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5th International Conference on

Alzheimer's Disease & Dementia

September 29-October 01, 2016 London, UK

Keynote Forum

(Day 1)

Claude M Wischik, J Alzheimers Dis Parkinsonism 2016, 6:5(Suppl)
http://dx.doi.org/10.4172/2161-0460.C1.020

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Claude M Wischik

TauRx Therapeutics, United Kingdom

Biography

Claude M Wischik holds the Chair in Old Age Psychiatry at the University of Aberdeen in Scotland, and is Executive Chairman of TauRx Pharmaceuticals. He studied medicine in Australia, completed his PhD at the Laboratory of Molecular Biology in Cambridge, and also higher psychiatric training in Cambridge. He was the first to identify Tau protein as the main constituent of the Alzheimer tangle and developed the first Tau Aggregation Inhibitors. He has published 121 papers and holds 11 patent groups based on his work with over 40 individual patents.

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Jerzy Leszek, J Alzheimers Dis Parkinsonism 2016, 6:5(Suppl)
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Jerzy Leszek

Wroclaw Medical University, Poland

Dendrimer's –New hope in the treatment of Alzheimer's disease

The lack of effective treatment for Alzheimer's disease(AD) stems mainly from the incomplete understanding of AD causes. Currently there are several hypotheses which try to explain the early molecular mechanisms of AD pathogenesis. Nanomedicine as a biomedical and pharmaceutical application of nanotechnology for making nanocarriers like dendrimers has shown great potential for the treatment of many CNS diseases.

Dendrimers are polymeric molecules chemically synthesized with well defined shape size and nanoscopic physicochemical properties reminiscent of proteins.

Recently an increasing number of studies have been focused on the potential of dendrimers to prevent aggregation and fibrillation of proteins involved in neurodegenerative disorders such as AD. Some of dendrimers were demonstrated to cross blood-brain barrier, which legitimized research on these compounds as potential drugs for neurological disorders. Recent our studies have revealed that dendrimers possess the intrinsic ability to localize in cells associated with neuroinflammation(activated microglia and astrocytes) and thus can be used as drug carriers in neuroinflammation therapy.

Above/mentioned findings may be of high significance in the context of potential application of dendrimers as drug carriers or active compounds per se. According to the opinion the author's of this presentation ,they are promising macromolecules for further investigations on their applicable in neurodegenerative disorders , for instance AD.

Biography

Jerzy Leszek is Professor of Psychiatry, Vice-Head of the Department of Psychiatry and Head of Alzheimer's Disease Lab at the Medical University in Wroclaw, Poland. He graduated at Medical University of Wroclaw in 1979, was awarded a doctorate in Wroclaw in 1981 and in 1999- examination for the degree of associate professor of psychiatry and since 2007 he is working as full professor of psychiatry at Wroclaw Medical University. He is author and co-author more than 280 papers (especially from old age psychiatry), some chapters to the books published in reputed Polish and international journals and serving as an editorial board member of several journals.

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Jordan L Holtzman
University of Minnesota, USA

Alternative Models for Drug Discovery in Alzheimer's Disease

Alzheimer's disease is described as a progressive dementia associated with the extracellular deposits in the brain of a garbage protein, β-amyloid (Aβ), and the intracellular deposits of tangles. The vast majority of the patients develop the disease in old age while a small portion have an early onset, familial form. Since the 1970's it has been generally assumed that the dementia seen in the two forms is due to the neurotoxicity of the Aβ. With the purification and sequencing of Aβ in the 1990's investigators were able to identify the mutations associated with the familial forms. A number of laboratories transfected mice with these mutated genes. Since these animals exhibited many of the pathological and clinical manifestations of the late onset disease, they have been used to screen for new therapeutic agents. Unfortunately, all 244 drugs identified in these models have failed in clinical trials in elderly patients. Work from my laboratory has suggested that plaque deposits of Aβ rather than causing the dementia is merely a biomarker for a decline in the capacity of the endoplasmic reticulum (ER) in the cells to catalyze the posttranslational processing of 40% of cellular proteins, including those synaptic, membrane proteins required for a functioning memory. This paradigm is based on our observation that in the CSF all Aβ is normally N-glycosylated and bound to ER proteins, ERp57 and calreticulin. These data suggest a new direction for drug discovery in which agents are screened for their ability to increase the levels in cell lines of fluorescently labeled components of the ER post-translational pathway. The development of these cell lines would permit rapid screening for potential therapeutic agents in plate readers.

Biography

Jordan L. Holtzman, M.D., Ph.D. is Professor, Departments of Pharmacology and Medicine and Division of Environmental Health Sciences, University of Minnesota, Minneapolis, USA. He was graduated from University of Chicago, the Pritzker School of Medicine in 1959. He completed internship from University Ill Rsc/Ed in 1960.

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Linda Levine Madori

St. Thomas Aquinas College, USA

Utilizing the TTAP method to enhance collective impact on a Dementia and gero-psychiatic unit; increasing interactions and involvement while decreasing falls

This paper will establish through a clinical research study of the use of the TTAP Method® on a Dementia and Gero-Psychiatric unit for a one year period (2014). All healthcare staff were taught to engage in "*Emotive Conversations*" through the use of this replicable multimodel approach with those individuals afflicted with middle to late stages of dementia, and other psychiatric diagnosis. Data collected over a 365 day period from 1800 patients demonstrates increased mood,increased overall time staff spent engaged with patients and decrease in patient falls. This research supports how channeling change through collective impact can significantly effect patients and staff on a gero-psyciatric unit, while successfully impacting healthcare.

