EDUCATING RIDA*

An underground scientific journey into the origins of spongiform disease

By Mark Purdey

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(ICELANDIC)
n. transmissible spongiform disease; wasting disease of ruminant animals, such as cattle and sheep

Since 1986, the infamous neurodegenerative syndrome known as Bovine Spongiform Encephalopathy (BSE) has blighted the heartbeat of British Agriculture. The disease has led to the annihilation of thousands of cattle, whilst its human analogue, new-varient Creutzfeld Jakob Disease (vCJD), has blighted the lives of a growing number of young people. Moreover, the spongiform epidemic has created a fierce battleground between nations, vested interests, political parties, farmers, victims and consumers.

But despite the severity of the BSE legacy, little genuine attempt has been made to crack the causal riddle of these diseases, thereby leaving us devoid of insight into measures that would best cure, control and, better still, prevent this terrible illness.

Hard evidence has been amassed so far which indicates that vCJD and BSE could both result from separate exposure of cattle and humans to the same set of toxic environmental factors

This story is an attempt to shine a ray of light over the whole debacle. It charts my own eco-detective escapades and original field investigations, which ran in tandem with the laboratory research of Cambridge University biochemist, Dr. David Brown. These works have gone a long way towards unearthing the truth underpinning the original cause of these grotesque diseases.

Hard evidence has been amassed so far which indicates that vCJD and BSE could both result from separate exposure of cattle and humans to the same set of toxic environmental factors—excess manganese and oxidizing agents—and not from the ingestion of beef by humans and animal by-products by cows. If this hypothesis continues to accumulate corroborative evidence, a radical upheaval of the status quo mindset can be expected.

Despite the fact that my theory has been substantiated both by field and by laboratory findings, Dr. Brown's and my own published works have largely been dismissed, with all funding proposals irrationally rebuffed at peer review. Contrary to the recommendations to UK government by the 1999 BSE Inquiry Report, rejection of our grant proposals continues to the present day, including one submission aimed at developing a feasible cure for vCJD!

THE LONE VOYAGER

My story begins in 1984 with the Ministry of Agriculture, Fisheries and Food (MAFF) Warble Fly Order, which called for compulsory treatment of UK cattle with toxic organophosphate insecticides. I fought the order for my own pedigree Jersey cattle herd and won a precedent High Court Judicial Review ruling against MAFF, debarring their enforced insecticide treatment of my cows.

The insecticides applied to the backlines of UK cattle was called Phosmet, a systemic acting chemical that, amongst a myriad of toxicological effects, disturbs the crucial balance of metals in the brain. I was therefore not surprised to witness BSE rearing its ugly head in UK cattle in 1986. In my opinion, this was a direct legacy of the UK government's warble fly mandate that exclusively enforced an annual dose rate that was four times higher than the application rates employed in the few other countries that used this type of insecticide.

(Phosmet, by the way, was produced by ICI Zeneca, which held the patent on it until March 1996, the same month that BSE was announced in humans. The patent was then sold to an unknown company in the Arizona desert called Gowans.)

I was a working dairy farmer with first-

TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

Transmissible Spongiform Encephalopathies (TSEs) are a group of progressive neurodegenerative diseases that emerge as three basic types: the inherited familial forms, the traditional, long-standing sporadic forms, and the new variant forms.

The inherited familial forms are strongly linked to a mutation on the prion protein gene, although environmental factors are involved in triggering the TSE. The profile of these TSEs is characterized by a slow degeneration of the central nervous system, ultimately ending in dementia, motor difficulties and death. ***Gerstmann-Straussler-Scheinker*** disease progresses slowly over about five years; ***Fatal Familial Insomnia*** begins with bizarre sleep and sexual disturbances and rapidly progresses to a fatal chronic insomnia lasting just a few months.

The traditional forms of TSEs appear in older mammals and have a variety of names depending on the species of animal: ***scrapie*** in sheep and goats, ***chronic wasting disease*** in deer and elk, ***transmissible mink encephalopathy*** in mink, and ***sporadic Creutzfeldt Jakob disease (CJD)*** in humans. These sporadic forms of TSE exhibit a brain pathology that is characterised by spongiform degeneration and loss of neurons in many regions of the brain (such as the cortex), gliosis, shrunken basal ganglia and prion rods (fibrils of aggregated proteins, mainly composed of misfolded prion protein). Symptoms involve behavioural and cognitive disorders progressing to visual, motor and sensory disorders, then ataxia, muscle wasting, seizures, insanity and death.

