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"Historic and Recent Evidence that Spirochetes Are Able to Reproduce the Clinical, Pathological and Biological Hallmarks of Alzheimer's Disease."

Dr. Miklossy, founder, Prevention Alzheimer International Foundation and director, International Alzheimer Research Center in Switzerland (CH) also practices memory and Lyme disease consultation in Vigimed Medical Center, CH. She is board certified in neurology, psychiatry and psychotherapy (Faculty of Medicine, University of Debrecen, Hungary) and in neuropathology (Swiss Society of Neuropathology and Swiss Medical Federation). She has received the degrees of Private docent (Dr habil or DSc) and Maître d'Enseignement et de Recherche (MER) in the University Hospital Center of Lausanne (CHUV), University of Lausanne. She was head of the Neurodegeneration research group for more than ten years in the University Institute of Pathology, Lausanne, CH.

She has done molecular biology research and participated in the introduction of Alzheimer's research in the Center of Neurovirology, Department of Neuroscience, Temple University, Philadelphia.. She headed the neuropathology of the Kinsmen Laboratory of Neurological Research, in The University of British Columbia, Vancouver, Canada. She is on the board of directors or scientific advisory board of several international organizations or foundations.

For more than 25 years she is actively involved in research on Alzheimer's disease and Lyme disease in the framework of international collaborations. Her presentations on international meetings and her publications were repeatedly considered for CME and press releases.

Our research interests include the pathogenesis of Alzheimer's disease (AD) and other neurodegenerative and chronic inflammatory disorders. From 1993 my research

focused on the role of bacteria, particularly of spirochetes, in persistent chronic infection, inflammation and amyloidogenesis in AD.

A century ago, Fischer (1907) has been suggested and Alois Alzheimer and his colleagues cited his view on the possibility that microorganisms might play a role in senile plaque formation. Additionally, there is an example in the history of medicine that chronic bacterial infection, namely chronic spirochetal infection (Treponema pallidum can cause slowly progressive dementia & reproduce the pathological and biological hallmarks of AD.

Increasing amount of recent data indicate, as we have suggested in 1993, that several types of spirochetes, including Borrelia burgdorferi and periodontal pathogen spirochetes are involved in the pathogenesis of AD. Recently, reviewing all data available in the literature a statistically strongly significant association, with a high risk factor was found between spirochetes and AD, fulfilling Hill’s criteria in favor of a causal relationship.

Exposure of human and mammalian primary CNS cells and organotypic cultures to spirochetes, showed that similarly to Treponema pallidum, Borrelia burgdorferi reproduces the pathological and biological hallmarks of AD (increased AβPP, Aβ and (p)tau levels).

Now from three decades we are involved in Lyme disease research. We have published the first pathological confirmation of the meningovascular form of chronic or late Lyme neuroborreliosis leading to cerebral vascular infarcts. Together with other authors we contributed to the pathological confirmation of the other major form of chronic Lyme neuroborreliosis, which is identical to the atrophic form of general paresis associated with slowly progressive dementia caused by Treponema pallidum in syphilis. We presented evidences on the direct involvement of Borrelia burgdorferi in the major tertiary forms of chronic Lyme neuroborreliosis. On invitation we contributed with a chapter on the pathology and biology of dementia in syphilis and Lyme disease in the prestigious Handbook of Clinical Neurology.

We have published observations on the presence of various pleomorphic forms, including the more resistant cystic, granular and L forms of Borrelia burgdorferi, in pure Borrelia cultures in infected cell cultures and in brains of demented patients with clinically, serologically and pathologically confirmed Lyme neuroborreliosis. We have also shown that Borrelia burgdorferi spirochetes

cultivated from thebrains of these patients are virulent and invade neuronal and glial cells and cause apoptosis. Recently, reviewing descriptions and illustrations available on the pathology of Lyme neuroborreliosis from the past 30 years, we reported that the major late or chronic forms of neurosyphilis were pathologically confirmed in Lyme disease as well and Borrelia burgdorferi was cultivated from tertiary lesions by various authors. These observations definitely indicate that chronic Lyme disease exists and Borrelia burgdorferi, similarly to Treponema pallidum plays a direct role in the pathogenesis of the tertiary manifestations of chronic/late Lyme disease.

Conference Lecture Summary

That pathogens suppress, subvert or evade host defences and establish chronic or latent infection had received little attention in the past. Various spirochetes, including *Treponema pallidum (T. pallidum)*, *Borrelia burgdorferi (B. burgdorferi)* and several periodontal pathogen spirochetes have the ability to escape host defences and establish chronic infection. Various spirochetes, in an analogous way to *Treponema pallidum*, are involved in the pathogenesis of several chronic disorders including cerebrovascular disorders and in slowly progressive cognitive decline with dementia.

T. pallidum, B. burgdorferi, and periodontal pathogen Treponemes (*T. denticola, T. pectinovorum, T. amylovorum, T. maltophilum, T. medium, T. socranskii*) persisting in the brain cause dementia and beta amyloid deposition. The two major tertiary forms of chronic neuroborreliosis, namely the meningovascular form with cerebral infarcts and cognitive decline resulting in dementia have been clinically and pathologically confirmed more that 20 years ago, indicating that *Borrelia burgdorferi* can cause chronic Lyme disease and chronic neuroborreliosis.

Spirochetes, including *Borrelia burgdorferi* are able to reproduce *in vitro* and *in vivo* the pathological and biological hallmarks defining AD dementia. A strong statistically significant association between spirochetes and AD fulfills Hill’s criteria and confirms a causal relationship between spirochetes and dementia. Validation of these observations by historic and recent reports further confirm that senile plaques are made up by spirochetes and correspond to biofilms. That host pathogen interactions in chronic spirochetal infection are identical to those

occurring in AD indicates that escaping host immune reactions, spirochetes, including *Borrelia burgdorferi,* sustain chronic infection and cause, in addition to cerebral infarcts, slowly progressive dementia associated with amyloid deposition in the brain. Association of co-infecting pathogens and formation of multi-bacterial biofilms further aggravate the degenerative process and the outcome of dementia. Importantly, these observations indicate that Alzheimer’s dementia can be prevented.