27th International conference on

Neurology and Cognitive Neuroscience

October 18-19, 2018 | Warsaw, Poland

Keynote Forum

Day 1

Ricardo B Maccioni, J Neurol Neurophysiol 2018, Volume 9
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Ricardo B Maccioni

University of Chile, Chile

The neuroimmunology of Alzheimer's disease

Alzheimer's disease (AD) is a progressive neurodegenerative disease, characterized by behavioral disorders, loss of memory and cognitive impairment. AD represents around 12% of people over 65 worldwide. Cumulative evidence shows that innate immunity participates in the pathogenesis of AD. According to our theory of neuroimmunomodulation, microglial activation by the so called "damaged signals" triggers alterations in the cross-talks between microglia and neuronal cells, involving a cascade of pathological events leading to tau hyperphosphorylation and oligomerization, associated with cognitive impairment. This activation depends on the type and intensity of the stimulus. Generation of NF-kB occurs, promoting the expression and release of proinflammatory cytokines IL1β, IL-6, IL-12, IFNγ and tumor necrosis factor TNFα. As a consequence, short-lived cytotoxic factors, such as superoxide radicals, nitric oxide and ROS are released. Pathological tau hyperphosphorylations by p35 Cdk5 complex reduce tau-microtubule interactions, downregulating tau activity in stabilizing microtubules. In AD, a persistently active microglial condition seems to generate neuronal damage, with a consequent neuronal death, causing the release of pathological tau toward the extracellular environment. Released tau would subsequently cause reactivation of microglial cells, thus promoting a feedback mechanism and generating a continuous neuronal damage. However, from the pathophysiological point of view, AD is significantly more complex that just inducing a loss of memory. In fact, alterations in the dopaminergic pathway together with serotonin depletion in the elderly lead to late onset depressive phenomena according with recent evidences. These events seem to occur together with neuroimmunomodulatory changes responsible for a final tau oligomerization in the course of neurofibrillary tangles formation. This means that both affective disorders and mood changes are followed by neuroinflammatory processes that lead to intraneuronal alterations that lead to cognitive impairment. We integrate the cellular basis for the functional connections between emotional and cognitive phenomena and their pathological alterations in AD, underlying possible mechanisms for the role of consciousness. The previously summarized mechanisms could explain the onset of AD, opening a new projection to research on therapeutic agents that could modulate the interactions between tau and microglial cells.

Biography

Ricardo B Maccioni is a Professor of Neurology at the Medical School, and Professor of Neurosciences at University of Chile. He also serves as the Director of the Laboratory of Molecular Neurosciences, Scientific Director of the International Center for Biomedicine (ICC) and Senior Investigator of the Brain/Mind Program in Chile. He has served as an Associate Professor at the University of Colorado, Medical School, USA. He has completed his Doctoral degree in 1975, and was a Postdoctoral Fellow in the National Institutes of Health at the University of Colorado, Health Sciences Center and a Visiting Scholar at the Max Plank Institute for Biophysics in Germany. On the basis of these and numerous other findings, he is considered among the leading investigators in Alzheimer´s disease. He is co-author with George Perry of the recent book "*Current Hypotheses and Research Milestones in Alzheimer´s Disease*", among other 10 books in this medical field. He has made outstanding achievements in the training of 59 young scientists. He is the author of 146 publications in high-impact journals and 18 patents.

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Jerzy Leszek, J Neurol Neurophysiol 2018, Volume 9
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Jerzy Leszek

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Early identification and efficient therapy of neurodegenerative disorders nanotechnology as a tool

Alzheimer's disease (AD) is the most common cause of dementia among people aged 65 and older. The diagnosis of the sporadic AD is based on clinical exclusion criteria and is only definite at necropsy. So, biochemical markers for AD would be of great value for its early diagnosis. During the last decade, research efforts have focused on developing CSF biomarkers for AD. The diagnosis performance of the CSF biomarkers: Tau protein, the 42-amino acid form of beta-amyloid (A Beta 42) and amyloid precursor protein are of great importance. Since AD pathology is irreversible and present-day medications for AD only lower its associated symptoms, application of disease-modifying treatments could be successful only if AD early diagnosis is possible. The recently growing application of nanotechnology in molecular detection of biomarkers is promising for very early diagnosis of AD. From a practical point of view, one may perform a molecular detection process either inside the body (in vivo) or on the samples derived from the body (in vitro). In his presentation, author discusses the challenges of current treatment and diagnosis of AD and the development of biocompatible nanoparticles-provide the rational and potentials using nanoparticles for both drug carrier and imaging contrast agent for early diagnosis and treatment of AD and other neurodegenerative disorders like Parkinson disease.

