

# Mark Purdey

'Seeking the truth through science'

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## **The WASTING LANDS - the CWD epidemic in deer**



### **Mark Purdey**

Chronic Wasting Disease of deer is much the same as scrapie in sheep. It is a traditional variant of the spongiform encephalopathy (TSE) range of diseases; a previously unheard of neurodegenerative syndrome made famous by the outbreak of mad cow disease in the UK .

Seven per cent of the free ranging/captive deer and elk population residing along a 100 mile length of the Front Range of the Rocky Mountains in North Colorado and South East Wyoming have been affected with this disease for several years now. Originally identified as a TSE in the late 1970's by veterinary neuropathologist, Professor Beth Williams, the disease could have been endemic for many decades. Any rancher or hunter who had noticed these ailing deer hobbling around the place, had probably put such cases down to premature ageing or some 'weakling' wasting condition.

### ***The origins of hyper infectious hysteria.***

Despite the long term tradition of CWD haunting the Front Range foothills, a surge of near hysteria has bestruck the official US wildlife departments whose job it is to preside over CWD. Following in the footsteps of the official furore over mad cow disease in Europe, the US government has sadly adopted the same unproven hypothetical mindset on the origins of these diseases; that TSEs stem from exposure to hyper infectious 'prions' that are readily transmitted via body to body contact (saliva, etc), or via 'prion' contaminated feed. In this respect, blame has been conveniently offloaded onto the deer themselves – for sharing the same feed troughs, etc, – or onto the hunters for transporting the 'infectious' agent around with them from shooting region to region.

But why has such a deeply flawed and scientifically inept theoretical consensus been permitted 'gospel' status for such an unusually protracted period of time? The launch of any new theory into the notoriously sceptical scientific establishment invariably attracts a fair degree of healthy challenge. But strangely enough with TSEs, there has been an exception to this rule. This is largely because the UK government has been actively engaged in tailoring or outright suppressing, any publicity surrounding dissident scientific studies that invalidate or even begin to threaten any aspect of the official hypothesis. Furthermore, it is strange to witness the same old 'masters of complacency' in the higher echelons of UK officialdom, suddenly adopting a high degree of hypersensitivity over the way that they deal with their affairs. Such an incongruous style of official behaviour has betrayed a deep level of insecurity over anything that they are telling us on BSE.

For instance, nobody has been told that the British meat and bone meal (MBM) feed that was held responsible for the massive BSE epidemic in the UK has been exported by the cargo boat load, to cattle herds all over the world since the 1960s - yet the majority of those countries have never suffered a single case of BSE in their cattle herds to date. Nor does anyone know about the 40,000 cases of BSE that have appeared in UK cows that were born after the 1988 ban on MBM going into UK cattle feed. In this respect, nearly a quarter of the total cases of BSE in Britain cannot be explained by the conventional causal theory! In some BSE endemic countries, more than half of their total BSE cases were born after their respective MBM bans. Even my five year old son can see the stupidity of such an obvious 'cover story'.

To escape the embarrassment of the outright failure of control measures, the UK government set about hoodwinking the British public and their foreign interests by creating a second feed ban in 1996; whereupon they instructed their spin doctor journalists to misappropriate blame for the unaccountable 40,000 cases of BSE onto 'leakage of micro amounts of MBM left over in the feed silos getting into cattle feed'. They then gave full coverage to the fact that the government were now banning the inclusion of MBM going into feeds destined for *all* types of farm livestock. The 1988 ban was subsequently forgotten and conveniently erased from the public memory banks. But today, 22 cases of BSE have now emerged that were born after this second 1996 ban!

This whole hyper infectious myth has been based on the fact that TSEs can be transmitted in the laboratory; whereby TSE affected brain tissue is injected into misfortunate laboratory animals that subsequently contract TSE. The fact that classes of Alzheimer's and other neurodegenerative diseases can be transmitted in this way is completely ignored. But these transmission experiments prove nothing in terms of demonstrating whether TSEs are caused by a microbiological infectious agent or not. After all TSEs do not fulfil Koch's postulates; the conventional yardstick for assessing whether a given disease stems from infectious origins.

The 'all important' success of these 'trumped up' transmission experiments could have equally easily represented the fact that a highly toxic chemical or metal species which had originally contaminated and killed the initial TSE diseased animal was then being transmitted into a secondary host. Once again, this equally feasible alternative explanation has been ignored.

But one of the first lines of epidemiological inquiry aimed at investigating the origins of CWD ought to have addressed the question why the disease has not spread like wildfire, wiping out susceptible individuals of the deer population residing right across the entire Rocky mountain ranges. But whenever the likes of rancher and hunting folk who live and breathe with the deer has attempted to infiltrate the CWD debate, their intuitive and practical perspectives on the disease have invariably been rebuffed by the official and scientific policy makers.

