



Elevated levels of ferrimagnetic metals in foodchains supporting the Guam cluster of neurodegeneration: Do metal nucleated crystal contaminants evoke magnetic fields that initiate the progressive pathogenesis of neurodegeneration?

Mark Purdey*

High Barn Farm, Elworthy, Taunton, Somerset TA4 3PX, UK

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Summary Elevated levels of aluminium (Al), strontium (Sr), barium (Ba), iron (Fe), manganese (Mn) cations – combined with deficiencies of magnesium (Mg)/calcium (Ca) – have been observed in the foodchains that traditionally support the Chamorro populations affected by high incidence clusters of Alzheimer (AD), Parkinson-like (PD), motor neurone diseases and multiple sclerosis on the island of Guam. Soils drawn from the cluster region demonstrated an excessive fivefold increase in ‘magnetic susceptibility’ readings in relation to soils from disease free adjoining regions. A multifactorial aetiological hypothesis is proposed that pivots upon the combined exposure to high levels of natural/industrial sources of ferrimagnetic/ferroelectric compounds incorporating Al, Fe, Mn, Sr, Ba (e.g., via yam/seafood consumption or exposure to world war 2 (WW2) munitions) and to low levels of Mg/Ca in all S. Pacific locations where these clusters of neurodegenerative disease have simultaneously erupted. Once gut/blood brain barrier permeability is impaired, the increased uptake of Al, Fe, Sr, Ba, or Mn into the Mg/Ca depleted brain leads to rogue metal substitutions at the Mg/Ca vacated binding domains on various enzyme/proteoglycan groups, causing a broad ranging disruption in Mg/Ca dependent systems – such as the glutamine synthetase which prevents the accumulation of neurotoxic glutamate. The rogue metals chelate sulphate, disrupting sulphated-proteoglycan mediated inhibition of crystal proliferation, as well as its regulation of the Fibroblast growth factor receptor complex which disturbs the molecular conformation of those receptors and their regulation of transphosphorylation between intracellular kinase domains; ultimately collapsing proteoglycan mediated cell–cell signalling pathways which maintain the growth and structural integrity of the neuronal networks. The depression of Mg/Ca dependent systems in conjunction with the progressive ferrimagnetisation of the CNS due to an overload of rogue ferroelectric/ferrimagnetic metal contaminants, enables ‘seeding’ of metal-protein crystalline arrays that can proliferate in the proteoglycan depleted brain. The resulting magnetic field emissions initiate a free radical mediated progressive pathogenesis of neurodegeneration. The co-clustering of these various types of disease in select geographical pockets around the world suggests that all of these conditions share a common early life exposure to ferromagnetic metal nucleating agents in their multifactorial

* Tel.: +44 1984 656832.

E-mail address: tsepurdey@aol.com.

aetiology. Factors such as individual genetics, the species of metal involved, etc., dictate which specific class of disease will emerge as a delayed neurotoxic response to these environmental insults.

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Introduction

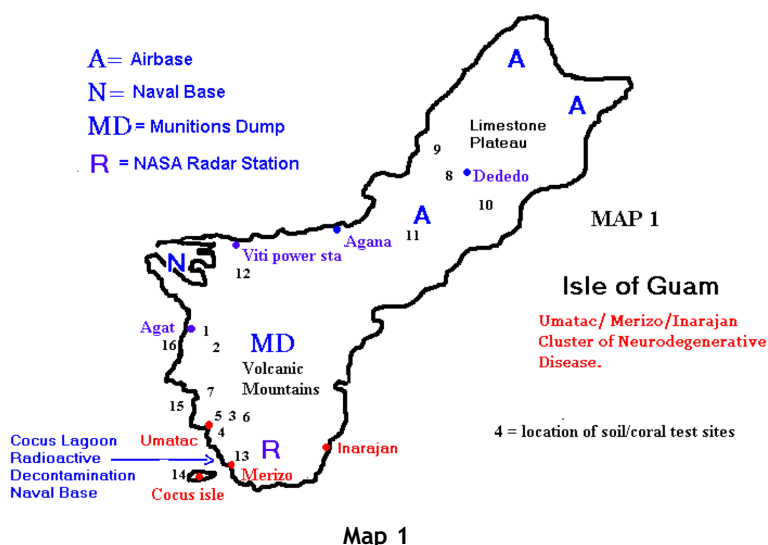
An environmental field study was designed to look at the levels of 46 metals and their magnetic susceptibility/radioactive status in the soils, vegetation/fruit and water supplies of the ecosystems which support the South Pacific Chamorro populations who have been affected by a high incidence clustering of AD, PD, amyotrophic lateral sclerosis (ALS), and, to a lesser extent, multiple sclerosis type diseases – collectively known as the Guam syndrome [1,2].

Whilst this syndrome had simultaneously erupted in several well defined locations across the South Pacific since the 1950s – the kii peninsula of Japan, West New Guinea, the islands of Rota and Guam [3,4] – it was decided to focus this pilot study on the ‘epicentre’ of the most intensive, clear cut cluster location on the isle of Guam, where the cases had emerged in three contiguous coastal villages located on the southernmost tip of the island – Umatac, Merizo and Inarajara [5] (see Map 1).

After the first cases of the epidemic erupted in the 1950s, the incidence rate of the disease was 50× higher than mean global rates [2] until the 1960s/1970s when the prevalence of the disease started to decline to reduced levels. In fact, few cases have yet to appear in any person born after

the mid 1950s [1]. The spatio-temporal epidemiology of the cluster indicates that this epidemic could represent a delayed neurotoxic reaction to the introduction of some alien toxic agent into the local environment during the 1940s/1950s. But the precise causal components have remained an enigma, becoming a widely debated topic of great controversy over the years.

One plausible aetiological theory focuses on the slow intoxication of the Chamorro population following the increased consumption of local cycad fruit during the impoverished years when the Japanese occupied Guam in WW2 [2]. Cycad represents a rather innocuous looking miniature palm tree that has outlived the dinosaurs and provided a staple flour product for the indigenous people who populate the South Pacific for centuries. The intoxication has been attributed to the ingestion of neurotoxic amino acids, beta-N-methylamino-L-alanine and/or methyl-azoxymethanol beta-D-glucoside (cycasin) that occurs naturally in cycad fruit [2,6]. And once cycad consumption had purportedly increased during the frugal WW2 period, the Chamorro’s genetically impaired expression of the antioxidant Mn/Zn superoxide dismutase and Apo-E detoxification enzymes [6] predisposed them to both inefficient degradation of these neurotoxic poisons as well as an inefficient anti-oxidisation of the free



radicals generated by this type of poisoning. However, cycad was eaten across the entire island of Guam [7], so this explanation fails to address the spatial distribution of the disease and the high concentration of cases in the South of Guam.

Another theory does seem to address the exclusive emergence of these neurodegenerative diseases in Southern Guam [8,9]. This explanation pivots upon a geochemical difference [8,9] between the miocene volcanic derived foodchain that has traditionally sustained the diseased Chamorro population in the south [10,11], and the limestone derived foodchain that has sustained the largely unaffected Chamarros in the North [10,11]. Westernisation of the traditional diet of the Chamarros since WW2 has run in tandem with a drastic reduction in their dependence upon local foods [2].

Several geochemical studies have already investigated this well defined correlation between the spatial epidemiology of Guam syndrome and the outcrop of volcanic terrain [8,9,11–13], and most researchers have arrived at the common conclusion that a bioaccumulation of Mn and Al, combined with low levels of Ca and Mg, played a causal role in the syndrome. Conclusions have suggested that chronic dietary deficiency of Ca/Mg since birth, induced excessive absorption of Al and Mn cations which accelerates oxidant mediated neuronal degeneration in susceptible populations [6].