The modern new variant forms appear as ***Bovine Spongiform Encephalopathy (BSE)*** in young cows, cats, antelopes and ***new variant Creutzfeldt Jakob disease (vCJD)*** in young humans. These new variant forms involve an accelerated, more aggressive course of the disease involving a more widespread unique neuropathology characterized by florid plaques (plaques surrounded by spongiform holes) with more pronounced psychiatric disorders.

hand experience of BSE erupting in cattle that had been purchased into my organic farm. But I was struck by the fact that no cases of BSE had ever emerged in home-reared cows on fully converted organic farms, despite those cattle having been permitted access to the feed that contained the meat and bone meal (MBM) ingredient as part of their 20 percent conventional feeding stuff allowance decreed in the organic standards.

The UK government was quick to blame the origins of BSE on the mysterious "scrapie agent," a malformed protein or "prion" found in the brains of all sheep who are suffering from the age-old neurodegenerative disease, scrapie. The "experts" argued that this supposedly "infectious" agent had jumped across from sheep into cows as a result of feeding cows with meat and bone feed that had been contaminated with scrapie-affected sheep brains. Relaxation of the rules governing the manufacture of MBM in the early 1980s was supposed to have initiated the BSE epidemic.

FLAWS IN THE CONVENTIONAL HYPOTHESIS

From the beginning the flaws in the Establishment's theory were evident:

1. Thousands of tons of the incriminated UK MBM feed was exported for cattle feed during the 1970s, 1980s and 1990s to areas that have remained BSE-free to date, such as South Africa, Sweden, Eastern Europe, Middle East, India and other Third World countries.

2. Changes in the temperature and manufacturing techniques of the MBM rendering process in the UK were blamed for permitting the survival of the scrapie agent in dead sheeps' brains, enabling the "agent" to jump across into cattle, thereby producing BSE. Yet in other scrapie-endemic countries, such as USA and Scandinavia, the exact same continuous flow system of rendering was adopted five years before the UK, yet these countries remained BSE-free.

3. Several US trials failed to invoke BSE in cattle after feeding or injecting them with massive doses of scrapie-contaminated brain tissue.

4. More than forty thousand cows born after the UK's 1988 ban on MBM inclusion in cattle feed have still developed BSE. Furthermore, a small number of cows born after the further additional 1996 ban on MBM inclusion in feed destined for all types of livestock have already developed BSE.

5. There have been no cases of BSE in the other ruminants, such as goats and sheep, susceptible to transmissible spongiform encephalopathy, despite the customary inclusion of an MBM protein source in their feeds.

6. Four of the original five kudu antelope that developed BSE at the London zoo had no possible access to MBM-containing feeds.

7. The UK government's former experimental farm at Liscombe on Exmoor was designed to raise suckler beef cattle on a pure grass-and-silage system without any resort to feeding concentrated feeds at all. Yet BSE struck down four animals on this holding.

8. The UK's mechanically processed meat products and baby foods blamed for causing vCJD in humans were exported all over the world to countries where vCJD has not erupted. Likewise, the practise of "skull splitting" in small rural butchers was offered as an explanation for the growing number of vCJD clusters in rural areas. But this has been practised by the smaller butchers all over the UK for centuries without any outbreak of vCJD.

Despite the myriad of epidemiological flaws and millions of dollars worth of research failing to ascertain any association between the origin of these diseases and the scrapie agent, the whole propaganda myth that BSE was caused by scrapie has become gospel to the mainstream public and professional mentality.

It is easy to see how such a reductionist

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Suzanne Lupien

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mindset took hold: the media loved the theory because they could drum up a viral holocaust-horror scoop. The vegetarian lobby found themselves endowed with a powerful propaganda weapon on their plate, whilst the scientific institutions could carry on drawing generous funding for their hyperinfectious witch-hunt without the embarrassment of having to account for years of barking up the wrong tree. And the government could conveniently off-load the blame onto the vagaries of some naturally occurring phenomena for which no vested interest or official directive could ever be held accountable.

PRION ORIGINS:
THE QUEST FOR PRIMARY CAUSE.

It is well demonstrated that the central pathological hallmark of all types of spongiform disease is the presence of a malformed protein—known as the "prion"—in the nervous system of diseased mammals. But none as yet has explained how and why this "prion" is originally formed in the natural world.

I became interested in the possibility that the systemic OP warble fly insecticides—which had to be poured along backline of the cow just millimeters away from the prion protein-expressing cells in the spinal cord—may trigger this malformation in some way, thereby serving as the primary cause of the disease.