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### *Historical truths ignored.*



A global study of the most basic rudiments of the history of TSEs clearly demonstrates that this disease does NOT spread via animal to animal contact or via ingestion of the 'infected' by the 'uninfected'. For instance, when I was researching the most intensive global hotspot of sheep scrapie in the northernmost Icelandic fjords, I found that the Icelandic sheep farmers had adopted the custom of slaughtering any sheep the moment the first symptoms of scrapie emerged. This tradition had not evolved from any fear of the disease exploding in the sheep population - since scrapie has occurred at a consistent incidence rate for light years in Iceland - but was carried out because the hard pressed farmers thought it best to eat the sheep (brains and all!) before the wasting symptoms of scrapie reduced the poor beast to skin and bones.

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.

A historical study of official government attempts to control both scrapie in Iceland and CWD in Colorado reveals the repeated FAILURE of several wholesale slaughter programmes that were executed in these well renowned, long standing TSE hotspot areas. After the deer and sheep had been culled across the vast tracts of land implicated in these TSE endemic regions, the fresh livestock introduced after a four year fallow period simply started to go down with the disease all over again.

Disturbingly, it seems that the US authorities have failed to learn such a simple lesson, and are following the farcical footsteps of their European counterparts, channelling public funds back into renewed slaughter schemes in Colorado and Wisconsin - schemes that are ironically no different from those which have already failed!

The repeated failure of these trials clearly indicates that the cause of this disease lies in the particular ENVIRONMENT where these animals were pastured. The answer must lie with some specific idiosyncratic factors commonly shared by all of the ecosystems where these spongiform hotspots erupt. An analytical field study of these regions provided a golden opportunity to pinpoint the aetiological needle in the causal haystack.

### *Low Copper ; the primary environmental prerequisite of TSE ?*

As part of my eco-detective treks visiting isolated TSE clusters all over the world, I came to research CWD in Colorado in the early 1990s. I soon realised that if the deer had been roaming those canyons for long enough, CWD would be as old as the pre Cambrian hills that towered above me. I drew a criss-cross of soil samples right across the CWD endemic area, and I remember the flecks of mica and schist that caught the razor-sun rays, almost dazzling me a few times - a phenomena that I had become well accustomed to during my sampling sprees in so many regions around the world; intensive sunlight, and more importantly, these specific geo-elements which characterised the granite terrain that underpinned every single long standing TSE cluster zone that

I had visited to date. Furthermore, this observation virtually guaranteed that my environmental analyses would, once again, come back “zero copper” from the lab – the causal cornerstone of spongiform disease pathogenesis.

I was also intrigued to learn that the only spongiform susceptible species which had failed to go down with the disease in the CWD endemic area was the pronghorn antelope – an indigenous antelope that is well adapted to its centuries old occupation of Rocky Mountain terrain. The Pronghorn can conserve levels of copper and selenium in its body considerably more efficiently than any other species of cervidae. Perhaps its metabolic predisposition for copper conservation which the other species do not possess explains why the pronghorn has resisted CWD?

In this respect, it was of no surprise when I heard the recent news that CWD had now been identified in deer living around Mt Horeb in Wisconsin – another copper deficient granite stronghold that has withstood the erosive elements over time. But for how long has CWD been around in Wisconsin? The disease may have been there for years, but only just been identified because of the recent surge of political sensitivity and scientific intrigue surrounding this disease; thereby raising the ‘CWD awareness’ profile sufficiently to recognise the disease. But if CWD has only just emerged, it has to be considered that copper deficiency has blighted these granite terrains for centuries and cannot therefore be held as solely accountable for the recent eruption of CWD.

### ***Metal Detector.***

An ideal research study presented itself after I located two Icelandic valleys fifteen miles apart; where one valley played host to sheep flocks which were riddled with scrapie, whereas the other valley supported sheep flocks which were entirely scrapie free. Intriguingly, sheep from both valleys had been freely intermixing on the open mountain during summertime - once again discrediting the conventional theory which assumes that scrapie transmits via animal to animal contact.

So the answer to the causal question clearly lay with some unusual combination of environmental factors that are present in the scrapie valley, yet absent in the scrapie free valley.

I ended up carrying out many self funded field analyses in Iceland and other TSE cluster regions in Japan, Slovakia, New Guinea, Colorado, Italy, Sardinia, etc. After many cul de sacs and false leads, I believe that my observations have actually now identified those common toxic denominators – low copper/high manganese combined with high intensities of low frequency infrasound shock - the key factors which have subsequently been shown to produce the fully fledged prion in laboratory cell cultures - eg; the malformed prion protein which characterises the brains of all animals affected with spongiform disease.