Nonetheless, the multifactorial causal 'jigsaw' of prerequisites surrounding Guam syndrome still remains to be fully addressed by the geochemical theory. For instance, the presence of this indigenous metal imbalance in the local volcanic terrain fails to explain why the disease did not erupt until the 1950s. Nor does it explain the greatly reduced rates of the disease in the North of the island, where levels of the 'incriminated' Al and Mn cations in the topsoils are equally elevated [11]. It also needs to explain why incidence rates of the syndrome are elevated to 'cluster status' on the nearby island of Rota [3] whose topsoils are entirely based upon limestone bedrock [10] – like the relatively 'disease-free' North of Guam.

In this respect, the aim of this pilot study was to broaden the scope of previous geochemical studies, and take a comparative look at 46 different metals and identify whether any unique abnormal mineral imbalance and/or abnormal magnetic susceptibility/radioactive status existed in the soil, water and, most particularly, the foods of the affected population in the South in relation to the disease-free populations of the North. And if imbalances were identified, whether they could play a primary role in the multifactorial aetiology of Guam syndrome.

The study focused upon three contiguous coastal villages located on the southernmost tip of the island of Guam – Umatac, Merizo and Inarajara [5] (see Map 1) – which represent the epicentre of the cluster.

After assessing the traditional diet of the Chamorro people – ferrel pig/deer meat, oranges, tangerines, lemons, pineapple, fish, crabs, prawn, molluscs, cycad, yams, tapioca, taro, coconut milk – the levels of metals were analysed in the cycad, yam, taro, tapioca, shellfish, crab; as well as the betel nut, papula leaf and the coral lime which comprises the 'cocktail concoction' that is habitually chewed by the traditional members of the Chamorro population for its pleasant stimulatory effects.

The history of any toxic environmental influences that could potentiate metal intoxications were also investigated. This entailed investigations into industrial/military/agricultural/natural sources of noise, electromagnetic magnetic radiations, radioactive or organic chemical stress influences, which could have increased the permeability of the blood brain barrier [15–22] thereby allowing increased uptake of metal-transporter protein complexes into the brain [23], and/or generating metal mediated free radical chain reactions [24].

Procedures for soil, water and vegetation collection/analyses

Samples of soil, water and vegetation were collected in accord with the methods used by the author in previous studies [25] and were focused on the areas where the local populations harvested their food supplies. Areas around potential sources of metal pollution that could have posed a problem during the 1940s/1950s – such as an oil fired power station, the airport flight paths and a former naval radioactive decontamination station – were also analysed in this study. These soil samples were also analysed for their total magnetic susceptibility status in accord with the protocols previously employed by the author [26].

Results (sources for reference ranges of elements [27,28])

Soil analyses results

The results of the soil analyses indicate elevated levels of both Fe, Al, Mn and Sr compounds in the extensive outcrops of red laterite top soils that

surface along the western side of Guam [10]. However, the analysis of the 'Atate clay' class of red laterite top soil which is exclusive to the South Western 'cluster' region of Guam [10] demonstrated excessive levels of Mn at 12,756 ppm, probably associated with a black nodulous rock that predominated in the intensively mineralised local soils; that also demonstrated the presence of a 'cocktail' of other heavy metals – cobalt, chromium, copper, nickel, vanadium, zinc, lithium (see Table 1).

Sr levels were particularly high around the main airport and the Viti oil fired power plant at 1097 and 1170 ppm, respectively – suggesting a possible origin from artificial pollutant sources linked to the fuel emissions from the aircraft and power station. However, Sr levels were more intensively elevated in the southern areas of Guam, especially at the former radioactive naval decontamination station on the Western tip of Cocus island [29,30] where Sr levels in the topsoil averaged 4151 ppm, suggesting the possible presence of Sr 90/89 isotopes. Sr levels were also excessively elevated at 4211 and 3512 ppm in the coral/shellfish samples drawn from the Umatac and Agat coastlines respectively.

Ba was consistently sevenfold higher in the Umatac vicinity in relation to negligible levels recorded in the North of the island; perhaps linked to the renowned presence of Ba in soils derived from the Miocene era [31] – comprising the deposits of the Bolanos volcanic formation of South West Guam [10].

The levels of the other alkali earth metals, such as CaO, were markedly higher in the soils of Northern Guam (32.6%) in relation to the South (2.8%), whilst the distribution of MgO was surprisingly the reverse – 3.06% in the North and 0.4% in the South.

The levels of those metals that indicate the possibility of artificial radioactive contamination [32] such as Thorium, Uranium and Yttrium, were elevated at some locations in the North of the island around Dededo, whilst other toxic metals such as Arsenic, Lead and Cadmium were also exclusively elevated in the more industrialised regions of N Guam. Given the proximity of these sampling locations to the various military bases (see Map 1), some association could exist between the high levels of these toxic metals and the polluted discharges from the various military/aeroplane activities.

Water analyses results

The levels of metals in the water supplies used across Guam were largely free from any toxic levels

of metals, although slightly raised levels of arsenic (13 ppb) were identified in the former spring water supplies of Umatac, whilst raised levels of Fe, Al, Rubidium and arsenic were present in the river water at Merizo; at 53.09, 35.03, 59.5 and 20.2 ppm, respectively (see Table 2).

Levels of Mg at 0.1 ppm and Ca at 1.5 ppm in the former spring drinking water supplies that served Umatac confirmed the very low Ca/Mg status, and were still low at 4.8 and 14.4 ppm, respectively in the 'treated' town water supplies that currently provide the village with their drinking water. Interestingly, Mg and Ca levels were relatively raised at 74 ppm/12.6 ppm in the water supplies across the disease free North of Guam.

Vegetation/fruit analyses results

Levels of Ca in all of the vegetation and fruit sampled in the disease cluster region of Southern Guam were consistently 3.5-fold lower at 7930 ppm in relation to the elevated levels of 28749 ppm recorded in the North of Guam. Likewise the levels of Mg in S Guam (2053 ppm) – unlike its comparative profile in the soils – were generally 50% or more below the levels of Mg recorded in the vegetation from the North of Guam (4095 ppm), whilst the greater portion of the traditional staple foods consumed by the Chamorros – such as cycad, yams, taro and tapioca – were all deficient in Mg and Ca (see Table 3).

Ba, Mn, Al and Fe were considerably higher in the vegetation around Umatac/Merizo (at 49, 186, 185, 204 ppm, respectively) than in the vegetation in the North of Guam (at 3, 68, 47, 31 ppm, respectively).

Whilst there was no elevation in the levels of these potentially toxic metals in the cycads, betel nuts, taro or tapioca that were sampled, the yams were, by contrast, excessively high in Fe, Al (809 and 940 ppm, respectively) and marginally high in copper, nickel, titanium and cobalt. The levels of Fe and Al in Yams (98 and 166 ppm, respectively) collected from the disease-free north of Guam were much reduced.

Sr was at a high level (91.4 ppm) in the vegetation collected on Cocus island (where the Naval radioactive decontamination base was located during the 1940–1960s) [26,27], and, interestingly, copper was high (12.9 ppm) in the paprika leaves that are habitually chewed with the betel nut and coral lime by the Chamorros; which might account for the phenomena of the 'reddish bronze' colouring that tarnishes the tongues of those who indulge in this traditional practise.

