CNS 2015

July 18 - 23

Prague, Czech Republic

computational neuroscience

The Program Book

Contents

We are grateful to the following organizations for their support without which none of this would be possible:

Springer

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Overview

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- **Petr Maršálek** (Charles University in Prague).
- **Martin Zápotocký** (Czech Academy of Sciences).

Fundraising

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.

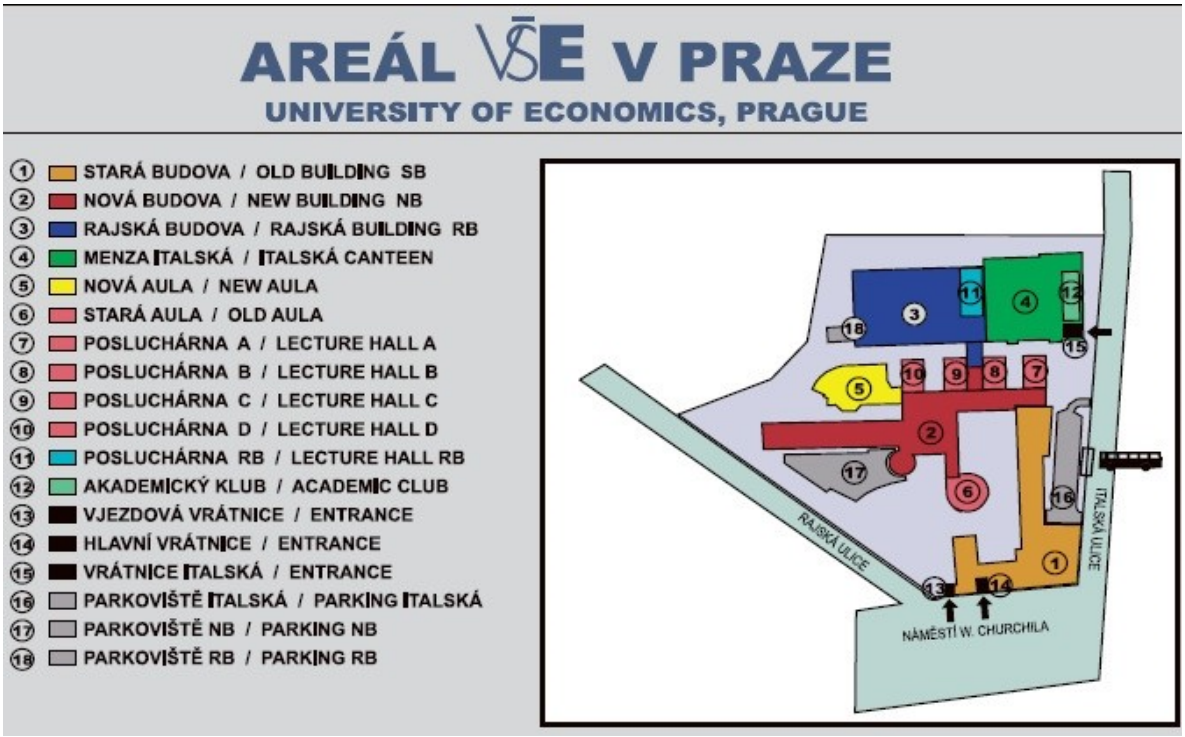
General Information

Meeting venue

University of Economics
W. Churchill Sq. 4
130 67 Prague 3
Czech Republic

The conference will be held on the campus of the University of Economics located in Žižkov - a city quarter situated just few minutes from the city centre, close to the range of cultural attractions that Prague offers.
The campus is well served by public transport. The main train station is in walking distance, as well as the subway line C, stop 'Hlavní nádraží', and the tram stop 'Husinecká'.

Plan of the campus:



Meeting room locations: See summary on back cover or single maps included in the corresponding sections (Tutorials, Main Meeting, Workshops).

Getting to the conference venue

From the Ruzyně (Vaclav Havel) Airport:

...by Public transport:

A.

Get on the bus #119 (the bus stop is situated across the road from the arrivals hall – follow the sign) and go to Nadrazi Veleslavin metro (and train) station. The bus from the airport runs every 20 minutes. Take the green metro line A to Mustek metro station (4 stations). As there are two exits, remember to follow the sign for Vaclavske Square. Take tram #9 in direction of Spojovaci and go to Husinecka tram stop (three stations) and then go down to W. Churchill Square. The whole journey takes about 50 minutes. Ticket: 32 CZK.

B.

Get on the bus #100 and go to Zlicín metro station. The bus from the airport runs every 30 minutes. Take the yellow metro line B to Namesti Republiky metro station (13 stations). As there are two exits, remember to follow the sign for Masarykovo nadrazi. Take tram #5 or #26 and go to Husinecka tram stop (two stations) and then go down to W. Churchill Square. The whole journey takes about 65 minutes. Ticket: 32 CZK.

...by Taxi:

The price for a taxi from the Ruzyne airport to the conference venue should be about 750 CZK (30 Euro).

From the Prague Main Train Station (Praha Hlavní nádraží):

...by public transport:

Get on the tram number 5 (in the direction of Olšanské hřbitovy), 9 (in the direction of Spojovací) or 26 (in the direction of Nádraží Hostivař). Go to Husinecká station (two stops), and then go down to W. Churchill square. The whole journey takes about 5 minutes. Ticket: 24 CZK.

...on foot:

The conference location is within a 5 minute walking distance. At the underground exit, go to the right across the street to the 'Bulhar' junction and then under the railway bridge via Seifert Street to the W. Churchill Square.

From Mustek metro station:

Go to the upper level of the underground system at Mustek and follow the sign for Vaclavske namesti â€“ Vodickova ulice to exit the system (there are two exits). Take tram #9 and go to Husinecka tram stop (three stations) and then go down to W. Churchill square.

From Náměstí republiky metro station:

Go to the upper level of the underground system at Namesti Republiky and follow the sign for Masarykovo nadrazi to exit the system. Take tram #5 or #26 and go to Husinecka tram stop (three stations) and then go down to W. Churchill Square.

Information for poster presentation

The poster area is located in the Atrium. Poster boards will be numbered. Fixing adhesive material will be available. Poster orientation is portrait and maximum net dimensions are 180 cm x 95 cm (A0 is smaller than 120 x 85 cm/ or 47 x 33 in).

Poster Sessions will be held on July 19 and 20 from 4 to 7 pm, and on July 21 from 3:30 to 6:30 pm. Poster set up:

Sunday, July 19, 2015: From 8 am to 4 pm
Monday, July 20, 2015: From 8 am to 4 pm
Tuesday, July 21, 2015: From 8 am to 3 pm

Poster should be removed:

Sunday, Monday, July 19 and 20, 2015: Next day morning latest
Tuesday, July 21, 2015: Before 7 pm

Posters that are not removed by the stated time will be discarded. The organisers are not responsible for loss or damage to posters not removed by the given times.

Registration and locations

Registration will be held in Rajská building, please follow the signage. The entrance is from Italská street, green building no. 4 on the plan of the campus.

Registration hours:

July 18: From 8 am to 6:30 pm
July 19: From 8 am to 7 pm
July 20: From 8 am to 7 pm
July 21: From 8 am to 6:30 pm
July 22: From 8:30 am to 6:30 pm
July 23: From 8:30 am to 6:30 pm

Locations (see also floor plans in the Tutorials, Main Meeting and Workshop sections):

What	Where
Tutorials	RB 113, RB 209, RB 210, RB 211, RB 212, RB 213
Keynote Lectures and Oral Sessions	Vencovského aula, with live audio/video transmission to Likešova aula
Workshops	NB C, NB D, RB 101, RB 113, RB 114, RB 209, RB 210, RB 211, RB 212, RB 213
OCNS board/program committee meetings	RB116
Welcome Reception	Academic Club, 3rd floor - July 18
Exhibition	Atrium, July 19-23
Coffee Breaks	Atrium

Good to Know

Detailed information is available on the official Czech Republic website at www.czech.cz

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 Prague.

Currency & Banking

The Czech Crown (CZK) is the official currency of the Czech Republic. Exchange of foreign currency is available at the Prague International Airport and at most hotels, banks and exchange offices throughout the city. International credit cards are accepted for payments in hotels, restaurants and shops.

Payment in cash (in EUR) is also available in some restaurants and shops, so please ask for details on-site if necessary. You can find the official exchange rates on the Czech National Bank website (http://www.cnb.cz/en).

Electricity

The Czech Republic uses a 230 volt 50 Hz system. Sockets follow the standard also used in France, Belgium, Denmark, Poland, Greece, Italy, Ireland and other countries and also have a grounding pin sticking out of the power socket, which is also known as a type E socket.

Shopping

Most shops in Prague are open from 9:00 to 18:00, Monday to Saturday. Shops in the city centre are usually open from 9:00 to 20:00, Monday to Sunday.

Time Zone

The Czech Republic is on Central European Time (CET), which is Greenwich Mean Time (GMT) + 1 hour. Note that April to October is daylight saving time, i.e. GMT + 2 hours.

Tipping

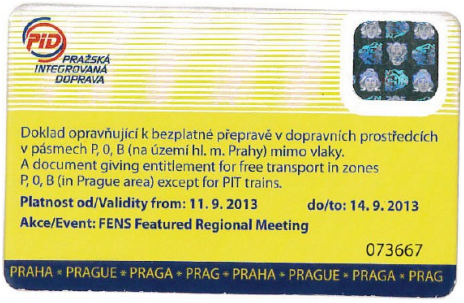
Service is usually included in the bill in most bars and restaurants, but tips are welcome. If you consider the service good enough to warrant a tip, we suggest about ten percent.

Tours

Tours are not a part of the meeting. If you wish to explore the city, check the possibilities in your hotel or choose the tour directly at www.premiant.cz/eng

Transportation

Each registered participant receives one free public transportation pass at the registration desk when registering. This ticket is valid within the dates of the meeting.



Prague has a very sophisticated public transportation system (Prague Integrated Transport, PIT, aka PID). It consists of the metro, trams, buses and the funicular ascending Petřín Hill. Prague's Metro system is quite new and efficient. During peak hours, trains run every 1 or 2 minutes and off peak at least every 10 minutes.

- **Metro:**

  The Metro system consists of three lines:
  Line A – (green), Depo Hostivař – Motol
  Line B – (yellow), Černý Most – Zličín
  Line C – (red), Letňany – Háje

  The Metro operates from 5:00 to 24:00.

- **Buses and trams:**

  Special lines No. 51–59 (trams) and 501–515 (buses) provide an all-night service.

Weather
The month of July is a typical summer month with daily high temperatures, which could exceed 30^{o} C, followed by thunderstorms and showers. You can check for current weather conditions in Prague at www.weather.com.

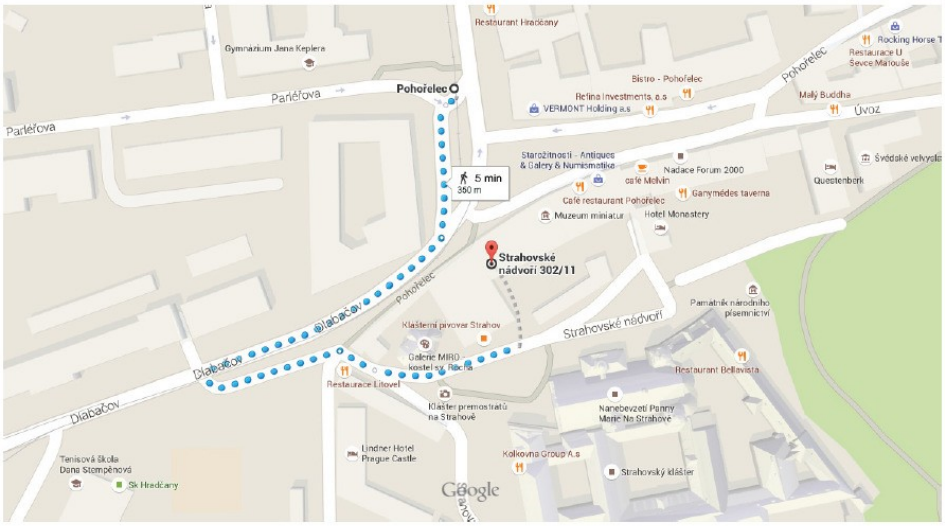
Important Telephone Numbers
112: General Emergency for Europe
150: Fire
155: Ambulance
156: Prague Municipal Police
158: Police

Free Wi-Fi
Free Wi-Fi is provided at the meeting venue. Each registered participant will receive his/hers own unique password.

Taxi Services
In the city centre, taxis are easy to flag down on the street, but we strongly recommend you use hotel taxis or call a taxi by phone through the radio taxi service. We recommend you use the following taxi companies:
AAA Taxi: +420 14 0 14
Profi Taxi: +420 14 0 15

Gala Dinner

Date: Tuesday, July 21, 2015
Time: 19:30
Venue: Velká klášterní restaurace (Strahov monastery restaurant)
Strahovské nádvoří 302, Prague 1 (www.klasternirestaurace.cz/en)
Recommended dress code: Casual
How to get there: Tram #22 from Malostranská metro station, line A to Pohořelec station. Travel time from the conference venue is about 40 minutes.



via Dlabačov **5 min**
Show terrain 350 m
Details

The Royal Canonry of the Premonstratensians at Strahov was established in the year 1140 by the Olomouc Bishop with assistance from the Czech Duke and subsequently Czech King Vladislav II. It is located near Prague Castle at the beginning of the Royal Way.

Strahov monastery restaurant offers Czech and international cuisine including traditional Bohemian specialities prepared according to age-old recipes. The wine served by the restaurant comes from its own Moravian Premonstratensian cellars and bears the name of the founder of the order – St. Norbert. You will enjoy a programme of folk music by the Small Band of Mr. Kettner with demonstrations of traditional Bohemian and Moravian dancing in period costumes.

Several steps from the restaurant you can enjoy a beautiful view over Prague with the Petrin hill on the right and Prague castle on the left. You can walk downhill to Lesser Town, Charles Bridge and the city centre.

CNS Party

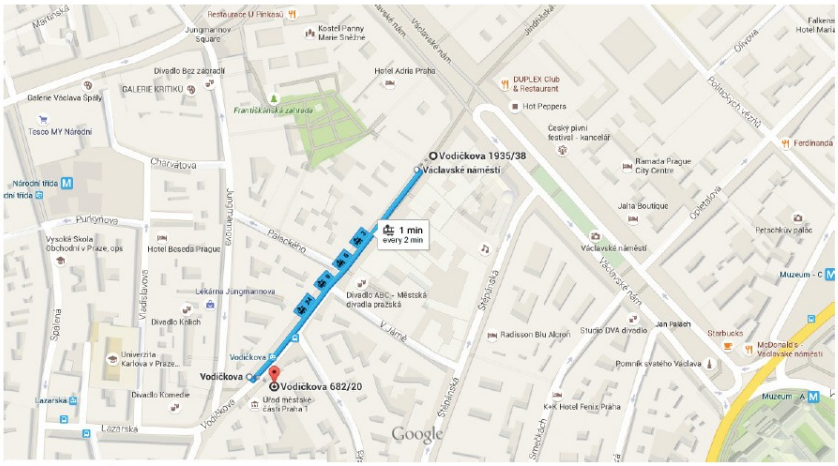
Date: Monday, July 20, 2015
Time: From 19:00 to 23:00
Venue: Novoměstský pivovar (New Town Brewery), Vodičkova 20, Prague 2 (http://npivovar.cz/en)
Recommended dress code: Casual

Each participant gets coupons for two free drinks (beer, wine or soda) and one pretzel. Also included is a mini-brewery guided tour in English language on a first-come first-served basis. Additional food and drinks can be ordered throughout the evening, but are not covered by OCNS. Main courses cost about 8 to 12 USD, beer (0.5 liters) is about 2 USD.
Following the slowly disappearing tradition of past CNS parties, participants are encouraged to bring their own unplugged musical instruments to tune with accordion players circulating among many brewery caves.

How to get there from the meeting venue/W. Churchill Square:

By tram No. 9...
...from Husinecká tram station to Vodickova tram station (3 tram stops are in-between: Hlavni nadrazi, Jindrisska, Vaclavske namesti. Travel time 12 min).

By walking...
...for about 2 km (approx. 25 min).



Restaurants

Selected restaurants close to the venue:

Seifertova Street

- **Restaurant Sklep**

  Czech and international cuisine, daily lunch menu
  www.restauracesklep.cz/en

- **Restaurace Lavička**

  Czech and international cuisine, vegetarian meals, daily lunch menu
  www.restaurace-lavicka.cz/en

- **Restaurace Poja**

  Czech cuisine, steaks, pasta, daily lunch menu
  www.restaurace-poja.cz

- **YES Burger**

  Burger restaurant
  http://yesburger.cz

- **Secret of Raw**

  raw food restaurant, daily lunch menu
  www.secretofraw.cz

- **Žizkovská galérka**

  stylish atmosphere of the 30’s years of the First Republic
  www.zizkovskagalerka.cz/en

- **Kuře v hodinách**

  Czech cuisine
  www.kurevhodinkach.eu

- **Kredenc restaurant**

  Czech and international cuisine, daily lunch menu
  www.kredencrestaurant.cz

- **Pizza Einstein**

  pizza, pasta, salads, daily lunch menu
  www.pizza-einstein.cz

City center, Václavské square area

- **Restaurace Jáma**

  American and mexican specialities
  www.jamapub.cz/en

- **U českých pánu**

  old style restaurant, Czech cuisine
  www.english.uceskychpanu.cz

- **Trilobite**

  Czech and international cuisine, daily lunch menu
  www.restauracetrilobit.cz

- **McDonald, KFC, Burger King**

  Fast food restaurants

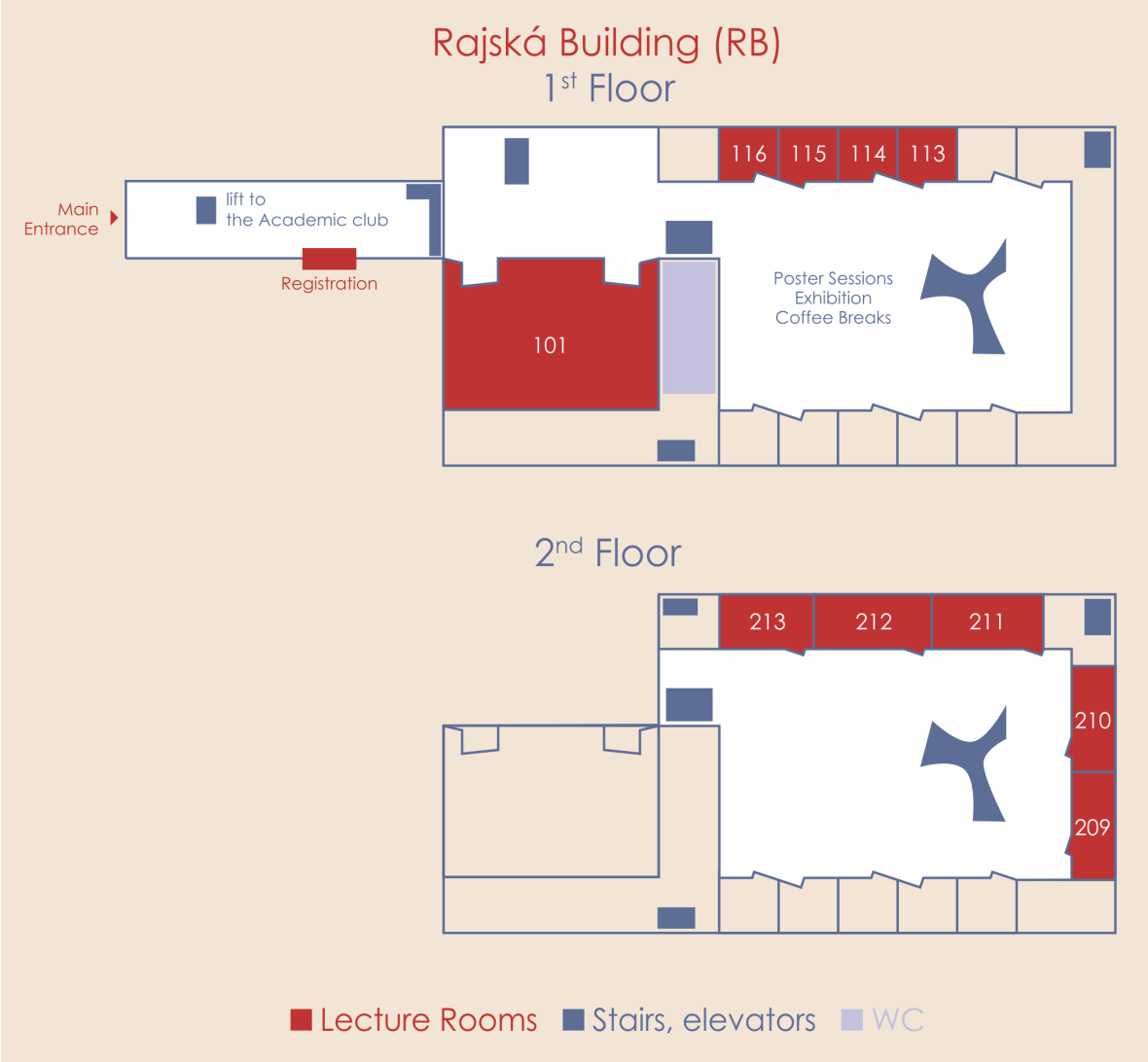
More Prague restaurant you can find at: www.restaurant-guide.cz/en

Where to go if you wish to take a quick break from the conference

- The park "Riegrovy sady" is located just a 5 minute walk from the conference venue. It is a mid-size, well maintained park, with several garden restaurants inside. To go there from the conference building, get out at the "Italska" exit (next to registration desk). Walk UPHILL on Italska street for 150 meters, turn left onto Vozova street. The entrance to the park will be on your right.
- The park and monument "Vitkov" is located about 1 km from the conference building. The park is placed on a tall hill, with a military monument and an enormous statue (15th century commander Jan Zizka on a horse) on the top of the hill. The main attraction is the beautiful view of Prague from the top of the hill. To reach the Vitkov park, walk via "namesti Winstona Churchilla" and "Kostnicke namesti". Please refer to a map, to avoid getting lost in the small streets.

