

# Educating *Rida*

## An Eco-Detective Journey

*Mark Purdey has spent the past 20 years on a quest to explain "industrially induced" diseases, including transmissible spongiform disease (rida in Icelandic). His eco-detective adventures have taken him around the globe, and his discoveries and convincing conclusions are definitely at odds with conventional "wisdom." Purdey was awarded the Acres U.S.A. Lifetime Achievement Award in 2001 in recognition of his untiring efforts.*

by Mark Purdey

The White Sands Missile Range is an extensive spread of U.S. military-controlled cactus country that spans the southernmost extremes of the San Andreas 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 12-foot-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 that have succumbed to this so called "hyperinfectious" disease. The truck is soon on its way, losing itself within the thousands of acres of parched military compound.

They've come to investigate yet another new eruption of chronic wasting disease — the deer equivalent of Mad Cow disease — in the United States. 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 bodily 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 happened one day in the New Mexico desert.

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 at this unique location, factors that 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 (wulfenite contains the copper-chelating metal molybdenum). The museum-quality black crystals lay scattered across the top of the terrain, twinkling out a kind of somber resonance under the desert sun. They emanate the haunted history of the place.

In addition, since the military have occupied the range, U.S. 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.

### 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 shockwaves of the BSE debacle have ricocheted around the world. Despite the severity of the Mad Cow legacy, however, little genuine attempt has been made to crack the causal riddle of these diseases, leaving us devoid of insight into measures that would best cure, control and, better still, prevent this disease.

There is a ray of light, however. My own eco-detective escapades and original field investigations have run in tandem with the laboratory quest of Cambridge University biochemist Dr. David Brown, and 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 — ferromagnetic 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.

Despite the conclusions of several field and laboratory studies providing strong support for this environmental hypothesis, the resulting publications have been dis-

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missed by the U.K. 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 U.K. 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 U.K. government's compulsory warble fly eradication regime in the High Courts in 1984. The decision exempted my farming business from treating our dairy herd with a systemic organodithiophosphorus (OP) insecticide — a toxic chemical derived from military nerve agents, which, among 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 U.K. cattle herd in 1985. In my opinion, it was a direct legacy of the government's compulsory warble fly campaign, a 1982 measure that had enforced twice-annual high-concentration application of systemic OP insecticides.

As considerably smaller outbreaks of BSE began to erupt across other European countries, and later in Japan, my investigations revealed the voluntary usage of these same types of systemic insecticide in those countries — albeit at half the dose rates applied in the U.K. These European outbreaks seemed to follow an EU campaign known as COST 811, 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 to the control of warbles.

In warble-free Japan, the BSE cases emerged in the specific herds that had imported breeding cattle from warble-infested North America. The Japanese had taken preventative measures by blanket-treating these herds with the same types of systemic OP that had been used in Europe. It should be pointed out that the United States had wisely adopted a less toxic approach for dealing with their warbles. They employed lower doses of “non-systemic” insecticides — for example, in-

secticides that were not designed to penetrate through the skin — and only treated individual cattle that were 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. I was struck, however, 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-bonemeal (MBM) ingredient as part of their 20 percent conventional feedstuff allowance decreed in the organic standards at that time.

### FLAWS IN THE CONVENTIONAL HYPOTHESIS

From then on I became deeply skeptical 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 led 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 tissue into unfortunate 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 transmissible Alzheimer's disease?

There are quite a few other major flaws in the conventional hypothesis, including:

1. Thousands of tons of the BSE-incriminated MBM feed were exported as cattle feed during the 1970s, '80s and '90s to countries that have remained BSE-free to date, *e.g.*, 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 U.K. were blamed for permitting the survival of the scrapie agent in the sheep brain material, thereby

enabling it to jump across into cattle, producing BSE. But none of these alterations were exclusive to U.K. plants. Other scrapie-endemic countries such as the United States and Scandinavia had adopted the same continuous-flow-system of rendering five years before the U.K., yet these countries had 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 United States 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 U.K.'s 1988 ban on MBM incorporation into cattle feed have nevertheless 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 U.K., 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 U.K. 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 U.K. 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, which has been offered as an explanation for the growing number of vCJD clusters in rural areas, had been practiced by small-scale butchers all over the U.K.

10. BSE fails to fulfill Koch's Postulates, the yardstick for gauging whether a given disease stems from infectious ori-

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gins. For instance, more than 15 percent of cattle slaughtered for displaying the classic symptoms of BSE did not demonstrate the presence of the “causal” prions at postmortem.