Table 1 Analyses of soils and corals drawn from disease cluster and disease free areas on Guam

Reference	Location	Matrix	MS	Al ₂ O ₃	Fe ₂ O ₃	MgO	CaO	Na ₂ O	K ₂ O	TiO ₂	P ₂ O ₅	MnO	Mn (ppm)	Ba	Co	Cr	Cu	Li	Ni	
Disease cluster	[3]	Umatac	Valley marsh	1520	12.30	8.51	2.81	11.55	0.46	0.62	0.73	0.14	0.220	1703	237	25	74	87	35	43
	[4]	Umatac	Valley silt	760	14.19	10.92	5.28	4.02	0.55	1.10	0.71	0.10	0.135	1045	187	32	99	118	27	58
	[5]	Umatac	Laterite	1400	26.06	11.60	1.27	0.52	0.04	0.23	0.88	0.04	1.648	12756	239	377	523	352	79	653
	[6]	Umatac	Jungle soil	1210	14.92	10.04	2.81	3.68	0.23	0.55	0.76	0.07	0.075	581	192	29	166	100	54	81
	[7]	Umatac	Red laterite	410	22.31	11.87	1.82	0.56	0.04	0.27	0.74	0.03	0.077	596	30	21	294	94	20	94
	[13]	Merizo	Laterite	1170	22.23	14.12	4.02	0.72	0.08	0.30	0.58	0.01	0.185	1432	55	52	459	103	41	429
Disease free	[1]	Agat	Red laterite	190	27.53	17.71	0.73	0.83	0.04	0.09	1.05	0.01	0.106	820	24	21	347	128	5	137
	[2]	Agat	Valley silt	220	16.47	10.29	5.81	1.58	2.48	0.47	0.65	0.04	0.121	937	46	34	242	98	10	132
	[8]	Dededo	Red laterite	250	23.27	10.44	0.19	23.95	0.02	0.00	1.06	0.20	0.128	991	21	10	603	21	2	84
	[9]	Dededo	Red laterite	540	17.14	7.76	0.22	29.68	0.02	0.00	0.77	0.23	0.201	1556	24	10	511	21	2	100
	[10]	Dededo	Limestone	-10	0.00	0.03	0.27	49.36	0.02	0.01	0.00	0.01	0.001	8	9	0	7	4	0	4
	[11]	Tiyan	Soil (airport)	160	16.85	9.44	0.39	21.09	0.03	0.04	0.68	1.19	0.267	2067	43	18	337	59	41	101
	[12]	Viti	Soil (power sta)	100	2.62	1.68	0.98	39.40	0.11	0.06	0.09	0.05	0.018	139	18	3	40	21	5	22
Cluster	[14]	Cocus Is	Coral/shellfish	3	0.02	0.03	0.68	49.73	0.55	0.01	0.000	0.010	0	16	12	1	6	5	4	6
	[15]	Umatac	Coral/shellfish	0	0.05	0.04	0.13	48.93	0.60	0.01	0.000	0.010	0	0	12	1	4	4	5	5
	[16]	Agat	Coral/shellfish	157	0.29	0.34	0.68	48.85	0.41	0.01	0.030	0.020	0	248	12	3	17	6	5	8
Disease cluster	[3]	Umatac	Valley marsh	1520	28	349	214	85	55	0.55	0.99	15	0.39	25	12	21	3	16	3.9	1.3
	[4]	Umatac	Valley silt	760	30	191	218	109	53	0.47	0.98	20	0.38	18	8	15	2	12	3.1	1.1
	[5]	Umatac	Laterite	1400	62	9	254	177	86	1.89	0.84	7	0.37	54	22	42	6	29	6.5	2.0
	[6]	Umatac	Jungle soil	1210	32	197	246	98	59	0.73	0.93	14	0.47	21	11	18	3	15	3.5	1.2
	[7]	Umatac	Red laterite	410	52	14	258	90	70	1.19	0.85	8	0.72	58	22	26	6	32	7.7	2.4
	[13]	Merizo	Laterite	1170	55	36	157	98	53	0.34	1.07	7	0.34	29	6	8	2	9	2.8	1.0
Disease free	[1]	Agat	Red laterite	190	59	10	363	60	86	0.64	0.98	5	0.48	10	2	7	1	3	1.1	0.4
	[2]	Agat	Valley silt	220	38	66	215	109	54	0.33	0.44	9	0.3	28	4	8	2	10	3.3	1.1
	[8]	Dededo	Red laterite	250	33	302	142	28	123	5.93	18.66	0	0.28	156	66	100	15	70	14.4	3.6
	[9]	Dededo	Red laterite	540	25	355	113	37	96	5.44	12.77	0	0.13	154	54	83	12	56	11.5	2.8
	[10]	Dededo	Limestone	-10	0	197	2	4	3	1.21	0.07	0	0.01	1	0	0	0	0.0	0.0	0.0
	[11]	Tiyan	Soil (airport)	160	29	1097	129	167	73	11.04	3.91	3	1.98	63	20	33	5	24	5.7	1.6
	[12]	Viti	Soil (power sta)	100	5	1170	28	45	11	1.78	0.17	2	0.1	5	1	2	0	2	0.5	0.2
Cluster	[14]	Cocus Is	Coral/shellfish	3	0	4151	1	4.00	3.00	2	0.02	0	0	0	0	0	0	0	0.1	0.0
	[15]	Umatac	Coral/shellfish	0	0	4211	2	3.00	3.00	1	0.02	0	0	0	0	0	0	0	0.1	0.0
	[16]	Agat	Coral/shellfish	157	1	3512	12	7.00	4.00	2	0.04	1	0	3	1	1	1	0.0	0.1	0.0
Disease cluster	[3]	Umatac	Valley marsh	1520	4.1	3.9	0.9	2.4	2.2	0.4	0.1	3.1	3.9	0.4	0.1	0.3	0.2	0.1	0.1	0.1
	[4]	Umatac	Valley silt	760	3.2	3.1	0.7	2.0	1.9	0.3	0.1	0.8	4.7	0.2	0.1	0.2	0.3	0.1	0.1	0.1
	[5]	Umatac	Laterite	1400	7.6	8.6	2.0	5.8	5.8	0.9	0.1	4.3	4.1	0.3	0.2	0.5	0.2	0.1	0.1	0.1

Table 1 (continued)

Reference	Location	Matrix	MS	Gd	Dy	Ho	Er	Yb	Lu	Ag	As	Pb	Cd	Tl	Mo	Sb	Bi
[6]	Umatac	Jungle soil	1210	3.8	3.6	0.8	2.3	2.2	0.3	0.2	2.2	4.9	0.2	0.1	0.3	0.2	0.1
[7]	Umatac	Red laterite	410	8.9	9.8	2.2	6.6	6.4	1.0	0.1	4.4	5.1	0.1	0.1	0.6	0.3	0.1
[13]	Merizo	Laterite	1170	3.5	4.1	1.0	2.9	2.9	0.5	0.1	2.4	3.0	0.2	0.1	0.4	0.2	0.1
<i>Disease free</i>																	
[1]	Agat	Red laterite	190	1.3	1.6	0.4	1.1	1.3	0.2	0.2	1.0	7.0	0.1	0.1	0.6	0.2	0.3
[2]	Agat	Valley silt	220	3.7	4.2	1.0	2.9	2.8	0.4	0.1	0.3	6.0	0.2	0.1	0.2	1.6	0.1
[8]	Dededo	Red laterite	250	15.6	14.5	3.5	10.1	7.8	1.2	0.4	35.9	44.8	3.6	0.4	2.7	3.8	1.7
[9]	Dededo	Red laterite	540	12.4	11.8	2.8	8.4	6.4	0.9	0.4	40.0	34.1	4.2	0.5	5.2	8.5	1.2
[10]	Dededo	Limestone	-10	0.1	0.1	0.0	0.1	0.0	0.0	0.0	7.3	0.3	0.1	0.0	0.1	0.0	0.0
[11]	Tiyan	Soil (airport)	160	6.1	6.2	1.4	4.3	3.5	0.5	0.2	32.6	70.9	2.3	0.7	2.7	6.2	0.8
[12]	Viti	Soil (power sta)	100	0.6	0.7	0.2	0.5	0.5	0.1	0.1	6.5	11.8	0.3	0.0	0.3	1.3	0.1
[14]	Cocus Is	Coral/shellfish	3	0.0	0.0	0.0	0.0	0.0	0.0	0.1	4.6	1.9	0.1	0.0	0.6	0.1	0.1
[15]	Umatac	Coral/shellfish	0	0.1	0.0	0.0	0.0	0.0	0.0	0.1	5.0	0.4	0.1	0.0	0.5	0	0.2
[16]	Agat	Coral/shellfish	157	0.3	0.3	0.1	0.2	0.2	0.0	0.1	5.0	0.5	0.1	0.0	1.0	0.1	0

MS = magnetic susceptibility in uCGS.