Program

Tutorials



T1 **Neural mass and neural field models**

Room RB 212, 18-Jul-15

Axel Hutt, INRIA Nancy, France
Jeremie Lefebre, University of Lausanne, Switzerland
Alistair Steyn-Ross, University of Wakaito, New Zealand
Nicolas Rougier, INRIA Bordeaux, France

T2 **Modeling and analysis of extracellular potentials**

Room RB 209, 18-Jul-15

Gaute T. Einevoll, Norwegian University of Life Sciences, Aas, Norway
Szymon Leski, Nencki Institute of Experimental Biology, Warsaw, Poland
Espen Hagen, Jülich Research Centre and JARA, Jülich, Germany

T3 **Modelling of spiking neural networks with the Brian simulator**

Room RB 211, 18-Jul-15

Dan Goodman, Imperical College London, UK
Pierre Yger, Institut de la Vision, Paris, France
Romain Brette, Institut de la Vision, Paris, France
Marcel Stimberg, Institut de la Vision, Paris, France

T4 **Theory of correlation transfer and correlation structure in recurrent networks**

Room RB 210, 18-Jul-15

Moritz Helias, Jülich Research Centre, Jülich, Germany
Farzad Farkhoo, Freie Universität Berlin, Berlin, Germany

T5 **Modeling of calcium dynamics and signaling pathways in neurons**

Room RB 213, 18-Jul-15

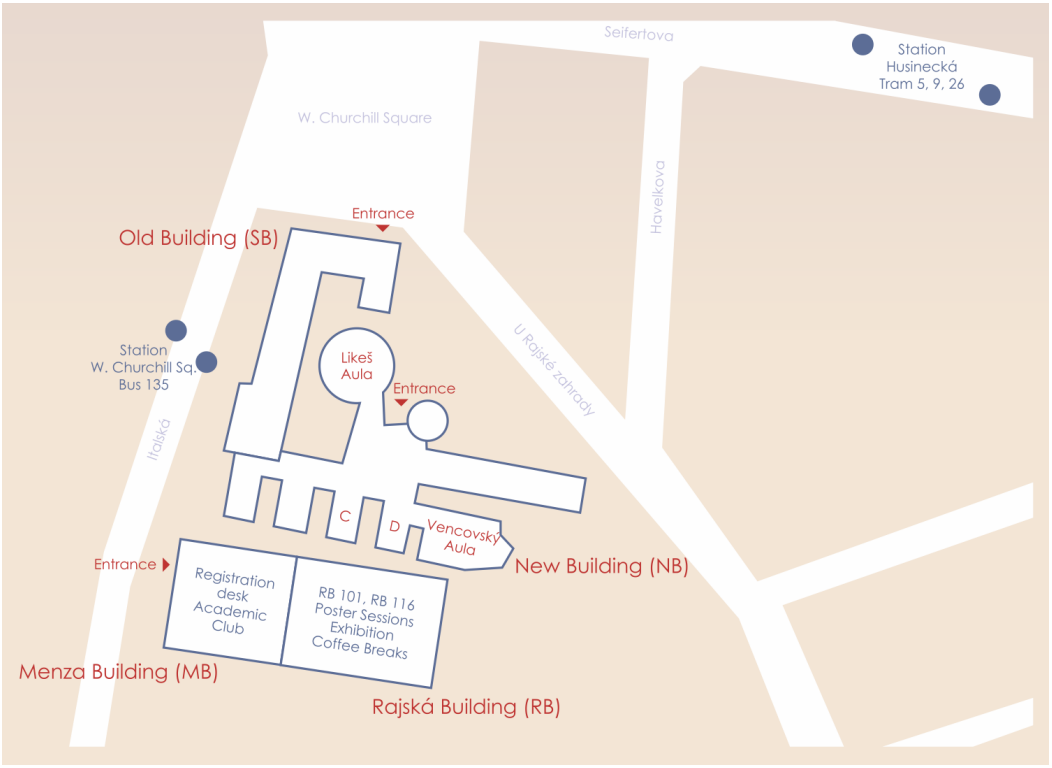
Kim Avrama Blackwell, Krasnow Institute for Advanced Study, George Mason University, Fairfax VA, USA

T6 **Interfaces in Computational Neuroscience Software: Combined use of the tools NEST, CSA and MUSIC**

Room RB 113, 18-Jul-15

Martin Jochen Eppler, Simulation Lab Neuroscience - Bernstein Facility for Simulation and Database Technology, Institute for Advanced Simulation, Jülich Aachen Research Alliance, Forschungszentrum Jülich, Jülich, Germany
Jan Moren, Neural Computation Unit, Okinawa Institute of Science and Technology, Okinawa, Japan
Mikael Djurfeldt, PDC center for high performance computing, KTH and INCF Stockholm, Sweden

Main Meeting



Saturday July 18

9:00 – 16:30		**Tutorials**
17:00 – 17:15		**Welcome and announcements**
17.15 – 18:15	K1	**Keynote 1:** ***Learning and variability in birdsong*** Adrienne Fairhall
18:15		**Welcome reception**

Sunday July 19

9:00 – 9:10		**Announcements**
9:10 – 10:10	K2	**Keynote 2:** ***Modeling Cortical Dynamics with Wilson-Cowan equations*** Jack Cowan
10:10 – 10:40		**Break**

Oral session I: Large Networks

10:40 – 11:00 O1 ***Limits to the scalability of cortical network models***
Sacha van Albada*, Moritz Helias, and Markus Diesmann

11:00 – 11:20 O2 ***The high-conductance state enables neural sampling in networks of LIF neurons***
Mihai A. Petrovici*, Ilja Bytschok, Johannes Bill, Johannes Schemmel, and Karlheinz Meier

11:20 – 11:40 O3 ***Quantifying the distance to criticality under subsampling***
Jens Wilting*, Viola Priesemann

11:40 – 12:00 O4 ***Large-scale analysis of brain-wide electrophysiological diversity reveals novel characterization of mammalian neuron types***
Shreejoy Tripathy*, Dmitry Tebaykin, Brenna Li, Ogan Mancarci, Lilah Toker, and Paul Pavlidis

12:00 – 13:30 **Break for lunch**

Oral session II: Synaptic plasticity

13:30 – 14:10 F1 **Featured oral 1:**
Complex synapses as efficient memory systems
Marcus K Benna*, Stefano Fusi

14:10 – 14:30 O5 ***Self-organization of computation in neural systems by interaction between homeostatic and synaptic plasticity***
Sakyasingha Dasgupta*, Christian Tetzlaff, Tomas Kulvicius, and Florentin Woergoetter

14:30 – 14:50 O6 ***A model for spatially periodic firing in the hippocampal formation based on interacting excitatory and inhibitory plasticity***
Simon Weber*, Henning Sprekeler

14:50 – 15:20 **Break**

Oral session III: Single-cell properties and modeling

15:20 – 15:40 O7 ***Whole-cell morphological properties of neurons constrain the nonrandom features of network connectivity***
Jugoslava Acimovic*, Mäki-Marttunen Tuomo, and Marja-Leena Linne

15:40 – 16:00 O8 ***Origin of the kink of somatic action potentials***
Maria Telenczuk*, Marcel Stimberg, and Romain Brette

16:00 – 19:00 **Poster session I: Posters P1 – P102**

Monday July 20

9:00 – 9:10 **Announcements**

9:10 – 10:10 K3 **Keynote 3:**
The Dynamics of Resting Fluctuations in the Brain
Gustavo Deco

10:10 – 10:40 **Break**

Oral session IV: Visual and auditory processing

10:40 – 11:00 O9 ***An accurate circuit-based description of retinal ganglion cell computation***
Yuwei Cui, Yanbin Wang, Jonathan Demb, and Daniel Butts*

11:00 – 11:20 O10 ***Contrast-dependent Modulation of Gamma Rhythm in V1: a Network Model***
Margarita Zachariou*, Mark Roberts, Eric Lowet, Peter de Weerd, and Avgis Hadjipapas

11:20 – 11:40 O11 ***Downstream changes in firing regularity following damage to the early auditory system***
Dan Goodman*, Alain de Cheveigné, Ian M. Winter, and Christian Lorenzi

11:40 – 12:00 O12 ***Towards a computational model of Dyslexia***
Sagi Jaffe-Dax*, Ofri Raviv, Nori Jacoby, Yonatan Loewenstein, and Merav Ahissar

12:00 – 14:00 **Break for lunch**

Oral session V: Neuromodulation and motor control

13:30 – 14:10 F2 **Featured oral 2:**
Closed-loop approach to tuning deep brain stimulation parameters for Parkinson's disease
Abbey Holt*, Max Shinn, and Theoden I Netoff

14:10 – 14:30 O13 ***Investigating the Effect of Electrical Brain Stimulation using a Connectome-based Brain Network Model***
Tim Kunze*, Alexander Hunold, Jens Haueisen, Viktor Jirsa, and Andreas Spiegler

14:30 – 14:50 O14 ***Closing the Loop: Optimal Stimulation of C. elegans Neuronal Network via Adaptive Control to Exhibit Full Body Movements***
Julia Santos*, Eli Shlizerman

14:50 – 15:20 **Break**

Oral session VI: Information theory and correlations

15:20 – 15:40 O15 ***Collective information storage in multiple synapses enables fast learning and slow forgetting***
Michael Fauth*, Florentin Woergoetter, and Christian Tetzlaff

15:40 – 16:00 O16 ***Limited range correlations, when modulated by firing rate, can substantially improve neural population coding***
Joel Zylberberg*, Jon Cafaro, Maxwell Turner, Fred Rieke, and Eric Shea-Brown

16:00 – 19:00 **Poster session II: Posters P103 – P205**

19:00 **CNS Party**

Tuesday July 21

9:00 – 9:10 **Announcements**

9:10 – 10:10 K4 **Keynote 4:**
From single neurons to populations: Modeling neuronal dynamics across different scales
Wulfram Gerstner

10:10 – 10:40 **Break**

Oral session VII: Oscillations and rhythms

10:40 – 11:00 O17 ***Multiple mechanisms of theta rhythm generation in a model of the hippocampus***
Ali Hummos*, Satish Nair

11:00 – 11:20 O18 ***Modelling phase precession in the hippocampus***
Angus Chadwick*, Mark van Rossum, and Matthew Nolan

11:20 – 12:00 F3 **Featured oral 3:**
Control of gamma vs beta competition in olfactory bulb by the balance between sensory input and centrifugal feedback control
François David, Emmanuelle Courtiol, Nathalie Buonviso, and Nicolas Fourcaud-Trocme*

12:00 – 13:30 **Break for lunch**

13:30 – 14:30 **OCNS Member Meeting**

Oral session VIII: Network structure and dynamics

14:30 – 14:50 O19 ***Self-Organization to sub-criticality***
Viola Priesemann*

14:50 – 15:10 O20 ***Large-scale brain dynamics: effect of connectivity resolution.***
Timothée Proix*, Andreas Spiegler, and Viktor Jirsa

15:10 – 15:30 **Break**

15:30 – 18:30 **Poster session III: Posters P206 – P305**

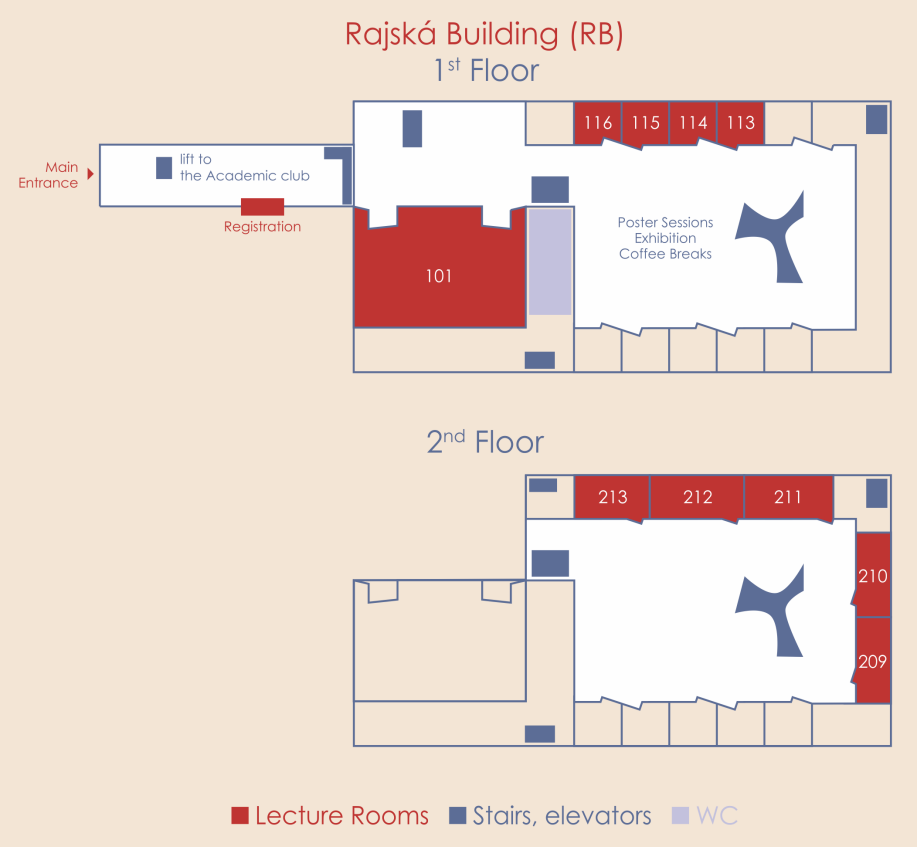
18:30 – 19:30 **Time to travel to banquet location (60 mins)**

19:30 **Banquet**

Wednesday July 22 and Thursday July 23

9:00 – 19:00 **Workshops**

Workshops



W1 **Invertebrates as Models of Cognition**

Room RB 113, Wednesday and Thursday

James Marshall, Department of Computer Science, University of Sheffield
Kevin Gurney, Department of Psychology, University of Sheffield
Eleni Vasiliki, Department of Computer Science, University of Sheffield
Thomas Nowotny, Department of Informatics, University of Sheffield

W2 **Methods of Information Theory in Computational Neuroscience**

Room NB C, Wednesday and Thursday

Alexander G Dimitrov, Washington State University
Michael C Gastpar, EPFL
Lubomir Kostal, Institute of Physiology CAS
Tatyana Sharpee, The Salk Institute
Simon R Schultz, Imperial College London

W3 **Stochastic neural dynamics**

Room NB D, Wednesday and Thursday

Peter J Thomas, Case Western Reserve University
Justus Schwabedal, Georgia State University

W4 **Methods of System Identification for Studying Information Processing in Sensory Systems**
Room RB 209, Wednesday

Aurel A Lazar, Columbia University
Mikko I Juusola, Department of Biomedical Science, University of Sheffield

W5 **Neuronal Oscillations: Computational models and dynamics mechanisms**
Room RB 101, Wednesday

Horacio G Rotstein, New Jersey Inst of Technology

W6 **Beyond the canon: temporal and spatial multiscale organization in cortex**
Room RB 210, Wednesday

Bill Lytton, SUNY Brooklyn
Wim van Drongelen, University of Chicago

W7 **Dendrite function and wiring: experiments and theory**
Room RB 211, Wednesday

Michiel Remme, Institute for Theoretical Biology, Humboldt University Berlin
Hermann Cuntz, Ernst Strüngmann Institute, Frankfurt
Benjamin Torben-Nielsen, Okinawa Institute of Science and Technology

W8 **Rate vs. temporal coding schemes: mutually exclusive or cooperatively coexisting**
Room RB 212, Wednesday

Milad Lankarany, Neuroscience and Mental Health, Hospital for Sick Children, Toronto
Steven A Prescott, Neuroscience and Mental Health, Hospital for Sick Children, Toronto

W9 **Spike initiation: models and experiments**
Room RB 213, Wednesday

Michele Giugliano, Universiteit Antwerpen
Romain Brette, Institut de la Vision, Paris

W10 **Neuromechanics and integrative motor control**
Room RB 114, Wednesday

Martin Zapotocky, Institute of Physiology of the Czech Academy of Sciences, Prague
Taishin Nomura, Dept Mechanical Science and Bioengineering, Osaka University

W11 **Computational Models of Midbrain Dopamine Neurons and Dopaminergic Signaling**
Room RB 209, Thursday

Carmen Canavier, LSU Health Sciences Center, New Orleans

W12 **Computation, Dysfunction, and the Brain**
Room RB 213, Thursday

Rowshanak Hashemiyoon, Dept Stereotactic and Functional Neurosurgery, University Hospital of Cologne
Michel Christoph, Switzerland Campus Biotech

W13 Synaptic plasticity and homeostasis

Room RB 101, Thursday

Pierre Yger, Institut de la Vision, Paris

Matthieu Gilson, Universitat Pompeu Febra, Barcelona

W14 High-performance computing in neuroscience - from physiologically realistic neurons to full-scale brain models

Room RB 210, Thursday

Wolfram Schenck, SimLab Neuroscience, Juelich Supercomputing Centre, Forschungszentrum Juelich, Germany

Alex Peyser, SimLab Neuroscience, Juelich Supercomputing Centre, Forschungszentrum Juelich, Germany

Markus Butz-Ostendorf, Juelich, Germany

W15 Metastable Dynamics of Neural Ensembles Underlying Cognition

Room RB 211, Thursday

Emili Balaguer-Ballester, Bournemouth University and Bernstein Center for Computational Neuroscience, University of Heidelberg

Maurizio Mattia, Istituto Superiore di Sanità Rome, Italy

Ruben Moreno-Bote, Fundacio Sant Joan de Deu Barcelona, Spain

W16 Open collaboration in computational neuroscience

Room RB 212, Thursday

Padraig Gleeson, University College London

W17 Postdoc and student career strategy workshop

Room RB 209, Wednesday

Jorge Meijas, Computational Lab of Cortical Dynamics, New York University, NY, USA

Abstracts

Tutorials

T1 **Neural mass and neural field models**

Room RB 212, 18-Jul-15

Axel Hutt, INRIA Nancy, France
Jeremie Lefebre, University of Lausanne, Switzerland
Alistair Steyn-Ross, University of Wakaito, New Zealand
Nicolas Rougier, INRIA Bordeaux, France

The brain exhibits dynamical processes on different spatial and temporal scales. Single neurons have a size of tens of micrometers and fire during few milliseconds, whereas macroscopic brain activity, such as encephalographic data or the BOLD response in functional Magnetic Resonance Imaging, evolve on a millimeter or centimeter scale during tens of milliseconds. To understand the relation between the two dynamical scales, the mesoscopic scale of neural populations between these scales is helpful. Moreover, it has been found experimentally that neural populations encode and decode cognitive functions. The tutorial presents a specific type of rate-coding models which is both mathematically tractable and verifiable experimentally. It starts with a physiological motivation of the model, followed by mathematical analysis techniques for neural mass models in the presence of noise, and applications to general anaesthesia and cognitive functions.

