(*Rida is the Icelandic for transmissible spongiform disease)

EDUCATING RIDA*

An underground, ecodetective journey into the origins of spongiform disease.

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The White Sands Missile Range is an extensive spread of US military controlled cacti country that spans the southernmost extremes of the San Andres mountain ridge. There is an eerie atmosphere to the place.

A Department of Natural Resources truck kicks up the dust across the droughted canyon, its engines reverberating in an agitated mode. It stops at the main entrance gates along the 12ft high perimeter fence. One of the wildlife officers gets out and walks to security, seemingly oblivious to the distant thump of a missile exploding across the range. He is clearly preoccupied with the more important task of slaughtering animals who have succumbed to this so called "hyperinfectious" disease. The truck is soon on its way, loosing itself within the thousands of acres of parched up military compound.

They've come to investigate yet another new eruption of chronic wasting disease (CWD) in the USA– the deer equivalent of 'mad cow disease'. This outbreak is particularly significant, in that it represents the first cases of a transmissible spongiform encephalopathy (TSE) disease recorded in a deer herd within the state of New Mexico. Furthermore, the affected herd has been confined behind a top security perimeter fence for several decades.

This latest epidemiological aberration delivers a serious challenge to the viability of the conventional consensus on the origins of CWD. It has rumbled the cornerstones of institutionalized 'expertise', bringing into question those veterinarians who have plumped for the assumption that some unconventional "hyperinfectious" agent is spreading via body to body contact through the deer populations.

So how did the 'infectious agent' jump the 500 mile gap between the long standing CWD hotspot zone in Colorado and the CWD-free deer residing within the White Sands Missile Range ? The 'experts' were baffled. But, true to form, this latest challenge to the official theory was conveniently obfuscated into oblivion; outcast as some illusory mirage that just happened one day in the New Mexico desert.

But the answer is only evident to those who care to scratch a bit deeper than the dust. For they cannot help but notice some overt environmental features that exclusively predominate this unique location. Factors which are invariably shared by every single TSE cluster location around the world;

Before the military came, White Sands was an industrious center for the mining of the manganese oxide and wulfenite ores (NB; wulfenite contains the copper chelating molybdenum metal). The museum quality black crystals lay scattered across the top of the terrain, twinkling out a kind of sombre resonance under the desert sun. They emanate the haunted history of the place.

And since the military have occupied the range, the US authorities have been actively engaged in monitoring the unique intensity of infrasonic shock bursts that are radiated by the explosions of their own missiles. The poor deer herd has played guinea pig to an unwitting experiment that has cracked the causal riddle of spongiform disease.

The BSE debacle.

Since 1986, the infamous novel neurodegenerative syndrome , BSE and vCJD , has insidiously blighted the heartbeat of British rural life. The disease has annihilated thousands of cattle and a growing number of young people, as well as creating a fierce battleground between nations, vested interests, political parties, farmers, victim support groups and consumers. More recently, the shock waves of the BSE debacle have ricocheted around the entire world.

But despite the severity of the mad cow 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 disease.

But this story shines a ray of light over the whole debacle. It charters my own eco-detective escapades and original field investigations which ran in tandem with the laboratory quest of Cambridge Uni biochemist, Dr David Brown. These combined works have gone some way towards unearthing the truth underpinning the original cause of these grotesque diseases.

Hard scientific evidence has been amassed which indicates that vCJD and BSE could both result from separate exposure of bovines and humans to the same package of toxic environmental factors – ferrimagnetic metals and low frequency sonic shock - and not from the ingestion of the one species by the other. If such a polemic hypothesis continues to accumulate momentum, a radical upheaval of the status quo mindset can be expected.

But despite the conclusions of several field and laboratory studies providing strong support for the environmental hypothesis, the resulting publications have been dismissed by

the UK government. Furthermore, contrary to the positive recommendations made in the 1999 BSE Inquiry and EU Commission BSE reports in respect of funding research into this theory, the irrational rejection of grant proposals by the UK government continues to the present day - including one submission aimed at developing a feasible cure for vCJD !

The Lone Voyager

My work first came to the fore after successfully quashing the UK government's compulsory warble fly eradication regime in the high courts in 1984. This exempted my farming business from treating our dairy herd with a systemic organo dithiophosphorus (OP) insecticide - a toxic chemical derived from the OP military nerve agents, which, amongst a myriad of toxicological effects, can chelate copper and open up the blood brain barrier ; thereby disturbing the overall crucial balance of metals in the brain. I was therefore not surprised to witness BSE rearing its ugly head in the UK cattle herd in 1985; which, in my opinion, was a direct legacy of the UK government's compulsory warble fly campaign – a 1982 measure that enforced the exclusive twice annual high concentration application of systemic acting OP insecticides.

As considerably smaller outbreaks of BSE began to erupt across other European countries, and later Japan, my investigations revealed the voluntary usage of these same types of systemic insecticide in those countries –albeit at half the dose rates as applied in the UK. These European outbreaks seemed to follow an EU campaign, known as COST 811, that was aimed at purging the remaining bastions of warble infestation on the European mainland – countries where outbreaks had continued because their respective authorities had adopted a more laid back, voluntary approach towards the control of warbles.

In warble-free Japan, the BSE cases emerged in the specific

herds which had imported breeding cattle from warble infested North America; and so the Japanese had taken preventative measures by blanket treating those herds with the same types systemic OP that had been used in Europe. It should be pointed out that the USA had wisely adopted a less toxic approach for dealing with their warbles. They employed lower doses of 'non systemic' acting insecticides – eg insecticides which were not designed to penetrate through the skin - whilst only treating the individual cattle that are warble infested .

I was a working dairy farmer with first 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 cows that had been born and raised on fully converted organic farms, despite those cattle having been permitted access to the feed that contained the incriminated meat and bone meal (MBM) ingredient - as part of their 20% conventional feedingstuff allowance decreed in the organic standards at that time.

From then on, I became deeply sceptical of the conventional consensus on the origins of BSE and its human equivalent vCJD. There were just too many radical flaws blighting the hypothesis that bovine ingestion of micro doses of scrapie contaminated MBM lead to BSE. Equally flawed was the follow up theory that human ingestion of BSE contaminated beef caused vCJD.