Mineral measurements; columns A1 to MnO in %/remainder in ppm.

Reference = Ref number of sampling location depicted on Map 1.

Magnetic susceptibility of soils (see Map 2)

The results of the magnetic susceptibility readings of the topsoils sampled across the North and South of Guam showed a very clearly defined differential; where, according to the reference ranges for various soils [33–36], readings were consistently elevated (averaging 1078 uCGS) in the disease cluster area of Southern Guam, and consistently fivefold lower (averaging 207 uCGS) around the disease-free Dededo region in the North of Guam. These results clearly demonstrate that magnetic susceptibility levels are at the top of the 'excessive' scale in the topsoils supporting the cluster area in the South of Guam, suggesting that this phenomena might represent a pathogenic relevance to Guam syndrome.

The elevated levels of Fe, Mn, Ti, Ni based compounds in these volcanic soils are probably responsible for causing the enhanced levels of magnetic susceptibility [33,34] in the south of the island, whilst other factors such as the propensity for an intensity of natural bush fires, the proximity to the coal fired power plant, WW2 napalm bomb assaults and thunder/lightning storms [33,34, p. 292/499,36] in this part of the island could have been contributory; since these phenomena are known induce thermal or shock induced remnant magnetism; by metamorphosing certain paramagnetically ordered metals into ferrimagnetically ordered species [34,37].

A hypothesis for the pathogenesis of Guam syndrome

A multifactorial aetiological hypothesis is proposed that pivots upon the combined exposure of the Chamorro population to high levels of either non-radioactive or radioactive sources of ferrimagnetic metal compounds – incorporating Al, Fe, Mn, Sr, Ba that results from exposure to yams/seafood in the diet or WW2/test munitions, etc. – and to low levels of Mg/Ca in all of the South Pacific locations where these clusters of neurodegenerative disease have simultaneously erupted.

Once the rogue metal replacement of vacant Mg/Ca domains has occurred then the invasive metal nucleators seed a proliferation of single domain ferrimagnetic crystal arrays whose magnetic field emissions initiate a chain reaction of free radical mediated degeneration of various tissues throughout the biosystem. Long term pathogenic damage is largely confined to the CNS, since the nervous

Table 2 Analyses of vegetation/fruit collected in disease/disease-free regions of Guam (elements in ppm)

	Sample	Tissue	Ca	Na	Mg	K	Fe	Al	B	Ba	Cd	Co	Cr
Cluster	Umatac	Pasture	7051	465	2955	19339	76	110	14	29	0.2	0.1	0.3
Cluster	Merizo	Pasture	9859	641	4629	11958	333	260	22	69	0.1	0.5	1.5
Dis-free	Viti	Pasture	8997	300	2393	9751	33	13	16	6	0.0	0.1	0.1
Dis-free	Agat	Pasture	5124	527	3246	17216	43	71	11	18	0.1	0.2	0.2
Dis-free	Dededo	Pasture	28749	3222	4944	7044	30	81	21	1	0.3	0.1	0.3
Cluster	Umatac	Yams	5763	6483	4647	18288	809	940	15	1	0.1	1.0	1.8
Cluster	Umatac	Yams	10474	3610	2914	9558	406	485	14	1	0.0	0.5	1.1
Dis-free	Dededo	Yams	341	248	672	14489	98	166	4	2	0.0	0.1	0.3
Cluster	Umatac	Paprika	9311	289	2965	38180	35	4	22	1	0.0	0.0	0.1
Cluster	Umatac	Cycad	449	221	684	7395	9	1	7	1	0.0	0.0	0.1
Cluster	Umatac	Betel	681	204	581	1571	215	186	2	1	0.0	0.3	1.1
Cluster	Umatac	Taro	1141	180	1027	10852	131	96	4	0	0.0	0.2	0.4
Cluster	Umatac	Tapioca	2867	458	1032	11799	133	118	4	1	0.0	0.2	0.2
Nuclear	Cocus Is	Pasture	8906	7910	4089	16112	12	3	18	1	0.0	0.0	0.2
Decontamination	Cocus Is	Pine	6265	1413	1348	5766	14	2	28	0	0.0	0.0	0.0
	Sample	Tissue	Cu	Li	Mn	Ni	Pb	Sr	Ti	V	Y	Zn	
Cluster	Umatac	Pasture	4.7	0.2	186.8	3.3	0.8	27.9	1.1	0.8	1.0	16.4	
Cluster	Merizo	Pasture	3.8	4.6	59.3	5.4	0.9	44.2	6.6	1.5	0.4	15.8	
Dis-free	Viti	Pasture	4.8	0.4	21.7	0.5	0.9	49.8	0.4	0.9	0.1	25.8	
Dis-free	Agat	Pasture	3.9	0.2	85.5	1.1	0.5	23.3	0.7	1.0	0.8	15.4	
Dis-free	Dededo	Pasture	6.3	0.1	115.0	1.2	1.6	26.6	0.7	1.4	0.3	38.6	
Cluster	Umatac	Yams	10.8	0.2	22.3	5.1	0.8	11.7	11.0	3.3	0.5	32.2	
Cluster	Umatac	Yams	7.8	0.0	20.4	2.8	1.5	25.9	8.5	1.5	0.2	41.0	
Dis-free	Dededo	Yams	8.1	0.1	11.6	0.6	0.1	2.8	1.3	0.5	0.1	10.3	
Cluster	Umatac	Paprika	12.9	0.1	25.6	3.9	1.0	18.8	0.1	1.0	0.0	25.7	
Cluster	Umatac	Cycad	1.4	0.3	6.4	0.3	0.1	2.1	0.1	0.3	0.1	6.1	
Cluster	Umatac	Betel	6.0	0.0	10.7	1.6	0.1	2.3	4.4	0.7	0.1	4.8	
Cluster	Umatac	Taro	5.1	0.2	8.0	0.6	0.2	3.3	1.3	0.5	0.0	48.9	
Cluster	Umatac	Tapioca	2.4	0.1	10.7	0.5	0.4	7.7	2.4	0.9	0.1	13.0	
Nuclear	Cocus Is	Pasture	4.2	0.1	6.1	0.4	0.9	91.4	0.1	1.2	0.0	32.5	
Decontamination	Cocus Is	Pine	3.7	0.1	4.2	0.6	0.6	11.2	0.4	0.5	0.0	17.9	

tissues are particularly sensitive to free radical assaults and are largely unable to regenerate after being subjected to oxidative insults [24].

Potential environmental sources of the rogue pathogenic metal nucleators

The traditional foodchain

The results of the vegetation analyses carried out in this study have demonstrated 2x lower levels of Mg in the vegetation of the south of Guam,

whilst the levels of Fe, Al, etc., are significantly more elevated in the pasture grasses of South in relation to the vegetation samples collected in the North – probably due to the acidic nature [14,28] of the volcanic soils in Southern Guam in respect of the alkaline limestone soils of the North.