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T2 Modeling and analysis of extracellular potentials

Room RB 209, 18-Jul-15

Gaute T. Einevoll, Norwegian University of Life Sciences, Aas, Norway
Szymon Leski, Nencki Institute of Experimental Biology, Warsaw, Poland
Espen Hagen, Jülich Research Centre and JARA, Jülich, Germany

While extracellular electrical recordings have been the main workhorse 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

- The biophysics of extracellular recordings in the brain,
- 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], and
- methods for
  - estimation of current source density (CSD) from LFP data, such as the iCSD [12-14] and kCSD methods [15], and
  - 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, and (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

- LFPy (software.incf.org/software/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
- tools for iCSD analysis, in particular,
  - CSDplotter (for linear multielectrodes [8]) (software.incf.org/software/csdplotter)
  - iCSD 2D (for 2D multishank electrodes [14]) (software.incf.org/software/icsd-2d)

Further, new results from applying the biophysical forward-modelling scheme to predict LFPs from comprehensive structured network models, in particular

- the Traub-model for thalamocortical activity [21], and
- the Potjans-Diesmann model of the early sensory cortex microcircuit using hybridLFPy (github.com/espenhgn/hybridLFPy) [22,23],

will be presented.

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T3 **Modelling of spiking neural networks with the Brian simulator**

Room RB 211, 18-Jul-15

Dan Goodman, Imperical College London, UK
Pierre Yger, Institut de la Vision, Paris, France
Romain Brette, Institut de la Vision, Paris, France
Marcel Stimberg, Institut de la Vision, Paris, France

Brian [1,2] is a simulator for spiking neural networks, written in the Python programming language. It focuses on making the writing of simulation code as quick as possible and on flexibility: new and non-standard models can be readily defined using mathematical notation[3]. This tutorial will be based on Brian 2, the current Brian version under development.

We will start by giving a general introduction to Brian 2 and discussing differences between Brian 1 and Brian 2, with specific recommendations on how to convert scripts between the two Brian versions. We will then focus on the specification of neuronal and synaptic models, discussing the various ways Brian offers to implement non-standard models. We will finish by demonstrating Brian's code generation facilities, including the newly introduced "standalone" mode, giving recommendations for improving the simulation performance.

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T4 Theory of correlation transfer and correlation structure in recurrent networks

Room RB 210, 18-Jul-15

Moritz Helias, Jülich Research Centre, Jülich, Germany
Farzad Farkhoo, Freie Universität Berlin, Berlin, Germany

In the first part of this tutorial, we introduce the mathematical tools to determine firing statistics of neurons receiving fluctuating input form a network. We show how one can apply an efficient Fokker-Planck method to derive the neurons' output statistics whenever the input can be assumed to be Gaussian white (iid) noise. We further study more realistic cases, where the input fluctuations depart from the iid assumptions. Using the integrate-and-fire neuron model, we will demonstrate how to compute the firing rate, auto-correlation and cross-correlation functions of the output spike trains. The transfer function of the output correlations given the time scale of the input correlations will be discussed [Moreno-Bote and Parga, 2006, Brunel et al 2001]. In particular, we will show that the output correlations are generally weaker than the input correlations and how the working regime of the neuron shapes the cross-correlation functions [Ostojic et al., 2009; Helias et al., 2013]. We conclude the first part by investigating the relation between neurons' pairwise correlation due common fluctuations and their firing rates [de la Rocha et al., 2007].

In the second part, we will consider correlations in recurrent random networks. Using a binary neuron model [Ginzburg & Sompolinsky, 1994], we explain how mean-field theory determines the stationary state and how the network-generated noise linearizes the single neuron response. The resulting linear equation for the fluctuations in recurrent networks is then solved to obtain the correlation structure in balanced random networks. We discuss two different points of view of the recently reported active suppression of correlations in balanced networks by fast tracking [Renart et al., 2010] and by negative feedback [Tetzlaff et al., 2012]. Finally, we consider extensions of the theory of correlations of linear Poisson spiking models [Hawkes, 1971, Pernice et al. 2011] to the leaky integrate-and-fire model [Trousdale et al. 2012, Pernice et al. 2012] and present a unifying view of linear response theory of weak correlations [Grytskyy et al, 2013].

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T5 Modeling of calcium dynamics and signaling pathways in neurons

Room RB 213, 18-Jul-15

Kim Avrama Blackwell, Krasnow Institute for Advanced Study, George Mason University, Fairfax VA, USA

Modeling signaling pathways in neurons is of increasing importance for understanding brain function. Biochemical and molecular mechanisms are crucial for the synaptic and intrinsic plasticity underlying learning and information processing, neuronal development, as well as pathological degeneration. Novel biosensors, live cell imaging and other techniques are increasing the quantity of data and revealing the complexity of molecular processes generating these phenomena.

The purpose of this tutorial is to introduce techniques for modeling calcium dynamics and signaling pathways in neurons. The first part presents the biological mechanisms (channels, diffusible second messengers, enzymes, kinases) that comprise signaling pathways and control calcium dynamics. The second part presents the mathematical equations used to model the components of these pathways. The third part of the tutorial provides an overview of some of the software packages available for such modeling, and explains how to develop deterministic and stochastic models using several of these software tools, including xppaut, genesis/Moose, smoldyn, and NeuroRD.

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T6 Interfaces in Computational Neuroscience Software: Combined use of the tools NEST, CSA and MUSIC

Room RB 113, 18-Jul-15

Martin Jochen Eppler, Simulation Lab Neuroscience - Bernstein Facility for Simulation and Database Technology, Institute for Advanced Simulation, Jülich Aachen Research Alliance, Forschungszentrum Jülich, Jülich, Germany

Jan Moren, Neural Computation Unit, Okinawa Institute of Science and Technology, Okinawa, Japan

Mikael Djurfeldt, PDC center for high performance computing, KTH and INCF Stockholm, Sweden

In this workshop we demonstrate how the MUSIC and ConnectionGenerator interfaces allow the NEST simulator to work as a module in a larger simulation and use external libraries for generation of connectivity.

Current simulation environments in computational neuroscience, such as NEURON, NEST or Genesis, each provide many tools needed by the user to carry out high-quality simulation studies. However, since models are described differently in each environment, and even may depend on specific features of the environment, it is hard to move models between environments and the modeler is stuck with the tools of the environment for which the model was developed.

This also makes it difficult to build larger simulations which re-use existing models as components. As systems grow more complex and encompass more subsystems they rapidly become unwieldy to develop. Monolithic systems make it infeasible to reuse separate model implementations for parts of the system.

Furthermore, in other fields of numerical computation, the modeler often has the freedom to assemble the tools of choice out of a set of mesh generators, solvers, etc. Again the monolithic structure of software in computational neuroscience prevents this. We are not free to choose among wiring routines, solvers or neuronal spike communication frameworks.

Standard model description languages, such as PyNN, NeuroML and NineML provide a partial solution by unifying the description of models, thereby improving reproducibility and making it easier to move the model between environments. Environments structured as frameworks, such as Genesis3 or MOOSE, also address the problems described above. Our aim with this workshop is to promote the use of generic interfaces in computational neuroscience software.

Interfaces allow for the use of alternative implementations of software components. In this tutorial, we demonstrate and teach the tools NEST (a network simulator), CSA (a connectivity description language) and MUSIC (a tool for simulations across multiple environments) and show how they interact through generic interfaces.

MUSIC is an interface and library which enables connecting separate models in real-time, even when they are implemented in separate simulator systems. The connections defined in MUSIC ports effectively implement an API for other models to use. This enables division of development of complex systems across areas and team members, and interfacing the model with outside data sources and sinks. The ConnectionGenerator interface allows to use different connection generating libraries in the simulators supporting the interface. This lets you plug in the library of choice for more freedom in describing your models.

Hands-on sessions will allow participants to work on a coupling between own code and either the ConnectionGenerator interface or MUSIC. Support is provided by the authors and experienced users of the interfaces.

Invited Presentations

Adrienne Fairhall
Department of Physiology and Biophysics,
University of Washington, Seattle, USA

K1 – Learning and variability in birdsong

The birdsong system has become a paradigmatic example of biological learning. We will discuss how detailed biological responses can help this system to implement reinforcement learning. In particular, we will discuss the potential role of a newly identified excitatory neural signal in basal ganglia and how it may help to modulate basal ganglia synchrony and the variability required for learning.

Jack Cowan
Mathematics Department, Neurology Department,
and Committee on Computational Neuroscience,
University of Chicago, Chicago, USA

K2 – Modeling Cortical Dynamics with Wilson-Cowan equations

Experimental data collected over the last decade indicates that there exist at least two distinct modes of cortical response to stimuli. In mode 1 a low intensity stimulus triggers a wave that propagates at a velocity of about 0.3 m/sec, with an amplitude that decays exponentially. In mode 2 a high intensity stimulus triggers a larger response that remains local, and does not propagate to neighboring regions. Other data indicate that unstimulated or resting cortex exhibits pair correlations between neighboring cells, the amplitudes of which decay slowly with distance, whereas stimulated cortex exhibits pair correlations whose amplitude falls of rapidly with distance. Here we show how the mean-field Wilson-Cowan equations can account precisely for the two modes of cortical response, and how stochastic Wilson-Cowan equations can account for the behavior of the pair correlations. We will present these results after outlining the basic properties of both the mean-field and stochastic equations.

Gustavo Deco
Center for Brain and Cognition,
Universitat Pompeu Fabra / ICREA, Barcelona, Spain

K3 – The Dynamics of Resting Fluctuations in the Brain

The grand average functional connectivity (FC) of a resting brain captures properly the well-structured spatial correlations between different brain areas. Whole-brain-models explicitly linking spontaneous local neuronal dynamics with the tractography based anatomical structure of the brain are able to explain the emergence of those spatial resting correlations. Nevertheless, resting activity is not only spatially structured but also shows a very stereotypical temporal structure which is characterized by rapid transitions switching between a few discrete FC states across time. In this talk, we introduce a powerful theoretical framework, which allows us to demonstrate that resting functional connectivity FC dynamics (FCD) constrains more strongly the dynamical working point of whole-brain models. Furthermore, using a very general neural mass model based on the normal form of a Hopf bifurcation we are able to demonstrate that the temporal dynamics of resting state fluctuations emerges at the edge of the transition between asynchronous to oscillatory behavior. Even more importantly, at that particular working point the global metastability of the whole brain is maximized. By optimizing the spectral characteristics of each local brain node, we discover the dynamical core of the brain, i.e., the set of nodes, which drives by oscillations the rest of the whole brain.

Wulfram Gerstner
Laboratory of Computational Neuroscience,
Brain Mind Institute, Lausanne EPFL, Switzerland

K4 – From single neurons to populations: Modeling neuronal dynamics across different scales

Can we replace a standard leaky integrate-and-fire model by something that is at the same level of complexity, but well-grounded on experimental data? Can we replace standard Wilson-Cowan type rate equations (or field equations) by other equations that are at the same level of complexity, but directly derivable from models of single neurons? In this talk, I will present a processing chain from experimental somatic single-electrode recordings to generalized integrate-and-fire models and from these to population rate equations. The parameters of the neuron models are directly extracted from experimental data, using ideas from Generalized Linear Models. Groups of similar neurons are arranged in populations with random connectivity. The population equations are derived using mean-field methods and can be interpreted as a generalized renewal process with adaptation and finite-size fluctuations.

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Contributed Talks

F1 Complex synapses as efficient memory systems

Marcus K Benna*, Stefano Fusi

Center for Theoretical Neuroscience, Columbia University, College of Physicians and Surgeons, New York, NY 10032, USA

The molecular machinery underlying memory consolidation at the level of synaptic connections is believed to employ a complex network of highly diverse biochemical processes that operate on a wide range of different timescales. An appropriate theoretical framework could help us identify their computational roles and understand how these intricate networks of interactions support synaptic memory formation and maintenance.

Here we construct a broad class of synaptic models that can efficiently harness biological complexity to store and preserve a huge number of memories, vastly outperforming other synaptic models of memory. The number of storable memories grows almost linearly with the number of synapses, which constitutes a substantial improvement over the square root scaling of previous models [1,2], especially when large neural systems are considered. This improvement is obtained without significantly reducing the initial memory strength, which still scales approximately like the square root of the number of synapses.

This is achieved by combining together multiple dynamical processes that operate on different timescales, to ensure the memory strength decays as slowly as the inverse square root of the age of the corresponding synaptic modification. Memories are initially stored in fast variables and then progressively transferred to slower ones. Importantly, in our case the interactions between fast and slow variables are bidirectional, in contrast to the unidirectional cascades of previous models.

The proposed models are robust to perturbations of parameters and can capture several properties of biological memories, which include delayed expression of synaptic potentiation and depression, synaptic metaplasticity, and spacing effects. We discuss predictions for the autocorrelation function of the synaptic efficacy that can be tested in plasticity experiments involving long sequences of synaptic modifications.

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F2 Closed-loop approach to tuning deep brain stimulation parameters for Parkinson's disease

Abbey Holt[1*], Max Shinn[2], and Theoden I Netoff[1,3]

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[3]*Department of Biomedical Engineering, University of Minnesota, Minneapolis, Minnesota, 55455, USA*

Deep brain stimulation (DBS) is used to treat motor symptoms of patients with Parkinson's disease (PD). However, tuning stimulation parameters is currently done using a time intensive trial-and-error process until maximum therapy is achieved with minimal side effects [1]. There is a need for a systematic approach to tuning parameters based on patient physiology. With the development of DBS electrodes that can simultaneously stimulate and record [2], a closed-loop approach may be taken. It is hypothesized that emergent oscillations in the basal ganglia network, particularly in the beta range (12-35 Hz) lead to motor symptoms of PD [3], and that DBS works by disrupting these oscillations. Our hypothesis is that stimulating at a specific phase in the pathological oscillation will optimally disrupt the oscillatory activity, and that this phase can be predicted from the phase response curve (PRC). Here, we use a computational network model of PD with an emergent pathological 34 Hz oscillation [5] to test this closed-loop approach to DBS and confirm the results *in vitro*. By stimulating at a specific phase in the beta oscillation we are able to modulate the power of the oscillation in the model. By stimulating soon after the peak in the oscillation, we disrupt the 34 Hz oscillation, while stimulating later in the period enhances it. Hence, the timing of stimulation affects how well the population of neurons desynchronized. Next, we test this concept *in vitro* by synchronizing patch-clamped neurons in the substantia nigra pars reticulata (an output nucleus of the basal ganglia) to an oscillatory input, such as a beta oscillation. We show that stimulating at a particular phase of the oscillatory input affects how well neurons synchronize or desynchronize to that input. Finally, we show it is possible to use the PRC to predict how stimulating at a specific phase will affect the neuron's ability to synchronize or desynchronize from the oscillatory input *in vitro*.

This work shows that stimulating at specific phases in an oscillation can synchronize or desynchronize neurons in a computational model and *in vitro.* By stimulating at specific phases of an emergent pathological oscillation in a closed-loop approach to DBS, we were able to suppress a pathological oscillation in a computational model of PD. In this approach, a frequency of 34 Hz was used for DBS, which is much lower than the value used clinically (>100 Hz). Through closed-loop stimulation, precisely timed stimuli with respect to the phase of the oscillation can dramatically decrease stimulus power needed for DBS. The ability to synchronize or desynchronize a neuron to an oscillatory input by stimulating at a certain phase was also validated *in vitro.* It is possible to predict the phase of stimulation to maximally disrupt neuronal synchronization to an external oscillatory input in single neurons using a PRC. We have previously shown a novel method to estimate a PRC from population data [4] in a computational model of PD. This suggests it may be possible to predict the phase at which to stimulate in order to optimally disrupt a pathological population oscillation in PD using the PRC, and apply this in a closed-loop approach to DBS.

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F3 Control of gamma vs beta competition in olfactory bulb by the balance between sensory input and centrifugal feedback control

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Gamma (40-80Hz) and beta (15-40Hz) oscillations and their associated neuronal assemblies are key features of neuronal sensory processing. However, the mechanisms involved in either their interaction and/or the switch between these different regimes in most sensory systems remain misunderstood. The mammalian olfactory bulb (OB) expresses both gamma and beta oscillations, which appear to be mutually exclusive, and a slower one related to the respiration (2-10Hz). Gamma oscillations have been linked to odorant physical properties (quality, intensity) while beta oscillations are strongly increased by odor experience (for reviews see [1, 2]). Importantly, the occurrence pattern of these two fast alternating oscillations is intermingled with the respiratory slow rhythm which provides a window for odor discrimination. Based on *in vivo* recordings and biophysical modeling of the mammalian olfactory bulb (OB), we explored how OB internal dynamics and the balance between sensory and centrifugal inputs control the occurrence and alternation of OB gamma and beta oscillations over a respiratory cycle.