The 'hyperinfectious hysterics' had based their hypothesis on the fact that TSEs could be transmitted via injections of TSE diseased brain tissues into misfortunate laboratory animals. Yet, various other neurodegenerative diseases , such as familial alzheimer's disease, have been transmitted in this way. So why is nobody freaking out about alzheimer's disease ?

The Flaws in the Conventional Hypothesis ;

1. Thousands of tons of the BSE incriminated meat and bone meal (MBM) feed were exported as cattle feed during the 1970s/1980s/1990s to countries that have remained BSE-free to date. - eg, South Africa, Sweden, Eastern Europe, Middle East, India, Third World, etc.

2. Relaxation in the temperature / manufacturing techniques of the MBM rendering process in the UK were blamed for permitting the survival of the scrapie agent in the sheep brain material; thereby enabling the "agent" to jump across into cattle, producing BSE. But none of these alterations were exclusive to the UK plants. For instance, other scrapie endemic countries such as USA and Scandinavea had adopted the same continuous flow system of rendering five years before the UK, yet these countries have remained BSE-free. Furthermore, the pathogenic, 'infectious' capacity of the scrapie agent remains active after heating to temperatures in excess of 500 degrees – way above the 150 degree temperatures employed in the supposedly 'safe' rendering processes operating in pre BSE days.

3. Several abhorrent live animal trials in the USA failed to induce BSE in cattle after feeding/injecting them with massive doses of scrapie contaminated brain tissue.

4. Forty thousand plus cows that were born after the UK's 1988 ban on MBM incorporation into cattle feed have still developed BSE.

5. Several countries such as Ireland, Portugal and France have witnessed a greater number of BSE cases in cows born after their respective bans on MBM, than in cows born before their

bans.

6. There have been no cases of BSE in other TSE-susceptible ruminants in the UK, such as goats and sheep, despite the customary inclusion of the same MBM protein source in their feeds.

7. Four of the original five kudu antelope that developed BSE at the London zoo had not had any possible access to MBM containing feeds.

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

9. The infamous mechanically retrieved meat products / baby foods blamed for causing vCJD in the UK were exported all over the world to countries where vCJD has not erupted to date. Likewise, the tradition of 'skull splitting' in small rural butchers, that has been offered as an explanation for the growing number of vCJD clusters in rural areas, had been practised by the smaller butchers all over the UK.

10. BSE fails to fulfill ' Koch's postulates'- the yardstick for gauging whether a given disease stems from infectious origins. For instance, more than 15% of cattle slaughtered for displaying the classic symptoms of BSE did not demonstrate the presence of the 'causal' prions at post mortem.

The reductionist mindset takes ahold.

Despite the myriad of epidemiological flaws and millions of pounds 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 became impregnated as ‘gospel’ into mainstream public/professional mentality.

But It is easy to see how the momentum of such a reductionist mindset took ahold ; The media loved the theory because they could drum up a viral holocaust-horror scoop. The farming industry could get their beef sales back on the road by deluding consumers that the causal agent had been eliminated. The vegetarian lobby found themselves landed 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 offload the blame onto the vagaries of some naturally occurring ‘nasty’ for which no vested interest or official directive could ever be held accountable.

In the early days, the world of TSE research had been confined to the rather cranky ranks of backstreet Institutions. Their researchers seemed more preoccupied with advancing acidic debate over the nature of the “infectious” agent than getting on with worthwhile research projects. It was these scientists who first fossilised the reductionist notion that TSEs stemmed from infectious origins.

But as soon as the positive evidence for the first case of BSE was back from the lab, a fast expanding clique of “expert” microbiologists swooped in, hijacking all the research grants and rapidly laying claim to full ownership and academic rights over this new strain of TSE. They coined the classy name ‘Prion disease’, and ran a host of sharp-suited symposiums set in expensive five star, Floridian hotels thousands of miles adrift from the English pasturelands – the hotbed of the real problem.

From then on, any investigations into the broader scientific perspectives surrounding TSEs were frozen out of the agendas of the funding bodies. Multidisciplinary research studies were forced to give way to research projects that conformed to the convergent assumption that the 'prion' would encapsulate all of the answers to this problem. The journals were soon bursting apart with a monotonous dirge of articles that bleated out yet another variation on the stereotype theme of the prion protein - prion protein genotypes, the biochemistry of the prion protein, along with a countless number of prion transmission live animal studies that had been duplicated by virtually every institution involved with TSE research – for no useful scientific purpose.

Once it became clear that the various feed bans had failed to halt BSE in the UK (eg; the 40,000 BSE cases in cattle born after the 1988 feed ban), the incestuous clique of 'expert' advisors were forced to come up with an ever increasing array of implausible reasons for explaining the continuation of BSE. And following on from their advice, an equally inept package of control measures were implemented whenever and wherever TSE reared its ugly head around the world.

Their final farcical solution entailed a wholesale annihilation programme of wild and domestic animal populations across specially designated TSE eradication zones. Despite the well publicised history of total failure of these control measures, this brave new wave of totalitarian overkill went ahead - gobbling up millions of healthy mammalian lives and millions of dollars of public funds.

Prion Origins; the quest for primary cause.

Whilst it is well established that the key pathological hallmark of the spongiform diseased brain is represented by a malformed version of the prion protein, known as a prion, there is no actual evidence in support of the assumption that the protein portion of

the prion represents the TSE infectious agent. Nor is there any evidence that TSEs are infectious diseases that can be spread via body to body contact. And furthermore, nobody has offered a credible explanation as to how these abnormally shaped 'prions' are initially created in the mammalian brain, nor have they explained how these prions are capable of inducing a cascade of self-replication in the TSE diseased brain.

I became interested in the possibility that the use of systemic organo phosphate (OP) warble fly insecticides may have triggered off this protein malformation in some way; thereby serving as one of the primary causes of the modern BSE / vCJD strains of this disease. For these oil based chemicals were designed to penetrate through the skin and metamorphose the internal environment of the cow into a poisonous medium so as to exterminate internal parasites. Farmers were forced to pour the chemical along the head / backline of the cow just millimeters from where the prion protein is manufactured in the cell lines of the spinal cord.