Despite the evidence for various artificial sources of metal contamination on Guam – e.g., the power plant at Viti, the combustion of aeroplane fuel around the airports and the dumping of munitions in the south central volcanic highlands of Guam (see Map 1) – it is difficult to envisage how the eco-dynamics of the metal emissions from any of these sources in the Northern/central parts

Table 3 Analyses of drinking and drainage waters in the disease and disease-free areas of Guam

	Location	Source	Ca (ppm)	Na (ppm)	Mg (ppm)	K (ppm)	Fe (ppm)	Al (ppm)	B (ppm)	Ba (ppm)	Cd (ppm)	Co (ppm)	Cr (ppm)	Cu (ppm)	Mn (ppm)	Ni (ppm)	Pb (ppm)	Sr (ppm)
Disease free	Agat	Stream	55.9	20.6	13.7	2.7	0.33	0.07	0.03	0.00	0.00	0.00	0.00	0.00	0.06	0.01	0.01	0.24
	Tiyat	Spring (D)	85.8	63.3	13.1	3.5	2.54	0.89	0.05	0.00	0.00	0.00	0.00	0.23	0.07	0.01	0.01	0.22
	Dededo	Tap (D)	81.5	66.8	11.2	3.3	0.17	0.04	0.05	0.00	0.00	0.00	0.00	0.01	0.01	0.01	0.01	0.13
Cluster zone	Umatac	Spring (D)	1.5	78.2	0.1	2.0	0.01	0.03	0.04	0.00	0.00	0.00	0.00	0.00	0.00	0.00	-0.01	0.01
	Umatac	River	36.6	23.9	9.1	4.9	0.34	0.22	0.02	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.00	0.17
	Umatac	Stream	14.4	14.7	7.7	3.8	0.23	0.21	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.06
	Umatac	Tap (D)	14.4	10.1	4.8	2.5	0.02	0.02	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.05
Merizo	River	42.7	26.4	53.0	6.5	53.09	35.03	0.02	0.04	0.01	0.05	0.05	0.15	0.07	1.60	0.17	0.02	0.15
	Cocus	Sea	291.0	10556	765.6	381.3	0.19	0.24	3.62	0.00	0.00	0.00	0.04	0.00	0.04	0.02	0.03	5.99
	Location	Source	Ti (ppm)	V (ppm)	Y (ppm)	Zn (ppm)	U (ppb)	Th (ppb)	Rb (ppb)	Cs (ppb)	Ag (ppb)	As (ppb)	Cd (ppb)	Tl (ppb)	Mo (ppb)	Sb (ppb)	Bi (ppb)	Sn (ppb)
Disease free	Agat	Stream	0.01	0.01	0.00	0.03	2.5	0.6	19.3	0.2	0.3	5.4	0.5	1.1	2.3	4.6	1.6	2.2
	Tiyat	Spring (D)	0.02	0.01	0.00	0.06	9.2	1	16.8	0.8	2.7	6.3	0.4	0.4	1.1	2.3	0.6	6.2
	Dededo	Tap (D)	0.00	0.00	0.00	0.03	5	0.7	15.2	0.9	0.2	3.9	0.2	0.1	1.8	3.2	0.3	2.3
Cluster zone	Umatac	Spring (D)	0.00	0.01	0.00	0.00	0.9	0.3	4.9	0.1	0.2	13.9	0.2	0.2	2.4	0.8	0.3	1.3
	Umatac	River	0.01	0.03	0.00	0.01	1.4	3.2	11.7	0.1	0.1	5.7	0.2	0.2	0.9	1	0.3	1.8
	Umatac	Stream	0.00	0.02	0.00	0.00	0.1	0.1	7.9	0.1	0.1	0.3	0.2	0.1	0.6	0.8	0.2	0.8
	Umatac	Tap (D)	0.01	0.00	0.00	0.17	0.2	0.1	13.1	0.1	1.1	0.3	0.7	0.1	0.7	0.7	0.3	1.9
Merizo	River	0.46	0.19	0.01	0.12	3.6	4	59.5	0.4	0.5	20.2	2.8	0.7	0.9	0.8	0.6	1.8	
	Cocus	Sea	0.00	0.27	0.00	0.08	20.7	0.5	569	2	0.5	0.4	0.4	0.2	71.3	5.3	1	27.9

(D) = drinking water.

of the island can be correlated with the spatio-temporal epidemiology of Guam syndrome concentrated in the south. In this respect, it seems that the elevated levels of Al, Mn, Fe, Sr, Ba recorded in both the soils and seabed of the cluster area, partially stem from the natural weathering of the red laterite and other volcanic derived soils in Southern Guam [10].

Whilst the majority of the traditional foods demonstrated low concentrations of these rogue metal cations, the analyses of the yam root crops demonstrated sufficiently bioconcentrated levels of ferrimagnetic metals to pose a toxicological risk to those who regularly consume the yams as well as the ferrel pigs which, in turn, consume the yams.

A similar scenario of metal bioconcentration – involving excess Mn – was also recorded by the author in the yams that had been grown on the islands of Groote (NE Australia) and the Azores [38] – locations where the respective indigenous people also consume yams as a staple food, and are also blighted with an excessive incidence rate of neurodegenerative disorders; especially the spinocerebellar ataxic condition, Machado Joseph Disease [39,40].

Since molluscs and other marine species are renowned for their ability to accumulate an intensity of ferrimagnetic metal crystals in their tissues, a more broad ranging analyses of the Chamorro's seafood diet (that thrives in a volcanic seabed ecology) [10] needs to be executed in the future.

World war two explosives

In the south west of Guam, the local consensus held by the Chamorro elders maintains that Guam syndrome resulted from early life exposure to some toxic substance that was released from the intensive number of bombs dropped into the waters during the World war 2 (WW2) US military invasion/liberation of Guam in July 1944 [41]. They maintain that their marine foodchain was contaminated as a result, thereby 'seeding' the disease.

This observation could prove to be entirely plausible, since the South Pacific clusters of neurodegenerative disease had all simultaneously emerged in the coastal districts of Western New Guinea, Kii Peninsula (S Japan) and Rota/Guam islands [1–4] – the main districts that were simultaneously under intensive bombardment with Ba, Sr and other metal based explosives [31] during this brief period of WW2 [42]. In fact, these military assaults can be connected back to the bombing sorties executed from one single aircraft carrier of

the US Navy – the USS Hornet (CV-12) – that carried out intensive bombardments on Guam and Rota islands on the 12th June 1944, and many further raids along the south coasts of Japan and New Guinea during June/July 1944 [42]. In this respect the neurotoxic agent could somehow be associated with a specific class of bomb that was despatched from this single warship. A likely candidate could entail the pioneer use of the Al based napalm bomb that was first used to 'burn off' the jungle during the WW2 invasion of these specific islands. In this respect, the South Pacific clusters could all commonly represent a delayed neurotoxic 'footprint' of the bombing sorties that had been executed with Ba and/or Al based explosives across the Pacific regions during WW2.

Furthermore, the simultaneous eruption of another cluster of neurodegenerative disease in the Pacific involved the Kuru form of TSE (similar to variant CJD (vCJD)). This was first reported in the 1950s by a mining prospector who had observed many cases of this unusual disease amongst the 'Fore' tribe who inhabited a small district in the highlands of New Guinea [43]. After studying the bell shaped curve of the kuru-like vCJD epidemic in the UK – where a five year period existed between the onset and peak of the epidemic cases [44] – It can be guesstimated that the onset of the kuru epidemic was initiated sometime during the late 1940s. In this respect, it is proposed that the first cases of kuru emerged *subsequent* to an unique local toxic event involving the accidental explosion of several bombs which occurred whilst the Fore tribe were looting several 'downed' US/Japanese WW2 bombers that had crash landed in their remote highland territories during the 1940s [43]. Furthermore, the Fore folk had also scavenged the Al/Mn alloy sheeting of the fuselages and crude uranium ballasting from the downed aeroplanes, subsequently moulding the metal into tools and cooking pans.

