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Review: Host selenium-deficient status influences evolution of some RNA viral infectious diseases

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Abstract

Antioxidant micronutrient selenium (Se) functions to help neutralize biologically induced oxidative stress. Others (Beck et al., Broome et al.) determined that Se-deficient individuals infected with benign strains of Coxsackievirus B3 (Keshan Disease), influenza A virus H3N2, or an oral live attenuated poliovirus used as vaccine developed impaired immunity against these RNA viruses; furthermore, each virus type rapidly mutated to nonstochastic, replicatable, long-lived**,** virulent forms in the Se-deficient host. These viral mutation rates under host Se-deficient conditions were orders of magnitude faster than RNA virus mutations in the ambient environment. Immunocompetence of the Se-deficient host improved with dietary-Se supplementation (for example, as blood plasma achieved >1.2 µMol Se in subjects vaccinated with polio vaccine), and the rapid RNA genomic mutations abated in the infected host. Nonetheless, virulent virus forms were transmissible and infectious, even to individuals with adequate Se-status.

Data from the literature, predominantly PubMed, identified human subsistence populations with blood-values of <0.4 µMol Se, where biogeochemical factors, including soil-Se concentrations, constrained Se availability from soils to foodcrops; subsistence diets there provided ~10 mcg Se/day, compared with the US RDI of 55 mcg Se/day. Se-poor regions, located in Asia and sub-Saharan Africa, were the geographic origins of some contemporary novel RNA viral infectious diseases (VIDs). Working from the literature, we tracked genomic changes from asymptomatic vectors to virulence in humans for IAV H2N2, H3N2 and H5N1 M1 and PB2 proteins, SARS-Coronavirus 'Spike' glucoprotein, HIV *nef* gene/protein, and the EBOV V35 protein in distinct Se-poor regions.

Biography

Michalann Harthill is an ecologist, retired from the US Geological Survey. She is the founder and president of GHI, Inc., established in 2008.