It was well recognised that OP insecticides exert their toxic effects in mammals by deforming the molecular shape of various nerve proteins like acetylcholinesterase; whereupon these malformed proteins cease to perform their proper function in the brain. But nobody had ever considered that a similar style molecular interaction may occur between OPs and the prion protein. Since the prion protein has been shown to bond up with copper in the healthy brain, I felt that the ability of these dithio insecticides to lock up copper in the treated animal may play some role in deforming the prion protein.

After many abortive attempts to coerce the Establishment into running the correct laboratory test, I eventually raised funds from wellwishers and personal loans to finance Dr Stephen Whatley of the Institute of Psychiatry in London to challenge brain cell cultures with the OP 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. Unfortunately, these experiments did not produce the key deformation of the prion protein that is seen in TSEs. I returned to square one, assuming that OPs in combination with a further factor X, could fulfill the final missing link in the causal jigsaw. Or perhaps OPs weren't involved at all !!

The Cluster Buster

Caught in the midst of a minefield multinational interests, medical spin docters and political propagandists, I grew exhausted by the vortex of professionals that had successfully hijacked and cul de sac'ed all UK scientific research into TSEs. Furthermore, I'd found myself frozen out into the cold, operating much like an underground scientist, tramping a covert journey around the rustic outbacks of the UK's dairylands to nail down the true cause of BSE.

So I expanded my horizons and embarked upon a refreshing ecodetective trek to analyse the unique environments around the world where traditional TSEs had erupted as high incidence clusters for many years. By scanning the overall characteristics of each cluster location, I attempted to pinpoint the common causal factors – the aetiological needles in the causal haystack.

Set against a backdrop of flamboyant and sometimes threatening scientific scenery, I embarked on a lone journey to the ends of the earth, sampling exotic corners of Colorado, Iceland, Slovakia, Calabria, Sardinia, Japan, etc; areas where an assortment of animals and humans had demonstrated a high incidence rate of TSE. My analytical results displayed

abnormally high levels of the metal manganese, and rockbottom levels of copper, selenium and zinc in all of these food chains in common. Levels of manganese returned to normal in adjoining disease-free areas.

I was also fascinated to discover these TSE affected populations lived in areas that were enduring 'front line' exposure to intensive shock bursts of low frequency infrasound – military and quarry explosions, volcanic and earthquake shocks, low flying supersonic jets, concorde overflights, etc. It took many further miles of investigation before the full relevance of this additional environmental facet could be integrated into the causal equation. But the role of the high manganese / low copper finding seemed very clear from the outset.

A specific environmental source of manganese could be pinpointed in every TSE cluster zone that I had investigated to date; where each TSE affected ecosystem could be directly connected to the atmospheric fall out of some naturally occurring or industrial source of combusted manganese oxide; eg stemming from volcanic, acid rain, steel/ glass/ ceramic /dye/ munitions factories, lead-free petrol refineries, the take off airspace beyond airports, etc. In this respect, it should be born in mind that atmospheric manganese, much like silver and aluminium, can be absorbed directly into the brain via the nasal-olfactory inhalatory route of intake (eg; the infamous cocaine snorter's route). Perhaps this highly efficient mode of uptake enables sufficient concentrations of manganese to accumulate in the brain and initiate TSE ?

Furthermore, many of the mammalian populations involved in the outbreaks of both traditional and new strain TSE could be linked to the consumption of high concentration manganese supplements for bone growth, etc. For instance, the clusters of chronic wasting disease in deer could all be linked to the areas where a 'spiced up' manganese mineral lick had been put down

by the deer hunters for addicting deer to their shooting territories. The manganese is added for promoting antler growth. In fact *all* species connected with TSEs – deer, humans, cows, goats, mink, sheep, zoo animals, cats, etc – are fed artificial manganese supplements in their feeds.

Disturbingly, manganese is also added to artificial milk substitute powders for calves (and human infants) at levels up to 1000 times those found in normal cow's milk respectively. This practice has been widespread in all countries affected with BSE to date.

Excess intakes of dietary manganese pose a great risk when fed to the immature mammal since the regulatory mechanisms of the blood brain barrier are underdeveloped at this stage; thereby permitting an excessive uptake of manganese and other metals into the brain.

The addition of manganese to artificial milk powders explains why European dairy cattle (who were invariably reared on this powder) suffered such high incidence rates of BSE in relation to the negligible rates of BSE encountered in beef suckler or organically reared cattle (who were invariably reared on natural cow's milk). The more extensively reared BSE-free cattle herds of Australasia have also never fed these mineral boosted milk powders.

Every storm cloud has a silver lining.

A few other TSE cluster hotspots had demonstrated the same low copper connection, but had measured abnormally high levels of other potentially toxic transition metals, such as silver, platinum, lithium, as well as manganese. These metals can also readily substitute at copper bonds on prion proteins .

Some of these TSE cluster zones were located in silver mining

areas, where local ecosystems were naturally high in silver, whilst other clusters were centered around ski resorts, reservoirs, airport flight paths, coastal districts, etc, where extensive aerial spraying of weather modifying silver iodide 'cloud seeding' chemicals had been used for inducing rainfall/snowfall and cloud/fog dispersion.

Metals like silver and platinum are also used as key ingredients in dental amalgam fillings, surgical depth electrodes / instruments, etc; perhaps explaining why dental treatment or surgical operations / electrode implantation are considered to be high risk prerequisites for triggering off CJD.

Manganese Breaketh Man.

The recent surge in the global incidence rates of TSEs, and other neurodegenerative diseases, seem to have run in tandem with the increased incorporation of high concentration manganese oxide additives into the bovine, human, pet and zoo animal food chains. This has resulted from the introduction of a multitude of applications involving free access mineral licks, supplement tablets, fertiliser and fungicide sprays, paints, petrol additives, etc. Or via increased consumption of trendy food products, such as soya, where the beans naturally bioaccumulate high levels of metals like manganese and aluminum from the soil.