Biography

Jerzy Leszek is a Professor of Psychiatry, Vice-Head of the Department of Psychiatry, Head of Alzheimer's Disease Lab at Medical University in Wroclaw, and Scientific Director of Alzheimer's Disease Center in Scinawa near Wroclaw, Poland. He completed his Graduation at Medical University of Wroclaw in 1980, was awarded a Doctorate in Wroclaw in 1981 and in 1999 examination for the degree of Associate Professor of Psychiatry and since 2005, he is working as Full Professor of Psychiatry at Wroclaw Medical University. He is the author and co-author of more than 540 papers (especially from old age psychiatry), and has published some chapters to the books in reputed international journals and serving as an Editorial Board Member of several journals. He is Editor-in-Chief of *Journal of Yoga and Physical Activity*. He is Scientific Editor and Co-author of first Polish academic handbook on Alzheimer's disease and ten another academic handbooks from old age psychiatry, member of scientific associations.

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Day 2

Wieslaw L Nowinski, J Neurol Neurophysiol 2018, Volume 9
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Wieslaw L Nowinski

University of Washington, USA

Cardinal Stefan Wyszyński University, Poland

Human brain atlases in neurology, neurosurgery and neuroeducation

We witness recently an explosion of brain-related initiatives. Our contribution has been in the creation of adult human brain atlases in health and disease, and the development of atlas-based solutions for education, research and clinical applications resulting in 35 brain atlases licensed to 67 companies and institutions, and being distributed in about 100 countries. Here, we address atlas usefulness in neuroeducation, neurosurgery, and neurology. Atlases are particularly useful in neuroeducation. Our most advanced 3D atlas "The Human Brain, Head and Neck in 2953 Pieces" created from 3/7 Tesla MRI and CT, has several novel features including: virtual 3D brain dissection, scene composing/decomposing, simultaneous display of surface and sectional neuroanatomy, continuous brain navigation, presentation of anatomy in context, correlation of neuroanatomy with terminology, quantification, and teaching materials preparation. In neurosurgery, we have introduced electronic brain atlases to clinical practice (used by 13 surgical companies), mainly for deep brain stimulation. They are useful for surgery planning, intra-operative support, and post-operative neurologic assessment. Additionally, the probabilistic functional atlas is useful for studying functional properties of cerebral structures. Our "3D Atlas of Neurologic Disorders" bridges neurology neuroanatomy and neuroradiology. It correlates brain damage with the resulting disorder and associated signs, symptoms and syndromes.

Recent Publications

1. Nowinski W L (2017) Human brain atlasing: past, present and future. The Neuroradiology Journal 30(6):504-519.
2. Nowinski W L, et al. (2015) The Human Brain, Head and Neck in 2953 Pieces. Thieme, New York.
3. Nowinski W L, et al. (2014) 3D Atlas of Neurologic Disorders. Thieme, New York.

Biography

Wieslaw L Nowinski is a Scientist, Innovator, Entrepreneur, Pioneer and Visionary. He is the Creator of world's most gorgeous human brain atlases. He has 568 publications, 121 patent applications filed and at least 76 granted (29 in US, 18 in EU), developed with his team 35 brain atlas products used in neurosurgery, neuroradiology, neurology, brain mapping, and neuroeducation, licensed to 67 companies and institutions, and distributed to about 100 countries. He has been conferred with 43 awards and honors, including 25 awards from leading medical societies. He was a Laureate (within top three) of European Inventor Award 2014 in Lifetime Achievement.

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Stephen D Skaper, J Neurol Neurophysiol 2018, Volume 9
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Stephen D Skaper

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Neuroinflammation, microglia and mast cells in the pathophysiology of neurocognitive disorders

Cells of the immune system and the central nervous system are capable of interacting with each other. The former cell populations respond to infection, tissue injury and trauma by releasing substances capable of provoking an inflammatory reaction. Inflammation is now recognized as a key feature in nervous system pathologies such as chronic pain, neurodegenerative diseases, stroke, spinal cord injury, and neuropsychiatric disorders such as anxiety/depression and schizophrenia. Neuroinflammation may also raise the brain's sensitivity to stress, thereby effecting stress-related neuropsychiatric disorders like anxiety or depression. The cytokine network plays a large part in how immune system cells influence the central nervous system. Further, inflammation resulting from activation of innate immune system cells in the periphery can impact on central nervous system behaviors, such as depression and cognitive performance. Here, we will present the current state of knowledge which implicates both microglia and mast cells, two of the principle innate immune cell populations, in neuroinflammation. Further, we shall make the case that dysregulation of microglia and mast cells may impact cognitive performance and, even more importantly, how their cell-cell interactions can work to not only promote but also amplify neuroinflammation. Finally, we will use this information to provide a starting point to propose therapeutic approaches based upon naturally-occurring lipid signaling molecules.

Biography

Stephen D Skaper has completed his PhD in Biochemistry at University of South Dakota and; Laurea in Chemistry at the University of Padua, Italy. He is Adjunct Professor in Department of Pharmaceutical and Pharmacological Sciences at University of Padua. Prior to this, he was a Senior Group Leader for Neurodegeneration Research, GlaxoSmithKline Research and Development Limited, UK, and also held academic research positions at the University of California, San Diego. He has authored/co-authored over 300 research papers, book chapters and monographs, and he is the Editor-in-Chief of CNS and Neurological Disorders Drug Targets. His research interests focus on the role of immune cells and their interactions in neuroinflammation.

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