In the Icelandic scrapie valley, the levels of copper in the pasture were rock bottom for natural reasons. Whereas the high levels of manganese had originated from volcanic emissions; with subsequent accumulation of manganese in the pasture grasses due to the characteristic wetness of the pastures in the scrapie valley - where the resulting soil acidity renders manganese freely available for uptake into the pasture grasses. The source of intensive infrasound in this valley specifically stems from the earthquakes and earth tremors that have consistently issued from the major tectonic fault line which runs past the head of the valley – the nearby town of Dalvik was flattened by one such earthquake in 1938.

I subsequently identified the same set of common toxic denominators in the Colorado CWD cluster area. My field survey and analyses revealed low copper throughout the deer’s food chain, in combination with a dietary ‘fetish’ of the densely populated local deer for consuming large quantities of pine needles – which analysed out at 2000 + ppm of available manganese. Another more disturbing issue surrounding manganese intake stemmed from the fact that deer hunters were being sold minerals that were intended to addict deer to their hunting territory. In this respect, the hunters have been unwittingly shooting their own industry in the foot, by putting down these dual purpose minerals that have been formulated to addict deer to their shooting grounds as well as for forcing the sturdy growth of their antlers. Guess which mineral is added for forcing antler growth? My contacts from Wisconsin also report use of these manganese minerals in their CWD hotspot region.

Intriguingly, the CWD endemic region of Colorado is also well noted for its high intensities of

natural radiations of low frequency infrasound. Not only does it lie along a major fault line that runs up the Front Range ridge – producing its fair share of mini earthquakes and tremors over the years – but several publications have highlighted the high intensities of infrasound that derive from the atmospheric turbulence and winds passing over the mountain ridges of this specific region. The large number of explosions from the intensive quarry blasting activities in this area should also be considered as relevant sources of artificial infrasound, just as the intensive testing of missiles down at Whitesands missile range in New Mexico and another testing range near Mt Horeb in Wisconsin may also serve as these relevant sources of artificial infrasound in these recently declared CWD outbreak regions.

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**The wasting lands**



### *Infrasonic shock waves, high manganese, low copper; what's the connection with CWD ?*

Whilst there seems to be a correlation between the presence of this package of environmental toxic denominators and the timing and distribution of CWD outbreaks in the USA, how can all of these factors prove relevant to the cause of these mystery spongiform lesions found in the brains of the victims of these diseases?.

An alteration in the normal molecular shape of a specific brain protein, known as the prion protein, has been shown to be a critical deciding factor in the development of TSEs; since a deformed prion protein hallmarks the brains of all those mammals who have died of TSEs, perhaps indicating that some loss of function of this protein is all part and parcel of the TSE disease process. Intriguingly, the prion protein has been shown to bond up with copper in the normal healthy brain, and I have hypothesised that the copper found attached to the prion protein plays a role in conducting the electromagnetic energy that is received from incoming sources of ultraviolet, geomagnetic, infrasonic radiations, etc, form the external environment. These energies are utilised for the bodies own balanced metabolism; for regulating circadian mediated functions such as immune defence, growth and repair of cells, sleep/wake cycles, etc.

But when copper is in short supply in the brain, due to certain environmental influences, the prion protein is capable of bonding onto certain alternative metals, such as manganese, bismuth or silver. But these foreign substitutes may not act in the overall best interests of the organism. For instance, manganese will store up electro energy instead of conducting it like copper; thereby blocking the flow of electromagnetic energy that is required for regulating certain vital body functions.

But one of the specific characteristics of manganese is that it can absorb the energy of sound – such as the high intensities of 'phonon' energy that are insidiously radiated with the inaudible low frequency range of sound, known as infrasound. But manganese can only absorb this energy when found in its specific 'trivalent' manganese form; whereupon the sound energy actually metamorphoses the atomic structure of the manganese so that it can become permanently magnetised. So whenever an individual who is carrying excessive levels of this freaky form of trivalent manganese in their brains enters into an external magnetic field, the manganese bonded prion proteins become permanently magnetised to explosive flash point levels; whereby self perpetuating, chain reactions of free radical mediated neurodegeneration burst forth, and TSE pathogenesis ensues.

Whilst high intensities of trivalent manganese may be 'manufactured' in the living brain via an 'oxidative transformation' of manganese 2+ in the retina by incoming ultraviolet radiation or other eco-oxidants, etc, it is also possible that an exclusive source of trivalent manganese has got into the food chains in TSE endemic areas; via its incorporation into animal feeds or mineral licks, etc, or via their natural presence in the indigenous geological bedrock of the TSE region. Such a scenario may explain why hunters who are feasting off deer shot in CWD regions who have thrived off mineral licks/pine needles containing trivalent manganese, will, in turn, become contaminated with trivalent manganese themselves; and thereafter rendered hyper susceptible to the low frequency infrasonic shocks in their hunting environment (eg; natural infrasound, rifle shots, etc

).The same eco-prerequisites that caused CWD in the deer are now primed and present in the human hunter. CJD could result.

