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1: Med Hypotheses. 2004;63(5):793-809.

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Erratum in:
Med Hypotheses. 2005;65(6):1207.

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

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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 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.

PMID: 15488650 [PubMed - indexed for MEDLINE]

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