
CNS 2016
25th Annual Computational Neuroscience Meeting,
July 2-7, Jeju Island, South Korea
http://www.cnsorg.org/

Contents

We are grateful to the following organizations for their support

without which none of this would be possible:

Overview

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OCNS, Inc is a US non-profit, 501(c)(3) serving organization supporting the Computational Neuroscience community internationally. We seek sponsorship from corporate and philantropic organizations for support of student travel and registration to the annual meeting, student awards and hosting of topical workshops. We can also host booth presentations from companies and book houses. For further information on how you can contribute please email http://sponsorship@cnsorg.org.

General Information

Meeting venue

Jungmun Sightseeing Complex
Seogwipo City, Jeju-do
South Korea
http://www.iccjeju.co.kr/EN/Main
Phone +82-64-735-1000

CNS 2016 will be held in the **International Convention Center Jeju (ICC Jeju), South Korea**. ICC Jeju is located in Jungmun Tourist Complex in Jeju island, which is a volcanic island and a world renowned resort in South Korea. Main Meeting, Poster session, and Banquet will be held on the 3rd floor in ICC Jeju. The meeting rooms for the tutorials and workshop will be on 3rd and 4th floor.

main conference hall (~ 600)

meeting room

meeting room

VIP Room

VIP room is prepared for invited speakers. The location of the VIP room is 304 on the third floor.

Getting to the conference venue

Direct International Flights to Jeju island

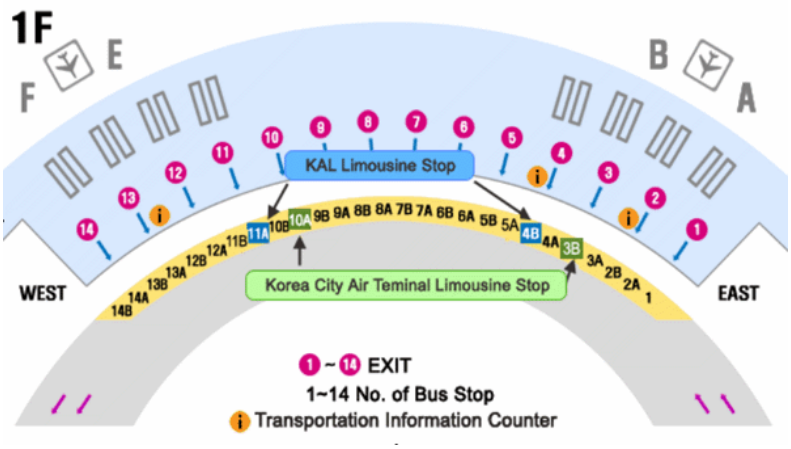
The most convenient way to travel to Jeju Island is by airplane. The flights link it with China, Japan, Taiwan and all major Korean cities are within one-hour flight. Jeju International Airport is conveniently located at the center of East Asia and is easily accessible from China, Japan, and Southeast Asia. The airport currently services 16 direct international flights and 13 domestic flights.

Transfer via Major Hub Airports

Visitors from countries without direct flights may easily travel to Jeju through major airports such as those in Tokyo, Osaka, Beijing, and Shanghai.

Transfer via Incheon International Airport, Korea

Incheon International Airport is the gateway to Korea. It was ranked the 9th highest international passenger traffic with 79 airlines connecting to 182 destination cities. Passengers can either transfer to a direct flight to Jeju from Incheon, or move to Gimpo Airport and transfer to a domestic flight from there. It is a one-hour-flight from Incheon or Gimpo to Jeju. To get to Gimpo Airport from Incheon, visitors can take a shuttle bus or high-speed rail. It takes approximately 30 minutes, and shuttle buses run every 5 minutes.



Transfer via Gimhae International Airport, Korea

You can access Gimhae Int'l Airport by international flights form China (Shanghai), Japan (Tokyo, Fukuoka, Nagoya and Osaka), Russia, Taiwan, Thailand, The Philippines and Vietnam. The domestic flights from Gimhae Int'l Airport, which is the second largest international airport in Korea, to Jeju Int'l Airport run between 08:00 to 22:00 at roughly 15 minutes intervals. 40 domestic flights are in service daily on average as of December 2013.

Visa-Free Entry

Visa-free Entry to Jeju: 180 Countries. (Only 11 countries are required to apply for a visa: Afghanistan, Cuba, Ghana, Iran, Iraq, Libya, Nigeria, Macedonia, Palestine, Sudan, and Syria). Duration of stay: up to 30 days

From Jeju International Airport to ICC Jeju (and hotels)

Take an Airport Limousine Bus from Jeju Int'l Airport to ICC Jeju and conference hotels. The limousine buses [Bus No. 600] run between 06:20 to 22:00 at roughly 15 minutes intervals, and the cost is KRW4,000 (about USD 3.5). It takes about 50∼60 minutes.

Taxi guide (Jungmun - Jeju International Airport)

Since the taxi fare is fixed, please confirm the distance before taking a taxi. Fare (Korean won): About 30,000 won (Maximum USD 30); Distance: 40km; Duration: 40–45 minutes. Advanced reservation is recommended if you plan to rent a car. Upon arrival, you can sign up for rental cars at the rent-a-car desk nearby airport exit.

Local information

Good to Know
Detailed local information is available on the Jeju tourism organization website at http://www.ijto.or.kr/english/

Official Language
The official language of the meeting is English. Interpreting is not provided.

Insurance
The organisers do not accept responsibility for individual medical, travel or personal insurance. All participants are strongly advised to take out their own personal insurance before travelling to Jeju.

Currency & Banking
Korea's official monetary unit is the won. Credit Card: Most of the businesses in Korea widely uses and accepts payment by credit cards at major hotels, department stores, large restaurants, and stores. Visa, Master, American Express and other credit cards can be used; however do check the service availability before making purchase as some stores may not be subject to this service. Exchanging Money: When you need to exchange your foreign currency into Korean won, visit a bank, exchange service center, or an authorized exchange. (Bank business hour: 09:00–16:00, closed on Saturdays, Sundays and public holidays.)
Currency Converter: www.xe.com/currencyconverter (Korean, English, Japanese, Chinese)

Time Zone
Korea Standard Time (KST) is 9 hours ahead of Greenwich mean time: GMT+9.

Electricity
The standard voltage in Korea is 220 volts. The outlet has two round holes and is the same type used in France, Germany, Austria, Greece, Turkey, and many other countries.

Weather
Jeju island has a mild oceanic climate throughout the year with the smallest annual temperature range in South Korea. The temperature for the hottest summer months (in July) averages no more than 33.0°C in and no less

than -2.7°C for winter. During the meeting in early July, the lowest temperature ranges from 21-23°C and the hottest temperature ranges between 26-28°C. (http://www.iccjeju.co.kr/EN/AboutJeju/Info)

Tourist Spots
With its natural beauty and unique island culture, Jeju provides with hundreds of tour options including pristine seas and fantastic rock cliffs, horses grazing on wide green meadows, and a variety of specialty museums. The island itself is an extinct volcano with its peak jutting skyward at the center and a broad, gentle littoral all the way around showing a very unique geographical condition. There are bountiful forests and ravines, fantastic rock formations and volcanic craters, and caves and grasslands that together paint a natural scene of breathtaking beauty. Sparkling seas and tiny islets surround Jeju, with rocks scattering amidst sandy beaches to create a magnificent view everywhere you look. Hallasan Mountain rises in the center of Jeju to 1950m above sea level. The rest of the island slopes down from its summit and is covered with dark gray volcanic rocks and volcanic ash soil. Relatively isolated from the rest of the world, the island's nature has been well preserved in its prehistoric state. http://english.jeju.go.kr/index.php/contents/AboutJeju/intro/intro.

Leisure
The island also offers a variety of leisure activities; snorkelling, scuba diving, kayaking, yachting, windsurfing hiking, golfing, fishing, horseback riding and paragliding, are available at numerous places throughout the island. Near Jungmun Resort Complex, where ICC Jeju is located, there are a variety of sight-seeing opportunities such as; the public 18-hole Jungmun Golf Course (about a 5-minute-ride), Yeomiji Botanical Garden (the largest indoor botanical garden in Asia), the Teddy Bear Museum, the Africa Museum, the Sound Island Museum, and Pacific Land. Scenic natural wonders include: Jungmun Beach, Cheonjeyeon Waterfall (according to the legend, seven nymphs guarding the heavenly emperor descended at night to bathe at night), and Jusangjeolli (natural stone pillars built up along the coast which are formed by magma piercing through cracks of the surface-rock. (http://www.iccjeju.co.kr/EN/AboutJeju/TourAndLeisure)

Welcome Reception

The welcome reception will be provided at the 6:30 pm of July 2 (Saturday), the first day of the conference, where finger foods and drinks are freely served at the "Ocean View" on the 5th floor in the Convention center (Jeju ICC). Additional order for wine will be charged. Attendees can enjoy social interactions with other participants during the reception.

Banquet

On the fourth day of the conference (July 5, Tuesday), we will prepare the banquet at 6:30 pm at the "Tamna Hall B" on the 5th floor in the Convention center (Jeju ICC). Dinner and wine will be served to the attendees who registered with the option of banquet included. Attendees can also purchase the banquet tickets for their companion when they pre-registered. If you havenâ€™t already purchased a ticket during registration, you can add one to your registration.
The banquet ticket will cost USD 50 per person. In the beginning of the banquet, the organizing committee will prepare a welcome ceremony. Then, there will be a celebration event with the performance of Korean traditional music and arts. During the event, the dinner and wine will be served.

Social night out

In the evening of the third day of the conference (6:30 pm, July 4, Monday), a social party will be prepared at the "Ocean View" on the 5th floor in the Convention center (Jeju ICC) where attendees can enjoy drinks and food with social interactions. Attendees purchase tickets for additional food and drinks on site.

Restaurant info

The Delizia restaurant is located on the third floor at the convention enter. It serves both Korean and Western cuisine, www.iccjeju.co.kr/Facilities/Delizia. You can have lunch or dinner here during the conference. Korean and Western restaurants are also in Booyoung Hotel which is just next to Jeju ICC. There are many restaurants within 10 mins walking distance from Jeju ICC.

Program

Tutorials

T1 **Subcellular modeling**

301A + 301B, 2-Jul-15, 9:00–16:30

Iain Hepburn, Okinawa Institute of Science and Technology, Japan
Andrew R Gallimore, Okinawa Institute of Science and Technology, Japan

T2 **Detailed modeling of structure and function at the cellular level**

302, 2-Jul-15, 9:00–16:30

Ben Torben-Nielsen, University of Hertfordshire, UK
Yann Le Franc, e-Science Data Factory, France

T3 **Simulation of large-scale neural network**

303A + 303B, 2-Jul-15, 9:00–16:30

Jun Igarashi, RIKEN and Okinawa Institute and Science and Technology, Japan
Hannah Bos, Julich Research Centre and JARA, Julich, Germany

T4 **Nonlinear dynamical analysis of brain datasets**

402A, 2-Jul-15, 9:00–12:00

Jaeseung Jeong, Korea Advanced Institute of Science and Technology, South Korea

T5 **Modeling and analysis of extracellular potentials**

402B, 2-Jul-15, 9:00–16:30

Gaute T. Einevoll, Norwegian University of Life Sciences & University of Oslo, Norway
Espen Hagen, Julich Research Centre and JARA, Julich, Germany

Main Meeting

Saturday July 2

9:00 –		**Registration (Halla Hall, 3rd floor)**
9:00 – 16:30		**Tutorials**
17:00 – 17:15		**Welcome and announcements**
17.15 – 18:15	K1	**Keynote 1: (Halla Hall)** ***Inferring learning rules in cortical circuits*** Nicolas Brunel
18:30		**Welcome reception (Ocean View, 5th floor)**

Sunday July 3

9:00 – 9:10		**Announcements**
9:10 – 10:10	K2	**Keynote 2:** ***Functional advantages of cell-type heterogeneity in neural circuits*** Tatyana O. Sharpee
10:10 – 10:40		**Break**

Oral session I: Oscillations and rhythms 1

10:40 – 11:00 O1 ***Assessing irregularity and coordination of spiking-bursting rhythms in central pattern generators***
Irene Elices*, David Arroyo, Rafael Levi, Francisco B. Rodriguez, and Pablo Varona

11:00 – 11:20 O2 ***Regulation of top-down processing by cortically-projecting parvalbumin positive neurons in basal forebrain***
Eunjin Hwang, Bowon Kim, Hio-Been Han, Tae Kim, James McKenna, Ritchie Brown, Robert W. McCarley, and Jee Hyun Choi*

11:20 – 12:00 F1 **Featured oral 1:**
Precise recruitment of spiking output at theta frequencies requires dendritic h-channels in multi-compartment models of hippocampal interneurons
Vladislav Sekulic*, Frances Skinner

12:00 – 13:30 **Break for lunch**

Oral session II: Visual and auditory processing

13:30 – 13:50 O3 ***Modeling auditory stream segregation, build-up and bistability***
James Rankin*, Pamela Osborn Popp, and John Rinzel

13:50 – 14:10 O4 ***Strong competition between tonotopic neural ensembles explains pitch-related dynamics of auditory cortex evoked fields***
Alejandro Tabas*, André Rupp, and Emili Balaguer-Ballester

14:10 – 14:30 O5 ***A simple model of retinal response to multi-electrode stimulation***
Matias Maturana, David B Grayden, Shaun Cloherty, Tatiana Kameneva, Michael Ibbotson, and Hamish Meffin*

14:30 – 14:50 O6 ***Noise correlations in V4 area correlate with behavioral performance in visual discriminaton task***
Veronika Koren*, Timm Lochmann, Valentin Dragoi, and Klaus Obermayer

14:50 – 15:20 **Break**

Oral session III: Single-cell properties and modeling

15:20 – 15:40 O7 ***Input-location dependent gain modulation in cerebellar nucleus neurons***
Maria Psarrou*, Maria Schilstra, Neil Davey, Ben Torben-Nielsen, and Volker Steuber

15:40 – 16:00 O8 ***Analytic solution of cable energy function for cortical axons and dendrites***
Huiwen Yu, Michael L. Hines, and Yuguo Yu*

16:00 – 19:00 **Poster session I: Posters P1 – P68 (3F lobby, 3rd floor)**

Monday July 4

9:00 – 9:10 **Announcements**

9:10 – 10:10 K3 **Keynote 3:**
Mesoscopic modeling of propagating waves in visual cortex
Alain Destexhe

10:10 – 10:40 **Break**

Oral session IV: Network reconstruction, estimation and visualization

10:40 – 11:00 O9 ***C. elegans Interactome: Interactive Visualization of Caenorhabditis elegans Worm Neuronal Network***
Jimin Kim*, Will Leahy, and Eli Shlizerman

11:00 – 11:20 O10 ***Is the Model Any Good? Objective Criteria for Computational Neuroscience Model Selection***
Justas Birgiolas*, Richard Gerkin, and Sharon Crook

11:20 – 12:00 F2 **Featured oral 2:**
Kernel methods in reconstruction of current sources from extracellular potentials for single cells and the whole brains
Daniel K Wojcik*, Chaitanya Chintaluri, Dorottya Cserpan, and Zoltan Somogyvari

12:00 – 13:30 **Break for lunch**

Oral session V: Oscillations and rhythms 2

13:30 – 13:50 O11 ***Cooperation and competition of gamma oscillation mechanisms***
Atthaphon Viriyopase*, Raoul-Martin Memmesheimer, and Stan Gielen

13:50 – 14:10 O12 ***A discrete structure of the brain waves***
Yuri Dabaghian*, Justin Devito, and Luca Perotti

14:10 – 14:50 F3 **Featured oral 3:**
The synchronized periods depend on intracellular transcriptional repression mechanisms in circadian clocks.
Jae Kyoung Kim*, Zachary Kilpatrick, Matthew Bennett, and Kresimir Josic

14:50 – 15:20 **Break**

Oral session VI: Synaptic plasticity

15:20 – 15:40 O13 ***Direction-specific silencing of the Drosophila gaze stabilization system***
Anmo Kim*, Lisa Fenk, Cheng Lyu, and Gaby Maimon

15:40 – 16:00 O14 ***What fruit fly think about values? —A model about olfactory associative learning***
Chang Zhao*, Yves Widmer, Simon Sprecher, and Walter Senn

16:00 – 18:30 **Poster session II: Posters P69 – P135 (3F lobby, 3rd floor)**

18:30 **Social night out (Ocean View, 5th floor)**

Tuesday July 5

9:00 – 9:10 **Announcements**

9:10 – 10:10 K4 **Keynote 4:**
Dynamics and Biomarkers of Mental Disorders
Mitsuo Kawato

10:10 – 10:40 **Break**

Oral session VII: Large networks

10:40 – 11:00 O15 ***Effects of ionic diffusion on power spectra of local field potentials (LFP)***
Geir Halnes*, Tuomo Mäki-Marttunen, Daniel Keller, Klas H. Pettersen, Ole Andreassen, and Gaute T. Einevoll

