

In the case of BSE where no viral cause has been identified, it is illogical to assume that one animal has to eat another in order to catch the same disease. Initially, the direction of any epidemiological research programme should follow elementary logic and investigate the most likely assumption that the various different species of mammals suffering from the same disease have all been exposed to the same causal factor in the environment. But it seems that nobody has investigated this route. Sheep did not cannibalize each other in order to catch scrapie, nor did wild deer in the Rocky Mountains cannibalize each other in order to catch their BSE-equivalent disease, chronic wasting disease.

The reductionist mindset of government scientists is betrayed by the narrow scope of questions that have been put to the relatives of the new variant Creutzfeld-Jakob Disease victims. The questionnaire is almost entirely focused on the carnivorous perspective of the victims' diets, and therefore tailored to suit their own hypothesis from the outset. The Establishment's assessment of nvCJD etiology seem to have completely ignored the fact that adolescent CJD was recorded well before the 1980s. And why do they move the goal posts every time a new challenge confronts their theory—like extending nvCJD's incubation period to tally with the long term vegetarian victim from Kent? Take note that they have completely ignored the case of the lifelong vegetarian nvCJD victim from France.

The British government's Spongiform Encephalitis Advisory Committee (SEAC), seems to have thrown aside one of its most relevant long standing observations on CJD epidemiology—people who are occupationally involved with pets and farm animals are at greater risk of developing CJD. And it is this observation that may well hold the key to the true cause of these diseases.

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Organophosphate pesticides linked to BSE

During the 1980s and early 1990s, cattle and cats (the species of animals that have developed BSE) were exclusively treated with systemically acting types of organophosphate (OP) insecticide which were designed to penetrate the entire physiological system of the animal, transforming the bloodstream into a toxic medium so as to kill off any unwanted parasites present. In the context of cattle, the use of these systemic OP's was subject to a compulsory government order for the eradication of warble fly. The UK government was unique in compelling a substantially higher biannual dose of this OP by comparison with the few other countries around the world that were following similar, less intensive measures to control this fly. Interestingly, these other countries, including Switzerland, France and Ireland, comprise the few other countries that are suffering from very small epidemics of BSE in their home-reared cows.

The National Farmers Union, the Meat and Livestock Commission and The British Veterinary Association formed a united front with MAFF (Ministry of Agriculture, Fish and Forestry) to ensure that all farmers complied with the law and treated their cattle. Systemic OP's are recognized as exerting their toxic effect by entering the central nervous system and deforming the molecular shape of various nerve proteins. These chemically-mutilated mutant proteins are subsequently rendered incapable of performing their proper function in the nerves.

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the malformation of another newly discovered brain protein called prion protein—the phenomenon that U.S. scientists have proposed as the cause of spongiform encephalopathies. Whilst some types of spongiform disease have been attributed to genetically acquired damage to the shape of the prion protein, the underlying cause of protein damage in the BSE and new variant CJD strain of the disease remains a mystery—amongst "open-minded" scientific circles, at any rate.

OP's are known to generate a highly reactive type of "free radical" in the tissues that they intoxicate. And it is this free radical legacy of OP poisoning which is capable of instigating a chain reaction of lethal attacks on nerve membranes and proteins in the central nerves of susceptible individuals.

Once tissues become 'infected' with free radical chain reactions, the introduction of freezing, heat or radioactive conditions to the affected cells does not arrest such an 'infection.' In fact, irradiation, heating and homogenizing of such tissue (brain tissue from spongiform affected animals is homogenized before it is inoculated into healthy animals in transmission trials) actually proliferates the free radical phenomena. This suggests that these free radicals may constitute the as yet unidentified "infectious" transmissible agent of these diseases.

Concerned members of the public helped me to fund a £14,000 experimental research project at the Department of Neuroscience, Institute of Psychiatry in London, where living tissue culture cells which express the prion protein were exposed to low doses of the OP chemical; so as to stimulate the context of a living cow undergoing OP treatment. Significantly some of the recognized changes of the prion protein, which appear in the early stages of spongiform disease, were induced in these OP-treated cells.

Clearly, these results go some way towards proving that OP's represent a primary or partial cause of BSE. Yet it was this very same simple test that the government had always assured me was too expensive for the taxpayer to fund and, besides, impossible to set up anyway, even with the most updated lab technology.

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In December 1996 Lord Lucas, MAFF's spokesman in the House of Lords, gave a written answer stating that the government had asked the SEAC committee to revisit the OP-BSE theory as a result of the recent research findings conducted at the Institute of Psychiatry.

After being invited to deliver my thesis to a SEAC meeting in April, 1997, I was disturbed that at no stage during the protracted inquiry that followed was the experimental evidence of the Institute's work addressed—the prime purpose behind this hearing. The committee dismissed the evidence that I presented, which had been drawn from independent peer-reviewed, published science literature. I was not surprised to learn that the outcome of this inquiry—the proceedings of which were described as "confidential" to any inquiring journalist—was a recommendation to government that any applications for funding research into the OP-BSE theory should not be supported.

I still shudder each time I visit our local farm stores and see the canisters of systemic OP products up for sale. Although the warble fly is eradicated and BSE is on the wane, farmers can still apply these chemicals in a voluntary capacity for controlling lice and other pests. I shudder further when I see the bottles of OP head lice shampoo and OP systemics for pets and gardens still in the shops for human use.

The real madness of the mad cow fracas would seem to lie with the deadlock that has kept these products on the open market for a full year since experimental evidence first linked their use to the cause of BSE. Perhaps the government is so scared of compensation claims that it employs everything at its disposal to prevent any degree of acceptance of the idea that their compulsory warble fly programme caused the biggest catastrophe in the history of British agriculture.

The brave new SEAC committee appears to be totally preoccupied with "effect" rather than "cause." Such a back-to-front approach betrays their sensitivity with anything to do with "cause." But how can any government program seriously hope to eradicate BSE or nvCJD if it has failed to eradicate, let alone recognize, the disease's true cause?