allied academies

13th World Congress on

Dementia and Alzheimer's Disease

September 16-17, 2019 | Paris, France

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Autoantibodies in Alzheimer´s disease

Alzheimer's disease (AD) is an age-related multifactorial progressive neurodegenerative disorder manifested by memory loss, spatial disorientation, and gradual deterioration of intellectual capacity. Its etiology is unknown. Pathological changes including synaptic and neuronal loss, oxidative damage, activated inflammatory cells and protein depositions such as extracellular amyloid plaques composed by misfolded amyloid beta (Aβ) peptide and intracellular neurofibrillary tangles comprised of hyperphosphorylated aggregates of the microtubule-associated protein Tau (τ) are observed.

Compromised blood brain barrier (often observed at AD) may permit increased contact of immune cells with components released from dying neuronal cells, as well as the transfer of various brain proteins into the blood and induction of autoimmune response against them. Immune system activation is frequently reported in patients with AD. Some autoantibodies are naturally occurring antibodies produced without extrinsic stimuli, originated from B1 cells and reacting with various components of neural system; however, antibodies derived after antigenic stimulation by "self" might be produced by B2 cells as well. Antibodies against Aβ, τ, neurofilament light and heavy chains, S100B, cholinergic, adrenergic and glutamatergic receptors, as well as some other brain-derived antigens were reported in patients with various neurodegenerations.It should be stresssed that some level of autoantibodies are comonly found at healthy individuals and incomplete and often controversial results are reported about CNS immune/autoimmune responses during AD. Although autoantibodies might be sometime causing or aggravating pathology, naturally occurring autoantibodies may maintain physiological homeostasis, play key roles in the clearance of self molecules and apoptotic cells, protect from pathologically altered structures like oxidatively damaged, aggregated, and non-functional lipids and proteins. It is concievable as well, that impaired/exhausted immune system of AD patients may contribute to pathology. It is worth mentioning that autoantibodies (or their specific profile) may serve as valuable biomarkers of various neurodegenerative diseases.

In our report we will focuss on autoantibodies against τ in AD, MCI and some other dementias, their charasterization in heatlhy subjects, AD, MCI patients and some other neurodegenerations, male/female differences and potential application of intravenous imunoglobulin (IVIG) treatent of neurodegenerative diseases.

Speaker Biography

Jan Ricny graduated from Faculty of Sciences, University of J.E. Purkyne, Brno, Czechoslovakia in 1975 and obtained PhD from Institute of Physiology, Czechoslovak Academy of Sciences, Prague in 1983. After postdoctoral training at McGill university at Montreal and Max-Planck Institut fur Biophysikalische Chemie at Gottingen works as researcher and senior researcher at Institute of Physiology of Academy of Sciences and National Institute of Mental Health, Czech Republic. Author of about 80 publications, H index 16. His main interests are cholinergic neurochemistry and neurodegenerative diseases.

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