11:00 – 11:20 O16 ***Large-scale cortical models towards understanding relationship between brain structure abnormalities and cognitive deficits***
Yasunori Yamada*

11:20 – 11:40 O17 ***Spatial coarse-graining the brain: Origin of minicolumns***
Moira Steyn-Ross*, Alistair Steyn-Ross

11:40 – 12:00 O18 ***Modeling large-scale cortical networks with laminar structure***
Jorge F Mejias*, John Murray, Henry Kennedy, and Xiao-Jing Wang

12:00 – 13:30 **Break for lunch**

13:30 – 14:20 **OCNS Member Meeting, room 400**

Oral session VIII: Information theory

14:20 – 14:40 O19 ***Information filtering by partial synchronous spikes in a neural population***
Alexandra Kruscha*, Jan Grewe, Jan Benda, and Benjamin Lindner

14:40 – 15:00 O20 ***Decoding context-dependent olfactory valence in Drosophila***
Laurent Badel*, Kazumi Ohta, Yoshiko Tsuchimoto, and Hokto Kazama

15:00 – 15:30 **Break**

15:30 – 18:30 **Poster session III: Posters P136 – P201 (3F lobby, 3rd floor)**

18:30 **Banquet (Tamna Hall B, 5th floor)**

Wednesday July 6 and Thursday July 7

9:00 – 19:00 **Workshops**

Workshops

W1 **Methods of Information Theory in Computational Neuroscience**

301A + 301B, Wednesday and Thursday, 9:00 – 16:30

Joseph T Lizier, The University of Sydney
Justin Dauwels, Nanyang Technological University
Taro Toyoizumi, RIKEN Brain Science Institute
Alexander G Dimitrov, Washington State University
Lubomir Kostal, Academy of Sciences of the Czech Republic

W2 **Connectome: Structure and Large Scale Dynamics**

302, Wednesday, 9:00 – 16:30

Leonardo L Gollo, QIMR Berghofer Medical Research Institute, Australia
James A. Roberts, QIMR Berghofer Medical Research Institute, Australia

W3 **Statistical Analysis for Neural Time Series**

302, Thursday, 9:00 – 16:30

Il Memming Park, Stony Brook University
Ian Stevenson, University of Connecticut

W4 **Multi-Area Models of Cortex**

402A, Thursday, 9:00 – 16:30

Sacha Jennifer van Albada, Institute of Neuroscience and Medicine (INM-6) Computational and Systems Neuroscience and Institute for Advanced Simulation (IAS-6) Theoretical Neuroscience and JARA BRAIN Institute I, Julich Research Centre, Julich, Germany
Gustavo Deco, Center for Brain and Cognition, Computational Neuroscience Group, Department of Information and Communication Technologies & Institució Catalana de la Recerca i Estudis Avançats (ICREA), Universitat Pompeu Fabra, Barcelona, Spain

W5 **Dynamical principles in Neural circuits**

402A, Wednesday, 9:00 – 12:00

Andrey Shilnikov, Georgia State University, USA
Akira Sakurai, Georgia State University, USA

W6 **Cortical Microcircuits: Understanding network structure and function in cortical processing**

303A + 303B, Wednesday, 9:00 – 12:00

Hamish Meffin, National Vision Research Institute, and Department of Optometry and Visual Science, The University of Melbourne
Anthony Burkitt, Department of Electrical and Electronic Engineering, The University of Melbourne

W7 **Recent advances and applications in real-time single-trial EEG analysis**

303A + 303B, Wednesday, 13:30 – 16:30

Tzyy-Ping Jung, University of California, San Diego
John K. Zao, Chiao-Tung University
Jee Hyun Choi, Korea Institute of Science and Technology

Abstracts

Tutorials

T1 Subcellular modeling

301A + 301B, 2-Jul-15, 9:00–16:30

Iain Hepburn, Okinawa Institute of Science and Technology, Japan
Andrew R Gallimore, Okinawa Institute of Science and Technology, Japan

Many important neural functions are controlled by complex networks of intracellular proteins and signalling molecules. A variety of modular signalling pathways connect and interact to form large networks possessing emergent properties irreducible to individual molecules or pathways. These include bistable and ultrasensitive switches, as well as feedback regulation, and synchronisation. These properties are essential for the induction and regulation of critical neural functions, such as long-term depression and potentiation. The complexity of these networks renders their analysis by inspection alone unfeasible, and we must turn to computational modelling to understand them.

The first half of this tutorial will focus on the structure and function of intracellular networks and deterministic methods for modelling and analysing them. We will use a number of important subcellular pathways to illustrate the key concepts and demonstrate the importance and utility of deterministic methods in their modelling and simulation. We will discuss both the biochemistry of these pathways and their mathematical representation. We will then discuss how these modular pathways connect and interact to form large networks. Important network motifs and their emergent properties will also be explained with specific examples given, as well as mathematical methods for their analysis. We will discuss a number of tools for simulating these differential equation models, but will use the open source software Copasi in the tutorial, owing to its ease of installation and use. Participants will have the opportunity to build and simulate their own signalling pathway model in Copasi. This part of the tutorial will serve as a good introduction to molecular systems modelling for those with little prior experience.

The second half of the tutorial will focus on more advanced modelling approaches based on several state of the art software packages. We will explain how the time evolution of real molecular systems can diverge from a differential equation-based description due to concepts such as probabilistic interactions in small volumes and spatial heterogeneity. We will describe mathematical approaches to modelling stochastic effects and diffusion and introduce a number of software tools that are based on such descriptions. These include particle-tracking packages such as MCell and Smoldyn, and voxel-based packages such as NeuroRD and STEPS. The features of the different software tools will be discussed and illustrated with specific practical examples. Finally, we will briefly discuss recent advances and expected near-future directions of the field, including massively parallel implementations and membrane potential coupling.

References

[1] Antunes, G., De Schutter, E. A Stochastic Signaling Network Mediates the Probabilistic Induction of Cerebellar Long-Term Depression. Journal of Neuroscience 32, 9288-9300, 2012.

[2] Bhalla, U.S., Iyengar, R. Emergent properties of networks of biological signaling pathways. Science 283, 381-387, 1999.

[3] Eungdamrong, N.J., Iyengar, R. Computational approaches for modeling regulatory cellular networks. Trends in Cell Biology 14, 661-669, 2004.

[4] Gallimore, A.R., Aricescu, A.R., Yuzakl, M., Calinescu, R. A Computational Model for the AMPA Receptor Phosphorylation Master Switch Regulating Cerebellar Long-Term Depression. Plos Computational Biology 12, 23, 2016.

[5] Kotaleski, J.H., Blackwell, K.T. Modelling the molecular mechanisms of synaptic plasticity using systems biology approaches. Nature Reviews Neuroscience 11, 239-251, 2010.

[6] Copasi: http://copasi.org/

[7] SimBiology (Matlab): http://uk.mathworks.com/products/simbiology/

[8] Genesis: http://www.genesis-sim.org/

[9] STEPS: http://steps.sourceforge.net/STEPS/default.php

[10] MCell: http://mcell.org/

[11] Smoldyn: http://www.smoldyn.org/

[12] NeuroRD: http://krasnow1.gmu.edu/CENlab/software.html

T2 Detailed modeling of structure and function at the cellular level

302, 2-Jul-15, 9:00–16:30

Ben Torben-Nielsen, University of Hertfordshire, UK
Yann Le Franc, e-Science Data Factory, France

In the morning session, we introduce the morphology of dendrites and axons, the specialised input and output arborisations of neurons. Their shape is pivotal for brain functioning for two reasons: First, overlap between dendrites and axons defines the micro-circuit. Second, the shape and membrane composition of dendrites define how inputs are transformed into relevant outputs. In this tutorial, we will start by explaining the importance of morphologies and how to quantify them (say, in order to distinguish healthy from pathological morphologies). We will touch on algorithmic synthesis of large numbers of unique neuronal morphologies for application in large-scale modelling efforts. We finish the morning session with a hands-on tutorial using btmorph [1] to analyse populations of neuronal morphologies.

In the afternoon session, we explain how neuronal dynamics takes place at the single neuron level and how dendrites turn input signals into an output. We briefly explain the conductance-based and compartmental-modelling paradigms to simulate the dynamics on neurons with detailed membrane composition and elaborate neuronal morphologies. We then proceed to show several free community resources to construct, simulate, share and analyse single neuron models. We end the afternoon session with a hands-on demonstration of how to construct and simulate detailed models of neurons using NEURON and python [2].

References

[1] Torben-Nielsen B. An efficient and extendable Python library to analyze neuronal morphologies. Neuroinformatics 12:619-622, 2014 .

[2] James G.K., Hines M., Hill S., Goodman P.H., Markram H.,1 Schürmann F. Component-Based Extension Framework for Large-Scale Parallel Simulations in NEURON. Front. Neuroinformatics, 3:1-12, 2009.

[3] Torben-Nielsen B., Cuntz H. Introduction to dendritic morphology, The computing dendrite, Springer, 2014.

[4] London M., Häusser M. Dendritic computation. Annu Rev Neurosci. 28:503-32, 2005.

[5] Parekh R., Ascoli G. Neuronal Morphology Goes Digital: A Research Hub for Cellular and System Neuroscience. Neuron 77(6): 1017–1038, 2013.

[6] Silver A. Neuronal arithmetic. Nature Reviews Neuroscience 11, 474-489, 2010.

T3 **Simulation of large-scale neural network**

303A + 303B, 2-Jul-15, 9:00–16:30

Jun Igarashi, RIKEN and Okinawa Institute and Science and Technology, Japan
Hannah Bos, Julich Research Centre and JARA, Julich, Germany

The first part of this tutorial is concerned with the emergence of large scale neuronal networks in neuroscience and the resulting challenges in software and hardware that are necessary to support large scale simulations. We will start by an introduction covering the development of networks examined in neuroscience and give an overview over existing large scale models. Subsequently we will give an overview over the history of supercomputers used for simulations of large scale networks. The introduction is followed by two lectures going into detail of the implementation of neuronal networks shedding light on the software as well as the hardware aspects. We will first discuss how a neural simulator can be implementation on the example of NEST [1]. The lecture concerned with the hardware aspect will introduce how calculation of neural network simulation is executed using processors and memory in a computer, with a story of recent representative semi-conductor chips and supercomputers. The second part of the tutorial focuses on hands-on exercise using NEST. The tutorial does not assume any prior knowledge in NEST. However, it is recommended that participants install NEST on their laptops beforehand [2]. We will start by introducing the basic commands of NEST and work our way up to the implementation of a random balanced network [3, 4]. The session is planned as an interactive mixture of lectures and exercise. At the end a final lecture on a basal ganglia-thalamo-cortical circuit model that helps to understand Parkinson'â€™s disease motor symptoms, will introduce an example of a large-scale network in more detail

References

[1] Gewaltig MO, Diesmann M. NEST (NEural Simulation Tool). Scholarpedia.2007;2(4):1430.

[2] http://www.nest-simulator.org/installation/

[3] Brunel N. Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons. J Comput Neurosci. 2000;8(3):183–208.

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T4 Nonlinear dynamical analysis of brain datasets

402A, 2-Jul-15, 9:00–12:00

Jaeseung Jeong, Korea Advanced Institute of Science and Technology, South Korea

Nonlinear dynamical analysis is an advanced method to analyze the time series based on the hypothesis that the time series is generated by nonlinear dynamical processes. This method reveals dynamical properties of the time series including dimensional complexity, sensitive dependence on initial conditions, dynamical nonstationarity that cannot be assessed by conventional linear spectral methods. For last three decades, nonlinear dynamical analysis of neural signals and the EEG has been used to successfully describe neuronal dynamics and diagnose neuropsychiatric disorders such as Alzheimer's disease, Epileptic seizure, Schizophrenia, Depression, Addiction, Post-traumatic stress disorder (PTSD), and Attention-deficit Hyperactivity disorder (ADHD) and to suggest potential treatments for them based on their disturbed brain dynamics.

In this tutorial, we introduce the basic ideas underlying the nonlinear dynamical analysis and define important concepts addressed in this analysis (e.g., Deterministic chaos, Embedding theorem, Delay coordinates, Surrogate data, etc.). Then, we briefly review main findings of neuronal signals and EEG abnormalities in various neuropsychiatric patients obtained from both conventional spectral analysis and nonlinear dynamical methods. Particularly, we address how nonlinear dynamical methods prominently contribute to Neuroscience, Psychiatry and Neurology as a biomarker of brain dynamics and a tool for diagnosing Alzheimer's disease, Schizophrenia, Epileptic seizure, and Attention-deficit Hyperactivity disorder (ADHD) in detail.

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T5 Modeling and analysis of extracellular potentials

402B, 2-Jul-15, 9:00–16:30

Gaute T. Einevoll, Norwegian University of Life Sciences & University of Oslo, Norway
Espen Hagen, Julich Research Centre and JARA, Julich, Germany

While extracellular electrical recordings have been one of the main workhorses in electrophysiology, the interpretation of such recordings is not trivial [1,2,3]. The recorded extracellular potentials in general stem from a complicated sum of contributions from all transmembrane currents of the neurons in the vicinity of the electrode contact. The duration of spikes, the extracellular signatures of neuronal action potentials, is so short that the high-frequency part of the recorded signal, the multi-unit activity (MUA), often can be sorted into spiking contributions from the individual neurons surrounding the electrode [4]. No such simplifying feature aids us in the interpretation of the low-frequency part, the local field potential (LFP). To take a full advantage of the new generation of silicon-based multielectrodes recording from tens, hundreds or thousands of positions simultaneously, we thus need to develop new data analysis methods grounded in the underlying biophysics [1,3,4]. This is the topic of the present tutorial.

In the first part of this tutorial we will go through

1. the biophysics of extracellular recordings in the brain,
2. a scheme for biophysically detailed modeling of extracellular potentials and the application to modeling single spikes [5-7], MUAs [8] and LFPs, both from single neurons [9] and populations of neurons [8,10,11],
3. methods for estimation of current source density (CSD) from LFP data, such as the iCSD [12-14] and kCSD methods [15],
4. decomposition of recorded signals in cortex into contributions from various laminar populations, i.e., (i) laminar population analysis (LPA) [16,17] based on joint modeling of LFP and MUA, or (ii) a scheme using LFP and known constraints on the synaptic connections [18].

In the second part, the participants will get demonstrations and, if wanted, hands-on experience with

1. LFPy (github.com/LFPy) [19], a versatile tool based on Python and the simulation program NEURON [20] (www.neuron.yale.edu) for calculation of extracellular potentials around neurons, and
2. new results from applying the biophysical forward-modelling scheme to predict LFPs from comprehensive point-neuron network models, in particular Potjans and Diesmann's model of the early sensory cortical microcircuit using hybridLFPy [22,23] will be presented.

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Invited Presentations

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K1 – Inferring learning rules in cortical circuits

Understanding the mechanisms of learning and memory is one of the major challenges in neuroscience. The dominant theory holds that information about sensory inputs is stored in cortical circuits thanks to synaptic plasticity. In spite of decades of research, the exact rules governing how synapses change as a function of the activity of pre- and post-synaptic neurons remain the subject of debate. In this talk, I will present two novel approaches for investigating the mechanisms of learning and memory. The first consists in inferring a learning rule from in vivo data, using experiments comparing the statistics of responses of neurons to large sets of novel and familiar stimuli. The second consists in exploring the consequences of an information optimization principle on the statistics of synaptic connectivity. I will show how methods from statistical physics can be used to characterize the statistics of connectivity in networks that optimize information storage, and compare the theoretical results with available data.

Tatyana O. Sharpee *Computational Neurobiology Laboratory*
The Salk Institute for Biological Studies,
San Diego, California, USA

K2 – Functional advantages of cell-type heterogeneity in neural circuits

Neural circuits are notorious for the complexity of their organization. Part of this complexity is related to the number of different cell types that work together to encode stimuli. I will discuss theoretical results that point to functional advantages of splitting neural populations into subtypes, both in feedforward and recurrent networks. There results outline a framework for categorizing neuronal types based on their functional properties. Such classification scheme could augment classification schemes based on molecular, anatomical, and electrophysiological properties.

Alain Destexhe *UNIC, CNRS, Gif sur Yvette,*
and The European Institute for Theoretical Neuroscience (EITN),
Paris, France

K3 – Mesoscopic modeling of propagating waves in visual cortex

Propagating waves are large-scale phenomena widely seen in the nervous system, in both anesthetized and awake or sleeping states. Recently, the presence of propagating waves at the scale of microns to millimeters was demonstrated in the primary visual cortex (V1) of macaque monkey. Using a combination of voltage-sensitive dye (VSD) imaging in awake monkey V1 and model-based analysis, we showed that virtually every visual input is followed by a propagating wave (Muller et al., Nat Comm 2014). The wave was confined within V1, and was consistent and repeatable for a given input. Interestingly, two propagating waves always interact in a suppressive fashion, and sum sublinearly. This is in agreement with the general suppressive effect seen in other circumstances in V1 (Bair et al., J Neurosci 2003; Reynaud et al., J Neurosci 2012).