In the OB, fast oscillations originate in the dendrodendritic interaction between excitatory mitral cells (MCs) and inhibitory granule cells (GCs). Experimental evidence have shown that GC dendritic arbor can operate in two modes: a local mode which effectively allows a weak inhibition between MCs without requiring GC spikes, and a global mode which induces a strong inhibition of MCs following GC spikes. We implemented these two inhibitory mechanisms in a parsimonious and flexible OB model based on generalized integrate-and-fire models. In granule non-spiking regime, the weak inhibition can sustain OB oscillation in the gamma frequency range with characteristics of an auto-entrainment process [3]. In contrast, in the granular spiking regime, MCs sufficiently excite the GCs such that the latter discharge and induce a strong inhibitory input which silences the MC population and generate beta oscillations, similarly to the PING regime [4]. Intrinsic properties of each type of oscillation are remarkably stable regarding most of tested network parameters. However their occurrence depends strongly on OB network sensory and centrifugal inputs (onto MCs and GCs respectively). In particular, sensory activation of MCs must be strong enough for the emergence of gamma oscillations, while sufficient centrifugal activation of GCs, to allow them to spike, is necessary to generate beta. Based on novel experimental data in anesthetized rat, we show that both inputs are slowly modulated by the respiratory rhythm but phase shifted by about a quarter cycle. In our model, this phase shift can account for the gamma-beta alternation observed *in vivo*. Finally, additional tests show that the model captures accurately the competition between gamma and beta oscillations when sensory or centrifugal inputs are modulated such as in different natural conditions involving odor characteristics (odor intensity) and behavior (odor experience, active sniffing). Overall the model approaches very closely OB dynamics observed *in vivo*, and can thus be used to interpret present and future experiments.

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O1 Limits to the scalability of cortical network models

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The size of the mammalian brain is inconveniently right in the middle between a few interacting particles and a mole of matter on a logarithmic scale. In physics, we learn that often in the limit where system size goes to infinity simple mathematical expressions can be obtained uncovering the mechanisms governing the dynamics at the large but finite system size in nature. In neuroscience, however, we found that such an ansatz may fail because correlations drop so slowly that the mechanism governing the behavior in the infinite size limit [1] is not the mechanism relevant at the scale of the brain circuit in question [2]. The direct simulation of networks at their natural size has historically been difficult due to the sheer number of neurons and synapses. Therefore, neuroscientists also routinely explore the other side of the logarithmic scale and investigate downscaled circuits. In summary, it seems that brain networks are often too small for the infinity limit and too large for simulations.

In this contribution, we assess the scalability of networks in the asynchronous irregular state with a focus on downscaling. By extending the theory of correlations in such networks [2,3,4,5] and verifying analytical predictions by direct simulations using NEST [6], we formally demonstrate that generally already second-order measures cannot be preserved. The underlying mathematical reason is a one-to-one mapping between correlation structure and effective connectivity, which depends both on the physical connectivity and on the working point of the neurons [7]. Correlations are relevant because they influence synaptic plasticity [8] and large-scale measurements of neuronal activity [9], and are related to information processing and behavior [10,11].

Our results show that the reducibility of asynchronous networks is fundamentally limited, indicating the importance of considering networks with realistic numbers of neurons and synapses. Fortunately, corresponding simulation technology is becoming available to neuroscience [12]. Both the investigation of the infinity limit and the exploration of downscaled networks remain powerful methods of computational neuroscience. However, researchers should make explicit the rationale they apply in up- or downscaling.

Acknowledgements
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O2 The high-conductance state enables neural sampling in networks of LIF neurons

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The apparent stochasticity of in-vivo neural circuits has long been hypothesized to represent a signature of ongoing stochastic inference in the brain [1, 2, 3]. More recently, a theoretical framework for neural sampling has been proposed, which explains how sample-based inference can be performed by networks of spiking neurons [4, 5]. One particular requirement of this approach is that the membrane potential of these neurons satisfies the so-called neural computability condition (NCC), which in turn leads to a logistic neural response function.

Analytical approaches to calculating this function have been the subject of many theoretical studies. In order to make the problem tractable, particular assumptions regarding the neural or synaptic parameters are usually made [6,7]. However, biologically significant activity regimes exist which are not covered by these approaches: Under strong synaptic bombardment, as is often the case in cortex, the neuron is shifted into a high-conductance state (HCS), which is characterized by a small membrane time constant. In this regime, synaptic time constants and refractory periods dominate membrane dynamics.

The HCS is also particularly interesting from a functional point of view. In [5], we have shown that LIF neurons that are shifted into a HCS by background synaptic bombardment can attain the correct firing statistics to sample from well-defined probability distributions (i.e., satisfy the NCC). In order to calculate the response function of neurons in this regime, we are required to consider a new approach.

The core idea of this approach is to separately consider two different “modes” of spiking dynamics: burst spiking and transient quiescence, in which the neuron does not spike for longer periods. For the bursting mode, we explicitly take into consideration the autocorrelation of the membrane potential before and after refractoriness by propagating the PDF of the effective membrane potential from spike to spike within a burst. For the membrane potential evolution between bursts, we consider an Ornstein-Uhlenbeck approximation. We find that our theoretical prediction of the neural response function closely matches simulation data. Moreover, in the HCS scenario, we show that the neural response function becomes symmetric and can be well approximated by a logistic function, thereby providing the correct dynamics in order to perform neural sampling. Such stochastic firing units can then be used to sample from arbitrary probability distributions over binary random variables [4, 5, 8, 9]. We hereby provide not only a normative framework for Bayesian inference in cortex, but also powerful applications of low-power, accelerated neuromorphic systems to highly relevant machine learning problems.

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O3 Quantifying the distance to criticality under subsampling

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Neuronal systems have been proposed to operate close to criticality. But how far from criticality are they precisely? We developed a novel method to determine the distance to criticality from data. Importantly, our method is reliable under subsampling, i.e. the experimental constraint that in many dynamical systems only a small fraction of all agents can be sampled. Thereby, our novel approach for the first time allows to determine the distance to criticality without bias from spiking activity in vivo, which in general is strongly subsampled.

In more detail, neuronal systems have been proposed to operate close to criticality, as power-law distributions of the avalanche size have been found for local field potentials from *in vitro* preparations [1], to human cortex [2]. Criticality is an attractive candidate state for neural dynamics, because in models criticality maximizes processing capacities [3]. However, it has been widely overlooked that criticality also comes with the risk of spontaneous runaway activity (epilepsy). Recent experiments suggest that spiking activity in rats, cats, and monkeys, is in a sub-critical regime, keeping a safety-margin from criticality [4]. Quantifying the precise distance to criticality may help to shed light on how the brain maximizes its information processing capacities without risking runaway activity.

In neural systems, critical dynamics is usually studied in the context of branching processes with continuous drive [1], because they approximate well the functional propagation of spiking activity on the network [4]. The dynamics of branching processes are determined by the expected number of spikes σ in postsynaptic neurons triggered by a single spike, showing either stationary dynamics (sub-critical, $\sigma < 1$) or transient growth (super-critical, $\sigma > 1$); for $\sigma = 1$ branching processes are critical and generate the characteristic power law scaling. Methods to infer σ from *fully sampled* systems are well established, however, *subsampling* [5] resulted in strongly biased estimates (Fig., empty symbols). To overcome this bias, we derived a novel measure, based on a multistep linear regression. This measure for the first time allows to quantify the distance to criticality even under strong subsampling (Fig., full symbols). Our method generalizes to auto-regressive processes with both additive and multiplicative noise, making it widely applicable. We validated our method by applying subsampling to simulated branching processes with invasion, and to a generic integrate-and-fire model. After validation, we applied this method to highly parallel spike recordings from macaque prefrontal cortex, cat visual cortex, and rat hippocampus. These analysis indicated that spiking activity is clearly subcritical ($0.97 < \sigma < 0.99$; $N = 10$ experiments), and not critical.

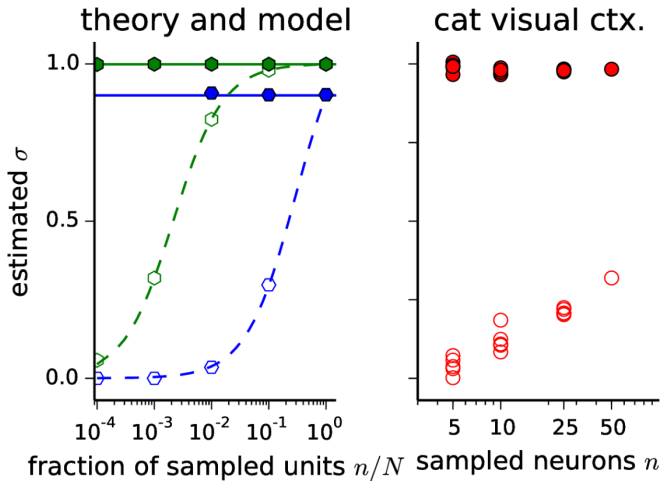

Figure 1: Estimated branching ratio σ in dependence of sampled units n of a system of size N, for conventional (empty symbols) and our novel (full) measures in theory and models and in spike recordings.

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O4 Large-scale analysis of brain-wide electrophysiological diversity reveals novel characterization of mammalian neuron types

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Brains achieve efficient function through implementing a division of labor, in which different neurons serve distinct computational roles. One striking way in which neuron types differ is in their electrophysiology properties. These properties arise through expression of combinations of ion channels that collectively define the computations that a neuron performs on its inputs and its role within its larger circuit. Though the electrophysiology of many neuron types has been previously characterized, these data exist across thousands of journal articles, making cross study neuron-to-neuron comparisons difficult.

Here, we present NeuroElectro, a public database where physiological properties for the majority of mammalian neuron types have been compiled through semi-automated literature text-mining and expert curation. The corresponding web application, at neuroelectro.org, provides a rich dynamic interface for visualizing and comparing physiological information across neuron types; conveniently linking extracted data back to its primary reference. Mining the database content after normalization for methodological differences, we show that there exist but 5-9 major neuron classes in terms of electrophysiological properties, which separate largely based on cell size and basal levels of excitability (**Figure 1**).

As an example of how this resource can help answer fundamental questions in neuroscience, we integrate NeuroElectro with neuronal gene expression from public datasets like the Allen Brain Atlas. We show that simple statistical models can accurately predict features of a neuron's electrophysiological phenotype given information of its gene expression alone. We further investigate these models to ask which genes, of the 20K in the genome, are most predictive of neuron physiology. We find that while ion channel-related genes provide significant predictive power, the most predictive gene classes surprisingly correspond to G-proteins and transcription factors, suggesting the involvement of hundreds of diverse genes in regulating a neuron's computational function.

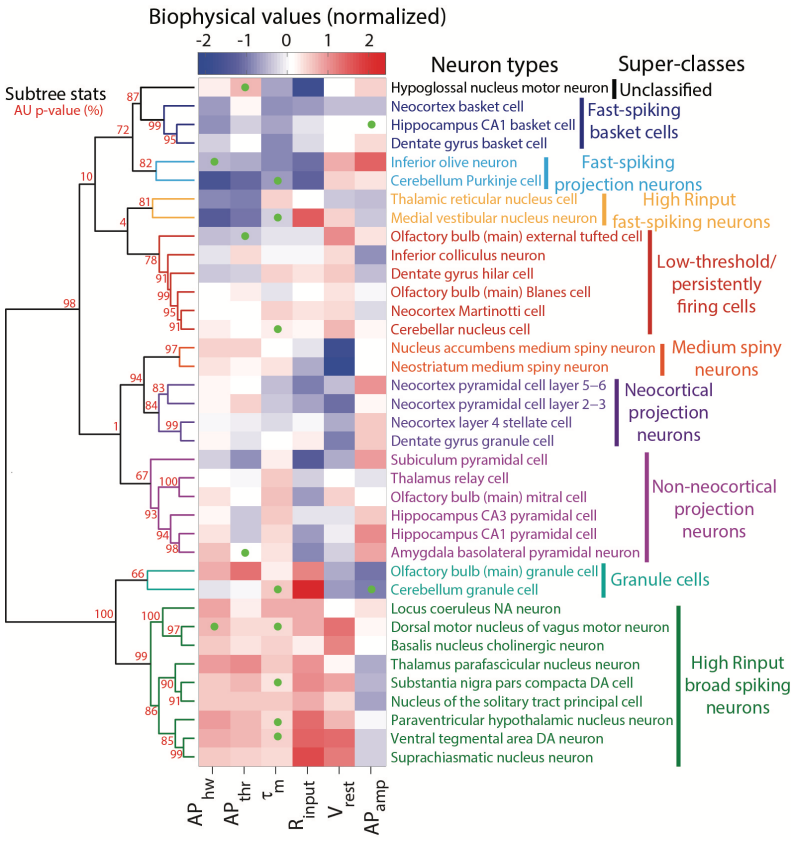

Figure 1: Hierarchical clustering of diverse neuron types on the basis of electrophysiological similarity. Electrophysiological parameters are obtained from the NeuroElectro database via literature-mining and are normalized to account for variability in experimental methodologies across studies.

O5 Self-organization of computation in neural systems by interaction between homeostatic and synaptic plasticity

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The ability to perform complex motor control tasks is essentially enabled by the nervous system via the self-organization of large groups of neurons into coherent dynamic activity patterns. During learning, this is brought about by synaptic plasticity, resulting in the formation of multiple functional networks – commonly termed as 'cell-assemblies'. A multitude of such cell assemblies provide the requisite machinery for non-linear computations needed for the mastery of a large number of motor skills. However, given the fact that there exists considerable overlap between the usage of the same neurons within such assemblies, for a wide range of motor tasks, creation and sustenance of such computationally powerful networks posses a challenging problem. How such interwoven assembly networks self-organize and how powerful assemblies can coexist therein, without catastrophically interfering with each other remains largely unknown. One the one side, it is already known that networks can be trained to perform complex nonlinear calculations [1], such that, if the network possesses a reservoir of rich, transient dynamics, desired outputs can be extracted from these reservoirs in order to enable motor control. On the other side, cell assemblies are created by Hebbian learning rules that strengthen a synapse if pre- and post-synaptic neurons are co-active within a small enough time window [2]. Therefore it appears relatively straightforward to combine these mechanisms in order to construct powerful assembly networks. However, given that the self-organization of neurons into cell assemblies by the processes of synaptic plasticity induces ordered or synchronized neuronal dynamics, which can destroy the required complexity of a reservoir network, such a combination remains a very challenging problem [3]. Furthermore, simultaneous creation of multiple cell as-

semblies can also lead to catastrophic interference if one cannot prevent them from growing into each other. In this study, we exploit for the first time the interaction between neuronal and synaptic processes acting on different time scales to enable, on a long time scale, the self-organized formation of assembly networks (Fig. 1), while on a shorter timescale, to conjointly perform several non-linear calculations needed for motor fine-control. Specifically, by the combination of synaptic plasticity and synaptic scaling [4], as a homeostatic mechanism, we demonstrate that such self-organization allows executing a difficult, six degrees of freedom, manipulation task with a robot where assemblies need to learn computing complex nonlinear transforms and - for execution - must cooperate with each other without interference. This mechanism, thus, permits for the first time, the guided self-organization of computationally powerful sub-structures in dynamic networks for behavior control. Furthermore, comparing our assembly network to networks with unchanging synapses ("static" networks) shows that it is indeed the embedding of a strongly connected assembly that creates the necessary computational power.

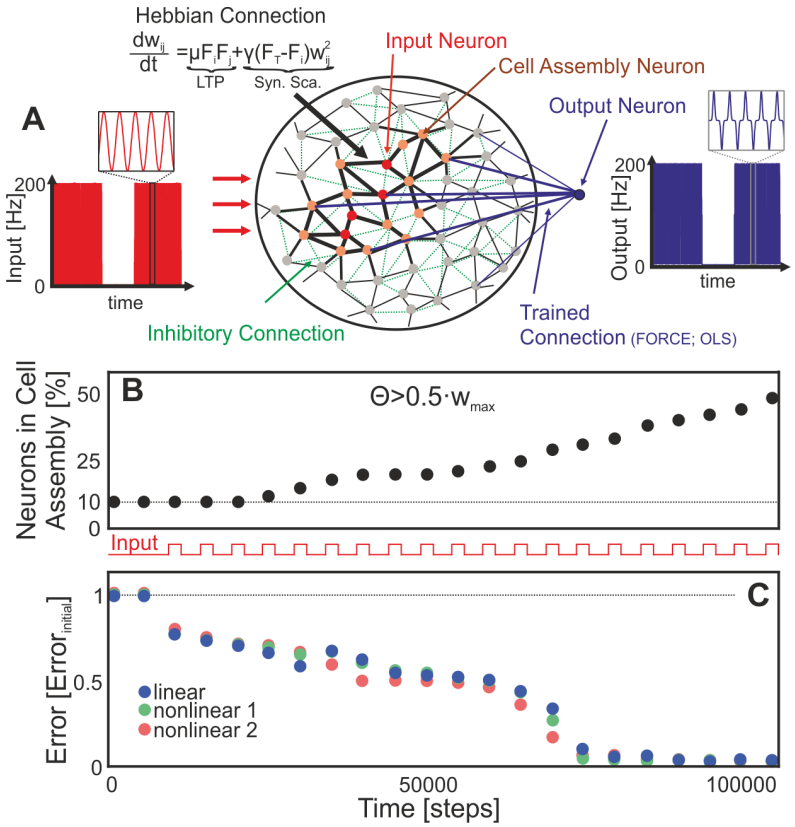

Figure 1: **Cell assembly size and computational performance are correlated.** (A) Input-driven formation of cell assemblies brought about by the interaction long-term potentiation (LTP) and synaptic scaling (Syn. Sca.). (B) With more learning trials the assembly grows and integrates more neurons. We measure this by arbitrarily defining assembly size by that set of neurons connected with efficacies larger than half the maximum weights. (C) Parallel to the outgrowth of the cell assembly the error of the system to perform several linear and non-linear calculations decreases.

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O6 A model for spatially periodic firing in the hippocampal formation based on interacting excitatory and inhibitory plasticity

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Neurons in the hippocampal formation exhibit a variety of spatially tuned firing patterns. The mechanisms by which these different patterns emerge are not fully resolved, although competing computational models exist for several of them. Here we present a new model thatcan generate all observed spatial firing patterns by a single mechanism. The model consists of a feedforward network with a single output neuron. Its essential ingredients are i) spatially tuned excitatory and inhibitory inputs [e.g., 1] and ii) interacting excitatory and inhibitory Hebbian plasticity. The inhibitory plasticity homeostatically controls the output firing rate by balancing excitation and inhibition [2]. We show in simulations and by a mathematical analysis that the output neuron develops periodic firing patterns along a stimulus dimension if inhibitory inputs are more broadly tuned than excitatory inputs along this dimension. More generally, depending on the relative spatial auto-correlation length of the excitatory and inhibitory inputs, the model exhibits firing patterns that are similar to those of place cells, grid cells (see Figure 1) or band cells (neurons that fire on spatially periodic bands [3]). For inputs with combined spatial and head direction tuning, the same mechanism leads to output firing patterns reminiscent of head direction cells and conjunctive cells (neurons that fire like grid cells in space but only at a particular head direction). A linear stability analysis of the homogeneous steady state accurately predicts the spatial periodicity obtained from simulations. The model combines the robust pattern formation of attractor models [e.g., 4], with the spatial (rather than neural) structure formation of models based on synaptic plasticity [5]. In contrast to attractor models [6], our model predicts that the grid spacing should be robust to global modifications in inhibitory synaptic strength, a distinction which could be experimentally verified.

In conclusion, we propose a feedforward network model that generates all known spatial firing patterns in the hippocampal formation through a single self-organizing mechanism.