Given the fact that long term manganese exposure is well known to induce a wide array of progressive neuropsychiatric degenerative disorders in factory welders and mine workers - dubbed as the 'manganese madness' syndrome - the idea that manganese could perform a front line role in the pathogenesis of TSEs is not out the way.

In fact, manganese toxicity can manifest itself in many forms of neurodegeneration. When I visited areas in the South Pacific, like the isle of Guam, where a raft of neurodegenerative

conditions involving Alzheimers, Parkinson's and Motor Neurone disease had run at a fifty fold higher incidence rate than the global average, I noticed that manganese mining used to be intensively carried out in all of these areas. After the mining ceased, the incidence rates of these diseases diminished accordingly.

Next, I visited Groote Eylandt, a once upon a time enchanted tropical island off the NE Australian coastline whose history has witnessed a bizarre degree of 'heaven and hells'. Not only do the island's soils play host to the mother of all manganese concentrations, but its flamboyant rainforest ecosystems have supported ideal hunter-gatherer grounds for some of the most pure bred, nomadic Aboriginal clans alive in Australia today. But, as I was soon to learn, the tropical charms of a Groote Eylandt of 'pick-your-own' coconuts and turquoise seas can be deceptive to the uninformed outsider.

I found that 3% of an indigenous Aboriginal clan residing around one of the largest open cast manganese mines in the world had recently been struck down by a bizarre Friedrich's ataxia -like syndrome - what appeared to be caused by a high manganese / low magnesium induced mutation in early life. Previously fit and healthy Aboriginees had found themselves rapidly transfigured into wasted, neurodegenerative wrecks. Their movements were more akin to a debilitated stick insect trying to cross ice, than a broad faced Aboriginal bushperson out stalking a wallaby.

Furthermore, the levels of unrestrained aggression / murder in this community had reached crisis proportions, where the rates of imprisonment are 20 fold higher than in any other Aboriginal community. Grotesque 'Hieronymous Bosch-style' brawls erupt on a weekly basis.

Interestingly, the link between manganese and aggression has been well established in many other parts of the world as well. For instance, manganese was recorded at elevated levels in the bodies of those who have been executed on death row for violent crimes. The scientist who carried out these studies, Professor Louis Gottschalk, reckons that 'manganese levels serve as a marker for violence'.

But, true to form, the official cause of this "drunken walking" syndrome has been conveniently scapegoated onto a mutation caused by faulty Aboriginal 'seed'. But those who had propounded this genetic-only causal hypothesis (funded by the mining corporation !) had excluded many important epidemiological perspectives; turning a blind eye to the fact that a handful of the white mineworkers on Groote were also beginning to show the early symptoms of Groote syndrome. One had already died of an 'undefined' neurodegenerative wasting condition.

To challenge the genetic dogma on the origins of this disease, I traveled several thousand miles to another isolated area where a cluster of this same ataxic condition exists amongst indigenous folk living on the islands of Flores and Sao Miguel in the Azores Archipelago. After drawing a range of environmental samples, I unearthed the same abnormal mineral ratio which I had found back on Groote Eylandt. In fact, the levels of manganese were so high in the black volcanic terrain of these islands, that the mining prospectors have considered it lucrative to mine the metal from the local seabeds.

Given the high intensity of various neurodegenerative diseases that have erupted around these manganese hotspot regions, I wondered whether the vastly increased exportation of manganese dioxide ores from these areas (into the steel, glass, dye, lead free fuel, paint, mineral feed supplement industries) across the developed world, had somehow seeded the 'madcow'

madness in the deer, sheep, cats, zoo animals, cows and young teenagers ?

Laboratory studies support the theory.

My observations of high manganese / low copper in TSE cluster areas lead to my connecting with the pioneering laboratory investigations of Dr David Brown at Cambridge Uni in the UK; a widely published biochemical expert who had pursued ground breaking studies which had elucidated interesting new facets of the elusive prion protein.

Dr Brown had demonstrated that the prion protein bonds to copper in the normal healthy brain. In this respect, his lab studies were complementary to my field studies, in that they provided the other half of the neccessary ground work upon which I formulated a holistic hypothesis on the origins of these diseases. I published a paper proposing that manganese could substitute itself at the vacant copper site on the prion protein; whereby this aberrant substitution by a foreign metal co partner could induce the all important deformation of the protein that is considered to be crucial to the development of TSE. In this respect, any TSE susceptible mammals who were self sufficient upon these high manganese / low copper TSE cluster foodchains would be at risk of developing TSE.

So David Brown ran the necessary cell culture experiments where he introduced manganese into copper depleted prion protein cell cultures. Amazingly, his experiments produced the key deformation of the prion protein which the earlier tests using OPs at the Institute of Psychiatry had failed to create. These experiments represented the first time that malformed prion protein had been created experimentally as a ‘de novo’ transformation.

Furthermore, follow up trials by the USA’s Prion Surveillance

Centre at Case Western Uni (Cleveland) looked at post mortem tissue samples taken from CJD brains. Their analyses revealed the same abnormal mineral ratio as identified in the TSE cluster environments – a tenfold higher level of manganese and 50% reduced levels of copper in relation to control brains. Interestingly, the manganese was bonded to the abnormal prions in these CJD tissues.

It seemed that the basic cornerstone of the environmental theory was beginning to establish itself, and an overall picture of the pathogenesis of this disease was panning out - that a high manganese / low copper imbalance somehow compromised the brain's ability to deal with acute shockbursts of sound and light – the other common characteristics of TSE ecosystems.

My investigations indicated that the traditional forms of TSEs - which tend to surface in elder mammals - represent a less intensive mode of exposure to naturally occurring environmental influences which bring about a high manganese / low copper ratio in the mammalian brain. Whereas, the more aggressive 'rapid attack' modern strains of TSE, BSE, vCJD , can be explained by the current trend of increased mammalian exposure to a modern cocktail of man made pollutants involving manganese compounds and copper chelating chemicals (the warble fly and headlice OP insecticides). These penetrate into the central nerves and give rise to the more virulent, accelerated version of TSE, where full blown symptoms of TSE erupt in much younger mammals than normal.