### THE REDUCTIONIST MINDSET

Despite the number of epidemiological flaws, and millions of pounds’ worth of research having failed 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 in the mainstream public/professional mentality.

It is easy to see how the momentum of such a reductionist mindset took hold. 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, while 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. The government, in turn, 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 fossilized the reductionist notion that TSEs stemmed from infectious origins.

But as soon as 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 pasture lands — 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 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, and 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 U.K. (*e.g.*, 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. Following 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 program of wild and domestic animal populations across specially designated TSE eradication zones. Despite the well-publicized 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

While 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 simply 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. 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 organophosphate 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. 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 recognized 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 molecular interaction may occur between OPs and the prion protein. Since the prion protein has been shown to bond 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 well-wishers 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 U.K. 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

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missing link in the causal jigsaw. Or perhaps OPs weren't involved at all!

Caught in the midst of a minefield of multinational interests, medical spin doctors and political propagandists, I grew exhausted by the vortex of professionals who had successfully hijacked and cul-de-sac'ed all U.K. scientific research into transmissible spongiform encephalopathy (TSE) diseases. Furthermore, I'd found myself frozen out, operating much like an underground scientist, tramping a covert journey around the rustic outbacks of the U.K.'s dairy lands to nail down the true cause of BSE.

So I expanded my horizons and embarked upon a refreshing eco-detective trek to analyze 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 rock-bottom levels of copper, selenium and zinc in all of these food chains. Levels of manganese returned to normal in adjoining disease-free areas.

I was also fascinated to discover that 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 atmos-

pheric fall-out of some naturally occurring or industrial source of combusted manganese oxide — stemming, for example, 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 aluminum, can be absorbed directly into the brain via the nasal-olfactory inhalatory route of intake (that is, 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 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 1,000 times of those found in normal cow's milk. 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 (which were invariably reared on this powder) suffered such high incidence rates of BSE in relation to the negligible rates encountered in beef suckler or organically reared cattle (who were invariably reared on natural cow's milk). The more extensive BSE-free cattle herds of Australasia were also never fed these mineral-boosted milk powders.

## 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 and 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, while 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 Creutzfeldt-Jakob disease (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, fertilizer and fungicide sprays, paints, petrol additives, etc. — or via increased consumption of trendy food products such as soy, where the beans naturally bioaccumulate high levels of metals such as 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 the "manganese madness" syndrome — the idea that manganese could perform a front-line role in the pathogenesis of TSEs is not out the way.

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In fact, manganese toxicity can manifest itself in many forms of neurodegeneration. When I visited areas in the South Pacific such as the Isle of Guam, where a raft of neurodegenerative conditions involving Alzheimer's, Parkinson's and motor neurone disease had run at a 50-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 northeast Australian coastline with a history that 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. Nevertheless, 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 percent 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/low magnesium-induced mutation in early life. Previously fit and healthy Aborigines 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 healthy Aboriginal bushperson out stalking a wallaby.

Furthermore, the levels of unrestrained aggression and murder in this community had reached crisis proportions, where the rates of imprisonment are 20-fold higher than in any other Aboriginal community. Grotesque Hieronymus 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 "man-

gane levels serve as a marker for violence."

True to form, however, the official cause of this "drunken walking" syndrome has been conveniently scapegoated onto a mutation caused by faulty Aboriginal genetics. 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 that 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, and mineral feed supplement industries) across the developed world had somehow seeded the "Mad Cow Madness" in the deer, sheep, cats, zoo animals, cows and young teenagers?

### LABORATORY STUDIES

My observations of high manganese/low copper in TSE cluster areas led to my connecting with the pioneering laboratory investigations of Dr. David Brown at Cambridge University, a widely published biochemical expert who had pursued groundbreaking studies that 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 necessary groundwork 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 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 food chains would be at risk of developing TSE.

So David Brown ran the necessary cell-culture experiments, in which 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 Prion Surveillance Center at Case Western University in 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 percent 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 natu-

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rally occurring environmental influences which bring about a high-manganese/low-copper ratio in the mammalian brain. On the other hand, the more aggressive, “rapid attack” modern strains of TSE — BSE and 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.

Unfortunately, the abnormal shaped prions that Brown’s high-manganese/low-copper experiments had created 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 might provide that final clue.