Whilst Kuru has been putatively associated with the cannibalistic ingestion of a hitherto unidentified TSE 'infectious' agent in human brain [43], this hypothesis fails to explain the exclusive emergence of this epidemic within a select focal geographic location, since cannibalism was traditionally practised across the whole of New Guinea. However, it is possible that the long term sustainability of the Kuru epidemic amongst the Fore [43] could be explained by the fact that a toxic metal – originating from the unique arrival of these crashed bomber aircraft into their local environment – had intoxicated the Fore; either due to direct exposure, or indirectly through the bioconcentration of the metals via their cannibalistic consumption of the rogue metal contaminated brains of their colleagues.

The presence of the CJD class of TSE neurodegenerative disease is also evident upon the isle of Guam. One such case involved the former Senator of Guam who had purportedly lived off vegetables and fruit grown on soils that had been contaminated by the dumping of waste munitions after WW2 (personal communication; Robert Celestial, Guam).

Cocus island; a former radioactive decontamination naval base

In respect of the neurodegenerative cluster in the South of Guam, another interesting spatio-temporal correlation can be drawn between the radioactive decontamination of naval vessels in the Cocus and Agat naval bases during the 'Operation Crossroad' period of the late 1940s to the early 1960s [29,30] (see Map 1). These boats had been involved in the atomic bomb test detonations off Enewetak atoll and were returned to Guam for acid washing and sandblasting – the prescribed clean up technique which was carried out by the US navy in these coral lagoon harbours [29,30]. The radioactive metal pollutants would have been discharged directly into the seawater, so it was very likely that the marine foodchain had become contaminated with radionuclide metals that were discharged from the decks of the boats involved in the atomic weapon detonations – such as Sr 90, cesium 136 which could have successfully competed for bonding to any Ca, Mg, Na, K binding domains in the marine ecosystem [45]. One study offers some evidence of radionuclide contamination of the sea water off the western shores of Guam during this period [46].

Cocus island is an elongated coral atoll island located two miles off the coast of Guam, and it is interesting that the levels of Sr in both the vegetation and soils (91.4 and 4151 ppm) constituted the highest levels recorded in the Guam study. Further testing is currently in progress which will ascertain whether the Sr measurements in the coral/shellfish samples represent Sr in its radionuclide Sr 90/89 isotope or in its non-radioactive Sr 88 form [32].

Given the central location of Cocus lagoon to the three cluster villages (see Map 1), as well as the dependence of the local Chamorro population upon seafood that was sourced from these radioactive polluted waters, then it is plausible to suggest that the community affected with Guam syndrome would have been exposed to significant chronic doses of radionuclides during the 1940–1960 period, and for a considerable portion of the 'half life' decay period after that.

However, the collection and consumption of molluscs and shellfish from around the coastline

of Guam has been prohibited since the 1980s. These measures would have unwittingly helped to stem any problem of radioactive contamination that may have existed, thereby curtailing the incidence rate of Guam syndrome in future years.

More disturbingly, the coral from the local reefs used to be heated over fires on the beaches and then ground into a powder for mixing with the betel nut and papula leaf. This concoction was habitually chewed by the Chamorro people for its stimulatory effects. Since the group 2 of alkali earth metals generally share similar ligand binding affinities as a group, the potential capacity of Sr 89/90 to bind into the Ca domains in the coral matrix [45], suggests that the ingestion of any Sr 90 contaminated coral could have presented a major route of radioactive contamination for the local Chamorro population.

Eco-factors in Southern Guam that could increase permeability of the blood brain barrier, thereby enhancing uptake of toxic metal-transport protein complexes into the CNS

Several long term natural/artificial sources of low frequency noise were observed in the cluster area [47]; such as low flying military jets, intensive detonations of bombs during WW2 (as well as waste military ordnance in the southern central volcanic mountains of Guam), a prevalence of electric storms as well as earthquake eruptions that had reached 8 on the richter scale. A tectonic fault line also passes beneath Umatac village.

Whilst some of these sonic stress phenomena are also present in the North of the island – where the airbases are actually located – the impact of these sonic shock blasts are more intensive in the south, since military jets tend to practise at faster sub-sonic, or even supersonic speeds at greater as opposed to closer distances from the runways. The chronic exposure of the Chamorro people to these sonic stress phenomena [19], combined with the prolonged trauma of being held captive prisoner under the Japanese military occupation, could have served to increase the permeability of the blood brain barrier [16], thereby permitting an increased entry of the locally elevated levels of ferri-magnetic metals into their CNS [23].

The presence of a NASA radar tracking station in the mountains behind Merizo, as well as a wireless station near to Agat, represents other unique features of the cluster environment which could

potentially generate electromagnetic radiations that have been *controversially* associated with increasing the permeability of the blood brain barrier [15,20–22].

Rogue metal replacements induce a progressive ferrimagnetisation of the biosystem. Various possibilities for a non-radioactive or radioactive metal induced mechanism of pathogenesis are discussed

Once gut/blood brain barrier permeability is impaired, any increased uptake of Al, Fe, Sr, Ba, or Mn into the Mg/Ca depleted biosystem can lead to a substitution, or antagonism of the Mg/Ca depleted binding domains [48–51] on various enzyme, transporter protein or proteoglycan groups [52,53] in the brain and bone matrix. In this respect, the competitive binding and/or antagonistic properties of Al, Mn, Fe, Ba and Sr with already depleted Mg/Ca cations can lead to a disruption in the organisation of membrane protein networks and their interaction with glycoproteins, as well as the stabilisation of the structural changes involved in the cell–cell organisation [51]. More specifically, the Mg/Ca dependent sulphated proteoglycan mediated control of the Fibroblast growth factor receptor complex [54] would be disrupted, severing the crucial cell–cell signalling pathways that maintain the structural integrity of the neuronal networks. Furthermore, the body's S-proteoglycan mediated endogenous protection against crystal deposition would be disrupted.

The resulting depression in the activity of these enzyme groups – in combination with the progressive 'ferrimagnetization' of the CNS and skeletal matrix with an overload of 'non-radioactive' or 'radioactive' ferrimagnetically ordered metal micro crystal nucleators – may help to explain the basis for the resulting free radical mediated pathogenesis [24], involving mutations [55,56] and loss of cell–cell signalling [57], which have already been identified as key players in the pathogenesis of these neurodegenerative-cum-osteoporotic conditions.

Mg/Ca deficiency prerequisite

Environmental Mg/Ca deficiencies appear to represent a common pathogenic denominator in many of the neurodegenerative disease clusters that have erupted in the more traditional subsistent communities around the world [4,8,9,58,40].

The environmental analyses of this study confirmed the findings of other teams that the traditional foodchain of the Chamorro folk is deficient in Mg/Ca [8,9]. This problem was further exacerbated by the customary practise of adding large amounts of salt to their meals – since high dietary intakes of sodium disrupt the uptake of Mg across the gut wall [59]. Once Mg/Ca is deficient in the biosystem, then rogue metals such as Mn, Sr or Ba are able to substitute at the vacant Mg/Ca sites on enzymes/transporter proteins/proteoglycans (in the CNS/skeletal matrix, etc.) since the outer shells of these metal atoms possess a similar electronic arrangement/ionic radius to Mg/Ca cations [51], thereby enabling a successful ligation to Mg/Ca binding domains, which could disrupt or modify the function of these enzymes.

The free radicals generated by the magnetic fields [24,60–63] or radioactive decay [24,32] emitted by any rogue metal nanoparticles that manage to permeate the CNS and proliferate into single domain crystal arrays could initiate the mutations [24,32] that have been identified in these neurodegenerative diseases; for example, the specific mutations in the enzymes that mediate the metabolism of cholesterol/lipids [55] the guanosine triphosphate cell signalling [56] – enzyme systems that are normally Mg regulated in the healthy mammal [51].

The high prevalence of other disorders that are indicative of Mg/Ca deficiency on Guam, such as osteoporosis, diabetes and cardiac arrhythmias [64,65], may also relate to the Mg deficiency prerequisite. For instance, an Sr replacement of Ca/Mg domains in the bone matrix of the local Chamorro population could explain the high prevalence of osteoporosis that co-exists amongst the population affected by Guam syndrome [2,13].