To investigate possible mechanisms for this suppression we have designed mean-field models to directly integrate the VSD experiments. Because the VSD signal is primarily caused by the summed voltage of all membranes, it represents an ideal case for mean-field models. However, usual mean-field models are based on neuronal transfer functions such as the well-known sigmoid function, or functions estimated from very simple models. Any error in the transfer function may result in wrong predictions by the corresponding mean-field model. To palliate this caveat, we have obtained semi-analytic forms of the transfer function of more realistic neuron models. We found that the same mathematical template can capture the transfer function for models such as the integrate-and-fire (IF) model, the adaptive exponential (AdEx) model, up to Hodgkin-Huxley (HH) type models, all with conductance-based inputs.

Using these transfer functions we have built “realistic” mean-field models for networks with two populations of neurons, the regular-spiking (RS) excitatory neurons, showing spike frequency adaptation, and the fast-spiking (FS) inhibitory neurons. This mean-field model can reproduce the propagating waves in V1, due to horizontal interactions, as shown previously using IF networks. This mean-field model also reproduced the suppressive interactions between propagating waves. The mechanism of suppression was based on the preferential recruitment of inhibitory cells over excitatory cells by afferent activity, which acted through the conductance-based shunting effect of the two waves onto one another. The suppression was negligible in networks with identical models for excitatory and inhibitory cells (such as IF networks). This suggests that the suppressive effect is a general phenomenon due to the higher excitability of inhibitory neurons in cortex, in line with previous models (Ozeki et al., Neuron 2009).

Work done in collaboration with Yann Zerlaut (UNIC) for modeling, Sandrine Chemla and Frederic Chavane (CNRS, Marseille) for in vivo experiments. Supported by CNRS and the European Commission (Human Brain Project).

Mitsuo Kawato *ATR Computational Neuroscience Laboratories, 2-2 Hikaridai, Seika-cho, Soraku-gun, Kyoto 619-0288, Japan*

K4 – Dynamics and Biomarkers of Mental Disorders

Current diagnoses of mental disorders are made in a categorical way, as exemplified by DSM-5, but many difficulties have been encountered in such categorical regimes: the high percentage of comorbidities, usage of the same drug for multiple disorders, the lack of any validated animal model, and the situation where no epoch-making drug has been developed in the past 30 years. NIMH started RDoC (research domain criterion) to overcome these problems [1], and some successful results have been obtained, including common genetic risk loci [2] and common neuroanatomical changes for multiple disorders [3] as well as psychosis biotypes [4].
In contrast to the currently dominant molecular biology approach, which basically assumes one-to-one mapping between genes and disorders, I postulate the following dynamics-based view of psychiatric disorders. Our brain is a nonlinear dynamical system that can generate spontaneous spatiotemporal activities. The dynamical system is characterized by multiple stable attractors, only one of which corresponds to a healthy or typically developed state. The others are pathological states.
The most promising research approach within the above dynamical view is to combine resting-state functional magnetic resonance imaging, machine learning, big data, and sophisticated neurofeedback. Yahata et al. developed an ASD biomarker using only 16/9730 functional connections, and it did not generalize to MDD or ADHD but moderately to schizophrenia [5]. Yamashita's regression model of working memory ability from functional connections [6] generalized to schizophrenia and reproduced the severity of working-memory deficits of four psychiatric disorders (in preparation).
With the further development of machine learning algorithms and accumulation of reliable datasets, we hope to obtain a comprehensive landscape of many psychiatric and neurodevelopmental disorders. Guided by this full-spectrum structure, a tailor-made neurofeedback therapy should be optimized for each patient [7].

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Contributed Talks

F1 Precise recruitment of spiking output at theta frequencies requires dendritic h-channels in multi-compartment models of oriens-lacunosum/moleculare hippocampal interneurons

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The theta rhythm (4-12Hz) is a prominent network oscillation observed in the mammalian hippocampus and is correlated with spatial navigation and mnemonic processing. Inhibitory interneurons of the hippocampus fire action potentials at specific phases of the theta rhythm, pointing to distinct functional roles of interneurons in shaping this rhythmic activity. One hippocampal interneuron type, the oriens-lacunosum/moleculare (O-LM) cell, provides direct feedback inhibition and regulation of pyramidal cell activity in the CA1 region. O-LM cells express the hyperpolarization-activated, mixed-cation current (I_h) and, in vitro, demonstrate spontaneous firing at theta that is impaired upon blockade of I_h. Work using dynamic clamp has shown that in the presence of frequency-modulated artificial synaptic inputs, O-LM cells exhibit a spiking resonance at theta frequencies that is not dependent on I_h [1]. However, due to the somatic injection limitation of dynamic clamp, the study could not examine the potential contributions of putative dendritic I_h or the integration of dendritically-located synaptic inputs. To overcome this, we have used a database of previously developed multi-compartment computational models of O-LM cells [2]. We situated our OLM cell models in an in vivo-like context by injecting Poisson-based synaptic background activities throughout their dendritic arbors. Excitatory and inhibitory synaptic weights were tuned to produce similar baseline activity prior to modulation of the inhibitory synaptic process at various frequencies (2-30Hz). We found that models with dendritic inputs expressed enhanced resonant firing at theta frequencies compared to models with somatic inputs. We then performed detailed analyses on the outputs of the models with dendritic inputs to further elucidate these results with respect to I_h distributions. The ability of the models to be recruited at the modulated input frequencies was quantified using the rotation number, or average number of spikes across all input cycles. Models with somatodendritic I_h were recruited at >50% of the input cycles for a wider range of theta frequencies (3-9Hz) compared to models with somatic I_h only (3-4Hz). Models with somatodendritic I_h also exhibited a wider range of theta frequencies for which phase-locked output (vector strength>0.75) was observed (4-12Hz), compared to models with somatic I_h (3-5Hz). Finally, the phase of firing of models with somatodendritic I_h given 8-10Hz modulated input was delayed 180-230° relative to the time of release from inhibitory synaptic input. O-LM cells receive phasic inhibitory inputs at theta frequencies from a subpopulation of parvalbumin-positive GABAergic interneurons in the medial septum (MS) timed to the peak of hippocampal theta, as measured in the stratum pyramidale layer [3]. Furthermore, O-LM cells fire at the trough of hippocampal pyramidal layer theta in vivo [4], an approximate 180° phase delay from the MS inputs, corresponding to the phase delay in our models with somatodendritic I_h. Our results suggest that, given dendritic synaptic inputs, O-LM cells require somatodendritic I_h channel expression to be precisely recruited during the trough of hippocampal theta activity. Our strategy of leveraging model databases that encompass experimental cell type-specificity and variability allowed us to reveal critical biophysical factors that contribute to neuronal function within in vivo-like contexts.

Acknowledgements
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ing hippocampal oscillations in awake mice. *Proc Natl Acad Sci USA* 2012, 109(40):E2726-E2734.

F2 Kernel methods in reconstruction of current sources from extracellular potentials for single cells and the whole brains

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Extracellular recordings of electric potential, with a century old history, remain a popular tool for investigations of brain activity on all scales, from single neurons, through populations, to the whole brains, in animals and humans, in vitro and in vivo [1]. The specific information available in the recording depends on the physical settings of the system (brain+electrode). Smaller electrodes are usually more selective and are used to capture local information (spikes from single cells or LFP from populations) while larger electrodes are used for subdural recordings (on the cortex, ECoG), on the scalp (EEG) but also as depth electrodes in humans (called SEEG). The advantages of extracellular electric potential are the ease of recording and its stability. Its problem is interpretation: since electric field is long range one can observe neural activity several millimeters from its source [2-4]. As a consequence every recording reflects activity of many cells, populations and regions, depending on which level we focus. One way to overcome this problem is to reconstruct the distribution of current sources (CSD) underlying the measurement [5], typically done to identify activity on systems level from multiple LFP on regular grids [6].

We recently proposed a kernel-based method of CSD estimation from multiple LFP recordings from arbitrarily placed probes (i.e. not necessarily on a grid) which we called kernel Current Source Density method (kCSD) [7]. In this overview we present the original proposition as well as two recent developments, skCSD (single cell kCSD) and kESI (kernel Electrophysiological Source Imaging). skCSD assumes that we know which part of the recorded signal comes from a given cell and we have access to the morphology of the cell. This could be achieved by patching a cell, driving it externally while recording the potential on a multielectrode array, injecting a dye, and reconstructing the morphology. In this case we know that the sources must be located on the cell and this information can be successfully used in estimation. In kESI we consider simultaneous recordings with subdural ECoG (strip and grid electrodes) and with depth electrodes (SEEG). Such recordings are taken on some epileptic patients prepared for surgical removal of epileptogenic zone. When MR scan of the patient head is taken and the positions of the electrodes are known as well as the brain's shape, the idea of kCSD can be used to bound the possible distribution of sources facilitating localization of the foci.

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F3 The synchronized periods depend on intracellular transcriptional repression mechanisms in circadian clocks.

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In mammals, circadian ($\sim$24hr) rhythms are mainly regulated by a master circadian clock located in the suprachiasmatic nucleus (SCN) [1]. The SCN consists of 20,000 neurons, each of which generates own rhythms via intracellular transcriptional negative feedback loop involving PER-CRY and BMAL1-CLOCK. These individual rhythms of each neuron are synchronized through intercellular coupling via neurotransmitters including VIP [2]. In this talk, I will discuss that the synchronized periods via coupling signal strongly depend on the mechanism of intracellular transcription repression [3-4]. Specifically, using mathematical modeling and phase response curve analysis, we find that the synchronized period of SCN stays close to the population mean of cells' intrinsic periods ($\sim$24hr) if transcriptional repression occurs via protein sequestration. However, the synchronized period is far from the population mean when repression occurs via Hill-type regulation (e.g. phosphorylation-based repression). These results reveal the novel relationship between two major functions of the SCN-intracellular rhythm generation and intercellular synchronization of rhythms. Furthermore, this relationship provides an explanation for why the protein sequestration is commonly used in circadian clocks of multicellular organisms, which have a coupled master clock, but not in unicellular organisms [4].

Acknowledgements
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O1 Assessing irregularity and coordination of spiking-bursting rhythms in central pattern generators

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Found in all nervous systems, central pattern generators (CPGs) are neural circuits that produce flexible rhythmic motor patterns. Their robust and highly coordinated spatio-temporal activity is generated in the absence of rhythmic input. Several invertebrate CPGs are among the best known neural circuits, as their neurons and connections have been identified and mapped. The crustacean pyloric CPG is one of these flagship neural networks [1, 2]. Experimental and computational studies of CPGs typically examine their rhythmic output in periodic spiking-bursting regimes. Aiming to understand the fast rhythm negotiation of CPG neurons, here we present experimental and theoretical analyses of the pyloric CPG activity in situations where irregular yet coordinated rhythms are produced. In particular, we focus our study in the context of two sources of rhythm irregularity: in-

trinsic damage in the preparation, and irregularity induced by ethanol. The analysis of non-periodic regimes can unveil important properties of the robust dynamics controlling rhythm coordination in this system.

Adult male and female shore crabs (Carcinus maenas) were used for the experimental recordings. The isolated stomatrogastric ganglion was kept in Carcinus maenas saline. Membrane potentials were recorded intracellularly from the LP and PD cells, two mutually inhibitory neurons that form a half-center oscillator in the pyloric CPG. Extracellular electrodes allowed monitoring the overall CPG rhythm. Conductance-based models of the pyloric CPG neurons and their associated graded synapses as described in [3, 4] were also used in this dual experimental and theoretical study.

Irregularity and coordination of the CPG rhythms were analyzed using measures characterizing the cells' instantaneous waveform, period, duty cycle, plateau, hyperpolarization and temporal structure of the spiking activity, as well as measures describing instantaneous phases among neurons in the irregular rhythms and their variability. Our results illustrate the strong robustness of the circuit to keep LP/PD phase relationships in intrinsic and induced irregularity conditions while allowing a large variety of burst waveforms, durations and hyperpolarization periods in these neurons. In spite of being electrically coupled to the pacemaker cell of the circuit, the PD neurons showed a wide flexibility to participate with larger burst durations in the CPG rhythm (and larger increase in variability), while the LP neuron was more restricted in sustaining long bursts in the conditions analyzed. The conductance-based models were used to explain the role of asymmetry in the dynamics of the neurons and synapses to shape the irregular activity observed experimentally. Taking into account the overall experimental and model analyses, we discuss the presence of preserved relationships in the non-periodic but coordinated bursting activity of the pyloric CPG, and their role in the fast rhythm negotiating properties of this circuit.

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O2 Regulation of top-down processing by cortically-projecting parvalbumin positive neurons in basal forebrain

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Particular behaviors are associated with different spatio-temporal patterns of cortical EEG oscillations. A recent study suggests that the cortically-projecting, parvalbumin-positive (PV+) inhibitory neurons in the basal forebrain (BF) play an important role in the state-dependent control of cortical oscillations, especially 40 Hz gamma oscillations [1]. However, the cortical topography of the gamma oscillations which are controlled by BF PV+ neurons and their relationship to behavior are unknown. Thus, in this study, we investigated the spatio-temporal patterns and the functional role of the cortical oscillations induced or entrained by BF PV+ neurons by combining optogenetic stimulation of BF PV+ neurons with high-density EEG [2, 3] in channelrhodopsin-2 (ChR2) transduced

PV-cre mice. First, we recorded the spatio-temporal responses in the cortex with respect to the stimulation of BF PV+ neurons at various frequencies. The topographic response patterns were distinctively different depending on the stimulation frequencies, and most importantly, stimulation of BF PV+ neurons at 40 Hz (gamma band frequency) induced a preferential enhancement of gamma band oscillations in prefrontal cortex (PFC) with a statistically significant increase in intracortical connectivity within PFC. Second, optogenetic stimulation of BF PV+ neurons was applied while the mice were exposed to auditory stimuli (AS) at 40 Hz. The time delay between optogenetic stimulation and AS was tested and the phase response to the AS was characterized. We found that the phase responses to the click sound in PFC were modulated by the optogenetic stimulation of BF PV+ neurons. More specifically, the advanced activation of BF PV+ neurons by $\pi/2$ (6.25 ms) with respect to AS sharpened the phase response to AS in PFC, while the anti-phasic activation (π, 12.5 ms) blunted the phase response. Interestingly, like PFC, the primary auditory cortex (A1) also showed sharpened phase response for the $\pi/2$ advanced optogenetic BF PV+ neuron activation during AS. Considering that no direct influence of BF PV+ neurons on A1 was apparent in the response to stimulation of BF PV+ neurons alone, the sharpened phase response curve of A1 suggests a top-down influence of the PFC. This result implies that the BF PV+ neurons may participate in regulating the top-down influence that PFC exerts on primary sensory cortices during attentive behaviors, and supports the idea that the modulating activities of BF PV+ neurons might be a potential target for restoring top-down cognitive functions as well as abnormal frontal gamma oscillations associated with psychiatric disorders.

Acknowledgements
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O3 Modeling auditory stream segregation, build-up and bistability

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With neuromechanistic modelling and psychoacoustic experiments we study the perceptual dynamics of auditory streaming (cocktail party problem). The stimulus is a sequence of two interleaved tones, A and B in a repeating triplet pattern: ABA_ABA_ ('_' is a silent gap). Initially, subjects hear a single integrated pattern, but after some seconds they hear segregated A_A_A_ and _B___B__ streams (build-up of streaming segregation). For long presentations, build-up is followed by irregular alternations between integrated and segregated (auditory bistability). We recently presented [1] the first neuromechanistic model of auditory bistability; it incorporates common competition mechanisms of mutual inhibition, slow adaptation and noise [2]. Our competition network is formulated to reside downstream of primary auditory cortex (A1). Neural responses in macaque A1 to triplet sequences [3] encode stimulus features and provide the inputs to our network (Fig 1A). In our model recurrent excitation with an NMDA-like timescale links responses across gaps between tones and between triplets. It captures the dynamics of perceptual alternations and the stimulus feature dependence of percept durations. To account for build-up we incorporate early adaptation of A1 responses [3] (Fig 1B, upper). Early responses in A1 are broadly tuned and do not reflect the frequency difference between the tones; later responses show a clear tonotopic dependence. This adaptation biases the initial percept towards integration, but occurs faster ($\sim$0.5s) than the gradual build-up process ($\sim$5-10s). The low initial probability of segregation gradually builds up to the stable probability of later bistable alternations (Fig 1B, lower). During build-up, a pause in presentation may cause partial reset to integrated [4]. Our extended model shows this behavior assuming that after a pause A1 responses recover

on the timescale of early adaptation. Moreover, the modeling results agree with our psychoacoustic experiments (compare filled and open circles in Fig 1B, lower).

Conclusions

For the first time, we offer an explanation of the discrepancy in the timescales of early A1 responses and the more gradual build-up process. Recovery of A1 responses can explain resetting for stimulus pauses. Our model offers, to date, the most complete account of the early and late dynamics for auditory streaming in the triplet paradigm.