But unfortunately the abnormal shaped prions, which Brown's high manganese / low copper experiments had created, had failed to demonstrate the bizarre 'infectious' multireplicating property that has been associated with the fully fledged prion in the TSE diseased brain. Could the further infrasonic shock factor that I had identified in the TSE cluster regions fulfill this

final missing piece in the causal jigsaw ? Perhaps some 'shock-induced' quantum facet of the manganese atom may provide that final clue .

Quantum capacities; the final key to the causal jigsaw ?

Modern health authorities could learn a lesson from the alchemists of the Byzantine era who regarded manganese as the black magic metal; whereby the quantum capacity of manganese to absorb light and sound energy, can induce a lethal 'Jekyll and Hyde' style conversion of this metal from innocuous to toxic form.

Interestingly, the initial pathological damage of TSE is manifested within the retina, eyelid, skin, auditory and optic nerve endings of the diseased mammal - areas that perform a front line role in neutralising the deleterious effects of incoming sound and light from the external environment. Furthermore, the normal copper bound prion protein is exclusively manufactured in these same tissues; the retina, spleen, lymphatic, tonsils, gut membranes, growth/ repair cells, myocardium, pineal, visual cortex, pituitary, sympathetic neurones, etc – tissues whose metabolism is regulated by the circadian daylight / darkness rhythm.

Lab animals cruelly subjected to GM prion protein 'knock out' experiments, have demonstrated that they are no longer able to regulate their sleep, sex and immune cycles – a sick way of demonstrating the role of the prion protein in mediating the circadian rhythm.

Copper prions as the conductors and manganese prions as the blockers of electromagnetic energy flow.

The simple fact that copper is employed in electric cables for

conduction of electrical energy, whereas manganese is employed in batteries for storing up electrical energy, offers a feasible explanation for both the function/ dysfunction of the prion protein, as well as the cause of TSEs;

Perhaps the copper element of the normal healthy prion protein plays a role in the conduction of electromagnetic energy along the circadian / auditory pathways of magnetic superexchange ; where a linear chain of paramagnetic copper atoms (bonded to prion proteins) provides a 'metal to metal to metal…' motorway which distributes the energy of light and sound around the body for energizing the cycles of sleep, sex, behaviour, heart beat, cell growth/ repair and immune response.

Whereas the abnormal manganese prion serves to block these pathways of electromagnetic superexchange; by storing up that electromagnetic energy to a level where the critical threshold of 'flash point' is exceeded; thereby detonating off neuropathogenic cluster bombs of free radical chain reactions that progressively degenerate the circadian pathways of the brain – and cause TSE.

In this respect, the blockage of the electromagnetic energy flow by such a manganese replacement of copper can precisely account for the clinical and pathological profiles of the TSE disease process.

It could be said that the discovery of the function of the prion protein may turn out to give further scientific substance to the existence of the electromagnetic meridians recognised by Chinese medicine - where the healthy copper prion performs a regulatory role in maintaining the electro-homeostatis along the acupuncture meridians and nodes.

Whilst David Brown's manganese experiments had created the infamous deformed prion protein, they had failed to produce the

all important pathogenic, 'infectious' prion. Furthermore, manganese is a paramagnetic metal (eg; a metal that can be temporarily magnetised) just like copper, so to some extent you would expect the manganese to effectively substitute for the electromagnetic conduction function of copper without too much negative repercussion. Perhaps some further modification of the manganese atom initiates a pathogenic capacity that is capable of kicking off the full blown pathogenesis of TSE ?

An infrasonic induced metamagnetic transformation of the manganese atom ?

One of the interesting properties of manganese is that it can absorb the energy of sound – well illustrated by its commercial use (like chromium, etc) in some audio tape materials for storing up a memory bank of sound recordings in magnetic form. But manganese can only couple up with phonon energy when it occurs in its trivalent, octahedra crystalline form.

So if atmospheres or soils containing manganese 3+ atoms are exposed to high intensities of infrasonic shock energy, the low frequency vibrations are capable of metamorphosing the actual atomic structure of the manganese atom itself, creating a kind of 'Jekyll and Hyde' like transformation of manganese from its normal paramagnetic to rogue ferrimagnetic form; eg from a temporary to permanently magnetisable form.

Once an ecosystem has been infrasonically adulterated, any manganese 3+ minerals that have been exposed to the full force of infrasonic shock – from military explosions, supersonic overflights, volcanic and tectonic eruptions, etc - would leave a permanent legacy of rogue ferrimagnetic manganese atoms that are free to infiltrate the foodchain; thereby contaminating any mammalian population that is self sufficient upon the local ecosystem in the years to come. If these adulterated foodchains

are simultaneously short of copper, then the mammalian population is put at high risk of developing TSE.

TSE clusters correlate to the epicentres of infrasound.

The overriding presence of this package of environmental prerequisites has been identified in every TSE cluster zone investigated to date. Besides the White Sands Missile Plant, another clear cut illustration of the infrasonic phenomena in a TSE cluster, involved an intensive outbreak of sheep scrapie after sheep had been first pastured on a block of former military controlled common land at Ashoro on the Hokkaido peninsula in Japan. This copper deficient land was previously utilised by the Japanese military during world war two. According to local people, intensive 'ground rocking' explosions were a daily occurrence.

And then there is the long established CWD hotspot zone that runs along the copper deficient Front Range foothill region of Fort Collins in Colorado. This area used to play host to the supersonic test flights of the military jets from nearbye Warren airbase during the late 60s / early 70s. When the complaints of the Fort Collins residents reached screaming point, the military were forced to move the flights to the plains in the North East; and CWD outbreaks have followed likewise.

Furthermore, my investigations have identified that the new wave of CJD, BSE and scrapie clusters that have erupted across southern Italy, Sardinia and Sicily since the mid 1990s are invariably located in areas beneath the flight paths radiating out of the intensive number of NATO bases recently sited within this strategically important military position within the Mediterranean basin (See map 1).

As a rule, the traditional forms of TSE result from exposures to the naturally occurring infrasonic shock waves that radiate from

geo-tectonic plate rift lines (earthquakes, volcanoes, etc,)(See map 2) whereas the new strain TSEs result from the more intensive low frequency shock bursts radiating from the supersonic military or concorde aircraft (See Map 3). The emergence of the respective types of TSE clusters along these teconic rift and flight path lines substantiate this idea well.