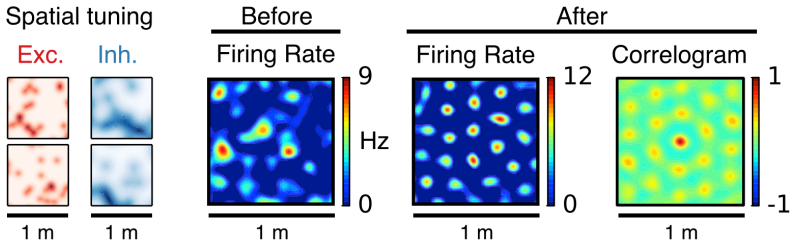

Figure 1: Example for the emergence of a grid cell. Columns from left to right: Spatial tuning of excitatory and inhibitory inputs (two examples each); spatial activity pattern of the output neuron before and after learning; auto-correlogram of activity after learning.

Acknowledgements
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O7 Whole-cell morphological properties of neurons constrain the nonrandom features of network connectivity

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We addressed the principles of micro-level organization of neuronal circuits and explored how the neuronal morphology constrains this organization. Several studies have demonstrated the non-trivial properties of the network connectivity using *in vitro* recordings from multiple neurons [1], [2], [3], yet it is unclear to what extent this structure reflects reorganization caused by synaptic plasticity, and what is imposed by the morphological constraints. Two recent articles explored this issue using the simulated neural circuits and demonstrated the specific structural properties in those circuits [4], [5].

We analyzed a model that emphasizes the role of single-cell morphology, a homogeneous population of neurons in a planar space without boundaries. Each neuron is composed of two displaced neurite fields defined on the limited support. A neurite field describes the likelihood of finding a neurite segment at a certain point in the plane. Using a proximity criterion (Peters' rule) the expected number of potential synapses is estimated between each pair of neurons. Alternatively, this number can be estimated from the realistic morphology of a simulated neuron, or from the morphologies reconstructed from *in vitro/in vivo* recordngs. The number of potential synapses depends on the axon-dendrite distance, which leads to a deffinition of the expected radius. An axon-dendrite pair that is expected to form at least one synapse must be on a distance not larger than the effective radius. All considered statistical measures of network connectivity are expressed as the functions of the effective radius normalized with the neuron size. In this study, we considered the standard graph theoretic measures of network connectivity, the motif counts, clustering coefficient, path length, and small-world coefficient. It has been demonstrated that they have a significant impact on the population activity in simulated networks [6].

Changing the normalized effective radius from small (0.3) to big (10) we vary the network properties between the two extremes. For the small values of the effective radius, the networks favor unidirectional connections and sparse local connectivity. The clustering coefficient and the path length are similar to those obtained in uniform random networks, i.e. in the networks independent of topology. For the large values of the effective radius, the local connectivity is dense with the majority of bidirectional connections. As the normalized effective radius increases, the clustering coefficient increases towards the values obtained for the networks with dominant local connectivity, while the path length remains close to the one of the uniform random networks. The normalized effective radius on the interval 1-2, provides the biggest variability of connectivity patterns and the optimized properties relevant for the information transfer.

Conclusions: We present a theoretical framework that relates neuromorphology with the connectivity in neuronal circuits, and that can be solved analytically. The normalized effective radius was found to be the key morphological property that dominantly affects considered connectivity measures. By tuning it we can obtain the networks with the biggest variability of local connectivity patterns. At the same time, those networks aquire the key characteristics of the small-world networks, known to optimize the information transfer.

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O8 Origin of the kink of somatic action potentials

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The Hodgkin and Huxley (1952) model of action potential (AP) generation accounts for many properties of APs observed experimentally and has been successfully used in modeling neurons of different types. In this model, however, the spike onset is much shallower than in experimental recordings from the soma suggesting different activation properties of sodium channels in the real tissue. To explain the origin of the observed sharpness (kink) in the spike onset three hypotheses were proposed: 1. Cooperative hypothesis: sodium channels cooperate in the axon initial segment, which makes their collective activation curve much sharper [2]. However, there is no experimental evidence for this hypothesis. 2. Active backpropagation hypothesis: spikes are initiated in the axon and backpropagate to the soma. The kink is caused by the sharpening of the axonal spike by active conductances during its backpropagation through the axon [3]. 3. Compartmentalization hypothesis: the kink comes from distal initiation and the current sink caused by the difference in the size of the soma and axon [1].

To find out what is truly happening in the cell during the action potential, we investigated the active backpropagation and compartmentalization hypotheses by means of computational modeling and theoretical analysis. In order to differentiate the hypotheses, we varied systematically the morphology of the neuron and distribution of the ionic channels along the cell, and tested how they contribute to the appearance of the kink. We show that the kink at spike onset is primarily due to compartmentalization rather than to active backpropagation.

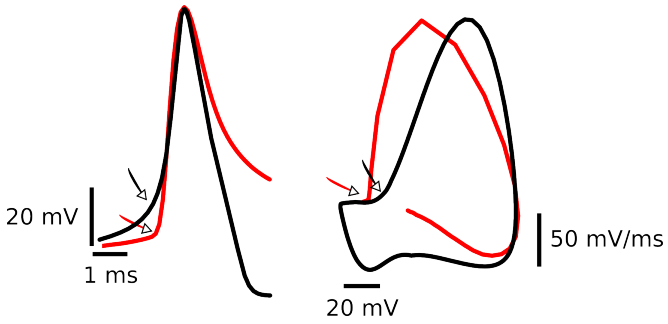

Figure 1: **Kink in the action potential**. Patch clamp recordings (red) from a cortical pyramidal cell and action potential produced by a Hodgkin Huxley type model (black). Left: Voltage-time relationship. Right: Phase plot of the same traces as in the left (dV/dt vs. V). Note, in both representations the onset of the action potential is much faster for the experimental recordings (red) than for the model (black).

Acknowledgements

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O9 An accurate circuit-based description of retinal ganglion cell computation

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Visual processing depends on computations performed by complex neural circuits. Although the circuitry in the retina has been extensively characterized, common “functional” models of how ganglion cell spike trains represent visual stimuli typically rely on linear descriptions of their receptive field [1]. Different types of nonlinear models have offered improvements in spike train prediction, but such improvements are often incremental, and in most cases not linked to known elements of the retinal circuit.

Here, we describe a new nonlinear model framework designed to represent key elements of the retinal circuit, which can predict recorded retinal ganglion cell spike trains with high temporal precision. We used recordings of both synaptic currents (via voltage-clamp recordings) and spike (via loose patch recordings) from the same ON Alpha ganglion cells in the mouse retina in order to build a two-stage nonlinear model. This model describes ganglion cell computation as sums and products of excitatory and inhibitory inputs [2]. Model parameters were estimated based on either intracellular or spike train data using a maximum-likelihood framework.

We found that excitatory synaptic currents to the ganglion cell are well described by an excitatory input combined with divisive suppression, both elements described by LN models fit to intracellular data. Using stimuli with center-surround structure, we demonstrate that this divisive suppression arises from the surround, and is the likely result of presynaptic inhibition mediated by amacrine cells [3], rather than synaptic depression [4]. We then extended this nonlinear model of synaptic currents to explain spike response of the ganglion cell by incorporating a spiking nonlinearity with spike refractoriness. All model parameters could be fit using the spike trains alone, resulting in a prediction of the excitatory currents that closely matched the models fit directly to the currents.

The resulting model had unprecedented ability to predict both synaptic current and spike trains (with 90% of the explainable variance) at one millisecond resolution on cross-validation datasets, capturing both fast transient responses in synaptic current, as well as the high precision of spike train responses. Furthermore, the model output automatically “adapted” to contrast, and could predict the responses across contrast levels with similar accuracy without any change in model parameters. Notably, the nonlinear structure of the model was particular to ON Alpha ganglion cells, and other retinal ganglion cell types had distinct computational structures, likely corresponding to different underlying connectivity within the retina governing their processing of vision.

Thus, by targeting a nonlinear model based on the specific computations performed by retinal circuit elements, we uncovered an extremely accurate description of retinal processing, and identified two-stage computational properties that can be linked to elements of the retinal circuit. In addition to providing an accurate description of ON Alpha cells, such computational framework also sets a foundation for understanding the different roles of the 20 ganglion cell types that comprise the input to the rest of the visual system.

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O10 Contrast-dependent Modulation of Gamma Rhythm in V1: a Network Model

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In our empirical data comprising of single-unit and LFP recordings in macaque area V1 and source reconstructed human MEG localized to visual cortex we have observed a robust increase in gamma oscillation frequency with increasing luminance contrast. In addition, at high grating contrasts, a robust decay in gamma power was observed in the LFP [1] but not the MEG. These phenomena are key to understanding the functional role of network frequencies and for investigating the stability of gamma oscillations at both local and macroscopic levels. However, even at the most basic level of spatially- undifferentiated neuronal models, it is not fully understood how excitatory (E) and inhibitory (I) neurons interact to generate the observed network gamma oscillations in the macaque single-unit and LFP data. For example we could obtain the frequency shift and power decay in a network where the rhythm is produced by excitatory neurons that fired more frequently than inhibitory neurons, and in another more neurophysiologically plausible network composed of excitatory neurons showing sparse firing [2, 3] and inhibitory neurons showing faster firing [4]. Moreover, it is unknown how increasing excitatory afferent drive (of which luminance contrast is a proxy) modulates the interactions between E and I populations (as well as interactions within each population) to account for changes in frequency and power. We aimed to replicate the empirical data from macaque visual cortex and to further investigate the stability of the observed gamma oscillation. Here, we present an undifferentiated V1 network PING model, with realistic neuronal features as determined and validated from the analysis of a large number of V1 neurons obtained in 3 rhesus monkeys. The model when perturbed by increasing afferent input, exhibits the core characteristics of the empirical data, that is, (1) a monotonic increase in LFP frequency, (2) a non-monotonic LFP power modulation with decay at high inputs, (3) a largely non-saturating increase in average unit firing rate. In addition, the model exhibits realistic single unit behavior across a range of inputs. In terms of the frequency shift, we have observed remarkable scaling behaviour: while the frequency of oscillations changes dramatically with input, the absolute average phase at which inhibitory and excitatory neurons fire in each oscillation cycle and the average relative phase to each other remain constant. This scaling may on one hand underlie the stability of the gamma oscillation locally and on other hand facilitate communication through coherence in the gamma range [5] across varying stimulus conditions, by preserving the timing and relative ordering of population firing irrespective of the oscillation frequency [6]. Our results suggest that the observed power decline results from a primary (functional) decoupling among inhibitory neurons. Further analysis highlighted that the functional decoupling is related to the balance of inhibition/excitation. In further steps, we intend to test these predictions in the empirical data, and then proceed to a differentiated V1 columnar model to investigate the divergences between human MEG and macaque LFP/spiking responses.

Acknowledgements
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O11 Downstream changes in firing regularity following damage to the early auditory system

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We demonstrate how an abstract mathematical model that approximates a wide range of more detailed models can be used to make predictions about hearing loss-related changes in neural behaviour.

One consequence of neurosensory hearing loss (noise-induced and aging-related) is a reduced ability to understand speech, particularly in noisy environments, and sometimes beyond what would be predicted from reduced audibility. Indeed, this type of speech deficit can occur in listeners with near-normal hearing thresholds [1]. A promising avenue of investigation to explain this comes from experimental results in mice showing that there can be a permanent loss of auditory nerve fibres (ANFs) following “temporary” noise-induced hearing loss (i.e. when thresholds return to normal after a few weeks) [2]. The downstream consequences of this loss of fibres has not yet been systematically investigated (although see [3]). We predict, using a theoretical analysis that applies to a wide range of neural models, that the regularity of the spike trains of many neurons in the cochlear nucleus (the next structure after the auditory nerve) will decrease following a reduction in the number of input cells.

We present a mathematical analysis of the stationary behaviour of “chopper” cells in the ventral cochlear nucleus, approximating them by a stochastic process that is entirely characterised by its mean, standard deviation and time constants. Furthermore, these constants can be straightforwardly related to physiologically significant parameters including the number of inputs and their average firing rates. From this approximation, we can compute the regularity of the chopper cell spike trains measured as the coefficient of variation of their interspike intervals (CV).

One simple prediction of this model is that when the intensity of a stimulus changes, leading to a change in the average firing rate of the ANF inputs, there will be a corresponding change in the regularity of the chopper cell spike train. This prediction poses problems for the widely used scheme for classifying chopper cells as sustained or transient based on their ongoing CVs as it implies that the classification could be level-dependent. We present a re-analysis of an existing experimental data set that demonstrates that ongoing CV is indeed level-dependent in the majority of chopper cells, and that in some cells this leads to a level-dependence in their classification.

Assuming a homeostatic regulation of long term firing rates, a loss of ANFs will lead to an increase in the standard deviation of the stochastic process and a consequent increase in the CV of the chopper cell. Some choppers that were previously classified as sustained will become transient, a substantial change in their behaviour that is highly likely to disrupt auditory processing. While the function of chopper cells is still debated, one suggested role is in the coding of temporal envelope [4], which is widely agreed to be essential for understanding speech. Loss of ANFs could therefore lead to a disruption of the processing of temporal envelope, and consequently degrade speech intelligibility. We briefly conclude by discussing the challenges of testing this hypothesis experimentally.

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O12 Towards a computational model of Dyslexia

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Dyslexics are diagnosed for their poor reading skills. Yet, they characteristically also suffer from poor verbal memory, and often from poor auditory skills. We now hypothesize that Dyslexia can be understood computationally as a deficit in integrating prior information with noisy observations. To test this hypothesis we analyzed performance in two tones pitch discrimination task using a two-parameter computational model. One parameter captures the internal noise in representing the current event and the other captures the impact of recently acquired prior information [1]. We found that Dyslexics' perceptual deficit can be accounted for by inadequate adjustment of these components: low weighting of their implicit memory in relation to their internal noise (Figure 1). Using ERP measurements we found evidence for Dyslexics' deficient automatic integration of experiment's statistics (Figure 2). Taken together, these results suggest that Dyslexia can be understood as a well-defined computational deficit.

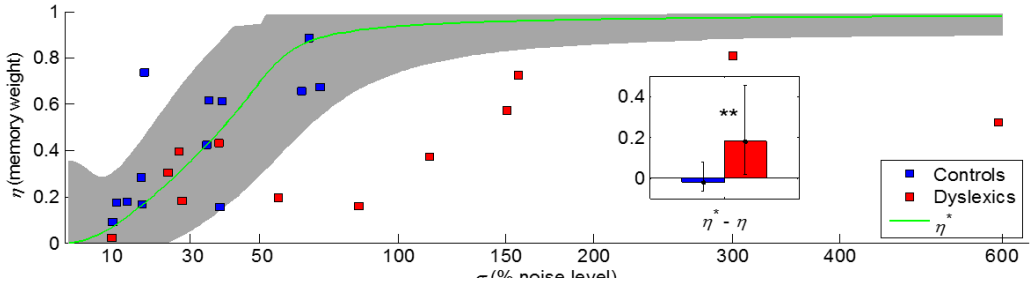

Figure 1: Estimated parameters of the Implicit Memory Model. Estimated values of η (weighting of implicit memory) as a function of estimated values of σ (percentage of internal noise) of Controls (blue) and Dyslexics (red). The optimal weighting η^* is plotted in green. Gray area depicts the confidence interval of 2.5% below the best performance. **Inset.** Median deviation from optimal weighting of previous trials. Dyslexics' deviation is larger than Controls' (Mann-Whitney test, Z = 2.5, P < 0.01). Error bars denote inter-quartile range.

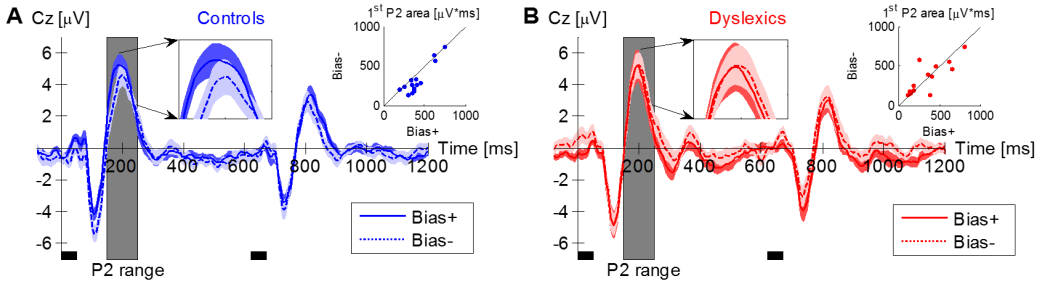

Figure 2: Grand Average ERPs to the two-tone stimulation. **A**. Controls **B**. Dyslexics. Trials are sorted according to the trial type, *Bias+* (where the impact of previous trials improves performance) and *Bias-* (where the impact of previous trials impairs performance). Controls' P2 after the first tone differs between the two trial types. Dyslexics' evoked responses did not differ between the two trial types. Filled areas denote cross-subject SEM. Small black rectangles under the plots denote the temporal location of the two tones in the trial. **Middle insets**. P2 region enlarged; **Top right insets**. Single subject P2 area in *Bias-* versus *Bias+* trials. The difference between the trial types is significantly larger among Controls than among Dyslexics (Condition X Group interaction: Mann-Whitney test, z = 2.5, P 0.05).

Acknowledgements
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O13 Investigating the Effect of Electrical Brain Stimulation using a Connectome-based Brain Network Model

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Transcranial direct current stimulation (tDCS) leads to positive effects in neurological and psychiatric diseases, such as depression, pain, or stroke, which outlast the treatment itself. Although numerous influencing stimulation parameters and factors are known, the mechanisms behind tDCS remain unclear. To reveal the mechanisms tDCS started to be considered to affect networks while (de)polarizing parts of the brain. We study here the ability of tDCS as a tool to bias functional networks by affecting the connections given the brain structure.

We used structural data, that is, a human connectome to construct a large-scale brain network model of 74 cerebral areas, each described by a Jansen and Rit model. The model was designed on the basis of the neuroinformatics platform *The Virtual Brain* to account for reproducibility of the simulations. The tDCS-induced currents on the cerebral areas were calculated using a finite element method model. Based on the dynamical repertoire of an isolated area [1], we analyzed the brain activity, that is, the spatiotemporal dynamics in terms of rhythms and baseline potentials during rest, during tDCS, and the change between both.

We identified the network states during rest and catalogued all states for further modeling studies. During tDCS, increased functional connectivity was found among a set of scalp EEG sensors, as reported in measurements [2], as well as among cerebral cortical areas (see Figure 1). Furthermore, tDCS led to sharpened frequency spectra and increased (anode) or decreased (cathode) power in the respective areas.

This study supports the notion that noninvasive brain stimulation is able to bias brain dynamics by affecting the competitive interplay of functional subnetworks. Our work constitutes a basis for further modeling studies to test target-oriented manipulation of functional networks (e.g. through adapted electrode montages) to improve pertinent treatment conditions. Furthermore, our approach emphasizes the role of structural data such as the network topology in emerging dynamics. Dynamics cannot necessarily be predicted from the structure but we found the structure especially important at transitions of network states.