The TTAP approach is formulated on the basic functional organization of the brain, neuroplasticity, including neurons, neurotransmitters and areas of the brain involved in transforming emotional and perceptual inputs into physiological responses and behaviors (Damasio, 1998, 1999; Golomb, J.,1996, Grober, E., 1999; Kandel, Schwartz & Jessel, 2000; LeDoux, 2000; Levine Madori, 2007-2014). All healthcare staff were given 15 hours of TTAP Certification Training which utilizes person centered themes within the therapeutic process to engaged participants in a twelve step process that incorporates medication & mindfulness, drawing, sculpture, movement, phototherapy and other forms of the creative arts into an ongoing enriching non-pharmaceutical approach. This method substantiates how conversation which is rich in emotions along with the expressive arts is quickly becoming a powerful way in which to break down "silo's" of responsibility in demanding and complex healthcare units while continually allowing for self-discovery (Cozolino, 2012, Luzebrink, 2013, Hass-Cohen, 2014).

Examples of other research studies utilizing this innovative method with the Alzheimer's population will be presented from the United States and Finland.

Biography

Linda Levine Madori is a two time Fulbright Scholar, Professor, Author, Researcher and Trainer of a non-pharmaceutical approach utilizing all the creative arts for brain stimulation and enhancing socialization found in her first book titled; *Therapeutic Thematic Arts Programming, in 2007 (TTAP Method.com). Her second book; Transcending Dementia through the TTAP Method; A New Psychology of Art, Brain and Cognition, expands on the current significant research demonstrating cost effectiveness utilizing this innovative multimodal approach for the geriatric and Alzheimer's population.*

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Zhicheng Xiao
Monash University, Australia

Neurodegeneration research: From molecules, big animal models to human beings

Appropriate connections or interactions among different neural cell types are essential for the correct and efficient functioning of the nervous system during development and regeneration after trauma or degeneration. The aim of my research is to understand the molecular events that mediate communication among neural cells in the nervous system during development, myelination, learning and memory, degeneration, and regeneration. These studies have yielded insights into the therapeutic potential of cell signalling molecules to ameliorate or even ablate the detrimental consequences of nervous system injury and neurodegenerative diseases, including stroke, traumatic brain injury, spinal cord injury, Alzheimer Disease (AD), and Multiple Sclerosis (MS). Using genome-wide chromatin immunoprecipitation approaches, we found that AICD is specifically recruited to the regulatory regions of several microRNA genes, and acts as a transcriptional regulator for miR-663, by which suppresses neuronal differentiation in human neural stem cells. We have generated transgenic pigs expressing mutant G93A hSOD1 and showing hind limb motor defects, which are germline transmissible, and motor neuron degeneration in dose- and age-dependent manners. Furthermore, in a case report we present the treatment of aggressive MS patient with multiple allogenic human umbilical cord-derived mesenchymal stem cell and autologous bone marrow-derived mesenchymal stem cells over a 4 y period. The treatments were tolerated well with no significant adverse events. Clinical and radiological disease appeared to be suppressed following the treatments and support the expansion of mesenchymal stem cell transplantation into clinical trials as a potential novel therapy for patients with aggressive MS.

Biography

Zhi-cheng Xiao, PhD. He received a Doctor of Natural Science Degree from Swiss Federal Institute of Technology, Zurich. He is current Professor in Monash University. He is the CEO & CFO of iRiccorgPharm, a premier Bio-Tech company. He has published more than 100 papers in reputed journals and serving as editorial board members of more than 10 journals.

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Xiaoliang Wang
Chinese Academy of Medical Sciences, China

Study on Diabetes Induced Dementia and the Mechanisms of Synaptic Plasticity in KK-Ay Mice

Diabetes mellitus (DM) may induce dementia, so-called diabetic encephalopathy. In the present study, the spontaneously obesity-induced Type 2 diabetic model, KK-Ay mice were used to study the relationship between spatial learning and memory deficits and the alteration of hippocampal synaptic plasticity.Our results showed that KK-Ay mice presented typical T2DM syndrome and deterioratedprogressivelyin Morris water maze from early stage (3 month old). Meanwhile, Aßdeposition and Tau phosphorylation increased in hippocampus. LTP (long term potentiation) was also impaired significantly. It is interesting that these deficits in KK-Ay mice could be relieved by diet intervention and anti-AD drugs. Further, we found that the underlying mechanisms of LTP impairment in KK-Ay mice might attribute to abnormal phosphorylation or expression of glutamate receptors subunits rather than alteration of basal synaptic transmission. The expression levels of NR1, NR2A and NR2B subunits of NMDA receptors (NMDARs) were unchanged while the Tyr-dependent phosphorylations of NR2A and NR2B subunits were significantly reduced in KK-Ay mice. The p-Src and CaMKII were also down regulated.In addition, AMPA receptor, GluR1 was decreased, and the GluR2 was significantly increased.In summary, ourresults suggest that deficits in learning and plasticity in KK-Ay mice may mainly arise from the abnormalof NR2 subunits, which were related to the activities of p-Src and CaMKII. It might be recovered by diet interventionand anti-AD treatment.

Biography

Wang,Xiaoliang has completed his MD from University of Essen, Germany in 1987. He returned to the Chinese Academy of Medical Sciences, Beijing in 1988 and promoted to full professor in 1993. He served as director of Institute of MateriaMedica, CAMS from 1997 to 2010. His research fields including neurodegenerative diseases, drug discovery and development. He has published 200 papers in reputed journals and has been serving as editorial board membersfor several journals.

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