### QUANTUM CAPACITIES

Modern health authorities could learn a lesson from the alchemists of the Byzantine era, who regarded manganese as the black magic metal: the quantum capacity of manganese to absorb light and sound energy can induce a lethal “Jekyll and Hyde” 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 neutralizing 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 AS CONDUCTOR & MANGANESE AS BLOCKER

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 and 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 highway that distributes the energy of light and sound around the body for energizing the cycles of sleep, sex, behavior, heartbeat, cell growth and repair, and immune response.

On the other hand, 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 flashpoint is exceeded, thereby detonating 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 recognized by Chinese medicine, where the healthy copper prion performs a regulatory role in maintaining the electro-homeostatis along the acupuncture meridians and nodes.

Whereas 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 (*i.e.*, a metal that can be temporarily magnetized) just like copper, so to some extent one 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?

### METAMAGNETIC TRANSFORMATION

One of the interesting properties of manganese is that it can absorb the energy of sound — well illustrated by its commercial use in some audiotape 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.

Thus, 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 transformation of manganese from its normal paramagnetic to a rogue ferrimagnetic form, that is, from a temporary to permanently magnetizable 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 food chain, thereby contaminating any mammalian population that is self-sufficient upon the local ecosystem in the years to come. If these adulterated food chains are simultaneously short of copper, then the mammalian population is put at high risk of developing TSE.

### TSE & 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

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illustration of the infrasonic phenomena in a TSE cluster involved an intensive outbreak of sheep scrapie after sheep had first been pastured on a block of former military-controlled common land at Ashoro on the Hokkaido Peninsula in Japan. This copper-deficient land was previously utilized by the Japanese military during World War II. 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 military jets from nearby Warren Air Force Base during the late '60s and early '70s. When the complaints of the Fort Collins residents reached screaming point, the military was forced to move the flights to the plains in the northeast, and CWD outbreaks have followed likewise.

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

As a rule, the traditional forms of TSE result from exposures to the naturally occurring infrasonic shock waves that radiate from geotectonic plate rift lines (earthquakes, volcanoes, etc.), whereas the new-strain TSEs result from the more intensive low-frequency shock bursts radiating from the supersonic military or Concorde aircraft. The emergence of the respective types of TSE clusters along these tectonic rift and flight path lines substantiate this idea well.

Perhaps it is no surprise that 99 percent 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. The afterburner turbofans employed by these supersonic airplanes radiate such a high intensity of low-frequency infrasound — in the 10 to 5 hertz range — that a 100-kilometer-wide carpet of acoustic shock waves is 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 United Kingdom. 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 hotspot locations.

It is perhaps no surprise that the infrasonic environs of Staten Island and Long Island in the United States — both under the take-off flight paths of JFK Airport, where Concorde and other aircraft land — have demonstrated the highest incidence cluster of traditional CJD in the country.

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. Rural and coastal ecosystems have become increasingly exposed to a toxic combination of manganese-based fertilizers and 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 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-infected animal products, since meat products are consumed equally by urban and rural populations alike.

## ‘INFECTIOUS’ FERRIMAGNETIC MANGANESE

Once the crucial supply of copper is curtailed in the brain — due to straightforward 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 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 in which a magnet is placed alongside a steel nail and the force field of the magnet is shown to magnetize that nail.

Thus, once an individual’s brain is contaminated by this freaky form of metamorphosed manganese, any subsequent exposure to external electromagnetic fields (*e.g.*, UV light, 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 — and 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 utilizing 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

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as the so-called infectious pathogenic agent in TSEs. Thus, whenever scientists inoculate unfortunate lab animals with TSE brain tissues (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 F — since ferrimagnetic metals will hold onto their magnetic charge until they are heated to temperatures beyond their respective Curie point temperature (550 F for manganese 3+).

### VALID THEORY

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

Intriguingly, tissues such as pituitary and retina which transmit TSE in the lab most efficiently are the same tissues in which manganese is recognized to concentrate most intensively in the body. Thus, 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. Whereas cannibalism may have played a role in the bioaccumulation of manganese — particularly if the pituitary tissues were ingested — the fact that virtually every tribe across New Guinea had adhered to a cannibalistic lifestyle, while remaining free of Kuru, needs to be addressed by those who promote this theory. 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 II?

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 with their scavenging of manganese-aluminum sheet metal from the fuselages of several Japanese bomber aircraft that had crashed in their area of the highlands during World War II. The Fore folk molded the metal to make tools, cooking pans and bowls, 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 has manufactured these metal aircraft panels since the late 1930s. During this period, the manganese-enriched emissions from the factory chimneys dispersed downwind, permeating the entire length of the valley. Intriguingly, a cluster of CJD cases have blighted the residents of the Fuji river basin for 50 years.

