More Letters

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regardless of whether it is true or not. Journalists aren't checking on the validity of medical reports, and they aren't interviewing opposing views. In the case of the recent vitamin C report, reporters did not interview the National Nutritional Foods Association, the Council for Responsible Nutrition, the Vitamin C Foundation, nor the American Healthcare Products Association.

But how long can the public be fooled? Why are the pharmaceutical companies so afraid of a simple vitamin? It's because high doses of vitamin C virtually eradicate the risk of developing cataracts, eliminate the need for blood pressure medication, reduce the need for anti-allergy drugs, reduce the risk of gall stones, and produce many other health benefits. The drug companies can't invent and patent a molecule as efficacious as vitamin C.

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Mad Cows or Mad Scientists?

Editor:

The world is going mad over Mad Cow disease. Hundreds of thousands of cows have been slaughtered, many people stopped eating beef from affected countries, governments have been criticized for not doing enough to stop the spread of the infection and trade wars have broken out as countries squabble over whose fault it is and where the next outbreaks are likely to occur and whether enough is being done to contain them.

Most believe 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) are caused by a Prion, a mutant protein. These semi-living prions are thought to be able to withstand temperatures that would kill the hardiest bacteria, viruses and parasites.

When organic dairy farmer Mark Purdey refused to use organophosphate pesticides (such as Phosmet) on his cows in 1982, as mandated by the British government to combat warble fly infestations, Mad Cow disease did not exist. He was simply concerned that the high doses poured along the backs of cows. specifically formulated to be absorbed into their bodies, would damage their health, particularly 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. After a long legal battle, Purdey won 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. Purdey started to publicly argue his theory that organophosphate pesticides were actually the cause, attracting attention, and seriously annoying the British scientific establishment and government who were starting to act as if the infectious theory was proven.

Purdey noted several inconsistencies in the infectious 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 rest in the world. When BSE was found in other countries it was in places like Brittany in France where organophosphate pesticides were first encouraged by the French government. As in the UK, BSE cases arose 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 the optional nature of the program. As further evidence, BSE cases in the UK started declining approximately after the warble fly eradication program was 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. Purdey noted that about 80% of the 82 cases were in rural areas, yet over 80% of Britons live in urban areas. One cluster in the Weald district of Kent is in a hop 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.

Purdey was not about to give up. He went on a tour of places in the world where spongiform encepalopathies 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 elevated Manganese levels and low

Copper, Selenium, Zinc and Iron, but not in similar areas where no illness was found. This was true in Colorado among deer and elk in an area of the front ranges where overpopulation often forces grazing on pine needles, which showed very high levels of Manganese, perhaps due to acid rain. In Iceland, he found Scrapie associated with similar 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 encepalopathies.

Purdey was not just randomly testing for mineral abnormalities. Copper is a constituent of the normal prion protein, and Manganese could be a replacement for it when Copper is deficient. 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 metals) to replace this element in the prion protein, but result in a non-functional conformation of the molecule. Organophosphates may also act to move Manganese from the normal 2+ form to the oxidative 3+ and 4+ forms.

David Brown, a researcher at Cambridge University changed his experiments to incorporate high Manganese/low Copper conditions and was able to reproduce all four protein changes *in vitro*, thus vindicating Purdey's theory.

Purdey and his supporters have not given up. He informed me that the British government, in a remarkable about-face, has asked him to lead a 3-year research study into clusters of spongiform encephalopathy around the world. It is critically important that the *true cause* of spongiform encephalopathies be determined, because the treatment is based on the believed cause, and not the effect. If Purdey is right, the health of cows could have been maintained through modifications in pest control and mineral supplementation regimens, and human victims may have been saved through chelation, supplementation and other therapies to right the Manganese/Copper imbalance.

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Further Reading:

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