It is well recognized that OP insecticides exert their toxic effects in mammals by deforming the molecular shape of various nerve proteins to the extent that they cease to perform their proper function in the brain. But none had ever considered that OPs could deform the prion protein in this way.

After many abortive attempts to coerce the Establishment into running the correct laboratory test, I eventually managed to raise funds from well-wishers and personal loans to finance Dr. Stephen Whatley of the Institute of Psychiatry in London to challenge brain cell cultures with Phosmet, the actual OP used at uniquely high doses on UK farms.

Amazingly, these trials demonstrated that the OP altered the cellular metabolism of prion protein in some of the ways observed in the early stages of spongiform disease, suggesting that Phosmet exposure may render mammals more susceptible to the disease. But these experiments did not produce the key deformation of the prion protein that represents the central hallmark of the transmissible spongiform encephalopathy (TSE) diseased brain. I returned to square one, assuming that OPs in combination with a further factor X could provide the final missing link.

THE CLUSTER BUSTER

I grew exhausted by the vortex of the mad politico-medico-multinational grand alliance that had successfully hijacked all UK scientific research into TSEs. I embarked upon a refreshing global trek to analyze the unique environments where traditional TSEs had erupted as high incidence clusters for many years. After tramping the world's most clear-cut TSE cluster zones in Colorado, Iceland, Slovakia, Calabria and Sardinia, where an assortment of animals and humans had developed TSE at exceptionally high rates, I discovered a common factor - abnormally high levels of the metal manganese, and rockbottom levels of copper, selenium and zinc in all of these food chains. Manganese levels were normal in adjoining disease-free areas.

BSE IN BRITAIN - A SAD HISTORY

The first official reports of BSE outbreak were in 1986 from the southeast of England, although many vets, farmers and slaughterers had suspected a trickle of cases from the late 1970s onwards. The disease has been an economic disaster for British farming interests as many cows were slaughtered and British beef was banned from Europe. The disease rapidly developed into a massive bell shaped epidemic which peaked in 1992 at 36,680 cases in the year and dwindled back to 1000 cases a year where the incidence rate stands at today. BSE has taken nearly 200,000 confirmed cases to date. The disease was largely concentrated in the South of England during early days; it erupted in some remote Scottish districts in later years due to the importation of the warble fly in cows being brought from Europe and who were then treated with organophosphates.

THE MEN FROM MANGANESE

A specific environmental source of manganese could be pinpointed in each cluster zone tested, where each habitat occupied by the TSE-affected species in question could be directly connected to the atmospheric fallout of some naturally occurring or industrial source of combusted manganese oxide, stemming from volcanic, acid rain; steel, glass, ceramic, dye and munitions factories; lead-free petrol refineries; the takeoff airspace beyond airports, and so forth.

My observations enabled me to construct a holistic hypothesis on the aetiology of TSEs, work that lead to my connecting with the pioneering laboratory studies of Dr. David Brown at Cambridge, a widely published biochemist who had single-mindedly pursued his groundbreaking studies on the elusive prion protein.

Dr. Brown demonstrated that in the normal healthy brain, the prion protein bonds to copper and that this copper-protein can exert an antioxidant function.

Brown's lab studies were complementary to my field studies, thereby providing the other half of the necessary ground work upon which I devised a hypothesis proposing that manganese could substitute itself at the vacant copper site on the prion protein; the substitution occurring in susceptible mammals who were entirely self sufficient upon high-manganese, low-copper food chains.

I considered that this manganese substitution could produce the all important deformation of the prion protein that is considered so crucial to the development of TSE. So David Brown ran the necessary cell culture experiments in which he introduced manganese into cells which manufacture prion protein. Remarkably, this experiment produced the key prion protein deformation which the earlier tests using OPs had failed to create.

Follow-up trials by Case Western University in Cleveland and a French team of scientists provided further confirmation. Both groups ran postmortem analyses of brain tissue taken from those who had died of conventional CJD. These tests revealed the same pattern of high-manganese, low-copper as identified in TSE food chains, a tenfold increase of manganese levels and 50 percent reduction in copper in relation to control brains drawn from those who had died of natural causes

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EVERY STORM CLOUD HAS A SILVER LINING

A few other TSE cluster hotspots had demonstrated the same low copper connection, but with high levels of silver, another transition metal, instead of manganese. Much like manganese, silver will also readily substitute at copper ligands on prion proteins. These environments were centered around ski resorts, reservoirs, airport flight paths and coastal districts where extensive aerial spraying of silver iodide "cloud seeding" chemicals had been used for inducing rainfall, snowfall and cloud or fog dispersion.

