References
1. Muldoon SF, Bridgeford EW, Bassett DS. Small-world propensity and weighted brain networks. Scientific report. 2016 Feb 25;6:22057.
2. Liu Q, Farahibozorg S, Porcaro C, Wenderoth N, Mantini D. Detecting large-scale networks in the human brain using high-density electroencephalography. *Human brain mapping* 2017 Sep;38(9):4631–43.
3. Joliot M, Jobard G, Naveau M, et al. AICHA: An atlas of intrinsic connectivity of homotopic areas. *Journal of neuroscience methods* 2015 Oct 30;254:46–59.
4. Siems M, Pape AA, Hipp JF, Siegel M. Measuring the cortical correlation structure of spontaneous oscillatory activity with EEG and MEG. *NeuroImage* 2016 Apr 1;129:345–55.

P194

Channelrhodopsin-2 model with improved computational efficiency

Ruben Schoeters[1], Thomas Tarnaud[1], Wout Joseph[1], Robrecht Raedt[2], Emmeric Tanghe[1], Luc Martens[1]
[1]University of Ghent, Waves - Information technology, Ghent, Belgium; [2]University of Ghent, 4Brain lab - Department of Neurology, Ghent, Belgium
Correspondence: Ruben Schoeters (ruben.schoeters@ugent.be)
BMC Neuroscience 2019, **20(Suppl 1)**:P194

Optogenetics is a neuromodulation technique that uses light to control neuronal activity. To this end, light sensitive ion channels or pumps (termed opsins) are genetically expressed into neurons. Channelrhodopsin-2 (ChR2) is an excitatory opsin consisting of seven transmembrane helices covalently bound with a retinal chromophore. Illumination of the opsin triggers a retinal 13 trans-cis isomerization followed by opening of the pore. UV/vis and difference infrared spectroscopy identified at least five different states in a single photocycle. Furthermore, electrophysiological recordings, retinal extraction and Raman measurements provide evidence for the existence of a second photocycle, which is widely adopted [1]. The place of transition between this dark- and light adapted photocycle is however still under debate (Fig 1, left). In-silico, the whole photocycle is predominantly modelled with a four-state branched model that consists of two open and closed states (Fig 1, middle). Moreover, an extra state-variable is typically used to model the time- and irradiance dependent activation [2]. Consequently, the model consists of four differential equations making it quite computational demanding.We proposed an alternative model that is based on the fast transient sodium model of Hodgkin and Huxley. However, instead of inactivation in the Hodgkin and Huxley model, the second state pair represents the light-dark adaptation (Fig 1, right). This model requires only two differential equations, thus reducing the number of equations with fifty percent. Furthermore, by using two light dependent rates in the light-dark adaptation cycle, we hypothesized no loss of ChR2 current features (i.e. a transient peak followed by a steady-state plateau and slow recovery from light adaptation, under voltage-clamp conditions). This hypothesis was tested and confirmed, by fitting our model to voltage-clamp recordings reported by [2]. For both the equilibrium and time constants, a logistics relationship was used to incorporate intensity and voltage dependence. However, these dependences were on a logarithmic and linear scale, respectively. Subsequently, the obtained model was compared against the 4-state branched model created by [2]. The computational efficiency was addressed in a cortex network model, consisting of 36 excitatory neurons containing the ChR2 current model and 12 inhibitory neurons. The simulation was solved with a global variable step and variable order solver (ode15s) in MATLAB. For a two second simulation containing one second of optical stimulation, an average (n = 10) of 220.73 s and 175.32 s computation time was required, for the configuration with the 4-state branched and own model, respectively. The proposed model results thus in a significant increase of computational efficiency.

