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Spiroplasma as a candidate causal agent of CJD is an infectious disease model of neuro degeneration

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reutzfeldt-Jakob disease in humans and other spongiform encephalopathies in animals including bovine spongiform rencephalopathy (Mad cow disease), scrapie in sheep, mink encephalopathy and chronic wasting disease (CWD) in cervids are all caused by a filterable transmissible agent. These transmissible spongiform encephalopathies (TSE) show buildup of miss-folded amyloid proteins in brain tissues (prion). Our studies show presence of spiroplasma, a wall-less bacterium, in all cases of TSE as shown by morphological and molecular studies. A novel spiroplasma has been grown out of brain and eye tissues from all cases of TSE examined into cell-free media. Inoculation of spiroplasma into rodents and ruminants induce clinical signs of neurodegeneration with spongiform changes in the brains. Experimental spiroplasmosis in deer show clinical signs remarkably similar to naturally occurring CWD. A breakthrough in understanding the role of spiroplasma in TSE is the proclivity of these bacteria to form biofilm on surfaces including mica and stainless steel. Spiroplasmain biofilm show similar resistant biologic properties as the TSE agent. The binding of spiroplasma to stainless steel and tendency of spiroplasma to infect corneal endothelia suggest the mechanism of iatrogenic transmissions of CJD via surgical instruments or corneal transplants. Spiroplasma in biofilm produce a functional amyloid (curli) which is known in other bacteria to miss-fold host proteins forming amyloids. Spiroplasma produces persistent infection in tissue cultures and has been shown to form alphasynuclein in vitro. Spiroplasma likely bind to clay in upper soil layers accounting for lateral transmission in ruminants. The experimental spiroplasmosis model inducing neurodegenerative disease may be important in deciphering an infectious cause for other neurodegenerative diseases such as Alzheimer's disease or Parkinson's disease.

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