MANGANESE IN HUMAN NUTRITION

Manganese in small amounts plays an important role in human nutrition. It forms an essential cofactor of numerous enzymes and is necessary for the utilization and balanced metabolism of many other nutrients. Manganese is a catalyst in the synthesis of fatty acids and cholesterol and is essential for the production of sex and thyroid hormones. Because manganese plays a role in the mitochondial "power stations" of the nerve neurones, deficiency can result in symptoms that are similar to those of overload—lack of coordination, irritability, psychological difficulties and even paralysis, convulsions, blindness and hearing loss.

Whole grains, egg yolks, nuts, seeds and green vegetables provide manganese if it is present in the soil. Manganese is poorly absorbed from food but readily absorbed into the brain via the inhalatory-nasal-olfactory route. Thus toxicity is much more likely from environmental atmospheric sources than from food, although diets high in manganese-containing foods, such as soy and tea, can exacerbate the condition of manganese overload, especially when fed in large amounts to infants or growing animals.

With an overabundance of manganese prions and loss of copper prions, the oxidative impact of UV energy received at the retina can no longer be quenched.

FURTHER DAYLIGHT ON TSEs

Another observation: each time my trek led me to a new TSE hotspot, I found myself face-to-face with the same type of high altitude, snow covered, pine tree terrain. Putting aside the common high-manganese, low-copper connection, this common geographical association with TSE-cluster regions continued to baffle me. Whenever I arrived at a fresh TSE location, I was always reminded of that first glimpse of the chronic wasting countryside of deer and elk in Colorado—the snow-peaked Rocky Mountains sawtoothing the July skyline beyond the parched Denver Plain.

It was after arriving in a village in Calabria, on the southern tip of Italy, that the relevance of this geographical connection to TSE finally gelled. There had been 20 cases of CJD in this village since 1995. I noted that the village had been recently constructed out of hideous bright white concrete sections—unusual for this area—and all were couched within a sun-parched, glaring basin of bare white sandstone terrain, producing all the prerequisites required for a most intensive ultra violet (UV) hotspot location. The pain of the UV in my eyes immediately connected me back to the high-UV / high ozone nature of high-altitude, snow-covered, coniferous terrain—the common geographical thread interlinking the Icelandic, Colorado and Slovak cluster ecosystems in my study—areas also impacted by the oxidizing effects of the ozone gas generated by the interaction of UV light with the terpine haze exuded from pine trees.

The UV prerequisite also explained other missing links in the science of traditional TSEs, such as the way in which initial pathological damage of TSE manifests itself within the retina, eyelid or skin of the affected mammal—external areas having to buttress front line exposure to sunlight. Furthermore, the normal copper-bound form of prion protein is found along the circadian pathways which conduct the electromagnetic energy generated by ultraviolet light around the brain; that is, in the retina, pineal gland, visual cortex, hypothalamus, pituitary and brain stem.

Prion protein is expressed in other tissues of the body which are also interconnected to the network that conducts electromagnetic energy, for instance in the spleen, lymphatic system, glial cells and nerve-growth-factor-mediated stem cells that proliferate during the growth and repair of neurons.

In this respect, the suggestion of an electro-conducting function of the copper prion protein may turn out to give further scientific substance to the existence of the electromagnetic meridians recognized by Chinese medicine, where the healthy copper prion performs a regulatory role in maintaining the electro-homeostasis along these meridians.

The hypothesis was falling into place: copper prions as the conductors and manganese prions as the blockers of electromagnetic energy flow.

The fact that copper is used in wires that carry electric currents, whereas manganese is used in batteries and light bulb filaments that store electrical energy, helps explain the underlying cause of prion diseases: healthy copper prions conduct the vital electro-energy of sunlight along the circadian pathways that innervate deep into the brain—in order to maintain the balanced cycles of sleep, sex and behavior whilst aberrant manganese-contaminated prions blockade and store up that incoming UV energy to an explosive flash point—to a level that detonates off neuropathogenic cluster bombs of free radical chain reactions along the circadian pathways.

