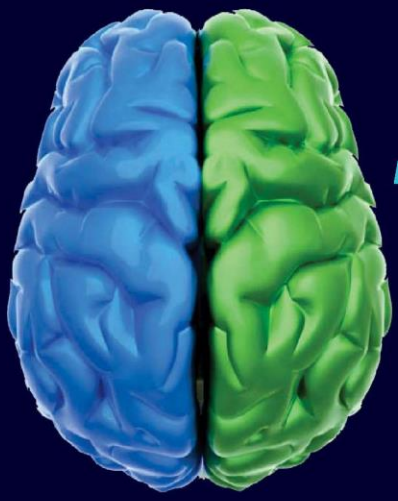

"Challenge the Brain, Change the Future"

August 30 (Wed)-31 (Thu), 2017
Grand Hilton, Seoul

The 20th Annual Meeting of
the Korean Society for Brain and Neural Science
Co-organized by the Korean Society for Neurodegenerative Disease

HOSTED BY · The Korean Society for Brain and Neural Science The Korean Society for Neurodegenerative Disease

SUPPORTED BY · National Research Foundation of Korea , PAVMED, Thermo Fisher Scientific, BT Inc., Nikon Instruments Korea, Leica Microsystems Ltd., ZEISS Korea

"Challenge the Brain, Change the Future" The 20th Annual Meeting of the Korean Society for Brain and Neural Science Co-organized by the Korean Society for Neurodegenerative Disease

P1-168

Effect of spike-timing-dependent plasticity on stochastic burst synchronization in A scale-free neural network

Sang-Yoon Kim, Woochang Lim

Institute for Computational Neuroscience and Department of Science Education, Daegu National University of Education, Daegu, Korea

We consider an excitatory population of subthreshold Izhikevich neurons which cannot fire spontaneously without noise. As the coupling strength passes a threshold, individual neurons exhibit noise-induced burstings. This neuronal population has adaptive dynamic synaptic strengths governed by the spike-timing-dependent plasticity (STDP). In the absence of STDP, stochastic burst synchronization (SBS) between noise-induced burstings of subthreshold neurons was previously found to occur over a large range of intermediate noise intensities through competition between the constructive and the destructive roles of noise. Here, we study the effect of additive STDP on the SBS by varying the noise intensity D in the Barabasi-Albert scale-free network (SFN) with symmetric preferential attachment with the same in- and out-degrees. This type of SFN exhibits a power-law degree distribution (i.e., scale-free property), and hence it becomes inhomogeneous one with a few "hubs" (i.e., super-connected nodes). Occurrence of a "Matthew effect" in synaptic plasticity is found to occur due to a positive feedback process. Good burst synchronization gets better via long-term potentiation (LTP) of synaptic weights, while bad burst synchronization gets worse via long-term depression (LTD). Consequently, a step-like rapid transition to SBS occurs by changing D, in contrast to the relatively smooth transition in the absence of STDP. Emergence of LTP and LTD of synaptic weights are investigated in details via microscopic studies based on both the distributions of time delays between the nearest burst onset times of the pre- and the post-synaptic neurons and the pair-correlations between the pre- and the post-synaptic IIBRs (instantaneous individual burst rates). We also investigate the effect of network architecture on SBS for a fixed D and a multiplicative STDP case depending on the states in comparison with the above additive STDP case (independent of the states).

Key Words: Stochastic burst synchronization, Spike-timing-dependent plasticity, Scale-Free network, Subthreshold neurons

P1-169

Neuronal dynamic routing model through coherent oscillations in complex network of electrical synapses

Sangyeol Kim[1,2,3], Iksoo Chang[1,3]

[1]Creative Research Initiatives Center for Proteome Biophysics, DGIST, Daegu, [2]Department of Physics, Pusan National University, Busan, [3]Department of Brain and Cognitive Sciences, DGIST, Daegu, Korea