Perhaps it is no surprise that 99 % of the total cases of vCJD and BSE to date are contained within Britain and France, both countries having exclusively developed the Concorde aircraft; thereby unwittingly exposing themselves to the most intensive source of artificial infrasound unleashed to date.

In this respect, all of the clusters of new variant CJD that have erupted in rural regions in the UK lie beneath the routine and charter flights paths of concorde and low flying military jets (Map 3). The afterburner turbofans employed by these supersonic aeroplanes radiate such a high intensity of low frequency infrasound - in the 10 to 5 hertz range- that a 100km wide carpet of acoustic shock waves are left in its wake – whether flying sub or supersonically. Racing pigeons who have flown into this shock carpet have failed to return home, having permanently lost their sense of magnetic orientation.

Some interesting epidemiological data on BSE clusters was first presented by a New Zealand epidemiologist, Dr Morris, to the BSE Inquiry. He showed that the most intensive clusters of BSE had always appeared on the extreme tips of the copper deficient west coast peninsulas in the UK. How could this pattern relate to MBM feeding ?

Intriguingly, the well used west coast test route for supersonic military and concorde aircraft ran precisely over these BSE hotspots locations (Map 4).

It is perhaps no surprise that the infrasonic environs of Staten island and Long island in the USA - both under the take off flight paths of JF Kennedy airport where Concorde and other aircraft land - has demonstrated the highest incidence cluster of traditional CJD in the USA (Map 5).

The pattern of emergence of both traditional and new variant TSE clusters in rural/coastal areas, as opposed to urban areas, substantiates the idea of an environmental origin hypothesis well. For rural / coastal ecosystems have become increasingly exposed to a toxic combination of manganese based fertilisers/ fungicides, copper chelating pesticide sprays, as well as the infrasound derived from the overflights of low flying turbojet aircraft - flights that have been largely prohibited over the urban areas of high population intensity.

Furthermore, this geographical pattern of TSE emergence helps to dispel the myth that CJD arises from ingestion of TSE affected animal products – since meat products are consumed equally by urban and rural populations alike.

Is the rogue ferrimagnetic manganese atom the 'infectious' TSE agent ?

So once the crucial supply of copper is curtailed in the brain - due to straight forward environmental copper deficiency or exposure to copper chelating OP insecticides, etc - the prion protein's metal bonds become vacant, rendering the protein vulnerable to bonding up with certain alternative metals , such as manganese, silver or lithium. But these foreign substitutes may not act in the overall best interests of the organism, particularly if the invasive metal is in 'ferrimagnetic' form.

For instance, once ferrimagnetic manganese substitutes at the vacant copper bonds on prion protein, the field inducing influence of its ferrimagnetically ordered atoms will

progressively corrupt the circadian mediated pathways of electromagnetic superexchange throughout the brain; whereby a status of permanent magnetic charge is spread from metal bond to metal bond, as a ‘domino-style’ contagious corruption that ‘jumps’ across the metal bonds, from prion to prion. This phenomena is well illustrated by the classic college physics experiment, where a magnet is placed alongside a steel nail and the force field of the magnet is shown to magnetise that nail.

So once an individual’s brain is contaminated by this freaky form of metamorphosed manganese, any subsequent exposure to external electromagnetic fields (eg, UV, sound waves, radar, cell phones, etc) will permanently charge up the ferrimagnetically ordered manganese prions. The metals rapidly become permanently saturated in magnetic charge, generating intensive magnetic fields, which, in turn, generate self perpetuating ‘cluster bombs’ of free radical mediated spongiform neurodegeneration. TSE ensues.

In this respect the TSE diseased brain can be likened to a solar powered battery on continuous charge; where the manganese loaded / copper depleted brain is no longer equipped to deal with the incoming surges of electromagnetic energy from the external environment. Instead of utilising this energy for the body’s own vital requirements, it becomes perverted into a potent force for neuronal suicide.

This theory explains why the so called ‘hyperinfectious’ property of the prion is a misnomer. It is the toxic ferrimagnetic metal component of the prion that serves as the so called ‘infectious’ pathogenic agent in TSEs. So whenever scientists inoculate misfortunate lab animals with TSE brain tissues (eg tissues contaminated with this rogue manganese atom) and effectively transmit TSE, they are actually transmitting ‘a magnetic field inducing capacity’ that is carried along with the

ferrimagnetically ordered manganese contaminant into the recipient animals, who, in turn, develop TSE.

Furthermore, the concept of the rogue ferrimagnetic manganese atom as the 'TSE agent' also explains why the so called 'infectious' pathogenic capacity of the prion can survive heating to temperatures in excess of 500 degrees – since ferrimagnetic metals will hold onto their magnetic charge until they are heated to temperatures beyond their respective 'curie point' temperature. (eg, 550 degrees for manganese 3+).

The theory addresses all.

Some would question how the toxic manganese theory of TSE origins can account for the well recognised 'iatrogenic' forms of TSE, where growth hormone treatment of humans - which utilises pituitary tissue as the pharmacological inoculant - can lead to a form of CJD.

But intriguingly, tissues such as pituitary and retina which transmit TSE in the lab most efficiently, are the same tissues in which manganese is recognised to concentrate most intensively in the body. So once an individual is contaminated with a rogue source of ferrimagnetic manganese, any subsequent use of their pituitary tissues in pharmaceuticals for 'growth hormone therapy' could spread the so called 'infectious' toxic agent, and initiate CJD.

Others would question how this theory can account for the outbreak of the kuru strain of TSE that exclusively erupted in an isolated tribe in the Fore region of the New Guinea Highlands. The conventional dogma blames this outbreak upon the Fore tribe's traditional practice of cannibalism. Whilst cannibalism may have played a role in the bioaccumulation of manganese - particularly if the pituitary tissues were ingested in these cannibalistic binges – the fact that virtually every tribe across

New Guinea had adhered to a cannibalistic lifestyle, whilst remaining free of Kuru, needs to be addressed by those who promote this theory. And furthermore, considering that cannibalism had been traditionally practiced for centuries across New Guinea, why did kuru fail to erupt until a few years after world war two ?