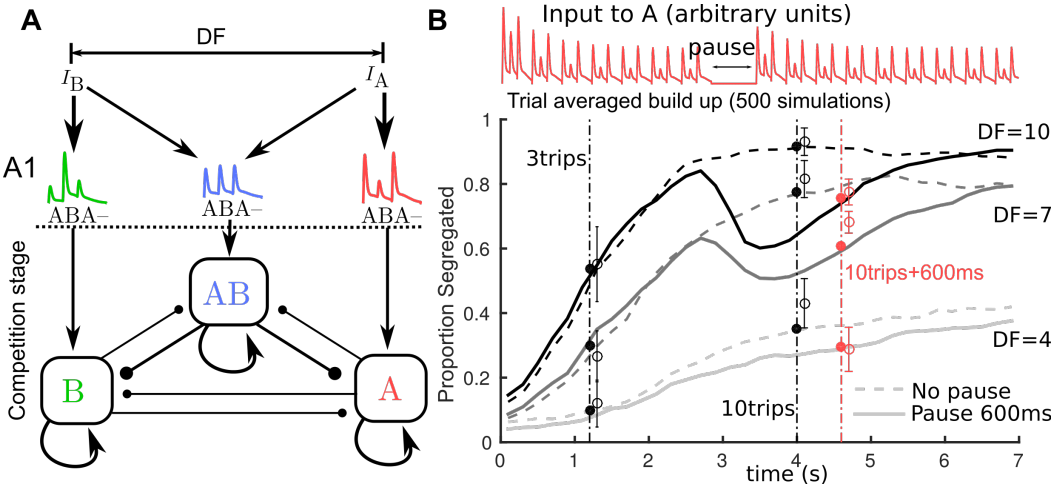

Figure 1: **A**. Model schematic: tone inputs I_A and I_B elicit pulsatile responses in A1, which are pooled as inputs to a three-population competition network. Central unit AB encodes integrated, peripheral units A and B encode segregated. Mutual inhibition between units and recurrent excitation are incorporated with adaptation and noise. **B**. A1 inputs show early initial adaptation, also if a pause is present. Build-up function shows proportion segregated increasing over time, here shown for three tone-frequency differences, DF, with no pause (dashed) or with a pause (solid curves). Time-snapshots from model (filled circles) agree with data (empty circles with SEM error bars, N=8).

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O4 Strong competition between tonotopic neural ensembles explains pitch-related dynamics of auditory cortex evoked fields

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Auditory evoked fields (AEFs) observed in MEG experiments systematically present a transient deflection known as the N100m, elicited around 100ms after the tone onset in the antero-lateral Heschl's Gyrus. The exact N100m's latency is correlated with the perceived pitch of a wide range of stimulus [1, 2], suggesting that the transient component reflects the processing of pitch in auditory cortex. However, the biophysical substrate of such precise relationship remains an enigma. Existing models of pitch, focused on perceptual phenomena, did not explain the mechanism generating cortical evoked fields during pitch processing in biophysical detail. In this work, we introduce a model of interacting neural ensembles describing, for the first time to our knowledge, how cortical pitch processing gives rise to observed human neuromagnetic responses and why its latency strongly

correlates with pitch. To provide a realistic cortical input, we used a recent model of the auditory periphery and realistic subcortical processing stages. Subcortical processing was based on a delay-and-multiply operation carried out in cochlear nucleus and inferior colliculus [3], resulting in realistic patterns of neural activation in response to the stimulus periodicities. Subcortical activation is transformed into a tonotopic receptive-field-like representation [4] by a novel cortical circuit composed by functional blocks characterised by a best frequency. Each block consist of an excitatory and an inhibitory population, modelled using mean-field approximations [5]. Blocks interact with each other through local AMPA- and NMDA- driven excitation and GABA-driven global inhibition [5]. The excitation-inhibition competition of the cortical model describes a general pitch processing mechanism that explains the N100m deflection as a transient state in the cortical dynamics. The deflection is rapidly triggered by a rise in the activity elicited by the subcortical input, peaks after the inhibition overcomes the input, and stabilises when model dynamics reach equilibrium, around 100ms after onset. As a direct consequence of the connectivity structure among blocks, the time necessary for the system to reach equilibrium depends on the encoded pitch of the tone. The model quantitatively predicts observed latencies of the N100m in agreement with available empirical data [1, 2] in a series of stimuli (see Figure 1), suggesting that the mechanism potentially accounts for the N100m dynamics.

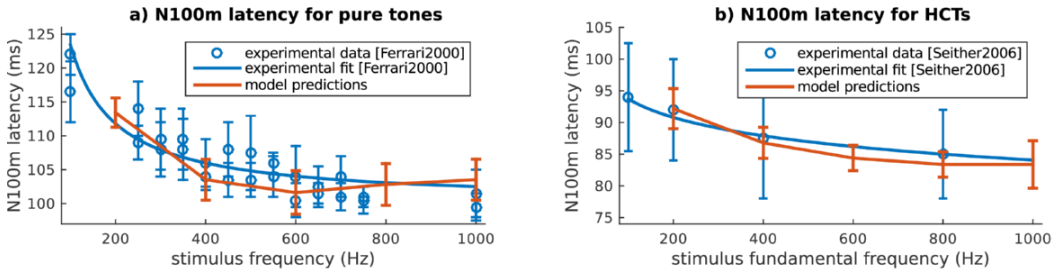

Figure 1: N100m predictions in comparison with available data [1, 2] for a range of pure tones (**A**) and HCTs (**B**).

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O5 A simple model of retinal response to multi-electrode stimulation

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Retinal implants can restore vision to patients suffering photoreceptor loss by stimulating surviving retinal ganglion cells (RGCs) via an array of microelectrodes implanted within the eye [1]. However, the acuity offered by existing devices is low, limiting the benefits to patients. Improvements may come by increasing the number of electrodes in new devices and providing patterned vision, which necessitates stimulation using multiple electrodes simultaneously. However, simultaneous stimulation poses a number of problems due to cross-talk between electrodes and uncertainty regarding the resulting activation pattern. Here, we present a model and methods for estimating the responses of RGCs to simultaneous electrical stimulation. Whole cell in vitro patch clamp recordings were obtained from 25 RGCs with various morphological types in rat retina. The retinae were placed onto an array of 20 stimulating electrodes. Biphasic current pulses with 500 μs phase duration and 50 μs interphase gap were applied simultaneously to all electrodes at a frequency of 10 Hz, with the amplitude of

current on each electrode sampled independently from a Gaussian distribution. A linear-nonlinear model was fit to the responses of each RGC using spike-triggered covariance analyses on 80% of the recorded data. The analysis revealed a single significant principle component corresponding to the electrical receptive field for each cell, with the second largest principle component having negligible effect on the neural response (Fig. 1a). This indicates that interactions between electrodes are approximately linear in their influence on the cells' responses. Furthermore, the spike-triggered ensemble showed two clusters (red and blue in Fig 1a) corresponding to stimulation that had a net effect that was either anodic first or cathodic first. The electrical receptive fields for both anodic first and cathodic first stimulation were highly similar (Fig. 1b). They consisted of a small number (1–4) of electrodes that were close to the cell body (green dot). The remaining 20% of data were used to validate the model. The average model prediction root-mean-square error was 7% over the 25 cells. The accuracy of the model indicates that the linear-nonlinear model is appropriate to describe the responses of RGCs to electrical stimulation.

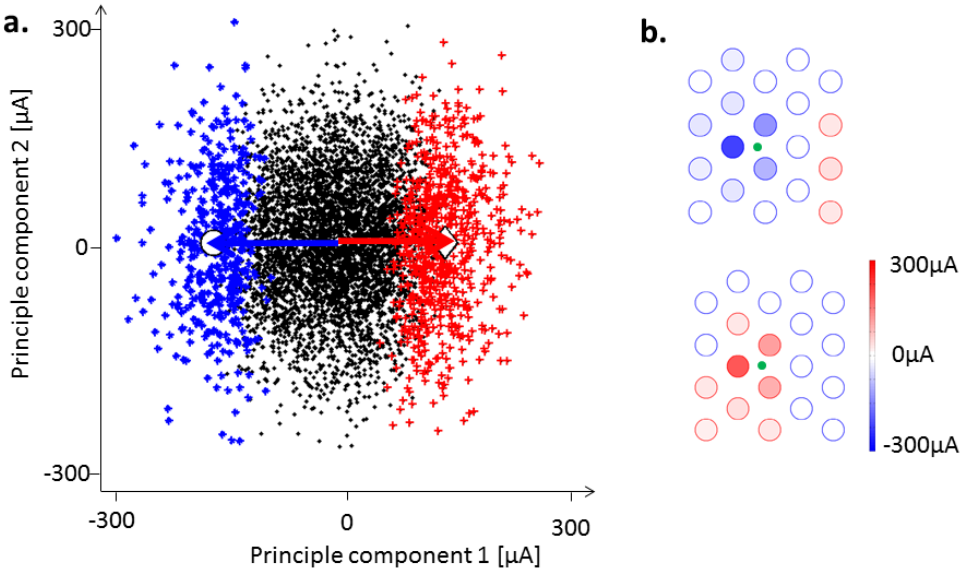

Figure 1: **A**. Spike triggered covariance showing the full set of stimuli (black dots) projected onto the first two principle components. Stimuli causing a spike formed two clusters: net cathodic first pulses (blue) and net anodic first pulse (red). **B**. Electrical receptive fields superimposed on the electrode array are shown for the cathodic first (blue) and anodic first clusters (red).

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O6 Noise correlations in V4 area correlate with behavioral performance in visual discrimination task

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Linking sensory coding and behavior is a fundamental question in neuroscience. We have addressed this issue in behaving monkey visual cortex (areas V1 and V4) while animals were trained to perform a visual discrimination task in which two successive images were either rotated with respect to each other or were the same. We hypothesized that the animal's performance in the visual discrimination task depends on the quality of stimulus coding in visual cortex. We tested this hypothesis by investigating the functional relevance of neuronal correlations in areas V1 and V4 in relation to behavioral performance. We measured two types of correlations: noise (spike count) correlations and correlations in spike timing. Surprisingly, both methods showed that correct responses are associated with significantly higher correlations in V4, but not V1, during the delay period between the two stimuli. This suggests that pair-wise interactions during the spontaneous activity preceding the arrival of the stimulus sets the stage for subsequent stimulus processing and importantly influences behavioral performance.

Experiments were conducted in 2 adult monkeys that were previously trained for the task. After 300 ms of fixation, the target stimulus, consisting of a naturalistic stimulus, is shown for 300 ms, and after a random delay period (500-1200 ms), a test stimulus is shown for 300 ms. The test can either be identical to the target stimulus (match) or rotated with respect to the target (non-match). Monkey responded by pressing a button and was rewarded for a correct response with fruit juice. Two linear arrays with 16 recording channels each were used to record population activity in areas V1 and V4. The difficulty of the task is calibrated individually to have 70% correct responses on average. The analysis is conducted on non-match condition, comparing activity in trials with correct responses with trials where the monkey responded incorrectly. Noise correlations were assessed as pair-wise correlations of spike counts (method 1) and of spike timing (method 2). For method 1, z-scores of spike counts of binned spike trains are computed in individual trials. r_sc is computed as Pearson correlation coefficient of z-scores in all available trials, balanced across correct/incorrect condition. For the method 2, cross-correlograms were computed, from which the cross-correlograms from shuffled trials are subtracted. Resulting function was summed around zero lag and normalized with sum of autocorrelograms [1].

While firing rates of single units or of the population did not significantly change for correct and incorrect responses, noise correlations during the delay period were significantly higher in V4 pairs, computed with both r_sc method (p=0.0005 in monkey 1, sign-rank test) and with r_ccg method (p=0.0001 and p= 0.0280 in monkey 1 and 2, respectively, 50 ms integration window). This result is robust to changes in the length of the bin (method 1) and to the length of the summation window (method 2). In agreement with [2], we confirm the importance of spontaneous activity preceding the stimulus on performance and suggest that higher correlations in V4 might be beneficial for successful read-out and reliable transmission of the information downstream.

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O7 Input-location dependent gain modulation in cerebellar nucleus neurons

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Gain modulation is a brain-wide principle of neuronal computation that describes how neurons integrate inputs from different presynaptic sources. A gain change is a multiplicative operation that is defined as a change in the sensitivity (or slope of the response amplitude) of a neuron to one set of inputs (driving input) which results from the activity of a second set of inputs (modulatory input) [1, 2].

Different cellular and network mechanisms have been proposed to underlie gain modulation [2, 3, 4]. It is well established that input features such as synaptic noise and plasticity can contribute to multiplicative gain changes [2, 3, 4]. However, the effect of neuronal morphology on gain modulation is relatively unexplored. Neuronal inputs to the soma and dendrites are integrated in a different manner: whilst dendritic saturation can introduce a strong non-linear relationship between dendritic excitation and somatic depolarization, the relationship between somatic excitation and depolarization is more linear. The non-linear integration of dendritic inputs can enhance the multiplicative effect of shunting inhibition in the presence of noise [3].

Neurons in the cerebellar nuclei (CN) provide the main gateway from the cerebellum to the rest of the brain. Understanding how inhibitory inputs from cerebellar Purkinje cells interact with excitatory inputs from mossy fibres to control output from the CN is at the center of understanding cerebellar computation. In the present study, we investigated the effect of inhibitory modulatory input on CN neuronal output when the excitatory driving input was delivered at different locations in the CN neuron. We used a morphologically realistic conductance based CN neuron model [5] and examined the change in output gain in the presence of distributed inhibitory input under two conditions: (a) when the excitatory input was confined to one compartment (the soma or a dendritic compartment) and, (b), when the excitatory input was distributed across particular dendritic regions at different distances from the soma. For both of these conditions, our results show that the arithmetic operation performed by inhibitory synaptic input depends on the location of the excitatory synaptic input. In the presence of distal dendritic excitatory inputs, the inhibitory input has a multiplicative effect on the CN neuronal output. In contrast, excitatory inputs at the soma or proximal dendrites close to the soma undergo additive operations in the presence of inhibitory input. Moreover, the amount of the multiplicative gain change correlates with the distance of the excitatory inputs from the soma, with increasing distances from the soma resulting in increased gain changes and decreased additive shifts along the input axis. These results indicate that the location of synaptic inputs affects in a systematic way whether the input undergoes a multiplicative or additive operation.

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O8 Analytic solution of cable energy function for cortical axons and dendrites

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Accurate estimation of action potential (AP)-related metabolic cost is essential for understanding energetic constraints on brain connections and signaling processes. Most previous energy estimates of the AP were obtained using the Na^+-counting method [1, 2], which seriously limits accurate assessment of metabolic cost of ionic currents that underlie AP generation. Moreover, the effects of axonal geometry and ion channel distribution on energy consumption related to AP propagation have not been systematically investigated. To address these issues, we return to the cable theory [3] that underlies our HH-type cortical axon model [4], which was constructed based on experimental measurements. Based on the cable equation that describes how ion currents flow along the cable as well as analysis of the electrochemical energy in the equivalent circuit, we derived the

electrochemical energy function for the cable model,

$$\frac{\partial^2 E}{\partial x \partial t} = I_{Na}(V - V_{Na}) + I_K(V - V_K) + I_L(V - V_L) - \frac{1}{2\pi a} i_a \frac{\partial V}{\partial x}$$

$$= g_{Na}^{max} m^3 h (V(x,t) - V_{Na})^2 + g_K^{max} n^4 (V(x,t) - V_K)^2 + g_L (V(x,t) - V_L)^2 + G_a (\frac{\partial V}{\partial x})^2$$

where g_{Na}^{max} (in a range of 50 to 650 mScm2), g_K^{max} (5 to 100 mScm2), and $g_L = 0.033$ mS/cm2 are the maximal sodium, maximal potassium, and leak conductance per unit membrane area, respectively; and $V_{Na} = 60$, $V_K = -90$, $V_L = -70$ mV are the reversal potentials of the sodium, potassium, and leak channels, respectively. The gate variables m, h, and n are dimensionless activation and inactivation variables, which describe the activation and inactivation processes of the sodium and potassium channels [4]. This equation describes the AP-related energy consumption rate per unit membrane area (cm^2s) at any axonal distance and any time. The individual terms on the right-hand side of the equation represent the contributions of the sodium, potassium, leak, and axial currents, respectively. Then we employed the cable energy function to calculate energy consumption for unbranched axons and axons with several degrees of branching (branching level, BL). Calculations based on this function distinguish between the contributions of each item toward total energy consumption. Our analytical approach predicts an inhomogeneous distribution of metabolic cost along an axon with either uniformly or nonuniformly distributed ion channels. The results show that the Na+-counting method severely underestimates energy cost in the cable model by 20%-70%. AP propagation along axons that differ in length may require over 15% more energy per unit of axon area than that required by a point model. However, actual energy cost can vary greatly depending on axonal branching complexity, ion channel density distributions, and AP conduction states. We also infer that the metabolic rate (i.e. energy consumption rate) of cortical axonal branches as a function of spatial volume exhibits a 3/4 power law relationship.