The membrane potentials of neurons propagate across electric synapses without directionality, threshold, and time delay in contrast to chemical synapses. Several experimental evidences and computational models have shown that electrical synapses are required to implement not only the movement of the subthreshold oscillation but also the emergence of both the rhythmicity in the global neural network and the spiking synchrony in the mammalian interneurons. How the complex network of neurons executes the simultaneous and coherent propagation of neuronal signals is the center of fundamental issues in Connectomics, yet is still far away from our systematic understanding theoretically and computationally. Also there are still debates on another issue, called 'Dynamic routing'; how the outgoing signal of the sensory-neurons, regulated by the modulation of firing-frequency, chooses the specific propagating path in various possible ways while the synaptic network is comparatively static.
In this work, we aim to suggest a physical mechanism on how 'Dynamic routing' operates with the propagation of coherent membrane potential oscillations in complex network of electrical synapses. Provided that the coherence length of oscillatory signals is longer than our system size, we employed the quantum mechanical formulation in order to describe the propagation of the subthreshold membrane potential across the complex network of electrical synapses with oscillatory incoming/outgoing signals. We could envisage the interference effect of all possible paths between input and output neurons, and investigated the propagation of coherent oscillatory signals among neurons in the complex network. We demonstrated the existence of allowed or forbidden propagation and the dynamic modulation of the propagation path depending on the wave-number of oscillatory signals in both the virtual network formed like square lattice and the real electrical synaptic network of C. elegans.

Key Words: Dynamic routing, Coherent oscillation, Complex network, Electrical synapse, Anderson's tight-binding hamiltonian

P1-170

Structural Insight to changes of thermostability in human prion protein induced by H187 mutants

Thi Hoa Nguyen[1,2], Sangyeol Kim[1,2,3], Mooseok Kang[1,2,3], Iksoo Chang[1,2]

[1]Creative Research Initiatives Center for Proteome Biophysics, DGIST, [2]Department of Brain and Cognitive Sciences, DGIST, Daegu, [3]Department of Physics, Pusan National University, Busan, Korea

Misfolding of prion protein (PrP) is known as one of key events in prion diseases, a group of lethal and infectious neurodegenerative diseases. It is well known that the misfolding of PrP is induced by acidic pH condition where its buried histidine employs protonation. However, despite of numerous intense studies, how the mutants of buried histidine affecting on changing PrP structure and thermostability remains unclear.
Here, we performed molecular dynamics simulations at various temperatures to investigate the changes in stability and structure of human PrP (residues 125-228) and its variants (H187F and H187R). We found that H187F (H1987R) mutant has higher (lower) thermostability comparing to wild-type. Also, the structural differences between wild-type and mutants in the pairwise interaction energy and atomic fluctuation of each amino acid were verified in this study.

Key Words: Prion protein, MD simulation, Thermostability

P1-171

Normalization of cortical thickness measurements across different T1 magnetic resonance imaging protocols by novel w-score standardization

Jinyong Chung[1], Kwangsun Yoo[1], Peter Lee[1], Chan Mi Kim[2], Jee Hoon Roh[2], Ji Eun Park[3], Sang Joon Kim[3], Sang Won Seo[4], Jeong-Hyeon Shin[5], Joon-Kyung Seong[5], Yong Jeong[1]

[1]Department of Bio and Brain Engineering, KI for Health Science and Technology, KAIST, Daejeon, [2]Department of Neurology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, [3]Department of Radiology and Research Institute of Radiology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, [4]Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, [5]School of Biomedical Engineering, Korea University, Seoul, Korea

Background: The use of different 3D T1-weighted magnetic resonance (T1 MR) imaging protocols induces image incompatibility across multicenter studies, negating the many advantages of multicenter studies. A few methods have been developed to address this problem, but significant image incompatibility still remains. Thus, we developed a novel method to improve image compatibility.
Methods: We developed a protocol-specific w-score standardization to control the protocol effect, which is applied to each protocol separately. We used three data sets. In dataset 1, brain T1 MR images of normal controls (NC) and patients with Alzheimer disease (AD) from two centers, acquired with different T1 MR protocols, were used (Protocols 1 and 2, n=45/group). In dataset 2, data from six subjects, who underwent MRI with two different protocols, were used with different repetition times, echo times, and slice thicknesses. In dataset 3, T1 MR images from a large number of healthy normal controls (Protocol 1: n=148, Protocol 2: n=343) were collected for w-score standardization.
Results: As expected, different protocols resulted in differing cortical thickness measurements in both NC and AD subjects. Different measurements were obtained for the same subject when imaged with different protocols. Multivariate pattern difference between measurements was observed between the protocols. Classification accuracy between two protocols was nearly 90%. After applying protocol-specific w-score standardization, the differences between the protocols substantially decreased. Most importantly, protocol-specific w-score standardization reduced both univariate and multivariate differences in the images while maintaining the AD disease effect. Compared to conventional regression methods, our method showed the best performance for in terms of controlling the protocol effect while preserving disease information.
Conclusions: Protocol-specific w-score standardization effectively resolved the concerns of conventional regression methods. It showed the best performance for improving the compatibility of a T1 MR post-processed feature, cortical thickness.

Key Words: T1 MR imaging, Cortical thickness, Image compatibility, W-score standardization, Alzheimer's disease