Despite publication of the hard evidence in support of my theory in prestigious scientific journals, the various U.K. authorities and their incestuous clique of "minder" advisors are blindly ignoring these findings. What's more, they are doing their utmost to publicly marginalize those of us who are trying to pursue this alternative research line, and are 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" physical disasters: arson, firearm intimidation, vandalization of my research library/communications, and insidious infiltration by a bizarre array of bogus Greens and phony free-lance journalists, along with a seductive approach by a scantily clad pseudo-stu-

dent 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, even as they were snooping into the current position of my research investigations. Once my work gained support from the likes of former Minister of Defense Tom King and H.R.H. The Prince of Wales, the physical aspects of this harassment abruptly ceased.

A recent demand to the U.K. government departments for my personal data revealed much of what had been going on behind the scenes. Repeated requests by Environment Minister Michael Meacher to personally meet with me had been deliberately stymied by his own officials. Other documents revealed how the British Agrochemical Association had been organizing 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 favor of some aspects of my own hypothesis, the U.K. government responded by setting up a further mini-inquiry to reexamine 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 stated that the use of OP warblecides had ceased in the United Kingdom by 1982, and that warblecides had been routinely used on Jersey Island. So, demands 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?

Ironically, the truthful picture of the compulsory twice-annual OP warblecide treatment program was that it was first introduced in 1982 in the United Kingdom, while 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

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globally circulated publication, they pleaded “qualified privilege” of the expert committee, and then spun out legal communications beyond the one-year post-publication mark, thereby exempting themselves from my claim.

After the broadcast of the *BBC Correspondent* film “Mad Cows and an Englishman,” which chartered my investigations, the government tried to appease mounting public interest by inviting me to resubmit an application to them for funding. I did so, and after sitting on my application for a year and a half, they homed in on the most fastidious, nitpicking comments in the peer-review appraisal, trumping them up as a sound scientific basis for their rejection. Immediately afterwards, the author of what seemed to be the most irrational, irrelevant critique found himself promoted to the government’s expert TSE Surveillance Steering Committee, presumably as a reward.

The U.K. 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 stance of the establishment toward the environmental perspectives of TSEs probably have more to do with protecting academic egos, professional reputations and the vested interests of the TSE institutions/key advisors than with promoting sound scientific argument. Another reason must undoubtedly 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.

Delving a bit 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 is spinning the propaganda myths that beef, lamb, venison, game and organic food (grown using 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 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 payroll of the multinational chemical consortiums — corporations that have invested billions of dollars 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 that have traditionally been 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 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 practice to slaughter the affected animal instantly, eating the flesh (brains and all!) before the poor animal has had time to waste away.

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 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. History has shown, however, that TSE 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, regardless of slaughter programs.

Such extreme mammaliageddon 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 U.S. deer invoked an official overreaction of unprecedented proportion — a wholesale slaughter policy of indigenous deer herds was enacted throughout all CWD regions across the United States, leaving many of the Native American tribes without their traditional source of dietary protein. While 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” advisers — the hysterics who dreamt up the hyperinfectious hypothesis in the first place. By burying or incinerating the evidence of their own control measures — *e.g.*, thousands of slaughtered animal carcasses — and then fallowing the land, the experts are placing themselves in a foolproof

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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.

Who is questioning the scientific reasoning for executing this final, farcical solution on these poor creatures? 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 programs being enacted after 1 percent or more animals in a flock prove positive to the TSE genotype test — an endemic phenomenon that has existed for countless years without ill effect. Annihilation of a herd of water buffalo in Vancouver, sheep flocks in Vermont, 400,000 cows slaughtered in Germany, plus thousands of scrapie-susceptible traditional sheep and goat herds erased from the European hillsides — all these healthy animals were killed for no good reason.

Along with the sad threat to the survival of some indigenous wild and domestic animal breeds, we shall also lose 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 that 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 flavors the landscapes 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 outbacks from the homogenized 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 cost (Agenda 2000) is fueled by 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 for the multinational carrot. PR tactics used to promote "important" government policy increasingly capitalize 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 iceberg 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 toward 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 learned from the intuitive wisdoms of the people on the ground. At the same time, it alerts us to the insidious and unscientific techniques that the incestuous clique of official experts employs 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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