My investigations suggest that the cause of Kuru stems from the same template of eco-factors; the Fore tribe's self sufficient lifestyle on copper deficient soils, coupled to their scavenging of manganese-aluminum sheet metal from the fuselages of several Japanese bomber aircraft which had crashed in their area of the highlands during world war two. The Fore folk moulded the metal to make tools, cooking pans and bowels which consequently contaminated their foods. They also accidentally exploded some of the bombs that were still on board the crashed aircraft. These explosions, well remembered by the surviving Fore folk, infrasonically irradiated their local environment.

This story goes on. At the mouth of the Fuji river valley in Japan lies a manganese-aluminum alloy factory that had manufactured these metal aircraft panels since the late 1930s. During this period, the manganese enriched emissions from the factory chimneys have dispersed downwind, permeating the entire length of the valley. Intriguingly, a cluster of CJD has blighted the residents of the Fuji river basin for 50 years.

Animal Pharm.

Despite publication of the hard evidence in support of this theory in prestigious scientific journals (see references below) the various UK authorities and their incestuous clique of 'minder' advisors are blindly ignoring these findings.

Whatsmore, they are doing their utmost to publicly marginalise those of us who are trying to pursue this alternative research

line; and using public money to implement their tactics of suppression into the bargain.

For 18 years now, I have found my work and personal integrity subjected to a steady derisory trickle of ridicule and dirty tricks. During the 1980s my farm and family became the victims of a raft of ‘once in a lifetime’ type physical disasters ; arson, firearm intimidation, vandalisation of my research library / communications, and insidious infiltration by a bizarre array of bogus greens and phoney free lance journos, along with a seductive approach by a scantily clad pseudo student who was supposedly doing her dissertation on my theory. After becoming suspicious, my investigations revealed that she was not even registered at the college where she purported to be studying !

At the end of the day, it transpired that the true objective of these ‘agent provocateurs’ was to subtly set about discrediting my social and scientific esteem, whilst finding out the current position of my research investigations. Once my work gained support from the likes of the former Minister of Defence, Tom King, and HRH Prince of Wales, the physical aspects of this harassment abruptly ceased.

A recent demand to the UK Gov Departments for my personal data revealed much of what had been going on behind the scenes. Repeated requests by Environment Minister Micheal Meacher to personally meet with me had been deliberately stymied by his own officials. Other documents revealed how The British Agrochemical Association had been organising a ‘joint initiative’ with the Ministry of Agriculture’s own grant funding department to channel public funds into a live animal trial that had been deliberately designed to refute my theory.

Since the BSE Inquiry had rejected the official scrapie-BSE hypothesis and found in favour of some aspects of my own hypothesis, the UK Government responded by setting up a

further mini Inquiry to relook at the origins of BSE. The resulting publication known as the 'Gabriel Horn report' employed a judicious selection of misrepresentation and outright bogus disinformation in order to discredit the validity of my theory. For example, they had stated that the use of OP warblecides had ceased in the UK by 1982, and that warblecides had been routinely used on Jersey island. So, according to the Horne Report, if OPs were the cause, why were all of the cows that developed BSE born after 1982, and why were BSE rates so low on Jersey?

But ironically, the truthful picture of the compulsory 2x annual OP warblecide treatment programme was that it was *first* introduced in 1982 in the UK, whilst only *one cow* on Jersey was formally subjected to compulsory OP warblecide treatment.

When I attempted to sue the government for defamation / loss of income resulting from the bogus statements in this globally circulated publication, they pleaded 'qualified priviledge' of the expert committee, and then spun out the legal communications beyond the one year post publication mark; thereby exempting themselves from my claim.

And after broadcasting of the BBC Correspondent film 'Mad Cows and An Englishman' which chartered my investigations, the government tried to appease the mounting public interest by inviting me to resubmit an application to them for funding. After sitting on my application for a year and a half, they homed in on the most fastidious, nit picking comments in the peer review appraisal; trumping them up as a sound scientific basis for their rejection. Immediately after, the author of the most irrational, irrelevant critique found himself promoted to the government's expert 'TSE surveillence steering committee'; presumably as a reward !
The UK government's tactics have impaired the whole healthy evolution of this new scientific perspective on TSEs.

A multinational masterplan ?

The epidemiological and experimental evidence amassed to date points to the fact that TSEs are caused by a clear cut combination of genetic and toxic environmental factors. So why do the authorities continue to treat these diseases as if they solely stem from hyperinfectious origins?

The reasons for such an irrational, Pavlovian-like stance of the Establishment towards the environmental perspectives of TSEs probably hangs upon issues that are more to do with protecting academic egos, professional reputations and the vested interests of the TSE institutions / key advisors, than with promoting sound scientific arguement. Another reason must undoubtably stem from the fear of massive compensation claims should government mandated use of 'OP warblecides' or licensing of 'manganese additives' be held accountable at the end of the day.

But delving abit deeper; who are the key culprits that are currently capitalising on the fashionable scare stories which maintain that "BSE prions will exterminate us all" ? Who are spinning out the propaganda myths that beef, lamb, venison, game and organic food (grown from animal manure) are contaminated with prions; and are therefore unfit for human consumption?

The key scaremongers can invariably be traced back to a mere handful of sociopathic pseudo scientists who move back and forth between the upper echelons of government and corporation controlled institutions. These incestuous experts are singing for their supper. They are on the payrole of the multinational chemical consortiums; corporations who have invested billions of bucks in researching and developing their GM arable protein crops and the complementary package of pesticides to go with them. They have bought up oceans of acres of dirt cheap arable

land across Eastern Europe, the Third World and North/South America and they are clearly attempting to destroy anyone competing for “their” global protein market - Prime targets are the small mixed livestock farming sectors of agriculture who have traditionally maintained the mainstay of meat and milk protein production around the world.

The multinationals’ preference for a mono arable cropping use of land is easily understood; since every acre of grassland devoted to meat and milk production requires negligible inputs of pesticide / GM seeds in relation to every acre of farmland that is devoted to agrochemical-intensive arable protein production.