With an overabundance of manganese prions and loss of copper prions, the oxidative impact of UV energy received at the retina can no longer be quenched. Consequently, the energy flow of UV piles up, finding itself misappropriated into converting the accumulated store of innocuous manganese 2+ (antioxidant) into its lethal manganese 3+ or 4+ form (pro-oxidant). So any accumulations of abnormal manganese prion protein in the retina finds itself transformed from a safe to a lethal form.

In this respect eco-oxidants such as UV serve to unleash a kind of "Jekyll and Hyde" effect in the manganese-contaminated, copper-depleted mammal, which, in turn, kicks off a whole chain reaction of free radical assault on the central nervous system—ultimately resulting in a neurodegenerative meltdown that leads to spongiform disease.

THE COCKTAIL OF OXIDANTS AND NEW VARIANT TSEs

This theory explains the genesis of the traditional strains of TSE. But what about the causes of the much more aggressive modern day strains of TSE (BSE and vCJD) surfacing in younger mammals? Perhaps these "rapid attack'" new strain TSEs could result from our increased exposure to the more potent oxidizing effects of a cocktail of man-made environmental agents which can penetrate the central nervous system—contaminants such as the systemic organophosphates (head lice shampoos, warblefly pesticides), radar, ozone, increased UV (due to stratospheric ozone depletion), microwave mobile phones, Concorde's supersonic waves, and so forth, thereby serving as the lethal oxidative trigger that produces a more virulent, accelerated version of TSE with full-blown symptoms erupting in much younger mammals than normal.

TSEs could therefore be viewed as diseases that result from a breakdown of oxidative homeostasis within the organism, where TSE-susceptible mammals living in environments that are simultaneously challenged by high intensities of manganese and oxidizing agents, and by low levels of antioxidant metals (copper, selenium and zinc) which all combine to create circumstances where the central nerves are severely hyper-oxidized, thereby kicking off free radical chain reactions that are free to proliferate in the absence of antioxidant defence.

The pattern of emergence of both traditional and new variant CJD clusters in rural and coastal areas, as opposed to urban areas, substantiates this oxidative origin idea well. Furthermore, the 80 percent predominance of CJD cases erupting in rural and coastal areas helps to dispel the myth that vCJD arises from ingestion of BSE-affected beef products, as meat products are consumed equally by urban and rural populations.

Rural and coastal areas have become increasingly exposed to a toxic cocktail of oxidizing agents, such as UV light, ozone and systemic crop sprays; whereas town environments have ironically been spared. This is largely due to the shield of smog that envelopes the majority of urban airspaces and serves to scatter and absorb the incoming UV rays; thereby preventing the UV - exhaust gas interaction that yields the deadly consequences of ozone gas formation. It is perhaps no surprise that the hyperoxidative environs of Staten Island and Long Island, which plays host to an oxidative cocktail of Concorde takeoffs, radar, microwaves, coastal UV and ozone, demonstrates the most intensive cluster of CJD in the US.

MANGANESE BREAKETH MAN

The high-manganese connection to the epidemic of the new variant TSE correlates as convincingly as the eco-oxidant connection. Over the last two decades, increased amounts of the high concentration "manganese oxide" additive have been introduced into the bovine, human, pet and zoo animal food chains in Europe as mineral licks, tablets, fertilizer and fungicide sprays, paints, and petrol additives. Another "trendy" vector for manganese exposure is the increased consumption of soy, which bioaccumulates excessive levels of this metal from the soil, whilst containing low levels of copper.

Most disturbingly, manganese is added to artificial milk substitute powders for calf and human infant consumption at about 1000 times the levels found in normal cow and human breast milk. Excess dietary manganese poses a great risk to the immature mammal, since the homeostatic regulatory mechanisms of the blood brain barrier are underdeveloped at this early stage, thereby permitting an excessive uptake of manganese and other metals into the brain. The dubious practise of adding soy as a protein booster to these powders only serves to exacerbate the manganese toxicity problem further!

MEAT AND BONE MEAL (MBM) FEEDING VERSUS SOY

Protein sources have always been in keen demand for feed concentrate ingredients in confinement dairies and feed lot operations in the developed world, where rations demand a 14-18 percent protein concentration. Waste animal protein derived from the rendered down remains of butchered livestock (alongside various plant protein sources) has been used in animal feeding stuffs since the 1920s—surprisingly, with no known ill effects! The BSE outbreak was blamed on changes in MBM manufacturing methods (such as use of lower temperatures and cessation of solvent extraction) and resulted in the substitution of large amounts of processed soy meal, as well as some fish proteins, for cattle feed. In fact, the BSE epidemic has been a boon for soybean growers and manufacturers. Soy has been used for many years as a principle ration for chickens and salmon (both meats allowed in the politically correct modern lowfat diet) but was not normally given in large amounts to ruminant animals because of the damage it inflicted on their livers. Now that MBM is banned in the US and Europe, the multinational-controlled GM soy industry has a large pool of new customers, not only among confinement dairies and large feedlot operations, but also among thousands of new vegetarians, anxious to avoid "mad cow" disease.