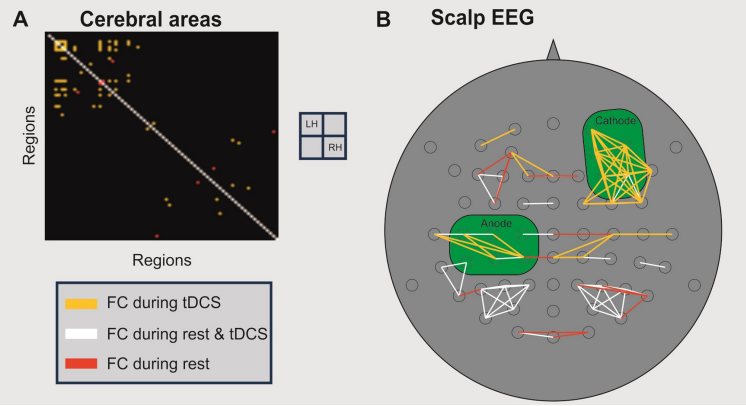

Figure 1: New functional connections are established during tDCS: among cortical areas, Panel **A**; and among scalp EEG electrodes, Panel **B**.

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O14 Closing the Loop: Optimal Stimulation of C. elegans Neuronal Network via Adaptive Control to Exhibit Full Body Movements

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The *Caenorhabditis elegans (C. elegans)* worm is a well-studied biological organism model. The nervous system of *C. elegans* is particularly appealing to study, since it is a tractable fully functional neuronal network for which electro-physical connectivity map (connectome) is fully resolved [1,2]. In a recent work, we succeeded in establishing a computational dynamical model for the *C. elegans* nervous system and showed that robust oscillatory movements in motor neurons along the body can be invoked by constant current excitation of command sensory neurons (e.g. PLM neurons associated with forward crawling) and that their activation corresponds to low-dimensional Hopf bifurcation [3]. While these first results validated the model, it is exciting to learn how the nervous system transforms its oscillatory dynamics to the muscles to support robust full body movements (e.g. forward crawling) [5]. Moreover, using methods generically applicable to other neuronal circuits, it is intriguing to understand the optimal sensory stimulations that cause these movements to persist.

We explore these questions by modeling the *C. elegans* musculature as a viscoelastic rod with discrete rigid segments [5], and map the neuronal dynamics such that they activate the muscles and deform the rod (Fig. 1A). When motor neuron activity stimulates muscles [2], this activation is translated into force applied to the rod, which moves in accordance with the physical properties of *C. elegans*. By stimulating the command PLM neurons, we establish for the first time that motor neuron dynamics are indeed producing coherent oscillatory full body movements that resemble forward crawling (Fig. 1B, videos available here).

We utilize our computational full body model to determine the appropriate sensory input for behavior, such as crawling, to persist after explicit external stimulation (touch) has ceased, as observed in experiments [5]. Since such persistence could be explained by a feedback loop between the environment and sensory neurons (Fig. 1C), we propose an adaptive control algorithm that extends existing recursive least squares-based algorithms (e.g. FORCE [6]). Our algorithm finds weights for synaptic input using a low-dimensional projection of motor neuron dynamics, and is capable of finding sensory input patterns that will lead to the desired movement.

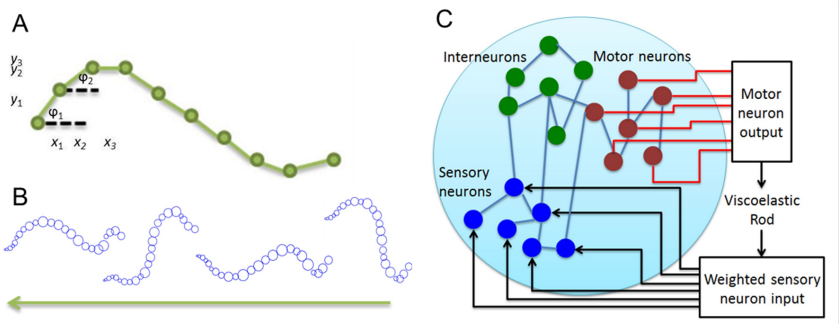

Figure 1: **A:** Structure of viscoelastic rod **B:** Viscoelastic rod-based simulation of *C. elegans* crawling during PLM excitation, videos available here **C:** Loop feeding transformed motor activity into sensory neurons

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O15 Collective information storage in multiple synapses enables fast learning and slow forgetting

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Most of the excitatory cortical synapses reside on dendritic spines. Although these spines undergo a remarkably high turnover [1, 2], they have been shown to be involved in learning and long-term memory. Along this line, it is unclear how information is preserved while its substrate (synapses or spines) is permanently changing.

Here, we use a simple stochastic model of structural plasticity to investigate this phenomenon : We assume a certain number of potential synaptic locations from one neuron to another.

At those locations, synapses (spines) are created with a constant probability and removed with a probability depending on the number of existing synapses and the stimulation of the neurons. From these two probabilities, the stationary distribution of the number of synapses between two neurons can be calculated.

Experimental measurements of these stationary probability distributions in the cortex show that the majority of connections has either zero or multiple synapses while one or two contacts are very improbable [e.g., 3-5]. Using information theoretic measures we show that, in our model, such bimodal distributions enable information storage over time scales many orders of magnitudes higher than the involved probabilities. Thus, in this system the conflict of rapid spine turnover (probabilities) and long-term memory is resolved by storing the information collaboratively in multiple synapses.

In the following, we will consider the bimodal stationary distributions as the working point of the system. Then, we can model external signals, as, e.g., increased or decreased activities during learning, as changes of the removal probabilities and stationary distributions (e.g., mediated by synaptic plasticity [6]).

For instance, for learning signals resulting to unimodal stationary distributions (only connected or only unconnected), we find that learning is orders of magnitude faster than forgetting. Along this line, we observe that retraining a task does not induce an increased overturn rate as during initial training, which has been similarly observed for dendritic spines in vivo [1, 2]. Our results clearly relate the difference in time scales to the shape of the stationary distribution and therefore reveal the functional advantage of the bimodal distribution found in experiment.

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O16 Limited range correlations, when modulated by firing rate, can substantially improve neural population coding

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Neural activities are unreliable indicators of features of the external world [1], and an open question is how our nervous systems function robustly in the presence of this noise. One possibility arises from the observation that variability is often correlated between neurons [2], leading to the important theoretical question of when and how noise correlations affect neural population codes. Much work has investigated this issue, leading to impressive insights about how the relationship between the statistical structures of signals vs noise affects neural population coding. Despite this progress, an important issue has been largely overlooked by the field: that of firing-rate-dependent correlations. Notably, the same pair of neurons can display different noise correlations in response to different stimuli; those correlations coefficients typically increase with increases in the neurons' firing rates [2,3].

In this paper, we investigate the role of so-called "limited range" correlations on population codes, either in the presence, or the absence of rate-modulation of the correlation coefficients. Limited-range correlations are frequently-observed population-wide correlation structures in which cells with similar tuning curves have positive noise correlations, and the correlations decrease with decreasing tuning curve similarity [2,4]. These patterns of noise correlation are typically harmful to population coding [5] (yielding worse population coding performance than would be obtained with the same tuning curves and noise variances for all cells, but no noise correlations); these effects are somewhat dependent on the degree of heterogeneity in the population's tuning curves [6,7].

Experimentally reported noise correlations are usually averaged over stimuli, thereby masking any stimulus dependence. Herein, we will demonstrate that, when correlation coefficients increase with the product of mean neural firing rates (as in [2,3]), the stimulus-averaged correlation coefficients will display limited-range structure. When the rate dependence of these correlations is ignored, those correlations appear to be quite harmful to the population code, in accordance with previous theoretical work [5]. Surprisingly, when the rate dependence is taken into account, the correlations can yield much better population coding than would be obtained in the presence of uncorrelated noise. These effects persist for either homogeneous or heterogeneous sets of neural tuning curves. One prior study [8] also found that rate-dependent correlations can have very different impacts on population codes than can rate-independent ones, but did not make the connection between firing-rate-dependent correlations and the frequently observed limited-range correlation structure. Overall, our results emphasize that, for understanding the impact of limited-range correlations on neural population coding, the firing-rate dependence of those correlations is a potentially important consideration. Thus, it is important to report not only stimulus-averaged correlation coefficients, but also the relationship between those correlation coefficients and the neural firing rates.

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O17 Multiple mechanisms of theta rhythm generation in a model of the hippocampus

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Hippocampal theta oscillations (4-12 Hz) are consistently recorded during memory tasks and spatial navigation. While computational models suggested specific mechanisms for theta generation, experimental inactivation of these mechanisms did not disrupt theta, precluding definitive conclusions about their roles. We investigated this discrepancy using a biophysical model of the hippocampus that included several of the components implicated in rhythm generation, all constrained by prior experimental results. The CA3 network model included recurrently connected pyramidal cells, and inhibitory basket cells (BC) and oriens-lacunosum moleculare (OLM) cells. The model was developed by matching experimental results characterizing neuronal firing patterns, synaptic dynamics, short-term synaptic plasticity and the three-dimensional organization of the hippocampus. The model revealed four mechanisms that generated theta oscillations: intrinsic theta resonance of pyramidal cells, recurrent connections between them, coupling between OLM and pyramidal cells, and, as a novel finding, the correlated input from entorhinal cortex. Consistent with experimental results, inactivation of any single mechanism did not disrupt the rhythm. Another novel finding was that the low and high cholinergic states differentially recruited theta generating mechanisms. Atropine -sensitive and -resistant forms of theta, however, corresponded to theta generated during low and high levels of network excitation, respectively. These findings provided an alternative interpretation of the atropine-based classification of theta oscillations, and suggested that the theta rhythm is an intrinsic property of the network. Any experimental manipulation or brain state that enhances or suppresses excitation might also, therefore, non-specifically enhance or suppress theta oscillations.

O18 Modelling Phase Precession in the Hippocampus

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The activity of cells in the rodent hippocampus is strongly modulated by both the location of the animal and the ongoing theta oscillation. Place cells, but not interneurons, show a strong spatial modulation of their firing rates, while both place cells and interneurons exhibit phase precession, a phenomenon whereby they spike at a faster frequency than the LFP theta oscillation, causing their spikes to shift to an earlier phase of this rhythm on each successive cycle [1, 2, 3]. Despite extensive research into this phenomenon, the mechanisms underlying phase precession remain unclear.

Place cells and interneurons are reciprocally connected in the CA1 region of the hippocampus. The interneurons receive pacemaker input from the medial septum, which entrains theta oscillations in the circuit. We tested whether a minimal model based on this architecture could produce phase precession in place cells and interneurons. Specifically, we simulated a single place cell and interneuron, which interact synaptically. The interneuron was driven with a constant depolarising input which generates tonic spiking, as well as a weak theta oscillation which entrains this spiking activity. The place cell received a depolarising input which is only active at a certain location in the environment, representing the place field.

We found that phase precession in both the place cell and interneuron emerges naturally in this model. When the animal is outside of the place field, the interneuron is fully entrained to the pacemaker theta oscillation, and the place cell is rhythmically inhibited, resulting in subthreshold theta oscillations. When the animal enters the place field, the place cell begins to spike, which perturbs the interneuron and causes it to transiently fire at a frequency higher than the pacemaker input. In turn, the spiking of the interneuron entrains the place cell, generating phase precession in the coupled pair.

Generalisation of this model to the network level reveals important constraints. In particular, as there are far fewer interneurons than place cells in CA1, it is necessary that the same interneuron is coupled to multiple place cells. In our model, a single interneuron can successfully couple to multiple place cells to generate phase precession, provided that their place fields do not overlap. This poses constraints on the possible place field mappings in such a network, and places limits on the fraction of place cells which can be active in a single environment. When working within these limits, the network can flexibly generate phase precession, both in linear and open environments, across a vast number of distinct place field mappings.

Our model has several advantages over existing models. First, our model generates phase precession through the intrinsic dynamics of the circuit, without the need for velocity controlled oscillators upstream. Second, our model can generate omnidirectional phase precession in open environments, without additional inputs from head direction cells. Finally, our model generates phase precession independently in each cell, and therefore allows spatial representations to be flexibly remapped without detriment to the temporal coding of spatial trajectories in the population [4].

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O19 Self-organization to sub-criticality

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Human brains possess sophisticated information processing capabilities, which rely on the interactions of billions of neurons. However, it is unclear how these capabilities arise from the collective spiking dynamics. A popular hypothesis is that neural networks assume a critical state [1,2], because in models criticality maximizes information processing capabilities [3,4]. However, it has been largely overlooked that criticality in neural networks also comes with the risk of spontaneous runaway activity [5], which has been linked to epilepsy. Does the brain indeed assume a critical state, despite the risk of instability? To revisit this question, we analyzed spiking activity from awake animals, instead of more coarse measures of neural activity (population spikes, LPF, EEG, BOLD) as in most previous studies. In all recordings (rats hippocampus, cats visual cortex, and monkey prefrontal cortex), spiking activity resembled a sub-critical state, not criticality proper [6]. We confirmed these results using a novel mathematical approach that is robust to subsampling effects [7] [see Wilting & Priesemann, conference proceedings CNS 2015]. While 'self-organization' to criticality has been widely studied (e.g.[5,8]), it is unclear what mechanism allows self-organize to sub-criticality instead. Here, we demonstrate that homeostatic plasticity [9] assures that networks assume a slightly sub-critical state, independently of the initial configuration. Surprisingly, increasing the external input (stimuli) altered the set-point of the network to a more sub-critical state. Our results suggest that homeostasis allows the brain to maintain a safety margin to criticality. Thereby the brain may lose processing capability, but avoids instability.

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O20 Large-scale brain dynamics: effect of connectivity resolution.

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Large-scale brain dynamics recently started to be modeled numerically based on both heterogeneous large-scale networks build from diffusion MRI, that is, a connectome, and local homogeneous connectivity kernel representing intracortical synaptic connections. However, topological properties of a connectome can significantly change with resolution and parcellation [1]. Furthermore, the sampling of the cerebral surfaces, resulting in a geometric model, and, in this way, the local connectivity kernel play crucial roles in the formation of spatial patterns on the cerebral surfaces as well as on the sensor level (e.g., EEG electrodes) by a forward calculation [2]. However, the effect of sampling and parcellation on modeling brain dynamics has not been studied so far. Here, we investigate qualitatively and quantitatively: (i) how different parcellation resolutions affect the dynamics of the network; and (ii) how the local connectivity affects the network dynamics. To do so, we used the neuroinformatics platform for large-scale brain simulations, called *The Virtual Brain* (TVB) [3] and developed a preprocessing pipeline to incorporate experimental data (e.g., structural MRI, diffusion-weighted MRI) in TVB [4].

We prepared ten individual models based on ten randomly selected subjects from the Human Connectome Project dataset [5]. For each individual model we performed simulations under two conditions during rest: (i) noise driven, using a bistable neural mass model, and (ii) after stimulation, using a excitable neural mass model. We investigated the effect of heterogeneous and homogeneous connectivity on large-scale brain dynamics by different numbers of regions in the parcellation (70 to 2240) and by varying the local connectivity coupling strength. To introduce experimental data (i.e., structural and diffusion MRI) into TVB we tackled issues such as surface downsampling (for achieving moderate simulation times) and mapping between surface and parcellation (to consistently use heterogeneous and homogeneous connectivity) by developing the Surface and Connectivity Reconstruction with an Imaging Pipeline for TVB Simulations, short SCRIPTS [4].

When considering slow dynamics, the major fiber bundles best reflected in the coarsest parcellation appeared to be mainly responsible for the emergence of the network attractors with limited changes over different parcellations and different local coupling strengths. For fast dynamics, new qualitative solutions appeared, but only in the presence of delays.

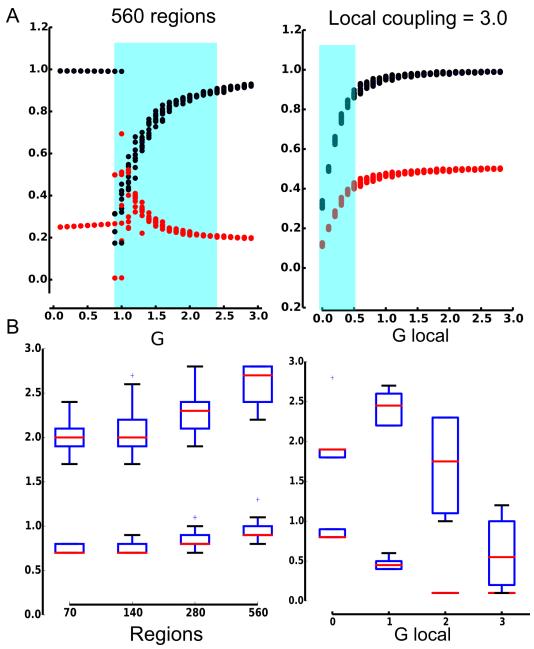

Figure 1: **A.** Spatial attractors. For each value of the global (G) or local (G_local) coupling parameter, correlation with in-strength (blue points) and with s-core (red points) for ten different initial conditions. The blue square indicates the critical interval. **B.** Values for the beginning and end of the critical range as a function of G or G_local.

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Workshops

W1 **Invertebrates as Models of Cognition**

Room RB 113, Wednesday and Thursday

James Marshall, Department of Computer Science, University of Sheffield
Kevin Gurney, Department of Psychology, University of Sheffield
Eleni Vasiliki, Department of Computer Science, University of Sheffield
Thomas Nowotny, Department of Informatics, University of Sheffield

This workshop will discuss how invertebrate brains may provide useful models of the bases of cognition, in spite of their limited size and apparently limited cognitive abilities. Important aspects of the discussion will be how minimal cognitive substrates (micro-brains) can, in conjunction with embodiment, lead to surprisingly sophisticated behaviours, and what lessons we can draw from these examples for the general understanding of cognition. The workshop should be exciting to the computational neuroscience community, because it brings together ideas of full-brain models, embodied cognition, and promising new technologies in the form of GPU super-computing and autonomous robotics.

The invited speakers cover a variety of interesting topics ranging from the visual navigation of ants to odour processing in bees. Furthermore, the mixture of speakers from both the computational and experimental sides provide an important ingredient for future progress in the field.

Speakers:

- Jeremy Niven (University of Sussex): TBA
- Esin Yavuz (University of Sussex): TBA
- Alex Cope (University of Sheffield): Modelling Honeybee Vision at Multiple Levels of Detail
- Chelsea Sabo (University of Sheffield): Embodiment of Honeybee Cognition and Behaviour
- Jean-Marc Devaud (Universite Paul Sabatier): The Crucial Role of Inhibitory Transmission for the Resolution of Ambiguities during Olfactory Reversal Learning
- Martin Nawrot (Free University of Berlin): TBA
- Aurore Averguès-Weber (Université Paul Sabatier): TBA
- Lianne Meah (University of Sheffield): An Approach to Modelling Decision-Making using the Honeybee
- James Turner (University of Sussex): TBA
- Lars Chittka (Queen Mary, University of London): TBA
- Andy Philippides (University of Sussex): TBA
- Natalie Hempel de Ibarra (University of Exeter): TBA
- Andrew Straw (Research Institute of Molecular Pathology): A Dynamical Systems Approach to Cognition in Drosophila: from Known Visual Circuits to Models of Behavioral Switching
- Barbara Webb (University of Edinburgh): TBA
- Giovanni Galizia (University of Konstanz): System Identification in the Insect Olfactory System: Getting the Input Right using Computational Tools
- Andrew Barron (Macquarie University): TBA
- Jeri Wright (University of Newcastle): TBA

W2 Methods of Information Theory in Computational Neuroscience

Room NB C, Wednesday and Thursday

Alexander G Dimitrov, Washington State University
Michael C Gastpar, EPFL
Lubomir Kostal, Institute of Physiology CAS
Tatyana Sharpee, The Salk Institute
Simon R Schultz, Imperial College London

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:

- Dan Butts (University of Maryland)
- Justin Dauwels (Nanyang Technological University): Variational Inference for Graphical Models of Multivariate Piecewise-Stationary Time Series
- Alexander Dimitrov (Washington State University, Vancouver)
- Michael Gastpar (Ecole Polytechnique Federale de Lausanne)
- Cornelius Glackin (University of Hertfordshire): Information and decision-making
- Jaroslav Hlinka (Institute of Computer Science CAS, Prague)
- Lubomir Kostal (Institute of Physiology CAS, Prague): Efficient information transmission and stimulus coding in neuronal models
- Robert Legenstein (Graz University of Technology): Bayesian learning through stochastic synaptic plasticity
- Ilya Nemenman (Emory University)
- Barani Raman (Washington University, St. Louis): Dissecting behavioral relevant features of population neural activity in a simple olfactory system
- Tatyana Sharpee (The Salk Institute)
- Simon Schultz (Imperial College London)
- Adria Tauste (Universitat Pompeu Fabra): Measuring neuronal information transfer during task performance via the directed information
- Gasper Tkacik (Institute of Science and Technology, Klosterneuburg): Beyond sensory bottleneck: Efficient coding of elements of visual form
- Joel Zylberberg (University of Washington, Seattle): Correlations and the propagation of information through neural circuits

W3 **Stochastic neural dynamics**

Room NB D, Wednesday and Thursday

Peter J Thomas, Case Western Reserve University
Justus Schwabedal, Georgia State University

Naturally occurring neural activity shows broad-band fluctuations and unpredictable transitions in its dynamics. Such randomness can be an integral aspect of neuronal function; examples range from discrete fluctuations of ion channels to sudden sleep stage transitions involving the entire brain.

