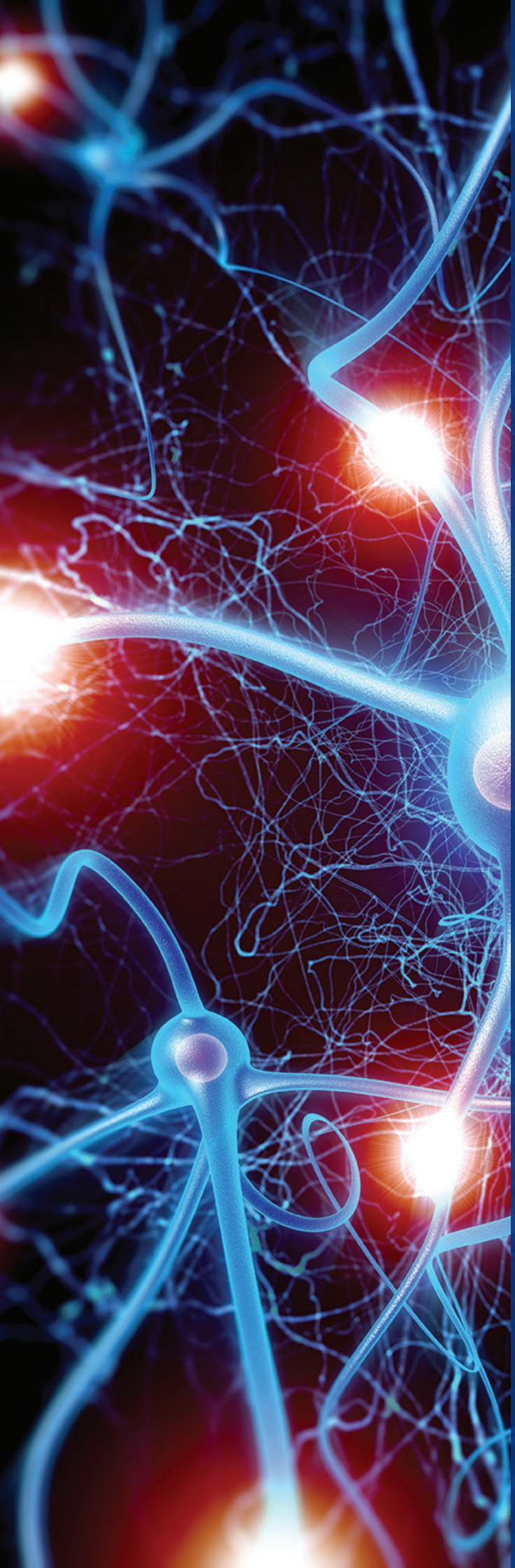


conferenceseries.com
1307th Conference

October 2017 | Volume 8 | Issue 6 | ISSN: 2171-6625
Journal of Neurology & Neuroscience

Proceedings of

17TH Global Neuroscience Conference

October 16-17, 2017 Osaka, Japan

Conference Series
One Commerce Center-1201
Orange St. #600, Wilmington, Zip 19899, Delaware, USA
Toll Free: 1-888-843-8169
Kemp House, 152 City Road, London EC1V 2NX, UK
Toll Free: +0-800-014-8923
Toll Free: Japan: 81-345780247 | Singapore: 800-852-6126 / 65 8008526126 |
USA/Canada: 1-650-889-4686
Email: neurosciencemeet@neuroconferences.com

conferenceseries.com
1308th Conference

17th Global Neuroscience Conference

October 16-17, 2017 Osaka, Japan

Keynote Forum

(Day 1)

conferenceseries.com

J Zhu, J Neurol Neurosci 2017, 8:6
DOI: 10.21767/2171-6625-C1-004

17TH GLOBAL NEUROSCIENCE CONFERENCE

OCTOBER 16-17, 2017 OSAKA, JAPAN

J Zhu

University of South Carolina, USA

Allosteric modulatory effects on HIV-1 Tat protein-induced inhibition of human dopamine transporter function