Furthermore, various conditions stemming from the disordered metabolism of insulin (that can result from deficiencies in the activities of the Mg-ATP activated pyruvate or lipoamide dehydrogenase enzyme groups [66]) have been observed as both a separate disease entity or as an additional complicating factor in those suffering from the early stages of Guam syndrome.

Glutamine synthetase is another Mg activated enzyme that can be replaced by Ba, Mn and other metal species [49]. Once its activity is depressed during contexts of Mg deficiency, then the highly neurotoxic glutamate molecule can accumulate in the brain [67]. Elevated levels of glutamate have been associated with the common classes of neurodegenerative diseases [68].

This observation of low Ca/Mg also provides a part explanation for the disturbance of the

sulphated proteoglycan-FGF co-receptors in these types of neurodegenerative disease. These receptors demonstrate a co-requirement for both Ca/Mg cations and the proteoglycan, if viable binding of FGF to its tyrosine kinase receptor is to succeed [54].

Sr and Ba replacement as a potential prerequisite in the pathogenesis of Guam syndrome

Contamination of the Ca/Mg depleted biosystem with Sr or Ba ions resulting from chronic exposure to natural or military munition sources could lead to a Ba/Sr substitution of Ca domains in a diverse array of metabolic contexts – for instance in molecules like calmodulin [28] which activates the protein kinases. Ba replacement will cause the Ca channels to ‘scramble’ into a state of rapid fire, inducing an ‘overdrive’ of the cholinergic and monoaminergic neuronal systems [31,67]. This represents one avenue through which this mode of metal poisoning will overburden the biosystem with oxidant mediated free radical chain reactions, which accelerate the aging process and ultimately propel the victim into a state of early senescence.

Ba competitively blocks passive cellular potassium ion efflux which results in intracellular potassium accumulation and extracellular hyperkalemia disturbance [67]. The effects of Ba on the sodium–potassium ion pump, as well as the Ca channels, accounts for the membrane depolarisation that has been shown to evoke the release of glutamate which has been observed during contexts of Ba intoxication [69]. It also needs to be considered how the build up of neurotoxic glutamate could result from the Ba replacement that can occur on the Mg binding domains of glutamine synthetase [49].

The soluble, reactive Ba/Sr ions are well recognised to conjugate with sulphates, thereby starving the nervous system of one of its most crucial structural caretakers – the sulphated proteoglycan group of molecules [51,54,66]. Once deprived of their sulphate co-partner, the synthesis of a metabolically viable proteoglycan molecule is impaired [54], whereby the proteoglycans will cease to perform their crucial function in the cell–cell signalling pathways that regulate the growth and maintenance of the complex infrastructure of neuronal networking [54]. Neurodegenerative wasting ensues.

Once the turn over of the syndecan, perlecan and glypican types of sulfated proteoglycan are

depressed within the biosystem, then the proteoglycan-dependent fibroblast growth factor-2 (FGF) signalling system is disrupted, causing an overall collapse in the proliferation and migration of the oligodendrocyte/astrocyte type 2 progenitor cells that are essential precursors of both developing and mature oligodendrocyte/astrocyte glial cells [70,71].

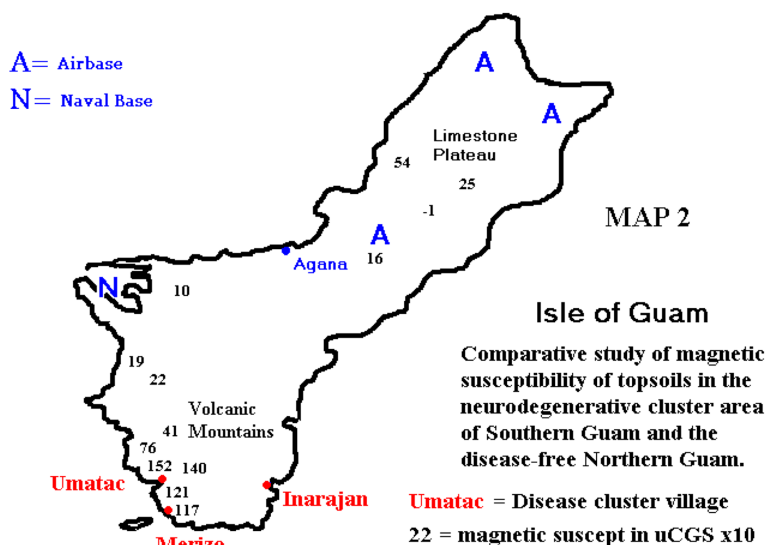
Neurodegeneration of the all important glial cell formations represent a key pathological feature of many types of neurodegenerative condition; such as AD, ALS, PD, MS and TSEs; perhaps explaining why these particular neurodegenerative diseases have a tendency to co-cluster in animal/human populations who are residing within these high Ba/Sr ecosystems [72].

A Ba/Sr induced depletion of endogenous S-proteoglycan turnover would also assist in achieving a similar style block in 5HT binding to a raft of 5HT receptor types; a pathogenic disruption that can also lead to a blockage in cell–cell communication with the adhesion molecules [54,72]. Furthermore, Loss of cell surface proteoglycan binding to superoxide dismutase would result in the proliferation of superoxide generated oxidative stress [72]. There is evidence for both disturbances in 5HT uptake and increased superoxide generated oxidative stress in the pathogenesis of many of these neurodegenerative diseases [24,73–76].

Interestingly, loss of activity of various sulphated proteoglycan molecules has been shown to be integrally associated with some of the key pathogenic mechanisms underpinning the causes of AD, PD, MS, ALS, TSEs, etc. [54,77]. Furthermore, in the context of this theory, the role of S-proteoglycans in protecting the organism against crystal proliferation plus the role of Sr/Ba ions in promoting mineralisation, may also be critical.

Chronic contamination of the CNS with ferrimagnetically ordered metal nucleators generate magnetic fields that initiate a free radical mediated progressive pathogenesis of neurodegeneration (see Fig. 1)

There is an extensive literature indicating that clusters of the various neurodegenerative diseases are located in environments that are intensively polluted by emissions from volcanoes, metal processing plants, steel mills, foundries, power plants, welding related industries [78]. Interestingly, the airborne emissions from these specific sources contain high levels of ferrimagnetic metal particulates [33].



Map 2

The fivefold increase in magnetic susceptibility of the volcanic soils that sustain the cluster area in Southern Guam – in relation to the disease-free North of Guam (see Map 2) – suggests that the uptake of ferroelectric and/or ferrimagnetically ordered metal nanoparticles from these soils into the biosystem could represent one of the key primary prerequisites that underpins the pathogenesis of Guam syndrome. In this respect, the central pathogenic mechanism could entail the progressive substitution of the diamagnetic/paramagnetic ordered metal centres in the CNS with an overload of rogue ferrimagnetic metal nucleators, which subsequently seed the multireplication of a myriad of metal-protein crystal arrays.

Since ferrimagnetic metal crystals interact over a million times more strongly with low level ‘earth-strength’ magnetic fields than diamagnetic/paramagnetic metal crystals [79], then the survival of the ferrimagnetically contaminated individual will be considerably compromised in respect of their capacity to neutralise the emissions of low level magnetic fields from their external and internal environments.

Many of these ferrimagnetic nanoparticles would initially be absorbed into the central nerves as single domain microcrystals [63], etc., that could subsequently proliferate into substantial metal-protein crystal arrays once they are lodged in the tissues. But this proliferating process can only proceed once the body’s protection against crystal deposition [83] – e.g., the ‘crystal inhibiting’ S-proteoglycan molecules[84] – has been inactivated following exposure to metals that bond to sulphates (Fig. 1).