To understand brain function as well as dysfunction, it is therefore necessary to develop analysis and modeling techniques of neuronal dynamics that explicitly incorporate such random components. Recent examples include application of techniques derived from the theory of stochastic processes and statistical physics to the analysis and modeling of stochastic neuronal oscillations; and progress in the analysis of stochastic network models going beyond mean-field descriptions.

This workshop will bring together leading theorists and applied researchers in the field to inspire and communicate such novel approaches in the study of neural activity.

Speakers:

1. Gerrit Ansmann (University of Bonn (DE)): Randomness and pattern switchings on complex networks of excitable units.
2. Andrea Barreiro (Southern Methodist University (US)): How single-neuron dynamics modulate correlated activity in neural circuits.
3. Wilhelm Braun (School of Mathematical Sciences and Centre for Mathematical Medicine and Biology, University of Nottingham (UK)): Integrate-and-fire neurons with stochastic thresholds.
4. Heather Brooks (University of Utah (US)): Quasicycles in the stochastic hybrid Morris-Lecar neural model.
5. Nicolas Brunel (University of Chicago (US)): Effects of neuronal morphology on firing rate dynamics.
6. Jack Cowan (University of Chicago (US)): Stochastic Wilson-Cowan equations for networks of excitatory and inhibitory neurons.
7. Felix Droste (Bernstein Center for Computational Neuroscience, Berlin (DE)): An analytical approach to information transmission in the face of up and down states.
8. Farzad Farkhooi (Bernstein Center for Computational Neuroscience, Berlin (DE)): Renewal approach to the stability analysis of noisy spiking recurrent networks.
9. David Angulo Garcia (Consiglio Nazionale delle Ricerche - Istituto dei Sistemi Complessi (IT)): Stochastic mean-field formulation of the dynamics of diluted neural networks
10. Kreso Josic (University of Houston (US)): Evidence Accumulation in a Changing Environment.
11. Ryota Kobayashi (National Institute of Informatics (JP)): Estimation of excitatory and inhibitory input rate from a single voltage trace.
12. Guillaume Lajoie (Max-Planck-Institute of Dynamics and Self-Organization (DE) and University of Washington (US)): Chaos-induced noise in recurrent networks: Structured deterministic randomness and its impact on information processing.
13. Enrica Pirozzi (University of Naples Federico II (IT)): Stochastic modeling of neuronal firing activity by generalized Ornstein-Uhlenbeck processes.
14. Stefan Rotter (Albert Ludwigs University of Freiburg and Bernstein Center Freiburg): Inhibition-dominated random networks for stimulus processing in rodent visual cortex.
15. Laura Sacerdote (University of Torino (IT)): A Leaky Integrate-and-Fire neuronal model with Gamma distributed interspike intervals.
16. Justus Schwabedal (Max-Planck-Institute for the Physics of Complex Systems (DE)): Inference of Phase Diffusion.
17. Peter Thomas (Case Western Reserve University (US) and Bernstein Center for Computational Neuroscience, Berlin (DE)): Asymptotic Phase for Stochastic Oscillators.
18. Jonathan Toubol (Collège de France (FR)): On the dynamics of large spiking neuronal networks.
19. John White (University of Utah (US)): Biophysical mechanisms of noise resistance in neurons.

W4 Methods of System Identification for Studying Information Processing in Sensory Systems

Room RB 209, Wednesday

Aurel A Lazar, Columbia University

Mikko I Juusola, Department of Biomedical Science, University of Sheffield

A functional characterization of an unknown system typically begins by making observations about the response of that system to input signals. The knowledge obtained from such observations can then be used to derive a quantitative model of the system in a process called system identification. The goal of system identification is to use a given input/output data set to derive a function that maps an arbitrary system input into an appropriate output.

In neurobiology, system identification has been applied to a variety of sensory systems, ranging from insects to vertebrates. Depending on the level of abstraction, the identified neural models vary from detailed mechanistic models to purely phenomenological models. Also known as reverse engineering, system identification is at the core of the BRAIN Initiative.

The workshop will provide a state of the art forum for discussing methods of system identification applied to the visual, auditory, olfactory and somatosensory systems in insects and vertebrates.

The lack of a deeper understanding of how sensory systems encode stimulus information has hindered the progress in understanding sensory signal processing in higher brain centers. Evaluations of various systems identification methods and a comparative analysis across insects and vertebrates may reveal common neural encoding principles and future research directions.

The workshop is targeted towards systems, computational and theoretical neuroscientists with interest in the representation and processing of stimuli in sensory systems in insects and vertebrates.

Speakers:

- Eugenia Chiappe (Champalimaud Neuroscience Programme, Champalimaud, Lisbon): Integration of Walking Direction and Speed Sensitivity in Cell-Specific Motion-Sensitive Visual Neurons
- Thomas R. Clandinin (Stanford University, Palo Alto): TBA
- Martin Egelhaaf (University of Bielefeld, Bielefeld): Motion as a Source of Environmental Information: A Fresh View on Biological Motion Computation and its Role for Solving Spatial Vision Tasks
- C. Giovanni Galizia (University of Konstanz, Konstanz): Honeybee Odor Processing: Neural Networks for Odor Identity and Evaluation in a World with many Odors and Fast Timescales
- Mikko I. Juusola (University of Sheffield, Sheffield): Saccadic Bursts Drive Maximal Visual Encoding, Improving Vision
- Aurel A. Lazar (Columbia University, New York): Projection Neurons in Drosophila Antennal Lobes Signal the Acceleration of Odor Concentrations
- Chung-Chuan Lo (National Tsing Hua University, Hsinchu): High-Level Information Processing of Sensory Signals in Nervous Systems
- Thomas Nowotny (University of Sussex, Brighton): Closed-Loop Computational Electrophysiology
- Dong Song (University of Southern California, Los Angeles): Understand Brain Functions from Spikes: A Nonlinear Dynamical System Identification Approach
- Andrew D. Straw (Research Institute of Molecular Pathology, Vienna): System Identification of Drosophila Optomotor Behavior Using Free Flight Virtual Reality

W5 **Neuronal Oscillations: Computational models and dynamics mechanisms**

Room RB 101, Wednesday

Horacio G Rotstein, New Jersey Inst of Technology

Oscillatory activity at various frequency ranges have been observed in various areas of the brain (hippocampus, entorhinal cortex, olfactory bulb among others), and are believed to be important for cognitive functions such as learning, memory, navigation and attention. These rhythms have bee studied at the single cell level, as the result of the interaction of a neuron's intrinsic properties, at the network, as the result of the interaction between the participating neurons and neuronal populations in a given brain region, and at higher levels of organization involving several of these regions. The advances in this field have benefited from the interaction between experimental and theoretical approaches.

The purpose of this workshop is to bring together both experimentalists and theorists with the goal of discussing their results and ideas on the underlying mechanisms that govern the generation of these rhythms at various levels of organization, and their functional implications for cognition.

Speakers:

- Nicolas Brunel (University of Chicago, USA): TBA
- Francesco Battaglia (Radboud Universiteit, Nijmegen, The Netherlands): TBA
- Carmen Canavier (Louisiana State University, USA): Resonant interneurons can increase robustness of gamma oscillations.
- Laura Colgin (The University of Texas at Austin, USA, tentative): TBA
- Mark Cunningham (Newcastle University, UK): TBA
- Vassilis Cutsuridis (Foundation for Science and Technology Greece, Greece): TBA
- Alain Destexhe (CNRS, France, tentative): TBA
- David Hansel (CNRS, tentative): TBA
- Stephanie Jones (Brown University, USA): TBA
- Stephen Keeley (NYU, USA): TBA
- Paola Malerba (UC Riverside, USA, tentative): TBA
- Ole Paulsen (Cambridge University, UK): optogenetic induction of hippocampal gamma oscillations.
- Horacio G. Rotstein (New Jersey Institute of Technology, USA): Inhibition-based theta resonance in a hippocampal network: a modeling study.
- Susanne Schreiber (Humboldt University Berlin, Germany): TBA
- Roger Traub (IBM, USA): TBA

W6 **Beyond the canon: temporal and spatial multiscale organization in cortex**

Room RB 210, Wednesday

Bill Lytton, SUNY Brooklyn

Wim van Drongelen, University of Chicago

In addition to new knowledge, recent years have brought a growing appreciation of the complexity of cortical organization across temporal and spatial scales, requiring a reconceptualization of prior models. We can no longer think of cells simply as independent distinct processing entities that can be slotted into circuitry and governed by a clock, in the way that transistors are placed into and driven by a circuit board.

Indeed, there is an intriguing overlapping of scales in cortex. Spatially, cell and network scales are dramatically intermixed in e.g. layer 5 pyramidal cells: these apical dendrites reach upward across layers of circuit wiring, receiving dynamically distinct inputs and making dynamically distinct subcellular responses at different circuit layers. Temporally, feedforward and feedback circuits are not distinct but interact with and through different cortical layers (and cells and dendrites), with responses at different temporal scales governed by imposed and intrinsic oscillations ranging from milliseconds (fast gamma) to seconds (sub-delta). Here again the activity of individual cells or subcircuits cannot be abstracted from this complex of multiscale activity. Rather than thinking hierarchically up the great chain of embeddings (molecule to spine to dendrite to cell to circuit to area ...), we may need to transform to a different representational frame. Perhaps one can begin to identify distinct spatiotemporal functional/dynamical modes that manifest across scales.

Speakers:

- Giorgio Ascoli (George Mason U) “Much ADO about BIG memory: A neural mechanism for Background Information-Gated learning based on Axonal-Dendritic Overlaps”
- Maxim Bazhenov (U California, Riverside) “Sleep Slow Oscillation And Memory Consolidation”
- Jack Cowan (U Chicago) cowang1@gmail.com “Modeling the functional architecture of the visual cortex, and beyond”
- Gaute Einevoll (NMBU) “Bridging scales with local field potentials (LFPs)”
- Bill Lytton (SUNY Brooklyn) “Multiscale or beyond?”
- Henry Markram (or an understudy, Human Brain Project) TBA
- Jorge Mejias (NYU) “Large-scale cortical network models with laminar structure: frequency-specific feedforward and feedback interactions”
- Stefan Mihalas (Allen Institute) “Multi-scale approaches to elucidating the computations of a cortical column”
- Cathy Schevon (Columbia U) “Spatial properties of evolving seizures : what we can learn from human microelectrode recordings”
- Wim van Drongelen (U Chicago) “The effect of local spike trains on macroscopic epileptiform activity in time and frequency domains”

W7 **Dendrite function and wiring: experiments and theory**

Room RB 211, Wednesday

Michiel Remme, Institute for Theoretical Biology, Humboldt University Berlin
Hermann Cuntz, Ernst Strüngmann Institute, Frankfurt
Benjamin Torben-Nielsen, Okinawa Institute of Science and Technology

Neuronal dendritic trees are complex structures that endow the cell with powerful computing capabilities and allow for high neural interconnectivity. Studying the function of dendritic structures has a long tradition in theoretical neuroscience, starting with the pioneering work by Wilfrid Rall in the 1950s. Recent advances in experimental techniques allow us to study dendrites with a new perspective and in greater detail. For example, dendritic function can now be studied in awake, behaving animals. Also, owing to the precise characterization of neural circuits, the role of the single dendrite can be studied in the context of its connectivity. The goal of the workshop is to provide a resume of the state-of-the-art in experimental, computational and mathematical investigations into the functions of dendrites in a variety of neural systems.

Speakers:

- Michael Häusser (University College London): tba
- Giorgio Ascoli (George Mason University): Reconstructing dendrites: from development to computation
- Claudia Clopath (Imperial College London): Synaptic plasticity across dendritic location
- Peter Jedlicka (Goethe University, Frankfurt): Biologically realistic models of dendritic and synaptic plasticity in the hippocampus
- Greg Jefferis (University of Cambridge): NBLAST: Rapid, sensitive comparison of neuronal structure and construction of neuron family databases
- Daniel Justus (German Center for Neurodegenerative Diseases DZNE, Bonn): Locomotion-speed dependent disinhibition of inputs to CA1 pyramidal neurons is mediated by a medial septal glutamatergic circuit
- Athanasia Papoutsi (IMBB-FORTH, Heraklion-Crete): Modeling the interplay of dendritic spikes and network connectivity in persistent activity
- Arnd Roth (University College London): Untangling cerebellar circuits with scanning electron microscopy and focused ion beam milling
- Balázs Ujfalussy (Institute of Experimental Medicine HAS, Budapest): Discovery of presynaptic ensembles by structural and intrinsic plasticity in dendritic branches
- Katharina Wilmes (Humboldt University Berlin): Local dendritic inhibition as a simple pathway-specific switch for Hebbian synaptic plasticity

W8 Rate vs. temporal coding schemes: mutually exclusive or cooperatively coexisting

Room RB 212, Wednesday

Milad Lankarany, Neuroscience and Mental Health, Hospital for Sick Children, Toronto
Steven A Prescott, Neuroscience and Mental Health, Hospital for Sick Children, Toronto

Deciphering how the brain processes information requires that we understand the diverse neural coding strategies used by different brain areas. Those strategies are often divided into rate and temporal codes (Shadlen and Newsome, J Neurosci 1998; Softky and Koch, J Neurosci 1993). This division has been the source of much debate and has often involved championing one strategy by rejecting the other. Although distinct, the two strategies are not necessarily mutually exclusive. Mounting evidence calls for a concerted effort to reconcile the data favoring each side.

The traditional view is that information is transmitted by the firing rates of individual neurons, and that this is best achieved by neurons operating independently of each other. However, it has long been observed that neighboring neurons exhibit correlated spiking; on the surface, these correlations ought to reduce information capacity, but more detailed consideration reveals that this is not always the case. Moreover, correlations can exist across many different timescales: Correlations that involve synchronization of spikes are liable to have very different effects than less precise correlations spanning 100s of milliseconds. Moreover, whether neurons should ideally operate independently depends on how one frames the problem: Rate coding may benefit from uncorrelated spiking whereas temporal coding relies on it – precisely timed spikes may be resilient to disruption by noise only when they occur synchronously across a set of neurons.

This workshop will explore the possibility that different coding strategies co-exist, invigorating an old debate with a new, more conciliatory approach. It is intended for a broad audience and will ideally attract audience members from diverse backgrounds. There is deliberately no focus on any one brain area (e.g. hippocampus or visual cortex) so that insights from different fields can be brought together and the assumptions implicit in any one field will be challenged. Invited speakers will cover a broad range of topics, addressing how information is encoded by large neural networks, but also how that encoding is impacted by the biophysical properties of neurons and synapses. Speakers will address both experimental and theoretical issues. The workshop will finish with an open forum aimed at discussing issues spurred by the preceding talks.

Speakers:

- Thomas Akam: Oscillatory Multiplexing of Population Codes for Selective Communication in Neural Circuits
- Sonja Gruen: etection of sequences of synchronized spiking activities
- Sliman Bensmaia: The Importance of Spike Timing in Tactile Coding
- Milad Lankarany: Multiplex Coding using Asynchronous and Synchronous Spikes
- Daniel Butts: Temporal precision and information in the awake cortex
- Alain Destexhe: Unexpected Roles of Inhibition in the Awake Brain
- Sungho Hong: Multiplexed coding by cerebellar Purkinje neurons
- Julijana Gjorgjieva: Two Time-scales of Information Transmission in Developing Cortical Neurons
- Jorge Mejias: Neural heterogeneity on rate and temporal coding
- Mario Mulansky: Time-resolved and parameter-free measures of spike train synchrony
- Robert Rosenbaum: Rates, correlations and high-dimensional dynamics in spatially extended balanced networks
- Constantinos Melachrinos: Deciphering the role of dendritic morphology on temporal coding in the Pre-Frontal Cortex

W9 **Spike initiation: models and experiments**

Room RB 213, Wednesday

Michele Giugliano, Universiteit Antwerpen
Romain Brette, Institut de la Vision, Paris

This workshop will gather leading experimentalists and theoreticians to discuss a pivotal element for our quantitative understanding of neuronal excitability: the initiation of action potentials. Phenomenological, electrophysiological, and computational aspects of spike initiation will be discussed by addressing the following questions:

- What models can quantitatively account for experimental observations?
- How is spike initiation regulated at different time scales?
- What is the role of the various ionic channels expressed in the axonal initial segment?
- What is the significance of neuronal morphology and spatial distribution of channels?
- What are the functional consequences of spike initiation properties?

Speakers:

- Romain Brette (Institut de la Vision, Paris): The compartmentalization of spike initiation
- Michele Giugliano (Universiteit Antwerpen, Antwerp): TBA
- Andreas Neef (Max Planck Inst. for Dyn. & Self-Org., Göttingen): The biophysical basis of the high-bandwidth information encoding in cortical neurons
- Christian A. Pozzorini (École Polytech. fédérale de Lausanne, Lausanne): Enhanced sensitivity to rapid input fluctuations by nonlinear threshold dynamics
- William J. Spain (University of Washington, Seattle): Functional consequences of adaptation of spike-threshold-accommodation
- Maarten Kole (Netherlands Inst. for neuroscience, Amsterdam): Role of branch geometry in action potential initiation
- Martina Michalikova (Humboldt-Universitiät, Berlin): Spikelets in pyramidal neurons: axonal (output) spikes that do not activate the soma
- Florence Cotel (Queensland Brain Institute, St Lucia): Serotonin induces central fatigue by inhibiting action potential initiation in motoneurons
- Farzan Nadim (New Jersey Institute of Technology, Newark): The synaptic effects of ectopic spike initiation and the history dependence of axonal conduction

W10 Neuromechanics and integrative motor control

Room RB 114, Wednesday

Martin Zapotocky, Institute of Physiology of the Czech Academy of Sciences, Prague

Taishin Nomura, Dept Mechanical Science and Bioengineering, Osaka University

Coordinated movement arises from the interaction of the nervous system, the body, and the environment. The mutual coupling between biomechanics and neural activity has received growing attention in recent years. This has lead to computational models that successfully capture coordinated movement on the level of a whole organism - e.g., crawling, swimming, or postural control. Simultaneously, very significant advances in robotics were inspired by known neural control strategies (e.g., coupled central pattern generators). In addition, recent computational studies have demonstrated how coordinated motor behavior may self-organize from neuromechanical interactions during development. The general principles of how the organization and activity of the nervous system adapts to the mechanics of the body and environment, however, are not yet agreed on.