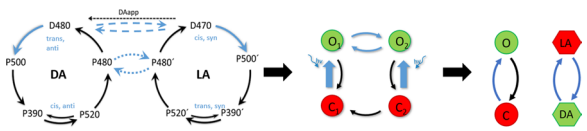

Fig. 1 The ChR2 photocycle based on UV/Vis and difference infrared spectroscopic measurements (left). The transition between the dark adapted (DA) and light adapted (LA) occurs either at the parent states (dashed step) or at the late intermediates (dotted step). A four state branching model (middle). The proposed model with opening and light-dark adaptation separately (right)

References
1. Bruun S, Stoeppler D, Keidal A, et al. Light-Dark Adaptation of Channelrhodopsin Involves Photoconversion between the all-trans and 13-cis Retinal Isomers. Biochemistry 2015, 54, 5389–5400.
2. Williams JC, Xu J, Lu Z, et al. Computational Optogenetics: Empirically-Derived Voltage- and Light-Sensitive Channelrhodopsin-2 Model. *PLoS Computational Biology* 2013, 9.

P195

A model of presynaptic KV7 channel function in hippocampal mossy fiber bouton

Elisabetta Giacalone[1], Michele Migliore[1], David Anthony Brown[2], Mala Shah[3], Katiuscia Martinello[3]
[1]CNRS, Biophysics Institute, Palermo, Italy; [2]Neuroscience Physiology and Pharmacology, University College London, Neuroscience Physiology and Pharmacology, London, United Kingdom; [3]UCL School of Pharmacy, University College London, School of Pharmacy, London, United Kingdom
Correspondence: Elisabetta Giacalone (elisabetta.giacalone@pa.ibf.cnr.it)
BMC Neuroscience 2019, **20(Suppl 1)**:P195

In hippocampus, a type of slowly-activated and non-inactivating K+ channels, belonging to the Kv7 family, are highly localized on initial segments of myelinated and unmyelinated axons where they influence neuronal excitability. Interestingly, immunohistochemistry shows that the Kv7.2 and Kv7.3 subunits are localized throughout hippocampal mossy fibers. Electrophysiological recordings from mature synaptic boutons showed that the Kv7/M- current is also present here, is active at rest and enhances the membrane conductance. The current also reduces the spike half-width and after depolarization (ADP) following a presynaptic spike. This is likely to have significant consequences for the modulation of excitatory neurotransmitter release from these boutons, and thus signal transmission, at DG-CA3 synapses.
In this poster, using a biophysical computational model of a mossy fiber bouton (MFB), we will discuss the mechanisms underlying the ADP observed after Kv7 channels block. The model is able to reproduce a number of experimental findings under control and after Kv7 current block by XE991. The results suggest that Kv7 conductance limits spike-induced rise in Ca2+concentration and regulates spike width and ADP amplitude. The model suggests that the ADP is caused by a relatively slow Ca2+-dependent mechanism, which can be conveniently modelled as a slow deactivation time constant of the Ca2+current in MFB. This is a new feature that has not been previously observed experimentally. Taken together, these results suggest that presynaptic Kv7 channels expression at the mossy fiber-CA3 synapse may have an important role in modulating synaptic transmission and signal coding in the hippocampus network.

P196

Characterization of the network dynamics of interconnected brain regions on-a-chip

Martina Brofiga, Paolo Massobrio, Sergio Martinoia
University of Genova, Department of Informatics, Bioengineering, Robotics, System Engineering (DIBRIS), Genova, Italy
Correspondence: Martina Brofiga (martina.brofiga@dibris.unige.it)
BMC Neuroscience 2019, **20(Suppl 1)**:P196

Neuronal networks are composed of different cell types precisely arranged into organizational schemes and connected via complex

BMC Neurosci 2019, **20**(Suppl 1):56
https://doi.org/10.1186/s12868-019-0538-0

BMC Neuroscience

MEETING ABSTRACTS

Open Access

28th Annual Computational Neuroscience Meeting: CNS*2019

Barcelona, Spain. 13–17 July 2019

Published: 14 November 2019

K1

Brain networks, adolescence and schizophrenia

Ed Bullmore
University of Cambridge, Department of Psychiatry, Cambridge, United Kingdom
Correspondence: Ed Bullmore (etb23@cam.ac.uk)
BMC Neuroscience 2019, **20(Suppl 1)**:K1

The adolescent transition from childhood to young adulthood is an important phase of human brain development and a period of increased risk for incidence of psychotic disorders. I will review some of the recent neuroimaging discoveries concerning adolescent development, focusing on an accelerated longitudinal study of ~300 healthy young people (aged 14–25 years) each scanned twice using MRI. Structural MRI, including putative markers of myelination, indicates changes in local anatomy and connectivity of association cortical network hubs during adolescence. Functional MRI indicates strengthening of initially weak connectivity of subcortical nuclei and association cortex. I will also discuss the relationships between intra-cortical myelination, brain networks and anatomical patterns of expression of risk genes for schizophrenia.