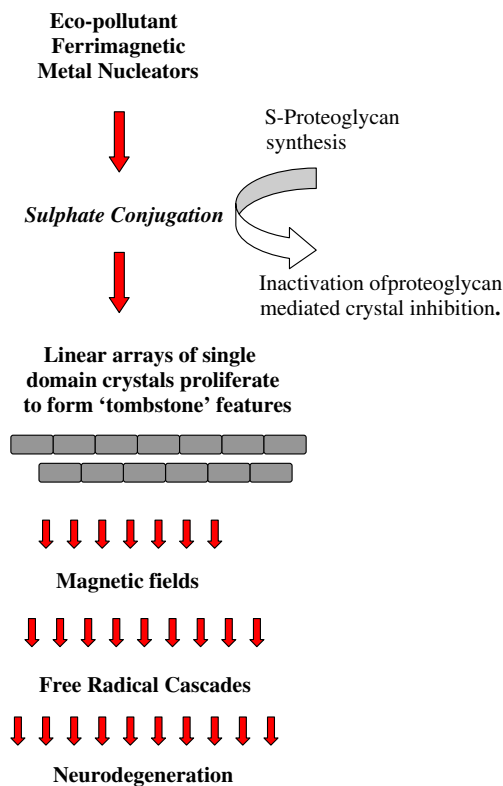


Figure 1 The ‘stack up’ of ferrimagnetic momentum; rogue metal nucleated crystal arrays induce magnetic fields that initiate a free radical mediated pathogenesis of neurodegeneration.

Each crystal splits to form elongated arrays of crystals, that generally align themselves into needle-like ‘nose to tail’ linear chains of single domain crystals [63,80], that are positioned in a parallel

orientation due to the magnetic moments already captured within each crystal domain [63]. The resulting needle like structures are aptly referred to as 'biological bar magnets'.

Interestingly, it is these small single domain types of crystal that have the potential to carry the highest amount of magnetic charge [63]. This type of crystal – involving magnetite, maghemite, greigite [79] – represents the kind of crystals that are invariably identified in the biological systems of bees, dolphins, bacteria, sharks, molluscs, arthropods, birds as well as the mammalian brain [63,80–82]. Furthermore, over 100 million ferrimagnetic crystals have been identified per gram of dura/pia matter drawn from the Alzheimer diseased and normal human brain [79].

These rogue ferrimagnetic metals could serve as the nucleating agents that have initiated the various aberrant amyloid/apatite crystalline deposits which researchers have consistently observed in neuropathological and osteopathological sections drawn from the victims of those who have died of these neurodegenerative diseases [13,85,86]. Furthermore, these ferrimagnetic crystals could constitute the basic chemical matrix of the heat resistant amyloid plaques, bunina bodies, fibrils, neurofibrillary tangles and other abnormal 'tombstone' structures that represent the idiosyncratic neuropathological hallmarks which serve to differentiate between the various classes of neurodegenerative disease [2]. The particular type of tombstone that develops is determined by a combination of the types of metal 'nucleator' and target protein that are involved.

The metal nucleated crystals steadily proliferate into arrays of single domain 'ferrimagnets'. Despite the ephemeral loss of magnetic moment that is drained at a time when energy is required for the formation of the new domain walls in the growth of crystal arrays [63] (the period that represents the protracted "incubation" stage of these diseases), the overall strength of the magnetic moments that are captured within the millions of single domains that make up the resultant arrays of pathogenic super-crystals is going to be considerably stronger than the total moments present in the single domain grains found in the healthy human.

The magnetic fields that are generated on the surfaces of the rogue super crystals will initiate free radical chain reactions that evoke deleterious oxidative pathogenic assaults [61,62] on surrounding membranes and proteins (e.g., around the magnetosomes [80] and circadian associated circuitry). The classic oxidant induced neuropathological profile results; one that has been consistently observed in the various classes of neurodegenerative disease [24] (Fig. 1).

In this respect, the intensity of ferrimagnetic crystal contamination within the brain is proportional to the degree of free radical damage that is generated as a result. The actual risk that this presents to the contaminated individual in respect of the actual development of neurological disease is further determined by the individual's genetic capacity to mount an adequate antioxidant defence.

Numerous studies have indicated that very subtle increases in the strength of magnetic field emissions within the biosystem will increase the generation of free radicals [60–62,87], which subsequently unleash increased chemical cascades that can result in severe pathogenic consequences [63]. Interestingly, major aberrations have been observed in the waggle dance/spatial orientation of the bee [80,88] as a result of subtle increases or decreases in the external magnetic field. Likewise, geographic orientation of migrating birds/homing pigeons has been seriously disrupted by alterations of as little as 0.1% in the external magnetic field [82]. Furthermore, it is interesting that the early clinical signs observed in AD, PD, CJD, etc., involve bouts of severe spatial and temporal disorientation. The actual amount of ferrimagnetic crystals lodged within the organism could also play a critical role here [82].

The fact that the mammalian nervous system has been shown to respond to subtle magnetic stimuli [80,89] further expands the emerging concept of a delicate metabolic interaction that exists between external magnetic fields, the overall quantity of ferrimagnetic crystals within the CNS and their combined capacity to generate internal magnetic fields that initiate free radical cascades. The metabolic association between the magnetic and nervous systems of the mammal strengthens the idea that a subtle upset in the homeostasis of any of the factors involved can unleash severe neurological repercussions.

Interestingly, the author has previously proposed that the metal microcrystals involved in nucleating the fibril 'tombstone' arrays of the TSE diseased brain are actually 'piezoelectric' in nature [90]; whereupon any incoming environmental electromagnetic or mechanical pressure wave energy (e.g., light, acoustic or tactile stimuli) that impacts the TSE diseased brain is transduced by the piezoelectric crystal pollutants into electrical energy; whereupon the 'super strength' magnetic fields that are generated around these crystals as a direct response to the acoustic challenges, etc., will initiate an intensive cascade of free radical mediated neurodegeneration; producing the 'fast track' clinical progression of disease as seen in TSEs. This concept might provide an explanation for the 'explosive' hypersensitive response of the BSE affected cow to the slightest degree of sonic

or tactile challenge encountered during the early clinical stages of the disease [91].

If a radioactive species of metal is involved in seeding these ferrimagnetic crystals – as may have been the case in the Guam cluster – then the production of the additional radicals generated by the radioactive decay would further accelerate the proliferation of oxidant mediated neurodegeneration [24], producing a fast track class of super-neurodegeneration.

Conclusions

Considering the diverse array of industrial, military, agricultural usage of these metals within the modern western environment, it is suggested that chronic, or acute-on-chronic early life exposure to these metals in the Ca/Mg depleted individual could represent a delayed neurotoxic mechanism that manifests as PD, ALS, AD, as well as the TSE types of neurodegenerative disease. Factors such as the individual genetic expression of metallo-transport proteins [51], the integrity of the blood brain barrier [23], the specific rogue metal nucleator and target proteins involved [74], all combine to determine which particular class of neurodegenerative wasting disease emerges at the end of the day.

Whilst treatment of at risk populations with Mg/Ca supplements (perhaps added to the water supplies) might offer the most straight forward means of preventing these neurodegenerative diseases, research also needs to be channelled into the development of therapeutic trials involving demagnetisation [34,35] ultrasonic crystal disintegration therapies or pharmaceutical compounds which inhibit the growth of crystals and/or serve as chelation treatments [67]; theoretically depleting the CNS of ferrimagnetically ordered metal nucleators and/or arresting any respective crystal formations. This line of research should run in tandem with a more detailed analyses of the ecosystems around the S Guam cluster, in order to ascertain which types of compound – silicate, sulphate, perovskite, oxide, titanate, etc. – are complexed with the ferrimagnetic metals, etc. And then to monitor the levels of those specific metal compounds in the clinical cases and post mortem victims of Guam syndrome.

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