Chronic barium intoxication disrupts sulphated proteoglycan synthesis: a hypothesis for the origins of multiple sclerosis.

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High level contamination by natural and industrial sources of the alkali earth metal, barium (Ba) has been identified in the ecosystems/workplaces that are associated with high incidence clustering of multiple sclerosis (MS) and other neurodegenerative diseases such as the transmissible spongiform encephalopathies (TSEs) and amyotrophic lateral sclerosis (ALS). Analyses of ecosystems supporting the most renowned MS clusters in Saskatchewan, Sardinia, Massachusetts, Colorado, Guam, NE Scotland demonstrated consistently elevated levels of Ba in soils (mean: 1428 ppm) and vegetation (mean: 74 ppm) in relation to mean levels of 345 and 19 ppm recorded in MS-free regions adjoining. The high levels of Ba stemmed from local quarrying for Ba ores and/or use of Ba in paper/foundry/welding/textile/oil and gas well related industries, as well as from the use of Ba as an atmospheric aerosol spray for enhancing/refracting the signalling of radio/radar waves along military jet flight paths, missile test ranges, etc. It is proposed that chronic contamination of the biosystem with the reactive types of Ba salts can initiate the pathogenesis of MS; due to the conjugation of Ba with free sulphate, which subsequently deprives the endogenous sulphated proteoglycan molecules (heparan sulfates) of their sulphate co partner, thereby disrupting synthesis of S-proteoglycans and their crucial role in the fibroblast growth factor (FGF) signalling which induces oligodendrocyte progenitors to maintain the growth and structural integrity of the myelin sheath. Loss of S-proteoglycan activity explains other key facets of MS pathogenesis; such as the aggregation of platelets and the proliferation of superoxide generated oxidative stress. Ba intoxications disturb the sodium-potassium ion pump--another key feature of the MS profile. The co-clustering of various neurodegenerative diseases in these Ba-contaminated ecosystems suggests that the pathogenesis of all of these diseases could pivot upon a common disruption of the sulphated proteoglycan-growth factor mediated signalling systems. Individual genetics dictates which specific disease emerges at the end of the day.

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