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O9 C. elegans Interactome: Interactive Visualization of Caenorhabditis elegans Worm Neuronal Network

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Modeling neuronal systems involves incorporating the two layers: a static map of neural connections (connectome), and biophysical processes that describe neural responses and interactions. Such a model is called the 'dynome' of a neuronal system as it integrates a dynamical system with the static connectome. Being closer to reproducing the activity of a neuronal system, investigation of the dynome has more potential to reveal neuronal pathways of the network than the static connectome [1]. However, since the two layers of the dynome are considered simultaneously, novel tools have to be developed for the dynome studies. Here we present a visualization methodology, called 'interactome', that allows to explore the dynome of a neuronal system interactively and in real-time, by viewing the dynamics overlaid on a graph representation of the connectome. We apply our methodology to the nervous system of Caenorhabditis Elegans (C. elegans) worm, which connectome is almost fully resolved [2], and a computational model of neural dynamics and interactions (gap and synaptic) based on biophysical experimental findings was recently introduced [3]. Integrated together, C. elegans dynome defines a unique set of neural dynamics of the worm. To visualize the dynome, we propose a dynamic force-directed graph layout of the connectome. The layout is implemented using D3 visualization platform [4], and is designed

to communicate with an integrator of the dynome. The two-way communication protocol between the layout and the integrator allows for stimulating (injecting current) into any subset of neurons at any time point (Fig 1B). It also allows for simultaneously viewing the response of the network on top of the layout visualized by resizing graph nodes (neurons) according to their voltage. In addition, we support structural changes in the connectome, such as ablation of neurons and connections. Our visualization and communication protocols thereby display the stimulated network in an interactive manner and permit to explore different regimes that the stimulations induce. Indeed, with the interactome we are able to recreate various experimental scenarios, such as stimulation of forward crawling (PLMAVB neurons andor ablation of AVB) and show that its visualization assists in identifying patterns of neurons in the stimulated network. As connectomes and dynomes of additional neuronal systems are being resolved, the interactome will enable exploring their functionality and inference to its underlying neural pathways [5].

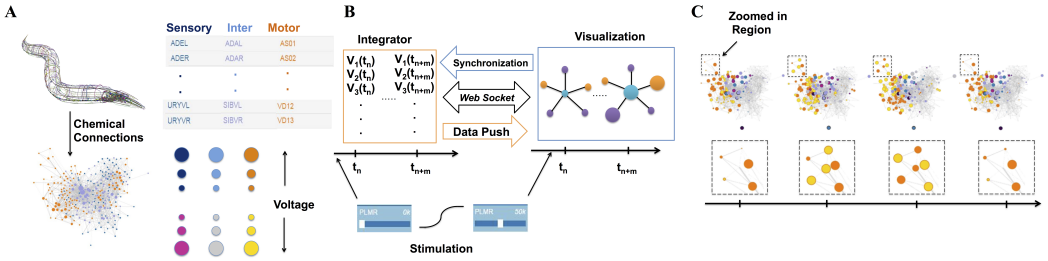

Figure 1: A. Visualization of C. Elegans dynome B. Communication diagram between the dynome and the layout C. Snapshots of visualization of C. elegans during the PLMAVB excitations (forward crawling).

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O10 Is the Model Any Good? Objective Criteria for Computational Neuroscience Model Selection

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Objectively evaluating and selecting computational models of biological neurons is an ongoing challenge in the field. Models vary in morphological detail, channel mechanisms, and synaptic transmission implementations. We present the results of an automated method for evaluating computational models against property values obtained from published cell electrophysiology studies. Seven published deterministic models of olfactory bulb mitral cells were selected from ModelDB [1] and simulated using NEURON's Python interface [2]. Passive and spike properties in response to step current stimulation pulses were computed using the NeuronUnit [3] package and compared to their respective, experimentally obtained means of olfactory bulb mitral cell properties found in the NeuroElectro database [4]. Results reveal that across all models, the resting potential and input resistance property means deviated the most from their experimentally measured means (R_{input} t-test p=0.02, V_{rest} Wilcoxon-test p=0.01). The time constant, spike half-width, spike amplitude, and spike threshold properties, in the order of decreasing average deviation, matched well with experimental data (p > 0.05) (Figure 1 Top).

In three models, the property deviations were, on average, outside the 95% CI of the experimental means (Figure 1 Bottom), but these averages were not significant (t-test p > 0.05). All other models were within the 95% CI, while the model of Chen et. al. had the lowest deviation [5].

Overall, the majority of these olfactory bulb mitral cell models display some properties that are not significantly different from their experimental means. However, the resting potential and input resistance properties signifi-

cantly differ from the experimental values. We demonstrate that NeuronUnit provides an objective method for evaluating the fitness of computational neuroscience cell models against publicly available data.

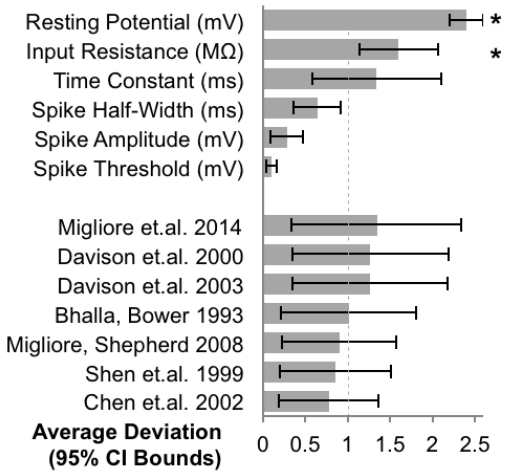

Figure 1: The average deviations of models and cell electrophysiology properties as measured in multiples of the 95% CI bounds of experimental data means. Dashed line represents 1 CI bound threshold. Top rows show average deviations across all models for each cell property. Bottom rows show deviations across all cell properties for each model.

Acknowledgements
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O11 Cooperation and competition of gamma oscillation mechanisms

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Neuronal oscillations in the gamma band (30–80 Hz) have been found in many cortical areas and have been associated to various sensory, motor, and cognitive tasks [1]. The two major mechanisms that have been suggested to underlie gamma oscillations [2] are "ING" (InterNeuronal Gamma), which is related to tonic excitation of reciprocally coupled inhibitory interneurons (I-cells, [3]), and "PING" (Pyramidal-InterNeuron Gamma), which is mediated by coupled populations of excitatory pyramidal cells (E-cells) and I-cells [4]. Gamma oscillations generated by different mechanisms may serve different biological functions. Using computer simulations and analytical methods, we therefore investigate which mechanism (ING or PING) will dominate the dynamics of a network when ING and PING interact and how the dominant mechanism may switch.

We find that ING and PING oscillations compete: The mechanism generating the higher oscillation frequency

"wins", it determines the frequency of the network oscillation and suppresses the other mechanism. For networks with type-I-phase-response-curve interneurons (cf. Figure 1D), the network oscillation frequency (green line corresponding to the network topology given in Figure 1C) is equal to or slightly above the higher of the ING (blue line) and PING (red line) frequencies in corresponding reduced networks that can generate only either of them, see Figure 1A for an only ING-generating reduced network and 1B for an only PING-generating reduced network. If the interneurons have type-II phase response curve, it is in between, see Figure 1E. We explain our computer simulation results by a theoretical model that allows a full theoretical analysis of the main results.

Our study suggests experimental approaches to decide whether oscillatory activity in networks of interacting excitatory and inhibitory neurons is dominated by ING or PING oscillations and whether the participating interneurons belong to the class I or II. Consider as an example networks with type-I interneurons where the external drive to the E-cells, $I_{0,E}$, is kept constant while the external drive to the I-cells, $I_{0,I}$, is varied. For both ING and PING dominated oscillations the frequency of the rhythm increases when $I_{0,I}$ increases (cf. Figure 1D). Observing such an increase does therefore not allow to determine the underlying mechanism. However, the rate of change of the frequency increase allows a distinction, as it increases for PING and decreases for ING (cf. Figure 1D). In networks with type-II interneurons, the non-monotonic dependence near the ING-PING transition may be a characteristic hallmark to detect the oscillation character (and the interneuron type): Decrease (increase) of the frequency when increasing $I_{0,E}$ indicates ING (PING), cf. Figure 1E.

Some experimental evidence is in line with these predictions. For example, Craig and McBain [5] reported that optogenetic silencing of pyramidal cells in CA3 of hippocampus, where the dominant in-vitro gamma oscillations are PING driven, led to a significant increase in the peak frequency of the oscillations, as predicted by our results (cf. the curves in Figure 1E at intermediate values of $I_{0,E}$). Using step-opsins [6], results as in Figure 1D and 1E could be obtained experimentally. This will allow a test of our results and predictions and may reveal how ING and PING oscillations interact.

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O12 A discrete structure of the brain waves

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A physiological interpretation of the biological rhythms, e.g., of the local field potentials (LFP) depends on the mathematical and computational approaches used for its analysis. Most existing mathematical methods of the LFP studies are based on braking the signal into a combination of simpler components, e.g., into sinusoidal harmonics of Fourier analysis or into wavelets of the Wavelet Analysis. However, a common feature of all these methods is that their prime components are presumed from the onset, and the goal of the subsequent analysis reduces to identifying the combination that best reproduces the original signal.

We propose a fundamentally new method, based on a number of deep theorems of complex function theory, in which the prime components of the signal are not presumed a priori, but discovered empirically [1]. Moreover, the new method is more flexible and more sensitive to the signal's structure than the standard Fourier method.

Applying this method reveals a fundamentally new structure in the hippocampal LFP signals in rats in mice. In particular, our results suggest that the LFP oscillations consist of a superposition of a small, discrete set of frequency modulated oscillatory processes, which we call "oscillons". Since these structures are discovered

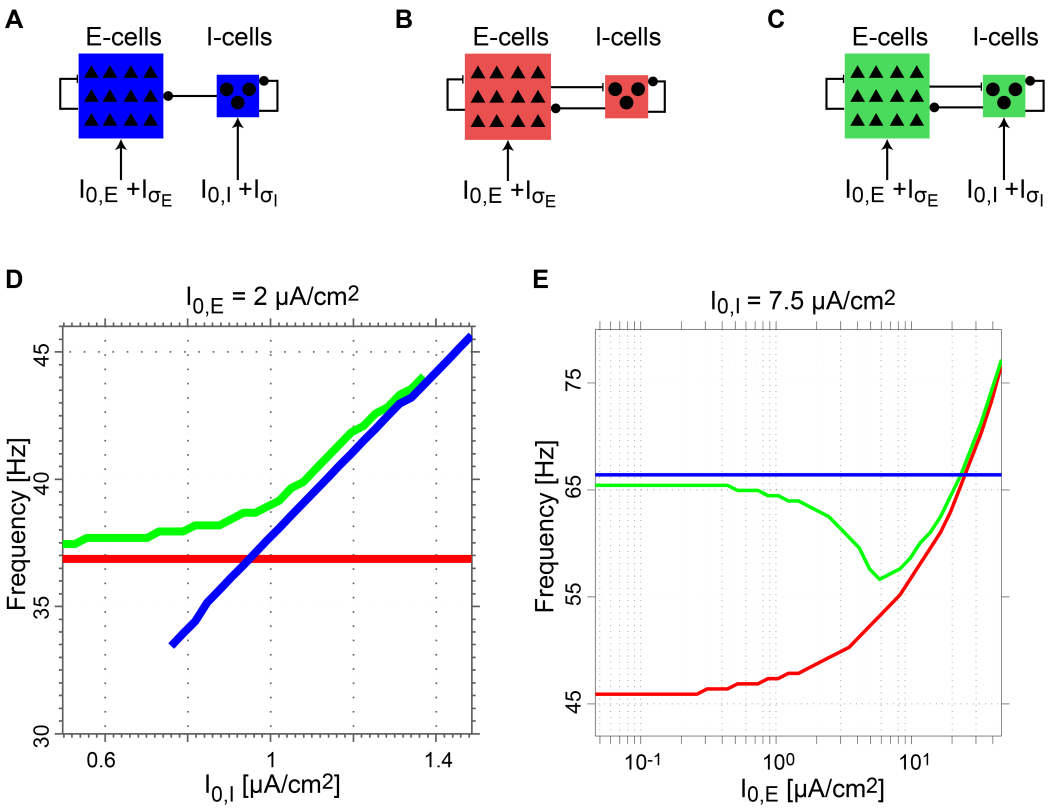

Figure 1: Oscillations in full and reduced networks of reciprocally coupled pyramidal cells and interneurons. **A**. and **B**. illustrate topologies of reduced networks that generate only "pure" ING or only "pure" PING, respectively, while **C**. highlights the topology of a "full" network that could in principle generate either ING or PING oscillations or mixtures of both. **D**, **E**. Frequency of pure ING-rhythm generated by the reduced network in (A) (blue line), pure PING-rhythm generated by the reduced network in (B) (red line), and rhythms generated by the full network in (C) (green line) as a function of mean current to I-cells $I_{0,I}$ and a function of mean current to E-cells $I_{0,E}$, respectively. (D) shows results for networks with type-I interneurons while (E) shows results for networks with type-II interneurons. Pyramidal cells are modeled as type-I Hodgkin-Huxley neurons.

empirically, we hypothesize that they may capture the signal's actual physical structure, i.e., the pattern of synchronous activity in neuronal ensembles. Proving this hypothesis will help enormously to advance a principal, theoretical understanding of the neuronal synchronization mechanisms. We anticipate that it will reveal new information about the structure of the LFP and other biological oscillations, which should provide insights into the underlying physiological phenomena and the organization of brains states that are currently poorly understood, e.g., sleep and epilepsy.

Acknowledgements
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O13 Direction-specific silencing of the Drosophila gaze stabilization system

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Many animals, including insects and humans, stabilize the visual image projected onto their retina by following a rotating landscape with their head or eyes. This stabilization reflex, also called the optomotor response, can pose a problem, however, when the animal intends to change its gaze. To resolve this paradox, von Holst and

Mittelstaedt proposed that a copy of the motor command, or efference copy, could be routed into the visual system to transiently silence this stabilization reflex when an animal changes its gaze [1]. Consistent with this idea, we recently demonstrated that a single identified neuron associated with the optomotor response receives silencing motor-related inputs during rapid flight turns, or saccades, in tethered, flying Drosophila [2].

Here, we expand on these results by comprehensively recording from a group of optomotor-mediating visual neurons in the fly visual system: three horizontal system (HS) and six vertical system (VS) cellsÂ. We found that the amplitude of motor-related inputs to each HS and VS cell correlates strongly with the strength of each cell's visual sensitivity to rotational motion stimuli around the primary turn axis, but not to the other axes (Figure 1). These results support the idea that flies send rotation-axis-specific efference copies to the visual system during saccades – silencing the stabilization reflex only for a specific axis, but leaving the others intact. This is important because saccades consist of stereotyped banked turns, which involve body rotations around all three primary axes of rotation. If the gaze stabilization system is impaired for only one of these axes, then the fly is expected to attempt to maintain gaze stability, through a combination of head and body movements, for the other two. This prediction is consistent with behavioral measurements of head and body kinematics during saccades in freely flying blow flies [3]. Together, these studies provide an integrative model of how efference copies counteract a specific aspect of visual feedback signals to tightly control the gaze stabilization system.

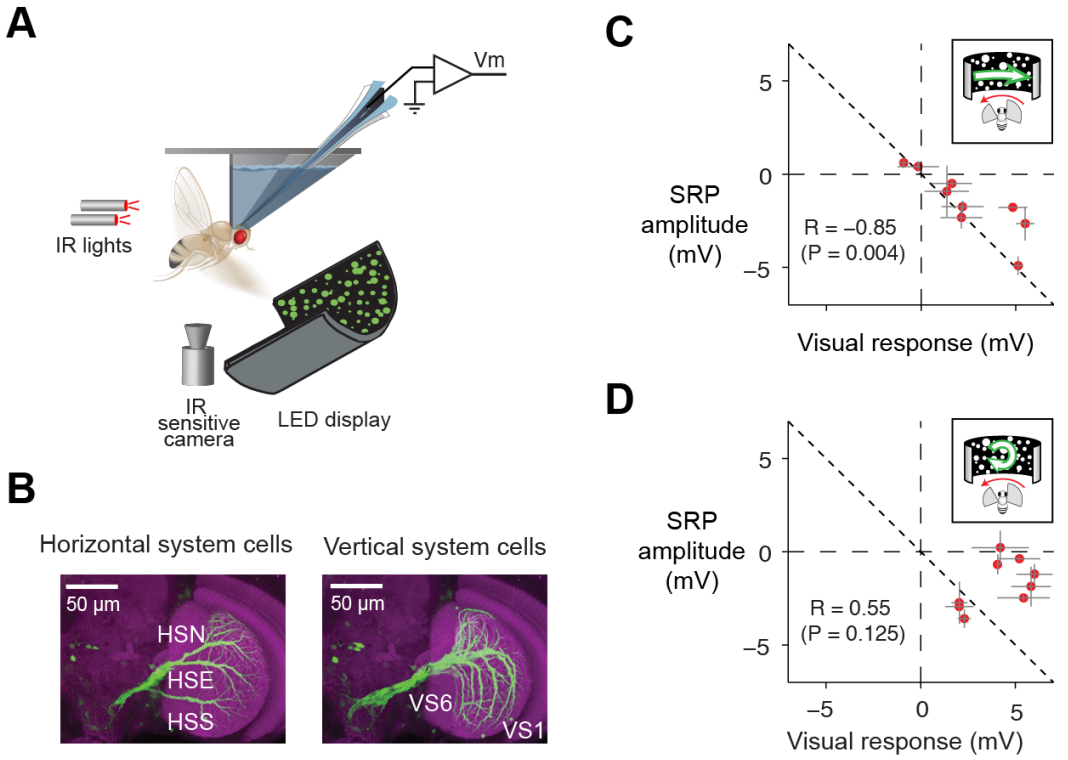

Figure 1: The amplitudes of saccade-related potentials (SRPs) to HS and VS cells are strongly correlated with each cell's visual sensitivity to rightward yaw motion stimuli. **A**. Experimental apparatus. **B**. Maximal-intensity z-projections of the lobula plate to visualize HS- or VS-cell neurites that are marked by a GAL4 enhancer trap line. **C**, **D**. The amplitude of saccade-related potentials (SRPs) were inversely correlated with visual responses, when measured under rightward yaw motion stimuli, but not under clockwise roll motion stimuli. Each sample point corresponds to each cell type. Error bars indicate SEM.