The inducible HIV-1 Tat Transgenic (iTat) mouse model recapitulates many aspects of neurocognitive impairments observed in HIV infected individuals. Tat and cocaine synergistically increase synaptic Dopamine (DA) levels by directly inhibiting DA Transporter (DAT) activity, ultimately leading to dopaminergic neuron damage. This study determined allosteric modulatory effects of SRI-30827 on HIV-1 Tat protein-mediated regulation of human DAT and Cocaine Condition Place Preference (CPP) in iTat mice. Results show that SRI-30827 attenuated Tat-induced inhibition of [3H]DA uptake and [3H] WIN35,428 binding in PC12 cells expressing human DAT. After a 7-d doxycycline (Dox) treatment, HPLC analysis revealed that DA content in the Prefrontal Cortex (PFC) and Nucleus accumbens (NAc) of iTat-Tg mice were increased by 92% and 37%, respectively, compared to control mice. Consistently, DA/DOPAC in the PFC and NAc of iTat-Tg mice was increased by 44% and 26%, respectively. We performed the patch clamp recording to measure Medium Spine Neurons (MSN) firing in brain NAc slices of iTat mice in the presence of DA and cocaine. Results show that that action potential frequency of NAc shell MSN was significantly increased in iTat mice compared to control mice. Further, action potential frequency of NAc shell neurons was decreased in response to 5 μM cocaine and further decreased when cocaine and 5 μM were applied together, which were completely attenuated in iTat mice. Finally, we found that ICV infusion of SRI-30827, a novel allosteric modulator, partially attenuated the potentiated cocaine-CPP in iTat mice. These findings suggest the hypothesis that Tat potentiates cocaine rewarding effect and allosteric modulator has potential for treatment of Tat-induced drug reward.

Biography

J Zhu research aims toward finding solutions to a newly recognized challenge in treatment for HIV-associated neurocognitive disorders (HANDs). About one-half of HIV-1-positive individuals suffer from HAND, which dramatically affects memory, learning, decision-making, planning and overall quality of life. Cocaine has been shown to exacerbate the severity of HAND. HAND is associated with HIV-1 viral proteins, which are present in the brain of HIV-1-infected patients. HIV-1 transactivator of transcription (Tat) protein--an HIV regulatory protein is thought to inhibit neuronal communication by acting directly on the human dopamine transporter, a membrane protein in the brain responsible for pumping the dopamine back into the cytosol and terminating dopamine signaling during neurotransmission. Dr. Zhu's project is to investigate how cocaine and Tat work to create binders that derail neuronal communication in the brain. The ultimate goal is to develop neuroprotective drugs and help HIV patients recover their neurological function.

zhuj@cop.sc.edu

Ming-Chao Huang, J Neurol Neurosci 2017, 8:6
DOI: 10.21767/2171-6625-C1-004

17TH GLOBAL NEUROSCIENCE CONFERENCE

Ming-Chao Huang

Taipei Veterans General Hospital, Taiwan

Leptin is essential for spinal microglia activation and the development of neuropathic pain after preganglionic cervical root avulsion

Preganglionic Cervical Root Avulsion (PCRA) affects both the peripheral and central nervous systems and is often associated with neuropathic pain. Unlike Peripheral Nerve Injuries (PNI), central lesions caused by disruption of cervical roots from the spinal cord following PCRA contribute to the generation of neuropathic pain. Leptin is involved in the development of neuropathic pain after PNI by affecting neurons. However, whether leptin is involved in microglial activation leading to neuropathic pain after PCRA is unknown. In this study the preganglionic avulsion of the left 6th-8th cervical roots was performed in C57B/6J mice and leptin-deficient mice. A leptin antagonist or leptin was administered to C57B/6J mice and leptin-deficient mice after injury, respectively. The expression pattern of spinal microglia was examined by immune-fluorescent staining. Von Frey filaments were used to test pain sensitivity. Our data showed that leptin is essential for the development of neuropathic pain after PCRA. Allodynia was absent in the leptin-deficient mice and the mice administered the leptin antagonist. We also found that leptin deficiency or the administration of its antagonist inhibited the development of microgliosis, the expression of CD86 and iNOS and Wallerian degeneration in the spinal cord. Moreover, the administration of exogenous leptin to leptin-deficient mice reversed these effects. We concluded that leptin is involved in the proliferation and activation of microglia, which in turn enhances the development of neuropathic pain. Blocking the effects of leptin might be a target for the treatment of neuropathic pain after PCRA.