Despite the scaremongering over the ‘hyperinfectious’ nature of the prion, a basic study of the epidemiological history of TSE clearly demonstrates that this disease does NOT originate from animal to animal contact or through ingestion of feeds contaminated with TSE brain material.

Why do the ‘experts’ blatantly refuse to consult the ‘down to earth’ wisdom of the Icelandic farmers and vets who have been living and breathing with scrapie TSE for light years. When the first hint of scrapie symptoms emerge in their sheep, it is customary practise to slaughter the affected animal instantly, eating the flesh (brains and all !) before the poor animal has had time to waste away.

So if scrapie or CWD can be passed onto humans via consumption - as the scientific authorities would have us believe - why have no cases of CJD erupted in these Icelandic sheep farmers? In fact, Iceland has only ever witnessed two cases of CJD in its entire medical history, and these victims had both hailed from the scrapie-free district in the far south of the country.

Despite the repeated failure of attempts to eradicate long established TSE hotspot regions in Colorado and Iceland by wholesale livestock slaughter / fallowing regimes enacted across the cluster zones, governments are still adopting this same slaughter strategy as a first choice means of control. But history has shown that TSEs will invariably re-erupt as soon as fresh livestock are introduced back into the slaughtered out areas; supporting the idea that the environmental causes of TSE are still well and truly wedded to the local food chain, irrespective of the slaughter programmes.

Such extreme mammaliogeddon measures do little more than remove the superficial evidence of the disease. They merely mislead the public into the illusory notion that TSE has been controlled (a good vote catching policy for any government)..

Despite these simple observations, a manic mindset has recently gripped the global authorities who have jumped to the assumption that TSEs stem solely from hyperinfectious origins.

For example, the recent discovery of new clusters of CWD in US deer has invoked an official overreaction of unprecedented proportion – a wholesale slaughter policy of indigenous deer herds has been enacted throughout all CWD regions across the USA, leaving many of the Native American tribes without their traditional source of dietary protein. Whilst studying in Wisconsin recently, I heard the story of a deer rancher who had retained some body tissues from one of their CWD affected deer - for reasons of independent post mortem - only to find himself subjected to a gunpoint raid by wildlife officials .

These draconian slaughter measures are invariably promoted by the same hardcore cell of global ‘expert’ advisors - the hysterics who dreamt up the hyperinfectious hypothesis in the first instance. By burying or incinerating the evidence of their own control measures – eg the thousands of slaughtered animal

carcases – and then fallowing the land, the experts are placing themselves in a foolproof position where the success or failure of their control measures can never be properly assessed. In this respect, they can guarantee keeping their professional reputations afloat for the remaining lifespan of their careers.

But who is questioning the scientific reasoning for executing this final farcical solution on these poor creatures . For the unilateral adoption of a policy of 'totalitarian overkill' of a few million healthy animals across the world has been received with almost complacent acceptance. Such perverse and senseless 'carry ons' have sadly become the daily 'non-stories' of our modern times. Reports pop up with ever increasing frequency of so called TSE precautionary control programmes being enacted after 1% or more animals in a flock prove positive to the TSE genotype test – an endemic phenomena that has existed for light years without ill effect. Annihilation of a herd of waterbuffalo in Vancouver, sheep flocks from Vermont, 400,000 cows slaughtered in Germany, plus thousands of scrapie susceptible traditional sheep and goat herds erased from the European hillsides – all healthy animals.

Along with the sad threat to the survival of some indigenous wild and domestic animal breeds, we shall also loose their valuable outputs of manure - the heartbeat of humus supply which protects the soil against the erosive forces of nature, and, more importantly, feeds the fertility of the earth that ultimately sustains all life on the planet.

Furthermore, these slaughter measures are imposing the death knell on the survival of traditional peasant cultures - lifestyles which have evolved to be symbiotically dependent upon the income generated from their livestock enterprises. We are saying farewell to one of the last bastions of our cultural identity; a holistic charm that flavours the landscapes that have been etched out by centuries of occupation under peasant family

farms. That delicate ethereal relationship that flows between the soil, crops, livestock and landscape is under threat, along with the aesthetically pleasing array of idiosyncrasies that go hand in hand with peasant lifestyles; the architecture, craft skills, folklore and dialects that have divided the rich rustic out backs apart from the homogenised, synthetica of the city.

The ‘all out’ slaughter tactic betrays a total lack of interest in the cause, prevention or cure of this grotesque disease. The Establishment’s current global agenda to depopulate livestock numbers at whatever the cost (Agenda 2000), is for reasons that have nothing whatsoever to do with illusory health risks to the human race, but more to do with envisioned profits from multinational GM proteins .

I cannot help but feel that the global leaders have sold out to the multinational carrot. PR tactics used to promote “important” government policy increasingly capitalises upon some emotive scare story as a means of manipulating public mentality into conforming with the overall global agenda of the corporations. In much the same way as a war for Iraqi oil has been presented under the pretext of a morally justified war to rid Iraq of weapons of mass destruction, so the corporations’ war to rid the world of livestock protein has been presented under the guise of ridding the world of the unhealthy ‘risks’ of hyperinfectious prions.

The Broader Picture.

The BSE debacle represents the mere tip of an iceburg of establishment ineptitude and socio-eco-irresponsibility. It displays a clear cut example of the far reaching extent to which the 'talons' of multinational monopolies can stretch to protect their global master plan on the fast expanding 'Health and Food chain Industry'. Can we afford to allow this insidious mode of food chain control to continue unregulated and unabated ?

There is an increasing groundswell of public unease concerning the unknown effects that our polluted environment is exerting on our health and long term survival. Public suspicion is mounting towards the transparent array of so called independent scientific experts and medical spin doctors who are called to advise governments and address the public on all aspects of the impact of chemical, metal or electromagnetic pollutants on our food chain.

This story returns us to the lessons that can be learnt from the intuitive wisdoms of the people on the "ground" . At the same time it alerts us to the insidious and unscientific techniques which the incestuous clique of official experts employ to marginalize and discredit those who dissent from the totalitarian line. It shows us the ill conceived basis on which the positions of the Establishment are truly based, along with the woeful degree of administrative complacency over issues which, in most cases, are matters of life and death for normal people.

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