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O14 What does the fruit fly think about values? — A model of olfactory associative learning

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Associative learning in the fruit fly olfactory system has been studied from the molecular to the behavior level [1,2]. Fruit flies are able to associate conditional stimuli such as odor with unconditional aversive stimuli such as electrical shocks, or appetitive stimuli such as sugar or water. The mushroom body in the fruit fly brain is considered to be crucial for olfactory learning [1,2]. The behavioral experiments show that the learning can not be explained simply by an additive Hebbian (i.e. correlation-based) learning rule. Instead, it depends on the timing between the conditional and unconditional stimulus presentation. Yarali and colleagues suggested a dynamic model on the molecular level to explain event timing in associative learning [3]. Here, we present new experiments together with a simple phenomenological model for learning that shows that associative olfactory learning in the fruit fly represents value learning that is incompatible with Hebbian learning.

In our model, the information of the conditional odor stimulus is conveyed by Kenyon cells from the projection neurons to the mushroom output neurons; the information of the unconditional shock stimulus is represented by dopaminergic neurons to the mushroom output neurons through direct or indirect pathways. The mushroom body output neurons encode the internal value (v) of the odor (o) by synaptic weights (w) that conveys the odor information, $v = w \cdot o$. The synaptic strength is updated according to the value learning rule, $\Delta w = \eta \cdot (s - v) \cdot \tilde{o}$, where s represents the (internal) strength of the shock stimulus, $\tilde{o}$ represents the synaptic odor trace, and η is the learning rate. The value associated with the odor determines the probability of escaping from that odor. This simple model reproduces the behavioral data and shows that olfactory conditioning in the fruit fly is in fact value learning. In contrast to the prediction of Hebbian learning, the escape probability for repeated odor-shock pairings is much lower than the escape probability for a single pairing with a correspondingly stronger shock.

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O15 Effects of ionic diffusion on power spectra of local field potentials (LFP)

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The local field potential (LFP) in the extracellular space (ECS) of the brain, is a standard measure of population activity in neural tissue. Computational models that simulate the relationship between the LFP and its underlying neurophysiological processes are commonly used in the interpretation such measurements. Standard methods, such as volume conductor theory [1], assume that ionic diffusion in the ECS has negligible impact on the LFP. This assumption could be challenged during endured periods of intense neural signalling, under which local ion concentrations in the ECS can change by several millimolars. Such concentration changes are indeed often accompanied by shifts in the ECS potential, which may be partially evoked by diffusive currents [2]. However, it is hitherto unclear whether putative diffusion-generated potential shifts are too slow to be picked up in LFP

recordings, which typically use electrode systems with cut-off frequencies at 0.1 Hz.

To explore possible effects of diffusion on the LFP, we developed a hybrid simulation framework: (1) The NEURON simulator was used to compute the ionic output currents from a small population of cortical layer-5 pyramidal neurons [3]. The neural model was tuned so that simulations over 100 seconds of biological time led to shifts in ECS concentrations by a few millimolars, similar to what has been seen in experiments [2]. (2) In parallel, a novel electrodiffusive simulation framework [4] was used to compute the resulting dynamics of the potential and ion concentrations in the ECS, accounting for the effect of electrical migration as well as diffusion. To explore the relative role of diffusion, we compared simulations where ECS diffusion was absent with simulations where ECS diffusion was included.

Our key findings were: (i) ECS diffusion shifted the local potential by up to 0.2 mV. (ii) The power spectral density (PSD) of the diffusion-evoked potential shifts followed a $1/f^2$ power law. (iii) Diffusion effects dominated the PSD of the ECS potential for frequencies up to 10 Hz (Figure 1). We conclude that for large, but physiologically realistic ECS concentration gradients, diffusion could affect the ECS potential well within the frequency range considered in recordings of the LFP.

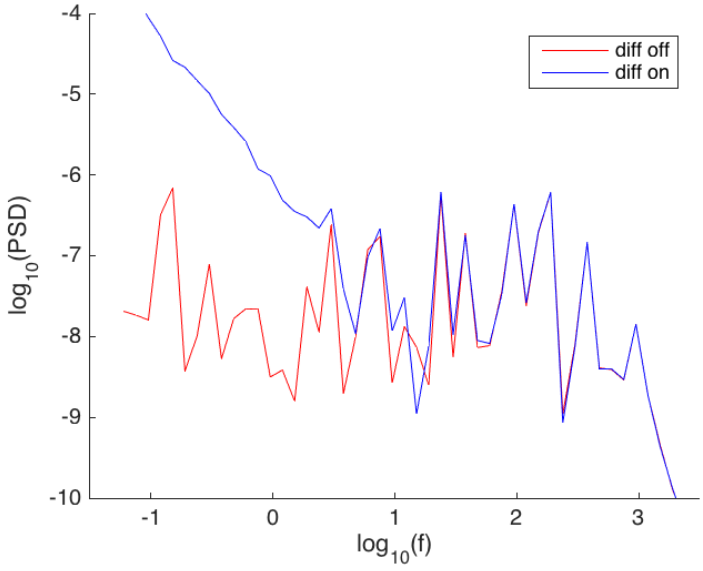

Figure 1: Power spectrum of ECS potential in a simulation including ECS diffusion (blue line) and a simulation without ECS diffusion (red line). Units for frequency and power are Hz and mV^2/Hz, respectively.

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O16 Large-scale cortical models towards understanding relationship between brain structure abnormalities and cognitive deficits

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Brain connectivity studies have revealed fundamental properties of normal brain network organization [1]. In parallel, they have reported structural connectivity abnormalities in brain diseases such as Alzheimer's disease

(AD) [1, 2]. However, how these structural abnormalities affect information processing and cognitive functions involved in brain diseases is still poorly understood. To deepen our understanding of this causal link, I developed two large-scale cortical models with normal and abnormal structural connectivity of diffusion tensor imaging on aging APOE-4 non-carriers and carriers in the USC Multimodal Connectivity Database [2, 3]. The possession of the APOE-4 allele is one of the major risk factors in developing later AD, and it has known abnormalities in structural connectivity characterized by lower network communication efficiency in terms of local interconnectivity and balance of integration and interconnectivity [2]. The two cortical models share other parameters and consist of 2.4 million spiking neurons and 4.8 billion synaptic connections. First, I demonstrate the biological relevance of the models by confirming that they reproduce normal patterns of cortical spontaneous activities in terms of the following distinctive properties observed in vivo [4]: low firing rates of individual neurons that approximate log-normal distributions, irregular spike trains following a Poisson distribution, a network balance between excitation and inhibition, and greater depolarization of the average membrane potentials. Next, to investigate how the difference in structural connectivity affects cortical information processing, I compare cortical response properties to an input during spontaneous activity between the cortical models. The results show that the cortical model with the abnormal structural connectivity decreased the degree of cortical response as well as the number of cortical regions responding to the input (Figure 1), suggesting that the structural connectivity abnormality observed in APOE-4 carriers might reduce cortical information propagation and lead to negative effects in information integration. Indeed, imaging studies support this suggestion by reporting structural abnormality with lower network communication efficiency observed in the structural connectivity of both APOE-4 carriers and AD patients [1, 2]. This computational approach allowing for manipulations and detailed analyses that are difficult or impossible in human studies can help to provide a causal understanding of how cognitive deficits in patients with brain diseases are associated with their underlying structural abnormalities.

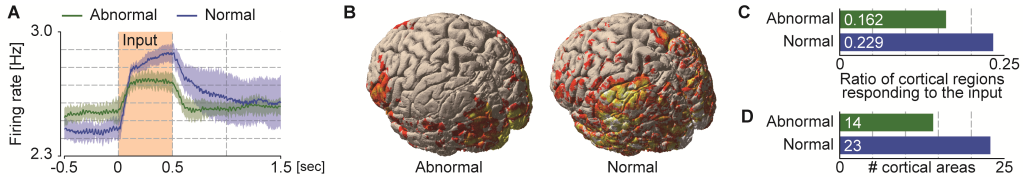

Figure 1: Responses to input to the left V1 in the two cortical models with normal/abnormal structural connectivity. (A) Average firing rates. (B, C and D) Cortical regions and cortical areas that significantly responded to the input.

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O17 Spatial coarse-graining the brain: Origin of minicolumns

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The seminal experiments of Mountcastle [1] over 60 years ago established the existence of cortical minicolumns: vertical column-like arrays of approximately 80–120 neurons aligned perpendicular to the pial surface, penetrating all six cortical layers. Minicolumns have been proposed as the fundamental unit for cortical organisation. Minicolumn formation is thought to rely on gene expression and thalamic activity, but exactly why neurons cluster into columns of diameter 30–50μm containing approximately 100 neurons is not known.

In this presentation we describe a mechanism for the formation of minicolumns via gap-junction diffusion-mediated coupling in a network of spiking neurons. We use our recently developed method of cortical "reblocking" (spatial coarse-graining) [2] to derive neuronal dynamics equations at different spatial scales. We are able to show that for sufficiently strong gap-junction coupling, there exists a minimum block size over which neural activity is expected to be coherent. This coherence region has cross-sectional area of order (40–60 μm^2), consistent with the areal extent of a minicolumn. Our scheme regrids a 2D continuum of spiking neurons using a spatial rescaling theory, established in the 1980s, that systematically eliminates high-wave-number modes [3]. The rescaled neural equations describe the bulk dynamics of a larger block of neurons giving "true" (rather than mean-field) population activity, encapsulating the inherent dynamics of a continuum of spiking neurons stimulated by incoming signals from neighbors, and buffeted by ion-channel and synaptic noise.

Our method relies on a perturbative expansion. In order for this coarse-graining expansion to converge, we require not only a sufficiently strong level of inhibitory gap-junction coupling, but also a sufficiently large blocking ratio B. The latter condition establishes a lower bound for the smallest "cortical block": the smallest group of neurons that can respond to input as a collective and cooperative unit. We find that this minimum block-size ratio lies between 4 and 6. In order to relate this 2D geometric result to the 3D extent of a 3-mm-thick layered cortex, we project the cortex onto a horizontal surface and count the number of neurons contained within each $l \times l$ grid micro-cell. Setting $l \sim 10\mu m$ and assuming an average of one interneuron per grid cell, a blocking ratio at the mid-value B = 5 implies that the side-length of a coherent "macro-cell" will be L = Bl = 50 μm containing ~ 25 inhibitory plus 100 excitatory neurons (assuming an i to e abundance ratio of 1:4) in cross-sectional area L^2. Thus the minicolumn volume will contain roughly 125 neurons. We argue that this is the smallest diffusively-coupled population size that can support cooperative dynamics, providing a natural mechanism defining the functional extent of a minicolumn.

We propose that minicolumns might form in the developing brain as follows: Inhibitory neurons migrate horizontally from the ganglionic eminence to form a dense gap-junction coupled substrate that permeates all layers of the cortex [4]. Progenitor excitatory cells ascend vertically from the ventricular zone, migrating through the inhibitory substrate of the cortical plate. Thalamic input provides low-level stimulus to activate spiking activity throughout the network. Inhibitory diffusive coupling allows a "coarse graining" such that neurons within a particular areal extent respond collectively to the same input. The minimum block size prescribed by the coarse graining imposes constraints on minicolumn geometry, leading to the spontaneous emergence of cylindrical columns of coherent activity, each column centered on an ascending chain of excitatory neurons and separated from neighboring chains by an annular surround of inhibition. This smallest aggregate is preferentially activated during early brain development, and activity-based plasticity then leads to the formation of tangible structural columns.

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O18 Modeling large-scale cortical networks with laminar structure

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Visual cortical areas in the macaque are organized according to an anatomical hierarchy, which is defined by specific patterns of anatomical projections in the feedforward and feedback directions [1, 2]. Recent macaque studies also suggest that signals ascending through the visual hierarchy are associated with gamma rhythms, and top-down signals with alpha/low beta rhythms [3, 4, 5]. It is not clear, however, how oscillations presumably originating at local populations can give rise to such frequency-specific large-scale interactions in a mechanistic way, or the role that anatomical projections patterns might have in this.

To address this question, we build a large-scale cortical network model with laminar structure, grounding our

model on a recently obtained anatomical connectivity matrix with weighted directed inter-areal projections and information about their laminar origin. The model involves several spatial scales –local or intra-laminar microcircuit, inter-laminar circuits, inter-areal interactions and large-scale cortical network – and a wide range of temporal scales – from slow alpha oscillations to gamma rhythms. At any given level, the model is constrained anatomically and then tested against electrophysiological observations, which provides useful information on the mechanisms modulating the oscillatory activity at different scales. As we ascend through the local to the inter-laminar and inter-areal levels, the model allows us to explore the sensory-driven enhancement of gamma rhythms, the inter-laminar phase-amplitude coupling, the relationship between alpha waves and local inhibition, and the frequency-specific inter-areal interactions in the feedforward and feedback directions [3, 4], revealing a possible link with the predictive coding framework.

When we embed our modeling framework into the anatomical connectivity matrix of 30 areas (which includes novel areas not present in previous studies [2, 6]), the model gives insight into the mechanisms of large-scale communication across the cortex, accounts for an anatomical and functional segregation of FF and FB interactions, and predicts the emergence of functional hierarchies, which recent studies have found in macaque [4] and human [5]. Interestingly, the functional hierarchies observed experimentally are highly dynamic, with areas moving across the hierarchy depending on the behavioral context [4]. In this regard, our model provides a strong prediction: we propose that these hierarchical jumps are triggered by laminar-specific modulations of input into cortical areas, suggesting a strong link between hierarchy dynamics and context-dependent computations driven by specific inputs.

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O19 Information filtering by partial synchronous spikes in a neural population

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Synchronous firing of neurons is a prominent feature in many brain areas. Here, we are interested in the information transmission by the synchronous spiking output of a noisy neuronal population, which receives a common time-dependent sensory stimulus. Earlier experimental [1] and theoretical [2] work revealed that synchronous spikes encode preferentially fast (high-frequency) components of the stimulus, i.e. synchrony can act as an information filter. In these studies a rather strict measure of synchrony was used: the entire population has to fire within a short time window. Here, we generalize the definition of the synchronous output, for which only a certain fraction γ of the population needs to be active simultaneously — a setup that seems to be of more biological relevance. We characterize the information transfer in dependence of this fraction and the population size, by the spectral coherence function between the stimulus and the partial synchronous output. We present two different analytical approaches to derive this frequency-resolved measure (one that is more suited for small population sizes, while the second one is applicable to larger populations). We show that there is a critical synchrony fraction, namely the probability at which a single neuron spikes within the predefined time window, which maximizes

the information transmission of the synchronous output. At this value, the partial synchronous output acts as a low-pass filter, whereas deviations from this critical fraction lead to a more and more pronounced band-pass filtering effect. We confirm our analytical findings by numerical simulations for the leaky integrate-and-fire neuron. We also show that these findings are supported by experimental recordungs of P-Units electroreceptors of weakly electric fish, where the filtering effect of the synchronous output occurs in real neurons as well.

Acknowledgements
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O20 Decoding context-dependent olfactory valence in Drosophila

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Many animals rely on olfactory cues to make perceptual decisions and navigate the environment. In the brain, odorant molecules are sensed by olfactory receptor neurons (ORNs), which convey olfactory information to the central brain in the form of sequences of action potentials. In many organisms, axons of ORNs expressing the same olfactory receptor converge to one or a few glomeruli in the first central region (the antennal lobe in insects and the olfactory bulb in fish and mammals) where they make contact with their postsynaptic targets. Therefore, each glomerulus can be considered as a processing unit that relays information from a specific type of receptor. Because different odorants recruit different sets of glomeruli, and most glomeruli respond to a wide array of odors, olfactory information at this stage of processing is contained in spatiotemporal patterns of glomerular activity. How these patterns are decoded by the brain to guide odor-evoked behavior, however, remains largely unknown.

