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Alzheimer's neuroborreliosis with trans-synaptic spread of infection and neurofibrillary tangles derived from intraneuronal spirochetes.

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In the realm of dementia, it is astonishing to note that neurofibrillary tangles (NFT) are microscopically identical in a childhood illness (SSPE) and in a dementia of late adult life (Alzheimer's disease). The words "Alzheimer-type" NFT in peer reviewed scientific articles written by acknowledged experts underscore the striking similarities in "tangles" in two different diseases. Subacute Sclerosing Panencephalitis (SSPE) is caused by infection with atypical measles virus. Alzheimer's disease has no known cause. There is little controversy in suggesting that all of the Tangles in SSPE infected neurons are produced by slow viral type variant of Measles infection. But the mere suggestion that infection might be a cause of Alzheimer's disease confounds the establishment. If a good case is to be made for infection in Alzheimer's disease, an excellent nerve cell infection model is needed. Monkeys have provided a very reasonable model. Recently, a primate neuroborreliosis brain infection model demonstrated that *Borrelia* injected into the skin of monkeys resulted in the appearance of *Borrelia* transcriptomes in brain neurons. If *Borrelia* can travel from skin to brain in the monkey, then why not look at human Alzheimer's tissues to see if the DNA of *Borrelia* is present in the human brain? The molecular detection tools perfected in animal neuroborreliosis studies have been applied to human Alzheimer's disease brain tissues. Seven of ten cases of Alzheimer's disease from McLean Hospital Brain Bank of Harvard University yielded positive signals for infectious DNA in a small pilot study. Alzheimer's diseased neurons analyzed with DNA probes, produced little "dots" of positive staining. Granulovacuolar bodies in Alzheimer's diseased neurons (little dots in a bubble), are one of the expected microscopic profiles of Alzheimer's disease. "Little dots" inside nerve cells are also signatures of viral infectious agents inside of nerve cells. So with the assistance of the microscope and the tools of

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Frequency of Alzheimer's neurofibrillary tangle in the brains of progressive supranuclear palsy, postencephalitic parkinsonism, Alzheimer's disease, senile dementia and non-demented elderly. [Ann N Y Acad Sci. 1981]

Neurofibrillary tangle distribution in the cerebral cortex of parkinsonism-dementia cases from Guam: differences with Alzheimer. [Brain Res. 1985]

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molecular biology, a new model of infection emerges as a cause of "Alzheimer's-type" neurofibrillary tangles. Here I hypothesize that it is chronic infection of human neurons in Alzheimer's disease that produces neurofibrillary tangles by a pathway similar to the chronic SSPE infection tangle pathway. In addition, transmission of infection from nerve to nerve is proposed to explain the evolution of Alzheimer's disease. Herein is offered a new view for the origins and for the progression of diseased nerves with tangle formations in Alzheimer's disease based on infection.

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