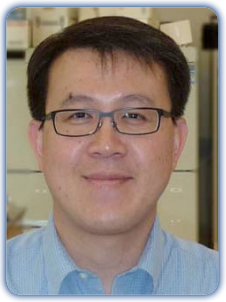
Biography

Ming-Chao Huang has completed his MD degree from Taipei Medical University, Taiwan in 1984 and his PhD degree from Tokyo Women's Medical University, Japan in 1996. He is a Neurosurgeon and is currently the Division Chief of Department of Neurosurgery, Taipei Veterans General Hospital, Taiwan. His research interests are nerve root injury (including basic mechanism and surgical repair), neuropathic pain (including basic study and treatment) and brain tumor (including medical and surgical treatment).

mchuang@vghtpe.gov.tw

Notes:

Wen-Hai Chou, J Neurol Neurosci 2017, 8:6
DOI: 10.21767/2171-6625-C1-004



Wen-Hai Chou

National Health Research Institutes, Taiwan

PKCε-ATF2 signaling in ischemia-induced neurodegeneration

Cardiac arrest continues to be the leading cause of death worldwide. Global cerebral ischemia that accompanies cardiac arrest is one of the major causes of morbidity and mortality. Out of many therapeutic approaches investigated, one of them is ischemic preconditioning, which is sufficient to protect brain tissues from subsequent lethal ischemic insult. PKCε peptide activator administered before, but not after, ischemia mediates preconditioning and confers neuro protection. However, the use of preconditioning as a therapeutic approach has not become standard clinical practice because the occurrence of cardiac arrest and cerebral ischemia is sudden and unpredictable. Thus, post-ischemic therapeutic targets have to be unraveled. The beneficial effects of PKCε peptide activators in ischemic preconditioning stimulate interests in understanding the molecular and cellular actions of PKCε after global cerebral ischemia. A detailed understanding of PKCε signaling pathways requires identification of its downstream targets. This study is to determine the downstream mediators of PKCε, so that novel therapeutic targets can be developed. We found that PKCε mediated the phosphorylation of Activating Transcription Factor 2 (ATF2) at threonine 52 in the hippocampus. ATF2 is a member of the Activator Protein 1 (AP1) transcription factor superfamily regulating normal growth and development as well as response to cellular stress. In response to global cerebral ischemia, PKCε expression was gradually decreased. This resulted in leakage of nuclear ATF2 to the mitochondria and subsequent ischemia-induced neurodegeneration. This study not only provides the first insight into the neuronal cell death regulated by PKCε and ATF2, but also establishes a strong base to develop new classes of therapeutic molecules to inhibit the leakage of ATF2 and reduce brain injury after cardiac arrest.

Biography

Wen-Hai Chou has completed his PhD from Department of Molecular Medicine, University of Texas Health Science Center, San Antonio and Post-Doctoral studies from University of California, San Francisco. He is an Assistant Investigator of National Health Research Institutes, Taiwan. He has published more than 22 papers in reputed journals including *Neuron, Journal of Clinical Investigation, Journal of Neuroscience* and *Journal of Biological Chemistry*.

wenhaichou@nhri.org.tw

Notes:

conferenceseries.com
1308th Conference

17th Global Neuroscience Conference

October 16-17, 2017 Osaka, Japan

Keynote Forum

(Day 2)

Wai Kwong Tang, J Neurol Neurosci 2017, 8:6
DOI: 10.21767/2171-6625-C1-004

Wai Kwong Tang

Chinese University of Hong Kong, China

Structural and functional MRI correlates of post stroke depression

Depression is common following an acute stroke. Post Stroke Depression (PSD) has notable impacts on the function recovery and quality of life of stroke survivors. Incidence decreased across time after stroke, but prevalence of PSD tend to be stable. Many studies have explored the association between lesion location and the incidence of PSD. For example, lesions in frontal lobe, basal ganglia and deep white matter have been related with PSD. Furthermore, cerebral microbleeds and functional changes in brain networks have also been implicated in the development of PSD. In this presentation, evidences of such association between the above structural and functional brain changes and PSD will be reviewed to learn the prevalence, course, vascular risk factors and treatment of PSD.

Biography

Wai Kwong Tang was appointed as Professor in the Department of Psychiatry, the Chinese University of Hong Kong in 2011. His main research areas are addictions and neuropsychiatry in stroke. He has published over 100 papers in renowned journals and has also contributed to the peer review of 40 journals. He has served the Editorial Boards of five scientific journals. He was also a recipient of the Young Researcher Award in 2007, awarded by the Chinese University of Hong Kong.