In Drosophila, attraction and aversion to specific odors have been linked to the activation of one or a few glomeruli (reviewed in [1]) in the antennal lobe (AL). These observations suggest a “labeled-line” coding strategy, in which individual glomeruli convey signals of specific ethological relevance, and their activation triggers the execution of hard-wired behavioral programs. However, because these studies used few odorants, and a small fraction of glomeruli were tested, it is unclear how the results generalize to broader odor sets, and whether similar conclusions hold for each of the âˆ¼ 50 glomeruli of the fly AL. Moreover, how compound signals from multiple glomeruli are integrated is poorly understood.

Here, we combine optical imaging, behavioral and statistical techniques to address these questions systematically. Using two-photon imaging, we monitor Ca^{2+} activity in the AL in response to 84 odors. We next screen behavioral responses to the same odorants. Comparing these data allows us to formulate a decoding model describing how olfactory behavior is determined by glomerular activity patterns in a quantitative manner. We find that a weighted sum of normalized glomerular responses recapitulates the observed behavior and predicts responses to novel odors, suggesting that odor valence is not determined solely by the activity a few privileged glomeruli. This conclusion is supported by genetic silencing and optogenetic activation of individual ORN types, which are found to evoke modest biases in behavior in agreement with model predictions. Finally, we test the model prediction that the relative valence of a pair of odors depends on the identity of other odors presented in the same experiment. We find that the relative valence indeed changes, and may even switch, suggesting that perceptual decisions can be modulated by the olfactory context. Surprisingly, our model correctly captured both the direction and the magnitude of the observed changes. These results indicate that the valence of olfactory stimuli is decoded from AL activity by pooling contributions over a large number of glomeruli, and highlight the ability of the olfactory system to adapt to the statistics of its environment, similarly to the visual and auditory systems.

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Workshops

W1 **Methods of Information Theory in Computational Neuroscience**

301A + 301B, Wednesday and Thursday, 9:00 – 16:30

Joseph T Lizier, The University of Sydney
Justin Dauwels, Nanyang Technological University
Taro Toyoizumi, RIKEN Brain Science Institute
Alexander G Dimitrov, Washington State University
Lubomir Kostal, Academy of Sciences of the Czech Republic

Methods originally developed in Information Theory have found wide applicability in computational neuroscience. Beyond these original methods there is a need to develop novel tools and approaches that are driven by problems arising in neuroscience.

A number of researchers in computational/systems neuroscience and in information/communication theory are investigating problems of information representation and processing. While the goals are often the same, these researchers bring different perspectives and points of view to a common set of neuroscience problems. Often they participate in different fora and their interaction is limited.

The goal of the workshop is to bring some of these researchers together to discuss challenges posed by neuroscience and to exchange ideas and present their latest work. The workshop is targeted towards computational and systems neuroscientists with interest in methods of information theory as well as information/communication theorists with interest in neuroscience.

Speakers:

- Lionel Barnett (University of Sussex)
- Demian Battaglia (Institute for Systems Neuroscience, Marseilles)
- John Beggs (Indiana University)
- Braden Brinkman (University of Washington, to be confirmed)
- Sakyasingha Dasgutpa, RIKEN Brain Science Insititute / IBM Research - Tokyo
- Justin Dauwels, Nanyang Technological University
- Joseph T. Lizier, The University of Sydney
- Mark McDonnell, University of South Australia
- Masafumi Oizumi, Monash University
- Rama Ratnam, University of Illinois at Urbana-Champaign (USA), and Advanced Digital Sciences Center, Illinois at Singapore (Singapore)
- Tatyana Sharpee, Salk Institute for Biological Studies
- Shigeru Shinomoto, Kyoto University
- Eli Shlizerman (University of Washington, to be confirmed)
- Adria Tauste (Universitat Pompeu Fabra)
- Taro Toyoizumi, RIKEN Brain Science Institute
- Michael Wibral, Goethe University
- Si Wu, Beijing Normal University

W2 Connectome: Structure and Large Scale Dynamics

302, Wednesday, 9:00 – 16:30

Leonardo L Gollo, QIMR Berghofer Medical Research Institute, Australia
James A. Roberts, QIMR Berghofer Medical Research Institute, Australia

Studies of the connectome are re-shaping the field of neuroscience. Networks have become a ubiquitous language. This is certainly reflected in computational neuroscience, where more and more groups are addressing problems at the large scale. However, the number of open questions is growing rapidly, so it is timely for computational neuroscientists to both direct our attention to the most important issues, and to grow capacity to take advantage of the opportunities that are unfolding. The workshop will present and highlight some of the important recent contributions on the structure of the connectome and the large-scale dynamics that it supports. We expect to have two round table sessions (closing the morning and the afternoon sessions) in which discussion will take place with the specific aim of exposing and highlighting the main issues and the interfaces where quantitative skills (abundant among computational neuroscientists) can be successfully applied to address exceptional emerging problems.

Speakers:

- Selen Atasoy (UPF)
- Ben D. Fulcher (Monash)
- Leonardo L. Gollo (QIMRB)
- Christopher J. Honey (Toronto)
- Jorge F. Mejias (NYU)
- Bratislav Misic (Indiana)
- James A. Roberts (QIMRB)
- Paula Sanz-Leon (Sydney)
- Andreas Spiegler (AMU)
- Andrew Zalesky (Melbourne)
- Changsong Zhou (HKBU)

W3 **Statistical Analysis for Neural Time Series**

302, Thursday, 9:00 – 16:30

Il Memming Park, Stony Brook University
Ian Stevenson, University of Connecticut

New technologies for recording from large groups of neurons provide an exciting opportunity for figuring out how the nervous system implements computations that underlie perception, cognition, and behavior. However, neural time series are complex and often high-dimensional, and there is a major bottleneck in statistical and computational methods for making sense of them. We aim to discuss statistical approaches for analyzing neural time series to increase our understanding of the neural code and computation. Scientific questions of interest include, but not limited to,

1. How can we incorporate neuroscience knowledge on the structure of the circuit or dynamics into neural data analysis?
2. How can we make efficient use of noisy, limited data? and
3. What machine learning tools can be applied to nonlinear neural time series?

Speakers:

- Shin Ishii (Kyoto University, Japan)
- Justin Dauwels (Nanyang Technological University, Singapore)
- Taro Toyoizumi (RIKEN Brain Science Institute, Japan)
- Sukbin Lim (NYU Shanghai, China)
- Eftychios Pnevmatikakis (Simons Center for Data Analysis, USA)
- Si Wu (Beijing Normal University, China)
- Shinsuke Koyama (Institute of Statistical Mathematics, Japan)
- Memming Park (Stony Brook University, USA)

W4 Multi-Area Models of Cortex

402A, Thursday, 9:00 – 16:30

Sacha Jennifer van Albada, Institute of Neuroscience and Medicine (INM-6) Computational and Systems Neuroscience and Institute for Advanced Simulation (IAS-6) Theoretical Neuroscience and JARA BRAIN Institute I, Julich Research Centre, Julich, Germany

Gustavo Deco, Center for Brain and Cognition, Computational Neuroscience Group, Department of Information and Communication Technologies & Institució Catalana de la Recerca i Estudis Avançats (ICREA), Universitat Pompeu Fabra, Barcelona, Spain

Cortical areas do not operate in isolation; rather, they interact extensively even during rest, and work together to produce function. Due to a lack of available human and computational resources as well as anatomical and physiological data, multi-area models of cortex are traditionally heavily simplified. Recent advances in computational resources, simulation technology and experimental data are expanding the options for large-scale cortical modeling. Through their integrative nature, large-scale brain models help identify gaps in experimental knowledge.

This workshop aims to provide an overview over current multi-area cortical modeling efforts, prominent experimental findings addressed by such models, and ways in which systematic knowledge can be gained from large-scale simulation studies, for instance with the help of mean-field theory.

The workshop targets modelers, theorists and experimentalists interested in multi-area cortical models, the underlying methodology, and the data needed to specify them.

Speakers:

- Andre M Bastos (MIT)
- Steven Bressler (Florida Atlantic University)
- Joana Cabral (University of Oxford)
- Martin Giese (University Clinic Tubingen)
- Matthieu Gilson (Pompeu Fabra University)
- Stefan Mihalas (Allen Institute for Brain Science)
- Paula Sanz-Leon (The University of Sydney)
- Maximilian Schmidt (Julich Research Centre)
- Xiao-Jing Wang (New York University)

W5 **Dynamical principles in Neural circuits**

402A, Wednesday, 9:00 – 12:00

Andrey Shilnikov, Georgia State University, USA
Akira Sakurai, Georgia State University, USA

The workshop will address the fundamental question of how circuit architectures infer and contribute to the dynamics of neural activity. Understanding generic mechanisms of the evolution of neural connectivity and transitions between different patterns of neural activity and modeling these processes are the fundamental challenges for applied mathematics and computational neuroscience. It will extend and generalize our understanding of dynamical principles in neural systems. Current and future findings will provide a systematic basis for comprehension of plausible biophysical mechanisms for the origination and regulation of rhythmic patterns including ones generated by central pattern generators.

Speakers:

- Yaroslav Molkov (Georgia State, USA)
- Thomas Nowotny (Essex, UK)
- Choongseok Park (NC A&T State, USA)
- Astrid Prinz (Emory, USA)
- Leonid Runchinsky (IUPI, SA)
- Akira Sakurai (Georgia State, USA)
- David Terman (Ohio State, USA)
- Krasimira Tsaneva-Atanasova (Exeter, UK)
- Kyle Wedgwood (Exeter, UK)

W6 **Cortical Microcircuits: Understanding network structure and function in cortical processing**

303A + 303B, Wednesday, 9:00 – 12:00

Hamish Meffin, National Vision Research Institute, and Department of Optometry and Visual Science, The University of Melbourne

Anthony Burkitt, Department of Electrical and Electronic Engineering, The University of Melbourne

Understanding how our brain computes and analyses sensory inputs from our external environment whilst enabling us to experience such rich and varied mental lives is one of the great scientific challenges of the 21st Century. Recent advances have uncovered much about the cerebral cortex, with its 2-4mm thick sheet of neurons having a consistent anatomical structure consisting of six well-characterised layers and network connectivity. This workshop aims to draw together some of the recent research in understanding these cortical microcircuits and the various approaches that are being pursued to analyse their structure and function.

Speakers:

- Michael Riemann (Blue Brain Project, EPFL, Switzerland)
- Andre Bastos (Picower Institute for Learning and Memory, MIT , USA)
- Hannah Bos (Institute of Neuroscience and Medicine, Research Centre Julich , Germany)
- Jorge Mejias (Center for Neural Science , New York University, USA)
- Abigail Morrison (Institute of Neuroscience and Medicine, Research Centre Julich , Germany)

W7 **Recent advances and applications in real-time single-trial EEG analysis**

303A + 303B, Wednesday, 13:30 – 16:30

Tzyy-Ping Jung, University of California, San Diego
John K. Zao, Chiao-Tung University
Jee Hyun Choi, Korea Institute of Science and Technology

Recent advances in wearable, dry-electrode electroencephalogram (EEG) system revolutionize the real time brain monitoring, yielding exciting new possibilities for clinical diagnostics and brain-computer interface outside the lab environment.

In this workshop, we will present the current state-of-the art in real time decoding of cognitive process in EEG signals. Talks will cover the analysis and measurement platform for the various representations of cognitive functions. Additionally, clinical and neuroscientific application will be presented. This workshop will take a broad view of contemporary EEG research and will be of interest to basic, translational, clinical investigators notwithstanding the engineers.

Speakers:

- Tzyy-Ping Jung (University of California, San Diego)
- John K. Zao (National Chiao Tung University)
- Jee Hyun Choi (Korea Institute of Science and Technology)
- Kyung Hwan Kim (Yonsei University)
- Chang-Hwan Im (Hanyang University)
- Sung Phil Kim (Ulsan National Institute of Science and Technology)
- Han-Jeong Hwang (Kumoh National Institute of Technology)

Posters

Poster Listing

Sunday Posters
Posters P1 – P68

P1 **Neural network as a scale-free network: The Role of a Hub**

Byungnam Kahng*

Department of Physics and Astronomy, Seoul National University, 08826, Korea

P2 **Hemodynamic Responses to Emotions and Decisions using Near-infrared Spectroscopy Optical Imaging**

Nicoladie D Tam*

Department of Biological Sciences, University of North Texas, Denton, TX 76203, USA

P3 **Phase Space Analysis of Hemodynamic Responses to Intentional Movement Directions using Functional Near-Infrared Spectroscopy (fNIRS) Optical Imaging Technique**

Nicoladie D Tam[1]*, Luca Pollonini[2], and George Zouridakis[3]

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[3]*Departments of Engineering Technology, Computer Science, and Electrical and Computer Engineering, University of Houston, Houston, TX, 77204, USA*

P4 **Modeling Jamming Avoidance of Weakly Electric Fish**

Jaehyun Soh, Daeeun Kim*

Biological Cybernetics, School of Electrical and Electronic Engineering, Yonsei University, Shinchon, Seoul, 120-749, South Korea,

P5 **Synergy and redundancy of retinal ganglion cells in prediction**

Minsu Yoo[1]*, Stephanie E Palmer[1,2]

[1]*Committee on Computational Neuroscience, University of Chicago, Chicago, IL, USA*
[2]*Department of Organismal Biology and Anatomy, University of Chicago, Chicago, IL, USA*

P6 **A neural field model with a third dimension representing cortical depth**

Viviana Culmone*, Ingo Bojak

School of Psychology, University of Reading, Reading, Berkshire, RG1 6AY, UK

P7 **Network analysis of a probabilistic connectivity model of the Xenopus tadpole spinal cord**

Andrea Ferrario*, Robert Merrison-Hort, and Roman Borisyuk

School of Computing and Mathematics, Plymouth University, Plymouth, PL4 8AA, United Kingdom

P8 The Recognition Dynamics in the Brain

Chang Sub Kim*

Department of Physics, Chonnam National University, Gwangju, 61186, Republic of Korea

P9 Multivariate Spike Train Analysis using a Positive Definite Kernel

Taro Tezuka*

Faculty of Library, Information and Media Science, University of Tsukuba, Tsukuba, 305-0821, Japan

P10 Synchronization of burst periods may govern slow brain dynamics during general anesthesia

Joo Pangyu*

Department of Physics, POSTECH, Pohang, 37673, Republic of Korea

P11 The ionic basis of heterogeneity affects stochastic synchrony

Young-Ah Rho[1,4]*, Shawn Burton[2,3], G. Bard Ermentrout[1,3], Jaeseung Jeong[4], and Nathaniel M Urban[2,3]

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P12 Circular statistics of noise in spike trains with a periodic component

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P13 Using fractional order dynamics to study non-Markovian neuronal activity

Fidel Santamaria*

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P14 Representations of directions in EEG-BCI using Gaussian readouts

Hoon-Hee Kim[1,2]*, Seok-Hyun Moon[3], Do-Won Lee[3], Sung-Beom Lee[3], Ji-Yong Lee[3], and Jaeseung Jeong[1,2]

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P15 Action Selection and Reinforcement Learning in Basal Ganglia during Reaching Movements

Yaroslav Molkov[1]*, Khaldoun Hamade[2], Wondimu Teka[3], William Barnett[1], Taegyo Kim[2], Sergey Markin[2], and Ilya Rybak[2]

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P16 Plasticity-driven self-organization under topological constraints accounts for non-random features of cortical synaptic wiring

Daniel Miner*, Jochen Triesch

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P17 Axon guidance: modeling axonal growth in T-Junction assay

Csaba Forro*, Harald Dermutz, Laszlo Demko, and Janos Voros

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P18 Modelling visual attention using spiking neural networks

Roberto A Vazquez*

Intelligent Systems Group, Faculty of Engineering, La Salle University, Mexico City, 06140, MEX

P19 Transient cell assembly networks encode persistent spatial memories

Yuri Dabaghian[1,2]*, Andrey Babichev[1,2]

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P20 Theory of population coupling and applications to describe high order correlations in large populations of interacting neurons

Haiping Huang*

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P21 Design of biologically-realistic simulations for motor control

Sergio Verduzco-Flores*

Computational Neuroscience Unit, Okinawa Institute of Science and Technology, Okinawa 1919-1, Japan

P22 Towards understanding the functional impact of the behavioural variability of neurons

Filipa Dos Santos*, Peter Andras

School of Computing and Mathematics, Keele University, ST5 5BG, UK

P23 Different oscillatory dynamics underlying gamma entrainment deficits in schizophrenia

Christoph Metzner[1]*, Achim Schweikard[2], and Bartosz Zurowski[3]

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[2]*Institute for Robotics and Cognitive Systems, University of Luebeck, Luebeck, Germany*
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P24 Memory recall and spike frequency adaptation.