Not only has this abnormal mineral imbalance been consistently identified in all of the ecosystems blighted by clusters of TSE, but studies on the brains of CJD casualties by Case Western University's US Prion Disease Surveillance Unit have identified a 10 fold higher level of manganese and 50% reduction of copper in relation to control brains. Furthermore, Dr David Brown at Cambridge University in the UK has produced the TSE-like malformed prion protein in cell culture experiments after adding manganese to copper deprived cells.

Despite publication of all of these complementary field and laboratory studies in prestigious scientific journals, the various European health authorities and their key TSE advisors are blindly ignoring these findings. Not only are they discarding such an important fresh direction in TSE research, but they are doing their utmost to publicly marginalize those of us who are trying to pursue this line; and using public money to implement their tactics of suppression into the bargain!

### ***The Genetic Connection.***

Whilst it is true that all types of TSE require components of genetic susceptibility in their causal interplay, the TSE susceptible individual still very much requires the additional exposure to these toxic environmental factors before the disease can ever begin to manifest itself; Witness, the large number of scrapie susceptible sheep that live in scrapie-free Australia but never develop the outward symptoms of scrapie. Yet whenever Aussie sheep are exported to countries where scrapie is endemic, symptoms of scrapie invariably break out - presumably because the environmental causal factors are absent in their native Australian terrain, yet fully present in the importing countries.

The same scenario is duplicated in respect of a CJD susceptible Greek-Italian population that lives in many regions across southern Italy. But CJD has only ever erupted (at an excessive incidence rate) in just one of the many hamlets where these people live - a hamlet that is exclusively exposed to the specific environmental prerequisites that initiate TSE.

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### *A multinational master plan ?*

Since all of the evidence points to the fact that TSEs are caused by a clear cut combination of genetic and toxic environmental factors, why do the authorities worldwide continue to handle these diseases as if they stem from highly infectious origins?

I can only assume that the rigid adherence of Establishment bodies to the reductionist mindset - regardless of whatever new evidence comes to light - merely betrays the current global agenda to depopulate livestock numbers for reasons that have nothing whatsoever to do with health risks to the human race.

The true picture is one of a mere handful of politically-motivated, sociopathic pseudo scientists who predominate the upper echelons of the UK's and EU's agricultural and scientific ministries. These incestuous "experts" are singing for their supper. They are on the pay role of the global corporations whose sole interest lies in forcing open a market place for their GM arable protein products. Powerful organisations who have no interest in making life easy for their competitors; eg; those of us who are trying to make a living out of selling livestock proteins.

We only have to ask ourselves who are the key culprits that are currently capitalising on the fashionable scare stories which maintain that "BSE prions will exterminate us all"? - the multinationals. Who is 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 multinationals!

We must remain aware that these corporations 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 going to attempt to smash anyone competing for "their" protein market who gets in the way.

Despite the scare mongering, a basic study of the history of CWD clearly demonstrates that this disease does NOT originate from deer to deer contact. Despite such a simple observation, a manic mindset has recently gripped the whole US nation who have jumped to the assumption that this disease stems solely from hyper infectious origins. Any evidence put forward for an environmental cause has been blindly ignored. In this respect, the recent discovery of another cluster of CWD in Wisconsin has invoked an official overreaction of unprecedented proportion – a wholesale slaughter policy has been enacted throughout CWD endemic regions across the USA.

But who is questioning the scientific reasoning of the US authorities for executing their final farcical solution on these poor creatures? For this latest turn towards a unilateral policy of 'totalitarian overkill' of a few thousand healthy deer has been received with almost complacent acceptance across the country. Such perverse and senseless 'carry-ons' have sadly become the daily 'non-

stories' of our modern times – particularly here in Europe; where reports pop up with ever increasing frequency of so called TSE precautionary control programmes initiating slaughter regimes all over the globe - annihilation of a herd of water buffalo in Vancouver, flocks of sheep from Vermont, flocks of sheep in Sardinia, 1000's of sheep flocks in Germany, 400,000 cows in Germany – all healthy animals. So what next ? The entire BSE-free British sheep flock on the grounds that BSE *might* just appear in British sheep?

The sad twist to this tale is that straightforward copper supplementation of deer in CWD risk areas may be all that is required to prevent manganese replacing the depleted copper at the critical prion protein bonding sites in the deer's brain; which, in turn, prevents the 'knock on' increase in susceptibility of the animal to environmental infrasound; that insidious eco-force to be reckoned with, capable of triggering off a melt down of self perpetuating, free radical 'cluster bombs' in the brains of manganese contaminated deer, thereby instigating CWD. Furthermore, copper supplementation may be all that is required to prevent the senseless slaughter.

**Mark Purdey - September 2002**

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