Some would question how the toxic manganese-oxidant theory of TSE origins can account for the well recognized "iatrogenic" forms of TSE, where growth hormone treatment of humans (which utilizes pituitary tissue as the inoculant) can lead to a form of CJD. The answer lies in the fact that tissues, such as pituitary and retina that are considered to transmit TSE in the lab most efficiently, are the exact same tissues in which manganese concentrates most intensively. Could the high manganese levels contained within these tissues act as the so-called infectious agent, particularly once the metal has been oxidized into its lethal pro-oxidant 3+ form?

FUTURE PATHWAYS

Despite the apparent reluctance of establishment bodies to address the works of David Brown and myself, we have both been independently driven to take this theory to its final conclusive stages.

But funding has not been forthcoming, despite recommendations by the UK's BSE Inquiry report, as well as MAFF's subsequent invitation asking me to resubmit proposals for research along these lines. Such a negative dismissal has thwarted the whole healthy evolution of this important new perspective on TSEs. Furthermore, establishment recalcitrance has blocked the development of a possible cure for new variant CJD, a study that David Brown tried to launch last year.

In light of recent French and other European threats to sue the UK for allegedly giving them BSE and vCJD, it is puzzling to witness the continuation of the dismissive mindset that UK authorities display towards any evidence that backs environmental involvement in TSEs; unbelievable, in fact, after studying the recent work of Professor Bounias, from Avignon. His study highlights the exact same spatial-temporal correlation between warble fly insecticide use and BSE emergence in France, as observed in the UK.

TO THE ENDS OF THE EARTH

Meanwhile, I have continued to expand my field investigations by designing a holistic environmental surveillance program involving metal and oxidant analyses of relevant water, soil, vegetation, atmosphere, blood and tissues that will be exercised in the variant CJD and BSE clusters that have recently erupted across the UK and Europe, and now in Japan. I have also been invited to study a cluster of mysterious progressive, fatal neurodegenerative diseases, known as Bird's disease, that has erupted amongst Aboriginal and Caucasian people living on Groote Eylandt, a remote island ecosystem off the Northern Australian Coast. The problem first developed after a mining corporation started the open cast mining of manganese on the island in the 1970s. A fine black manganese dust has reportedly coated the entire island.

True to form, the local authorities have conveniently scapegoated the emergence of this syndrome—which manifests as a motor neurone disease or a mystery dementia—onto a combination of Aboriginal genetics and a rare virus that was introduced by a Portuguese miner who came to work on the island three decades ago.

With permission from the local Aboriginal society, I hope to acquire brain sections from those who have died of the TSE-like "dementia" strain of this disease and see whether TSE-prion "tombstone" features can be detected.

I have also managed to persuade a local GP in Darwin to treat some of the early stage victims of Bird's disease with the manganese chelating drug EDTA. Until now, victims of this grotesque disease have been kept in total darkneww regarding the existence of a possible cure for a disease that has always been considered fatal.

KURU

Similar to CJD, kuru is a progressive neurologic disorder that occurs primarily in the Fore natives who inhabit a tiny pocket of the New Guinea highlands. Symptoms are much like vCJD and include an exaggerated startle response and emotional instability, with pathologic bursts of laughter. Advanced states are characterized by dementia. In the terminal state, the patient is generally totally placid, mute and unresponsive.

Until the early 1960s, the disease was prevalent, especially in women and children, but in recent years the incidence has declined. This was said to be due to the abandonment of ritual cannibalistic practices in which natives ate the flesh of the dead, a theory that fit in very well with the dogma that the bovine version was caused by consumption of infected meat and bone meal. But the entire native population across New Guinea were traditionally involved in cannibalism, so why Kuru in just one tiny region? A more likely explanation is the massive eruption of a local volcano in 1911 which showered the foodchain of the Fore region in a black manganese oxide ash—the decline in kuru paralleling the importation of more foodstuffs from the outside world. Both the early stage unmotivated laughter with psychosis, and the placid, unresponsive characteristics of advanced kuru are similar to the symptom profile of manganese poisoning.