K2

Neural circuits for mental simulation

Kenji Doya
Okinawa Institute of Science and Technology, Neural Computation Unit, Okinawa, Japan
Correspondence: Kenji Doya (doya@oist.jp)
BMC Neuroscience 2019, **20(Suppl 1)**:K2

The basic process of decision making is often explained by learning of values of possible actions by reinforcement learning. In our daily life, however, we rarely rely on pure trial-and-error and utilize any prior knowledge about the world to imagine what situation will happen before taking an action. How such "mental simulation" is implemented by neural circuits and how they are regulated to avoid delusion are exciting new topics of neuroscience. Here I report our works with functional MRI in humans and two-photon imaging in mice to clarify how action-dependent state transition models are learned and utilized in the brain.

K3

One network, many states: varying the excitability of the cerebral cortex

Maria V. Sanchez-Vives
IDIBAPS and ICREA, Systems Neuroscience, Barcelona, Spain
Correspondence: Maria V. Sanchez-Vives (msanche3@clinic.cat)
BMC Neuroscience 2019, **20(Suppl 1)**:K3

In the transition from deep sleep, anesthesia or coma states to wakefulness, there are profound changes in cortical interactions both in the temporal and the spatial domains. In a state of low excitability, the cortical network, both in vivo and in vitro, expresses it "default activity pattern", slow oscillations [1], a state of low complexity and high synchronization. Understanding the multiscale mechanisms that enable the emergence of complex brain dynamics associated with wakefulness and cognition while departing from low-complexity, highly synchronized states such as sleep, is key to the development of reliable monitors of brain state transitions and consciousness levels during physiological and pathological states. In this presentation I will discuss different experimental and computational approaches aimed at unraveling how the complexity of activity patterns emerges in the cortical network as it transitions across different brain states. Strategies such as varying anesthesia levels or sleep/awake transitions in vivo, or progressive variations in excitability by variable ionic levels, GABAergic antagonists, potassium blockers or electric fields in vitro, reveal some of the common features of these different cortical states, the gradual or abrupt transitions between them, and the emergence of dynamical richness, providing hints as to the underlying mechanisms.

Reference

1. Sanchez-Vives, M, Marcello M, Maurizio M. Shaping the default activity pattern of the cortical network. *Neuron* 94.5 (2017): 993–1001.

K4

Neural circuits for flexible memory and navigation

Ila Fiete
Massachusetts Institute of Technology, McGovern Institute, Cambridge, United States of America
Correspondence: Ila Fiete (fiete@mit.edu)
BMC Neuroscience 2019, **20(Suppl 1)**:K4

I will discuss the problems of memory and navigation from a computational and functional perspective: What is difficult about these problems, which features of the neural circuit architecture and dynamics enable their solutions, and how the neural solutions are uniquely robust, flexible, and efficient.

F1

The geometry of abstraction in hippocampus and pre-frontal cortex

Silvia Bernardi[1], Marcus K. Benna[2], Mattia Rigotti[3], Jérôme Munuera[4], Stefano Fusi[1], C. Daniel Salzman[1]
[1]Columbia University, Zuckerman Mind Brain Behavior Institute, New York, United States of America; [2]Columbia University, Center for Theoretical Neuroscience, Zuckerman Mind Brain Behavior Institute, New York, NY, United States of America; [3]IBM Research AI, Yorktown Heights, United States of America, [4]Columbia University, Centre National de la Recherche Scientifique (CNRS), École Normale Supérieure, Paris, France
Correspondence: Marcus K. Benna (mkb2162@columbia.edu)
BMC Neuroscience 2019, **20(Suppl 1)**:F1