James P Roach[1]*, Leonard Sander[2,3], and Michal Zochowski[2,3,4]

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[3]*Department of Physics, University of Michigan, Ann Arbor, MI 48109, USA*
[4]*Biophysics Program, University of Michigan, Ann Arbor, MI 48109, USA*

P25 Stability of neural networks and memory consolidation preferentially occur near criticality

Quinton M Skilling[1⋆], Nicolette Ognjanovski[2], Sara Aton[2], and Michal Zochowski[1,3]

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P26 Stochastic Oscillation in Self-Organized Critical States of Small Systems: Sensitive Resting State in Neural Systems

Shengjun Wang[1,2], Guang Ouyang[2], Jing Guang[3], Mingsha Zhang[3], Ky Michael Wong[4], and Changsong Zhou[2,5,6⋆]

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[3]State Key Laboratory of Cognitive Neuroscience and Learning, Beijing Normal University, Beijing, China
[4]Department of Physics, Hong Kong University of Science and Technology, Clear Water Bay, Hong Kong
[5]Beijing Computational Science Research Center, Beijing 100084, People's Republic of China
[6]Research Centre, HKBU Institute of Research and Continuing Education, Shenzhen, China

P27 Neurofield: A C++ library for fast simulation of 2D neural field models

Peter Robinson[1,2], Paula Sanz-Leon[1,2⋆], Peter Drysdale[1,2], Felix Fung[1,2], Romesh Abeysuriya[3], Chris Rennie[1,2], and Xuelong Zhao[1,2]

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[3]Department of Psychiatry, Medical Sciences Division, University of Oxford, Oxford, OX37JX, United Kingdom

P28 Action-based grounding: Beyond encoding/decoding in neural code

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[1]Department of Computer Science & Engineering, Texas A&M University, College Station, TX, 77845, USA
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P29 Neural computation in a dynamical system with multiple time scales

Yuanyuan Mi, Xiaohan Lin, and Si Wu

State Key Lab of Cognitive Neuroscience & Learning, IDG/McGovern Institute for Brain Research, Beijing Normal University, Beijing 100875, China

P30 Maximum entropy models for 3D layouts of orientation selectivity

Joscha Liedtke[1,2], Manuel Schottdorf[1,2⋆], and Fred Wolf[1,2]

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P31 A behavioral assay for probing computations underlying curiosity in rodents

Yoriko Yamamura⋆, Jeffery Wickens

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P32 Using statistical sampling to balance error function contributions to optimization of conductance-based models

Timothy Rumbell[1], Julia Ramsey[2], Amy Reyes[2], Danel Draguljic[2], Patrick Hof[3], Jennifer Luebke[4], and Christina M Weaver[2⋆]

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P33 Exploration and implementation of a self-growing and self-organizing neuron network building algorithm

Hu He[1], Xu Yang[2⋆], Hailin Ma[1], Zhiheng Xu[1], and Yuzhe Wang[1]

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P34 Disrupted resting state brain network in obese subjects: A data-driven graph theory analysis

Kwangyeol Baek[1,2⋆], Laurel Morris[1], Prantik Kundu[3], and Valerie Voon[1]

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P35 Dynamics of cooperative excitatory and inhibitory plasticity

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P36 Frequency-dependent oscillatory signal gating in feed-forward networks of integrate-and-fire neurons

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P37 Phenomenological neural model for adaptation of neurons in area IT

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P38 ICGenealogy: Towards a Common Topology of Neuronal Ion Channel Function and Genealogy in Model and Experiment

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P39 Temporal input discrimination from the interaction between dynamic synapses and neural subthreshold oscillations

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P40 Different roles for transient and sustained activity during active visual processing

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P41 Scale-free functional networks of 2D Ising model are highly robust against structural defects: Neuroscience implications

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P42 High frequency neuron can facilitate propagation of signal in neural networks

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P43 Investigating the effect of Alzheimer's disease related amyloidopathy on gamma oscillations in the CA1 region of the hippocampus

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P44 Long-tailed distributions of inhibitory and excitatory weights in a balanced network with eSTDP and iSTDP

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P45 Simulation of EMG recording from hand muscle due to TMS of motor cortex

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P46 Structure and dynamics of axon network formed in primary cell culture

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P47 Efficient signal processing and sampling in random networks that generate variability

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P48 Modeling the effect of riluzole on bursting in respiratory neural networks

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P49 Mapping relaxation training using effective connectivity analysis

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P50 Modeling neuron oscillation of implicit sequence learning

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P51 The role of cerebellar short-term synaptic plasticity in the pathology and medication of downbeat nystagmus

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P52 Nonlinear response of noisy neurons

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P53 Behavioral Embedding Suggests Multiple Chaotic Dimensions Underlie C. elegans Locomotion

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P54 Fast and scalable spike sorting for large and dense multi-electrodes recordings

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P55 Sufficient sampling rates for fast hand motion tracking

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P56 Linear Readout of Object Manifolds

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P57 Differentiating models of intrinsic bursting and rhythm generation of the respiratory pre-Bötzinger complex using phase response curves

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P58 The effect of inhibitory cell network interactions during theta rhythms on extracellular field potentials in CA1 hippocampus

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P59 Expansion recoding through sparse sampling in the cerebellar input layer speeds learning

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P60 A set of curated cortical models at multiple scales on Open Source Brain

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P61 A Synaptic Story of Dynamical Information Encoding in Neural Adaptation

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P62 Physical Modeling of Rule-observant Rodent Behavior

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P63 How Adaptation Makes Low Firing Rates Robust

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P64 Predictive coding in area V4 and prefrontal cortex explains dynamic discrimination of partially occluded shapes

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P65 Stability of FORCE learning on spiking and rate-based networks

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P66 Stabilising STDP in striatal neurons for reliable fast state recognition in noisy environments

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P67 Electrodiffusion in One- and Two-Compartment Neuron Models for Characterizing Cellular Effects of Electrical Stimulation

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P68 STDP improves speech recognition capabilities in spiking recurrent circuits parameterized via Differential Evolution Markov Chain Monte Carlo

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P69 Bidirectional transformation between dominant cortical neural activities and phase difference distributions

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P70 Maturation of sensory networks through homeostatic structural plasticity

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P71 Corticothalamic dynamics: structure, number of solutions and stability of steady-state solutions in the space of synaptic couplings

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P72 Optogenetic vs. electrical stimulation of the parkinsonian basal ganglia. Computational study

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P73 Exact spike-timing distribution reveals higher-order interactions

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P74 Neural Mechanism of Visual Perceptual Learning Using a Multi-layered Neural Network

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P75 Inferring collective spiking dynamics from mostly unobserved systems

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P76 How to infer distributions in the brain from subsampled observations

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P77 Influences of embedding and estimation strategies on the inferred memory of single spiking neurons

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P78 A nearest-neighbours based estimator for transfer entropy between spike trains

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P79 Active learning of psychometric functions with multinomial logistic models

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P80 Connectome harmonics reveal organizing principles behind brain's functional networks

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P81 Inferring low-dimensional network dynamics with variational latent Gaussian process

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P82 Computational investigation of energy landscapes in the resting state subcortical brain network

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P83 Local repulsive interaction between retinal ganglion cells can generate a consistent spatial periodicity of orientation map

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P84 Phase duration of bistable perception reveals intrinsic time scale of perceptual decision under noisy condition

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P85 Feedforward convergence between retina and primary visual cortex can determine the structure of orientation map

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P86 Quantitative Classification of Neural Network Activity Patterns in Imaging Data

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P87 Symmetry of spike-timing-dependent-plasticity kernels regulates volatility of memory

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P88 Effects of time-periodic coupling strength on the first-spike latency dynamics of a scale-free network of stochastic Hodgkin-Huxley neurons

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P89 Spectral properties of spiking responses in V1 and V4 change within the trial and are highly relevant for behavioral performance.

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P90 Methods for building accurate models of individual neurons

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P91 A full size mathematical model of the early olfactory system of honeybees

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P92 Stimulation-Induced Tuning of Ongoing Oscillations in Spiking Neural Networks

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P93 Decision-specific sequences of neural activity in balanced random networks driven by structured sensory input

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P94 Modulation of tuning induced by abrupt reduction of SST cell activity

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P95 The functional role of VIP cell activation during locomotion

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P96 Stochastic inference with spiking neural networks

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P97 Modelling orientation-selective electrical stimulation with retinal prostheses

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P98 Ion channel noise can explain firing correlation in auditory nerves

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P99 Limits of temporal encoding of thalamocortical inputs in a neocortical microcircuit

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P100 On the representation of arm reaching movements: a computational model

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P101 A computational model for investigating the role of cerebellum in acquisition and retention of motor behavior

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P102 The emergence of semantic categories from a large-scale brain network of semantic knowledge

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P103 Multiscale modeling of M1 multitarget pharmacotherapy for dystonia

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P104 Effect of network size on computational capacity

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P105 NetPyNE: a Python package for NEURON to facilitate development and parallel simulation of biological neuronal networks

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P106 Effect of Network Structure on Population Synchronization in A Scale-Free Network of Bursting Neurons

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P107 Inter-areal and Inter-regional Inhomogeneity in Co-axial Anisotropy of Cortical Point Spread in Human Visual Areas

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P108 Two Bayesian Quanta of Uncertainty Explain the Temporal Dynamics of Cortical Activity in the Non-Sensory Areas during Bistable Perception

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P109 Optimal and suboptimal integration of sensory and value information in perceptual decision making

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P110 A Bayesian Algorithm for Phoneme Perception and Its Neural Implementation

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P111 Complexity of EEG signals is reduced during unconsciousness induced by ketamine and propofol

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P112 Self-Organized Criticality of Neural Avalanche in a Neural Model on Complex Networks

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P113 Dynamic alterations in connection topology of the hippocampal network during ictal-like epileptiform activity in an in vitro rat model

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P114 Computational Model to Replicate Seizure Suppression Effect by Electrical Stimulation

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P115 Identifying excitatory and inhibitory synapses in neuronal networks from spike trains using Sorted Local Transfer Entropy

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P116 Neural network model for obstacle avoidance based on neuromorphic computational model of boundary vector cell and head direction cell

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P117 Dynamic gating of spike pattern propagation by Hebbian and anti-Hebbian spike timing-dependent plasticity in excitatory feedforward network model

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P118 Inferring characteristics of input correlations of cells exhibiting up-down state transitions in the rat striatum

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P119 Graph properties of the functional connected brain under the influence of Alzheimer's disease

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P120 Learning sparse representations in the olfactory bulb.

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P121 Functional classification of homologous basal-ganglia networks

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P122 Short Term Memory Based on Multistability

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P123 A physiologically plausible, computationally efficient model and simulation software for mammalian motor units

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P124 High-resolution current source density reconstruction by Gaussian interpolation for microelectrode array analysis of hippocampal network dynamics following theta-burst stimulation

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P125 Decoding laser-induced somatosensory information from EEG

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P126 Phase synchronization of alpha activity for EEG-based personal authentication

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P127 Altered small-world cortical network in patients with schizophrenia during an auditory oddball paradigm task: an EEG study

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P128 – Withdrawn –

P129 Investigating phase-lags in sEEG data using spatially distributed time delays in a large-scale brain network model

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P130 Epileptic seizures in the unfolding of a codimension-3 singularity

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P131 Incremental dimensional exploratory reasoning under multi-dimensional environment

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P132 A low-cost model of eye movements and memory in personal visual cognition

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P133 Complex network analysis of structural connectome of autism spectrum disorder patients

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P134 Cognitive motives and the neural correlates underlying human social information transmission, gossip.

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P135 EEG hyperscanning detects neural oscillation for the social interaction during the economic decision-making

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P136 Detecting purchase decision based on hyperfrontality of the EEG

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P137 Vulnerability-based critical neurons, synapses, and pathways in the Caenorhabditis elegans connectome

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P138 Motif analysis reveals functionally asymmetrical neurons in C. elegans

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P139 Computational approach to Preference-Based Serial Decision Dynamics: Do Temporal Discounting and Working Memory affect it?

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P140 – Withdrawn –

P141 Social stress induced neural network reconfiguration affects decision making and learning in zebrafish

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P142 Descriptive, generative, and hybrid approaches for neural connectivity inference from neural activity data

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P143 Optimal tDCS electrode montages to stimulate nonsuperficial cortical regions: a simulation study

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P144 Contrast dependent phase sensitivity of complex cells in primary visual cortex

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P145 Divergent-convergent synaptic connectivities accelerate coding in multilayered sensory systems

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P146 Swinging networks

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P147 Inferring dynamically relevant motifs from oscillatory stimuli: challenges, pitfalls, and solutions

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P148 Spatiotemporal mapping of brain network dynamics during cognitive tasks using magnetoencephalography and deep learning

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P149 Multiscale complexity analysis for the segmentation of MRI images

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P150 A neuro-computational model of emotional attention

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P151 Multi-site delayed feedback stimulation in parkinsonian networks

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P152 Bistability in Hodgkin-Huxley-type equations

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P153 Phase changes in postsynaptic spiking due to synaptic connectivity and short term plasticity: mathematical analysis of frequency dependency

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P154 Quantifying resilience patterns in brain networks: The importance of directionality

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P155 Dynamics of rate-model networks with separate excitatory and inhibitory populations

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P156 A model for multi-stable dynamics in action recognition modulated by integration of silhouette and shading cues

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P157 Spiking model for the interaction between action recognition and action execution

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P158 Surprise-modulated belief update: how to learn within changing environments?

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P159 A fast, stochastic and adaptive model of auditory nerve responses to cochlear implant stimulation

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P160 Quantitative comparison of graph theoretical measures of simulated and empirical functional brain networks

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P161 Determining discriminative properties of fMRI signals in schizophrenia using highly comparative time-series analysis

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P162 Emergence of narrowband LFP oscillations from completely asynchronous activity during seizures and high-frequency oscillations

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P163 Neuronal diversity in structure and function: cross-validation of anatomical and physiological classification of retinal ganglion cells in the mouse

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P164 Analysis and modelling of transient firing rate changes in area MT in response to rapid stimulus feature changes

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P165 Step-wise model fitting accounting for high-resolution spatial measurements: Construction of a layer V pyramidal cell model with reduced morphology

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P166 Contributions of schizophrenia-associated genes to neuron firing and cardiac pacemaking: a polygenic modeling approach

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P167 Local field potentials in a 4x4 mm2 multi-layered network model

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P168 A spiking network model explains multi-scale properties of cortical dynamics

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P169 Using joint weight-delay spike-timing dependent plasticity to find polychronous neuronal groups

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P170 Tensor decomposition reveals RSNs in simulated resting state fMRI

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P171 Getting in the Groove: testing a new model-based method for comparing task-evoked vs resting-state activity in fMRI data on music listening

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P172 STochastic Engine for Pathway Simulation (STEPS) on massively parallel processors

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P173 Toolkit Support for Complex Parallel Spatial Stochastic Reaction-Diffusion Simulation in STEPS

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P174 Modeling the generation and propagation of Purkinje cell dendritic spikes caused by parallel fiber synaptic input

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P175 Dendritic morphology determines how dendrites are organized into functional subunits

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P176 A model of Ca2+/calmodulin-dependent protein kinase II activity in Long Term Depression at Purkinje cells

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P177 Reward-modulated learning of population-encoded vectors for insect-like navigation in embodied agents

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P178 Data-driven neural models part II: connectivity patterns of human seizures

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P179 Data-driven neural models part I: state and parameter estimation

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P180 Spectral and spatial information processing in human auditory streaming.

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P181 A tuning curve for the global effects of local perturbations in neural activity: Mapping the systems-level susceptibility of the brain

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P182 Diverse homeostatic responses to visual deprivation mediated by neural ensembles

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P183 Opto-EEG: A novel method for functional connectome in mouse brain based on optogenetics and high density electroencephalography

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P184 Biphasic responses of frontal gamma network to repetitive sleep deprivation during REM sleep

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P185 Brain-state correlate and cortical connectivity for frontal gamma oscillations in top-down fashion assessed by auditory steady-state response

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P186 Neural field model of localized orientation selective activation in V1

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P187 An oscillatory network model of Head direction and Grid cells using locomotor inputs

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P188 A computational model of Hippocampus inspired by the functional architecture of Basal Ganglia

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P189 A computational architecture to model the microanatomy of the striatum and its functional properties

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P190 A scalable cortico-basal ganglia model to understand the neural dynamics of targeted reaching

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P191 Emergence of radial orientation selectivity from synaptic plasticity

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P192 How do hidden units shape effective connections between neurons?

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P193 Characterization of neural firing in the presence of astrocyte-synapse signaling

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P194 Metastability of spatiotemporal patterns in a large-scale network model of brain dynamics

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P195 Comparison of three methods to quantify detection and discrimination capacity estimated from neural population recordings

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P196 Quantifying the constraints for independent evoked and spontaneous nmda receptor mediated synaptic transmission at individual synapses

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P199 Gamma oscillation via adaptive exponential integrate-and-fire neurons

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P200 Visual face representations during memory retrieval compared to perception

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P201 Top-down modulation of sequential activity within packets modeled using avalanche dynamics

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