tangwk@cuhk.edu.hk

Fu-Zen Shaw, J Neurol Neurosci 2017, 8:6
DOI: 10.21767/2171-6625-C1-004

Fu-Zen Shaw

National Cheng Kung University, Taiwan

Neurofeedback training of frontoparietal alpha rhythm enhances episodic memory

Neurofeedback Training (NFT) of brain rhythm is an operant conditioning paradigm through a video or audio interface and has been widely used in clinic. Episodic memory is a prerequisite for successful life functioning. This study aims to explore trainability of alpha NFT with a sham-controlled experimental design and effect of alpha NFT on episodic memory. Participants were randomly assigned into a control group receiving feedback of 4-Hz amplitude randomly selected from 7-20 Hz or an alpha group receiving feedback of 8-12 Hz amplitude. The NFT contained 12 sessions and each session consisted of 6 min blocks. Picture recognition task with identifying exact shape and size of objects was used to assess episodic memory. Topographic distribution of trained alpha rhythm was categorized through the whole-head EEG recording. The Alpha group exhibited a linear increase in amplitude and duration of alpha rhythm throughout the NFT exclusively. The Alpha group exhibited significantly higher amplitude and longer total duration of alpha rhythm compared with those of the control group. Accuracy of the picture recognition task in the Alpha group was significantly improved after NFT compared with that of the control group. In particular, participants with increased alpha rhythm which primarily distributed in bilateral frontoparietal region exhibited significantly linear trend between alpha duration and accuracy of the picture recognition task. The evoked alpha rhythm in the occipital region seemed to be no effect on accuracy of the picture recognition task. The present study provides additional evidence on the trainability of alpha rhythm through NFT and also identifies functional localization of alpha rhythm in the frontoparietal region on enhancement of episodic memory. Our results suggest a non-pharmacological intervention on memory enhancement throughout a NFT of alpha rhythm.

Biography

Fu-Zen Shaw has his interest in establishment of animal models of epilepsy and fibromyalgia and also provides valuable non-pharmacological interventions for ameliorating seizures through closed-loop deep brain stimulation or for enhancing wellbeing of healthy people and patients with insomnia or mild cognitive impairment through self-training of brain rhythms. Recently, he has developed a neurofeedback platform to train alpha rhythm with a sham-controlled group to validate controllability, specificity and independence of the neurofeedback training. He also provides convinced evidence about trained alpha rhythm on enhancement of both working memory and semantic episodic memory.

fzshaw@gmail.com

Notes:

Kah-Leong Lim, J Neurol Neurosci 2017, 8:6
DOI: 10.21767/2171-6625-C1-004

17TH GLOBAL NEUROSCIENCE CONFERENCE

OCTOBER 16-17, 2017 OSAKA, JAPAN

Kah-Leong Lim

National University of Singapore, Singapore

Neuroprotective and neurorestorative strategies for Parkinson's disease

Parkinson Disease (PD) is a prevalent neurodegenerative disease affecting millions of predominantly elderly individuals worldwide. Despite intensive efforts devoted to drug discovery, the disease remains incurable. Compounding this problem is the current lack of a truly representative mammalian model of PD. Interestingly; the drosophila has emerged as a good system to model the salient features of the disease, including Dopaminergic (DA) neurodegeneration and associated locomotion defects. Taking advantage of this and also the utility of the drosophila as a tool for drug discovery, we have uncovered several neuroprotective compounds and associated targets. These include AMP Kinase (AMPK) activators that are relevant in human PD cases. Our results support the use of drosophila PD model as an intermediate *in vivo* host for phenotype-based drug screening. Because PD involves the degeneration of neurons in a rather circumscribed region in the brain, neuro-restorative therapy via cell replacement represents another strategy to treat the disease. Here, we have exploited the induced pluripotent stem cell (iPS) technology to derive transgene integration- and feeder-free iPS from cells lining the human umbilical cord, an immune-privileged organ that mediates interactions across the feto-maternal interface. Collectively designated as CLiPS (Cord Lining-derived iPS), we demonstrated that CLiPS-derived DA neuronal precursors transplanted into an immune-competent 6-hydroxydopamine mouse model of PD not only survived but also differentiated into mature DA neurons in the absence of pharmacological immunosuppression. Further, the engrafted mice showed functional motor recovery and restoration of dopamine level (illuminated via PET imaging). These results position CLiPS as a promising source of donor cells for allogeneic cell replacement therapy for PD (Supported by NMRC-TCR).

Biography

Kah-Leong Lim has completed his PhD from the Singapore Institute of Molecular and Cell Biology in 1999. He has completed his Postdoctoral training at the Department of Pathology in Harvard Medical School (2000-2001) and subsequently at the Department of Neurology in Johns Hopkins University School of Medicine (2001-2002), where he has worked on the topic of Parkinson's disease with Professor Ted Dawson. He is currently the Deputy Director of Research at the National Neuroscience Institute of Singapore and Director of Basic and Translational Research in the Singhealth Duke-NUS Neuroscience Academic Clinical Program.

Kah_Leong_Lim@nni.com.sg

Notes: