Organophosphates Implicated In Mad Cow Disease

Cover-up: Insecticide causes Mad Cows & nvCJD
by Fintan Dunne
Research Kathy Mc Mahon
ICI's nerve gas insecticide triggers BSE & human nvCJD

If Mark Purdey is right we are in big trouble. We are destroying our brains with insecticides.

His groundbreaking research into the cause of BSE in cattle and new variant CJD in humans, has been sidelined by United Kingdom officials. They attribute both diseases to ingestion of prion protein found in contaminated beef. But Purdey has evidence the government's anti-parasite campaigns unleashed a chemical holocaust for cattle resulting in BSE, and that human CJD is accelerated by the same chemical effects.

Could a chemical be that deadly? For fear of attack by Saddam Hussein, most Israeli hospitals have antidotes to a deadly nerve gas developed by Nazi chemists which contains organophosphate (OP) -- the same compound found in the insecticides suspected of driving BSE and CJD. The vast bulk of the cattle found staggering around in British fields with their brains burned out, have been treated for warble-fly with a constituent of nerve gas.

The CJD and BSE symptoms also mirror 'manganese madness', an irreversible fatal neuro-psychiatric degenerative syndrome that plagued manganese miners in the first half of the last century. Could manganese and organophosphate be causing these diseases?

Cambridge scientist David R. Brown is hot on the trail. His recent research has shown that the prion proteins linked to BSE can bond destructively with manganese found in animal feeds or mineral licks. His latest, as yet unpublished work has found a tenfold increase in the metal manganese in brains of CJD victims.

All this is fully consistent with the Purdey hypothesis. These manganese-tipped prions could be the principal cause of the neurological degeneration seen in BSE. But manganese is only the bullet -- organophosphate insecticide is the high-velocity gun. It fires manganese into the brain by depleting copper which the manganese then replaces.

Purdey says the manganese-tipped prions set off lethal chain reactions that neurologically burn through the animal. Phosmet organophosphate has been used at high doses in British warble fly campaigns. Privately, scientists will confirm that prions in the bovine spine -- along which this insecticide is applied -- can be damaged by ICI's Phosmet organophosphate insecticide. But few will state it publicly or publish it as scientific finding. In 1996, former ICI subsidiary Zeneca sold the phosmet patent to a PO Box company in Arizona called Gowan -- just one week before the UK government admitted to a link between BSE and nvCJD.

Bonding the prion

Cambridge University prion biochemist, David R. Brown is dismissive of the science behind the infectious model of BSE. He terms it "a very limited amount of science by a few assumed -- reputable scientists." He insists there is "no evidence an infectious agent is present in either meat or milk."

"Simple tests on udder walls of cows -- which could easily detect an infectious prion -- have not been done, why I don't understand."

A number of researchers have found that organophosphate (OP) in systemic warble fly insecticide can deform the prion molecule, rendering it ineffective at buffering free radical effects in the body. Worse still, the prion is then partial to bond with manganese and become a 'rogue' prion. A chain reaction whereby rogue prions turn others to rogues also, can explain the bovine spongiform disease mechanism.

Brown showed how prion protein bonds benignly with copper, but lethally with manganese. Even natural variations in relative environmental availability of manganese versus copper can trigger prion degradation. Chickens notoriously excrete most of the supplements fed to them -- including manganese. And their manganese-rich excreta have been blended into cattle feed in the UK.

Scientist and organic farmer, Mark Purdey gave evidence to the UK BSE inquiry, that warble fly insecticide was the cause of the disease. The scientist wheeled out to rubbish Purdey's evidence -- Dr. David Ray, later turned out to have been receiving funding from the insecticide manufacturer ICI.

A lobby group that includes Bayer, Monsanto, Novartis, Pfizer, Roche and Schering-Plough was behind the effort to discredit Purdey. In December 1999, the same Dr. David Ray was appointed to the UK Veterinary Products Committee

(VPC) -- a government body that licences animal medicines.

Purdey has been consistently denied even exploratory funding to extend his privately supported research. Yet the Purdey chemical poisoning model matches with the epidemiological spread of CJD clusters in humans. It also predicts the incidence of BSE-type diseases in animals. The accepted infectious model fits neither.

The pharmaceutical industry has key motives to deny the chemical source of BSE and CJD, because a spotlight on chemicals would expose the role the insecticides in Alzheimer's -- another neurodegenerative disease. That might lead to claims which would dwarf those from BSE and CJD litigants. In fact, two leading brain researchers into CJD and Alzheimers have died in suspicious circumstances in recent years.

In the United States, the Environmental Protection Agency is already reviewing Phosmet's safety. And the Centers for Disease Control in the US has recently conducted experiments on mice that confirm the organophosphate risk.

According to Purdey, not only is the EC beef slaughter campaign futile -- because BSE disease is mostly non-infectious, but unless the underlying chemical cause is addressed, BSE will simply reappear from chemical causes. A new warble fly campaign is already underway in France using the organophosphate insecticide.

His greater concern is that some lotions for scabies and head lice are now priming children and adults for CJD and Alzheimers in later life, and that manganese in unleaded petrol may prove as deadly as the lead it replaced.

Shining a light on spongiform

Speaking from his rural English Somerset farm -- as plans forge ahead for the European cattle cull, Purdey asks: "Why does CJD degeneration in humans begin in the retina, and why are CJD disease clusters found in high altitude locations?"

The question is rhetorical, and Purdey has an eye-opening answer. He argues that the prion molecule acts as a shock-adsorber of damaging energy from ultraviolet rays and other oxidizing agents.

Once this prion defence system is rendered ineffective by organophosphates, these oxidizing effects have an unmediated impact on tissues. Eventually, UV radiation damages the retina and oxidative stress destroys the brain tissues of CJD patients. This theory would expect to find higher CJD incidence in mountain regions -- where UV radiation levels are elevated. That prediction holds true.

A similar but accelerated mechanism could be driving BSE. ICI's Phosmet organophosphate warble fly insecticide -- applied on the backs of animals along the spinal column, similarly degrades prions. "Systemic versions of the insecticide are designed to make the entire cow carcass toxic to warble fly," explains Purdey. "Unfortunately it's toxic to prions too -- especially those prions located just millimeters from the point of application."

Since first postulating an environmental -- rather than infectious - -- theory of spongiform diseases, Purdey has built evidence from around the world that explains and predicts the incidence in humans and animals: a cluster of CJD in Slovakia, Eastern Europe -- around a manganese plant; Rocky Mountain deer with Chronic Wasting Disease (CWD), who were found to be eating pine needles rich in manganese; the futile slaughter of sheep in Cyprus -- only for BSE to reemerge within years.

"The reappearance of BSE in Cyprus obviously points to an environmental cause," says Purdey, who is sanguine when reflecting on the condemnation of him by mainstream scientists.

>From this research, any prudent person would conclude there is a significant risk attaching to the use of organophosphate in humans. Preparations for head lice and scabies are known to be overused in practice and might be priming users for CJ disease.

Purdey believes his bias for field work is the key to his success. He bemoans the "reductionism" of much lab-centered science. "I have travelled the world to investigate known clusters of spongiform disease - -- something mainstream researchers don't seem remotely interested in doing."

"I suppose they have mortgages and kids who need to go to university," he muses. "Privately, some were agreeing with me, but then they would denounce me publicly. It was quite strange really."

The money trail

Critical scientists like Purdey are unlikely to prevail. The pharmaceutical industry holds most research purse strings, and would hardly energetically explore an avenue of research that could expose them to litigation for causing BSE. The official theory is lavishly funded, alternative theories rarely, if at all.

There are more explosive implications to his -- and other's latest research. Purdey says similar organophosphate-induced protein deformation could also underlie Alzheimer's and related diseases. If that were true, the litigation fallout would destroy some pharmaceutical giants, and a lot of very influential noses would be out of joint.

Disturbingly, Purdey and other brain researchers seem to have had an undue share of unfortunate accidents. Purdey's house was burned down and his lawyer who was working with him on Mad Cow Disease was driven off the road by another vehicle and subsequently died. The veterinarian on the case also died in a car crash -- locally reported as: 'Mystery Vet Death Riddle.'

Dr. C.Bruton, a CJD specialist -- who had just produced a paper on a new strain of CJD -- was killed in a car crash before his work was announced to the public. Purdey speculates that Bruton might have known more than what was revealed in his last scientific paper.

In 1996, leading Alzheimer's researcher Tsunao Saitoh, 46 and his 13-year-old daughter were killed in La Jolla, California, in what a Reuters report described as a "very professionally done" shooting.

What Alzheimer's Disease, Mad Cow Disease, and CJD have in common, is abnormal brain proteins and a putative link to organophosphates. Other neurodegenerative diseases and even Gulf War syndrome among returning veterans has been attributed, in part to the insecticide. But the sidelined
scientists' suspicions are still largely ignored.

In their favour at the moment, is a growing unease on the part of the public. As BSE forges on and Governments panic, Science may be out to lunch on BSE, compromised by bovine spongythinking myopathy.

Mark Purdey funds his own research, testing/labs/travel to cluster sites. Donations to his research fund will help him carry on his work. Mark Purdey Research Fund, High Barn Farm, Elworthy, Nr Taunton, Somerset TA4 3PX, UK.

http://www.cjdalert.com

Note from Jonathan Campbell:

If organophosphates are indeed the causal factor in BSE and nvCJD, the agrochemical giants such as Monsanto, Syngenta, and Aventis have more to fear than litigation. As the toxic effects and persistence of organochlorine pesticides became known, the agrochemical industry shifted to organophosphates, which represent the majority of insecticides and herbicides in use today. They are the underpinning of highly mechanized, pesticidal agriculture, which is used to grow more than 90% of U.S. produce. Most non-organic produce today has measurable residues of organophosphate pesticides. Evidence of danger of these widely-used chemicals is a serious threat to a cornerstone of U.S. agribusiness.

Additionally most of the revenue and sales advantage of genetically modified crops - such as Roundup-Ready Soybeans - are based on the widescale use of organophosphorus herbicides such as Roundup and Liberty (Basta). Serious health concerns regarding this class of pesticides would
place the genetic engineering of crops into question.