The workshop brings together researchers with primary background in neuroscience and bio-robotics/neuroengineering, and aims to facilitate a fruitful interaction of approaches from these two fields. The speakers study a wide range of organisms/systems, spanning insects, higher organisms, and humanoid robots. The theoretical concepts and computational methods will provide a unifying framework.

Speakers:

- Shinya Aoi (Kyoto University): Exploring adaptive motor control in locomotion using neuromusculoskeletal models and legged robots
- Jan Bartussek (University of Rostock): Catch me if you can - embodiment of flight control in flies
- Gennady Cymbalyuk (Georgia State University, Atlanta): Cellular mechanisms governing dynamics of Central Pattern Generators
- Auke Ijspeert (EPFL, Lausanne): to be confirmed
- Shuhei Ikemoto (Osaka University): Mutual inhibition and co-contraction of a musculoskeletal robot arm using artificial muscle spindles
- Hugo Gravato Marques (Champalimaud Neuroscience Center, Lisbon): From muscle twitches to coordinated behaviour: a developmental approach
- Taishin Nomura (Osaka University): Stability vs variability of human bipedal standing and walking
- Segiy Yakovenko (West Virginia University): Hierarchical synergies for the control of locomotion
- Martin Zapotocky (Czech Academy of Sciences, Prague): Synchronization and frequency tuning in neuromechanical systems

W11 Computational Models of Midbrain Dopamine Neurons and Dopaminergic Signaling

Room RB 209, Thursday

Carmen Canavier, LSU Health Sciences Center, New Orleans

This workshop will pull together diverse approaches to modeling midbrain dopamine neurons, including simple one-compartment models that can be easily analyzed using phase-plane techniques as well multi-compartmental models that include the spatial extent of the dendritic tree or axonal arbor. Some models link models of electrical activity to cell metabolism, which is clearly important in Parkinson's disease, for example. We will emphasize the diversity of dopaminergic subpopulations and the need to tailor models to subpopulations. Different subpopulations have different inputs and distinct targets. There is also evidence for different dynamic range and bursting mechanisms in different subpopulations, which likely differentially encode motivational value, salience and novelty. We will also have speakers discuss higher level models of dopaminergic signaling, and how single neuron models might inform the next level of modeling.

Speakers:

- Carmen Canavier (LSU Health Sciences Center, New Orleans): Dynamic Diversity of Dopamine Neurons
- Guillaume Drion (Brandeis University, Waltham): Potential functional implications of a hidden variability in SNc DA neuron excitability
- Rebekah Evans (NIH-NINDS, Bethesda): T type calcium channels trigger a hyperpolarization induced after depolarization (HI-ADP) in SNc dopamine neurons
- Jean-Marc Fellous (University of Arizona, Tuscon): Experience-dependent reactivation of VTA neurons during sleep
- Boris Gutkin (Ecole Normale Superieure, Paris): VTA circuit models and nicotine/alcohol effects on DA dynamics
- Jinyoung Jang (Sunkyunkwan University, Seoul): Balance between the proximal dendritic compartment and the soma determines the spontaneous firing rate in midbrain dopamine neurons
- Alexey Kuznetsov (IUPUI, Indianapolis): A model for VTA circuitry: toolbox for the study of addictions
- Andy Oster (Eastern Washington University, Cheney): VTA Dopamine Neuron Dynamics: response to ramping applied currents
- Eleftheria Pissadaki (University of Oxford, Oxford): From the axons of the SNc dopamine neurons to their dendritic processes: further clues to susceptibility in Parkinson's disease

W12 Computation, Dysfunction, and the Brain

Room RB 213, Thursday

Rowshanak Hashemiyoon, Dept Stereotactic and Functional Neurosurgery, University Hospital of Cologne

Michel Christoph, Switzerland Campus Biotech

More and more, studies are emphasizing circuitry and network function in the brain. Investigations are focused on the changes of the functional and anatomical features in a healthy brain as compared to dysfunctional brain states; thus, studies of the healthy brain fuel insights into brain dysfunction, whilst observations of dysfunctional brain states give clues to normal brain function.

Knowledge garnered from both domains has given insight into the possible processes or mechanisms underlying a range of neurological disorders, including Parkinson's disease, Tourette syndrome, schizophrenia, and epilepsy. Theories of changes in neuronal coordination dynamics are adapted to improve therapeutic strategies. Observations of the outcomes from these applications in turn provide data about the neurophysiological and computational strategies employed by the brain. One new and exciting arena is the field of brain stimulation. Results from various investigations determine targets and parameters, while analyses of their outcomes elucidate circuitry, causality and network interactions.

For example, direct electrophysiological recordings from deep brain stimulation therapy in the human brain provide information of thalamic dynamics during severe pathology, and also how they change when responding to therapy. Meanwhile, modeling studies are used to define critical windows for therapeutic intervention in psychiatry to ensure optimal – and sometimes life-saving – results.

This workshop explores computation in both the healthy and dysfunctional brain to uncover what each state might reveal about the other. Findings from theoretical, experimental, and clinical studies will be interwoven to give a more complete understanding of the function and dysfunction of brain circuitry.

The main topics that we will discuss are:

- What are the changes in normal information processing that lead to the aberrations which define disease states such as observed in various neurological disorders?
- How can we apply those theories to improve therapeutics?
- What strategies can we use to optimize the yield from the neurobiological data from empirical and clinical studies to elucidate our understanding of normal v. abnormal function?

Speakers:

- Christoph Michel, University of Geneva, Geneva Temporal dynamics of neuronal networks and aberrations in neurological and psychiatric disorders
- Viktor Jirsa, Aix-Marseille University, Marseille Functional connectivity dynamics in large-scale brain networks
- Marc Goodfellow, University of Exeter, Exeter The importance of network structure in seizure generation
- Anthony Grace, University of Pittsburgh, Pittsburgh The circuit dynamics of dopamine system regulation and its disruption in psychiatric disorders
- Giacomo Koch, Foundation Santa Lucia, Rome Neuromodulation of cortico-cortical circuits with TMS: From basic neurophysiology to clinical application
- Rowshanak Hashemiyoon, University Hospital Cologne, Cologne How can DBS help us understand the circuit dynamics of normal v. abnormal function?
- Günter Schiepek, Paracelsus Medical University, Salzburg Discontinuous patterns of brain activation during psychothera

W13 Synaptic plasticity and homeostasis

Room RB 101, Thursday

Pierre Yger, Institut de la Vision, Paris

Matthieu Gilson, Universitat Pompeu Febra, Barcelona

This workshop will gather leading experimentalists and theoreticians to discuss latest results and models on synaptic plasticity and homeostasis in sensory cortices. Whether synaptic changes strongly depend on the exact timing of spikes, or on average

ring rates is still a matter of debate and may vary from area to area. However, it is robustly observed, in vitro and in vivo, that homeostatic mechanisms are important to regulate the global activity. The workshop will therefore be a unique opportunity to address various questions:

- How is homeostatic regulation expressed both in vitro and in vivo?
- How can it be combined with Hebbian forms of learning? Are those two mechanisms competing at a single synapse?
- What are the functional implications of those plasticity mechanisms?
- From a modeler's point of view, what are the di erent possible implementations of homeostasis, and what are the links with meta-plasticity?

Speakers:

- Pierre Yger (Institut de la Vision, France): Synaptic plasticity and Homeostasis: a review of concepts
- Christian Tetzla (BCCN Göttingen, Germany): The interaction of synaptic plasticity and scaling and their role in memory formation
- Alanna Watt (Mc Gill University, Canada): Adaptive regulation of Purkinje cell spiking in spinocerebellar ataxia type 6
- Taro Toyoizumi (RIKEN BSI, Japan): Modeling the dynamic interaction of Hebbian and homeostatic plasticity
- Sami El Boustani (MIT, USA): Investigation of single-cell plasticity in mouse V1
- Claudia Clopath (Imperial College, UK): Emergence of functional connections in neural networks with synaptic plasticity
- Per Jesper Sjöstrom (Mc Gill University, Canada): Neocortical optogenetic kindling: Emergent seizures after repeated hyperactivity
- Friedemann Zenke (EPFL, Switzerland): Hebbian and non-Hebbian plasticity orchestrated to form and retrieve memories in spiking neural networks
- Carlos Stein (EPFL, Switzerland): Theory of cortical plasticity as stable higher-order feature learning

W14 High-performance computing in neuroscience - from physiologically realistic neurons to full-scale brain models

Room RB 210, Thursday

Wolfram Schenck, SimLab Neuroscience, Juelich Supercomputing Centre, Forschungszentrum Juelich, Germany

Alex Peyser, SimLab Neuroscience, Juelich Supercomputing Centre, Forschungszentrum Juelich, Germany

Markus Butz-Ostendorf, Juelich, Germany

Supercomputing is increasingly available in neuroscience and boosts the ability to create models with a degree of detail and biological realism never seen before. By the recently available computational power, single cell models can now represent a highly detailed neuronal morphology, compartmentalized functional interactions between synapses on a single dendritic branch and even molecular processes on a sub-synaptic scale.

Biological neuronal network models, too, become more realistic as representing a large amount of different cell types in a realistic layered cortical organization predicting dynamics of spike trains in cortical networks. Different forms of synaptic and structural plasticity can be combined in one model allowing us to study interfering activity and connectivity dynamics on different spatio-temporal scales. The ultimate goal is to generate full-scale brain models on the world's high-end supercomputers. The hope is that physiologically realistic brain models will provide us deeper understanding of the healthy and diseased brain and offer novel tools to design new treatment strategies after brain lesions and for neurodegeneration.

The aim of this workshop is therefore to bring together the leading developers of high performance simulation and hardware tools in neuroscience with users from experimental fields and to demonstrate potential applications of the new techniques. Workshop speakers from the HPC domain are encouraged to present how their tools contribute to the scientific progress in neuroscience while experimentalists should point out the need for HPC resources in their workflow.

This workshop will be complemented by a special issue in Frontiers in Neuroanatomy entitled "Anatomy and plasticity in large-scale brain models" (URL: http://journal.frontiersin.org/ResearchTopic/3644).

Speakers:

- Steve B. Furber (Advanced Processors Technology Group, School of Computer Science, University of Manchester, UK): The SpiNNaker Brain Simulation Machine
- Elisabetta Chicca (Faculty of Technology, Cognitive Interaction Technology — Center of Excellence, Bielefeld University, Germany): Simulating plasticity with neuromorphic hardware
- Wolfram Schenck (Jülich Supercomputing Centre, Forschungszentrum Jülich, Germany): The Simulation Lab Neuroscience — A novel institution to support Neuroscientists in using HPC infrastructure
- Ben Torben-Nielsen (Computational Neuroscience Unit, Okinawa Institute of Science and Technology Graduate University, Onna son, Japan): NeuroMac — Simulating virtual dendritic morphologies
- Bill Lytton (Department of Physiology and Pharmacology, SUNY Downstate, Brooklyn, NY, USA) / Michael Hines (Department of Neurobiology, Yale University, New Haven, CT, USA): Modeling detailed neuronal morphologies and plasticity with NEURON
- Sacha van Albada (Institute of Neuroscience and Medicine (INM-6), Forschungszentrum Jülich, Germany): Large-scale model of the visual cortex in NEST
- Sebastian Rinke (German Research School for Simulation Sciences, Aachen, Germany) / Markus Butz (Jülich, Germany): RELeARN — Rewiring of full-scale cortical networks
- Karl Zilles (Institute of Neuroscience and Medicine (INM-1), Forschungszentrum Jülich, Germany): Polarized light imaging (PLI) — A new dimension in imaging full brain connectivity
- Markus Axer (Institute of Neuroscience and Medicine (INM-1), Forschungszentrum Jülich, Germany): Big brain — Big data. How to store and process PLI data.
- Petra Ritter (BrainModes Group, Charite, Berlin, Germany): The Virtual Brain

W15 Metastable Dynamics of Neural Ensembles Underlying Cognition

Room RB 211, Thursday

Emili Balaguer-Ballester, Bournemouth University and Bernstein Center for Computational Neuroscience, University of Heidelberg

Maurizio Mattia, Istituto Superiore di Sanità Rome, Italy

Ruben Moreno-Bote, Fundacio Sant Joan de Deu Barcelona, Spain

Is the traditional view on cortical activity dynamics, in which the cognitive flow of information wanders through multiple attractor states driven by task-dependent inputs, still a valid model? This picture has been recently challenged both empirically and from the modelling perspective. For instance, in several contemporary models, intrinsic activity fluctuations drive transitions between metastable states even in the absence of external stimuli. Thus, in these views, noise enriches the dynamical repertoire of available states and temporal scales which permit the flexible processing of task-related cognitive entities. In contrast, another proposed metaphor of transient brain dynamics consists of a sequence of metastable states composing arbitrary trajectories in the phase space which are reliably followed by the neural activity, even without the crucial intervention of noise. The interpretation of the collective dynamics of neuronal assemblies underlying perception and cognitive processing is a very active debate, touching the essence of our understanding of neural computation, and hence one of the most exciting topics in neuroscience. In this workshop we will address a range of modelling and data analysis approaches which focus on metastable nonlinear dynamics underlying perception and cognitive processing. The workshop will include a short symposium.

Speakers: (tentative titles)

- Sue Denham (Faculty of Health and Human Sciences, Plymouth University, UK): Perceptual multistability in audition: individual differences, models and unanswered questions.
- James Rankin (Center for Neural Science, New York University, USA): Differential effects of attention and stimulus manipulations in auditory bistability.
- Gemma Huget (Department of Applied Mathematics I, Universitat PolitÃ¨cnica de Catalunya, Spain): Noise and adaptation in multistable perception: a case study with tristable visual plaids.
- Daniel Durstewitz (Bernstein Center for Computational Neuroscience, Central Institute of Mental Health, Medical Faculty Mannheim/ Heidelberg University, Germany. School for Computing and Mathematics, Faculty of Science and Environment, Plymouth University, UK): Assembly dynamics in prefrontal-hippocampal networks.
- Thomas Nowotny(Centre for Computational Neuroscience and Robotics, School of Engineering and Informatics, University of Sussex, UK): Comparing winnerless competition and a barely stable fixed point as models of neural dynamics in insect olfaction.
- Pablo Varona (Grupo de Neurocomputación Biológica, Dpto. de Ingeniería Informática, Escuela Politécnica Superior, Universidad Autónoma de Madrid, Spain): Heteroclinic dynamics and cognitive functions.
- Christopher Buckley (School of Engineering and Informatics, University of Sussex, UK): The influence of closed-loop sensorimotor feedback on brain dynamics.
- Alberto Bernacchia (School of Engineering and Science, Jacobs University Bremen, Germany): Cortical dynamics explained by models of synaptic matrix ensembles.
- Maurzio Mattia (Istituto Superiore di Sanità, Rome, Italy): Heterogeneity in bistable cortical modules: evidence and advantages.
- Emili Balaguer-Ballester (Faculty of Science and Technology, Bournemouth University, UK. Bernstein Centre for Computational Neuroscience, University of Heidelberg, Germany): Modulation of prefrontal cortex metastable dynamics by amphetamine.

W16 Open collaboration in computational neuroscience

Room RB 212, Thursday

Padraig Gleeson, University College London

Building and analysing biophysically and anatomically detailed neuronal networks is a complex and time consuming task, which ideally involves researchers with a range of backgrounds and technical skills. However, most labs cannot expect to have all of these researchers present at any given time. This can lead to stalled projects, lost data/software and needless repetition of experimental and computational work.

A number of initiatives have been started which address these issues. Some are creating public resources with freely available data to constrain such models. Others are using best practices from open source software development to encourage building and sharing of models in a collaborative environment. This workshop will serve as an informative introduction to these projects as well as a discussion forum for getting feedback and gathering requirements from the community for the developers of these initiatives.

Speakers:

- Jan Antolik (CNRS): Sumatra/PyNN/Helmholtz/INCF MSM Program/NeuralEnsemble
- Nicholas Cain (Allen Brain Institute): Resources for Open Collaboration at the Allen Brain Institute
- Padraig Gleeson (University College London): NeuroML & the Open Source Brain Initiative
- Stephen Larson (MetaCell Ltd): OpenWorm
- Aurel A. Lazar (Columbia University): Neurokernel
- Bill Lytton (SUNY Downstate Medical Center): ModelDB
- Eilif Muller (EPFL): Human Brain Project resources for the integrative modelling community
- Adrian Quintana (University College London): Geppetto: online visualisation & simulation for neuronal models
- Shreejoy Tripathy (University of British Columbia): Neuroelectro.org

W17 Postdoc and student career strategy workshop

Room RB 209, Wednesday

Jorge Meijas, Computational Lab of Cortical Dynamics, New York University, NY, USA

The computational neuroscience (CNS) community is both international and interdisciplinary, and there are many possible roads to success in the field. However, the challenges faced by current or soon-to-be postdocs are also diverse, and excellent mentorship from primary investigators is an invaluable resource for the development of future leaders in research or industry. This workshop is intended to provide postdocs and students in CNS an opportunity to hear about several very successful career paths and/or strategies from current leaders in the CNS community. The workshop will consist of testimonial insights from junior faculty having recently transitioned from postdoc status, researchers working outside of their home countries, and senior faculty who have witnessed and steered search committees, reviewing boards, and indeed the field of computational neuroscience itself through both ’fat’ and ’lean’ funding periods and through its exciting continued development. Postdocs and students are encouraged to ask questions to the speakers and participate in the discussion of topics of universal interest or specific concerns. Our own concerns are often more universal that we realize until we voice them! You are all invited to join the discussion. We also kindly ask you to please spread the word to anyone who might be interested (your students or postdocs, colleagues, etc).

The list of faculty mentors for this year includes:

- Ingo Bojak (UK)
- Romain Brette (France)
- Claudia Clopath (UK)
- Udo Ernst (Germany)
- Pablo Varona (Spain)
- and others...

Posters

Poster Listing

Sunday Posters
Posters P1 – P102

P1 **Computational modeling of heterosynaptic plasticity in the hippocampus**

Peter Jedlicka[1⋆], Lubica Benuskova[2,4], and Wickliffe C. Abraham[3,4]

[1]*Institute of Clinical Neuroanatomy, Neuroscience Center, Goethe University Frankfurt, Frankfurt/Main, Germany*
[2]*Department of Computer Science, University of Otago, Dunedin, New Zealand*
[3]*Department of Psychology University of Otago, Dunedin, New Zealand*
[4]*Brain Health Research Centre, University of Otago, Dunedin, New Zealand*

P2 **The sensori-motor model of the hippocampal place cells**

Anu Aggarwal⋆

Electrical and Computer Engineering Department, University of Maryland, College Park, MD USA

P3 **Short desynchronization epochs in neural synchronization: detection, mechanisms, and functions**

Leonid Rubchinsky[1,2⋆], Sungwoo Ahn[1,3]

[1]*Department of Mathematical Sciences, Indiana University Purdue University Indianapolis, Indianapolis, IN, USA*
[2]*Stark Neurosciences Research Institute, Indiana University School of Medicine, Indianapolis, IN, USA*
[3]*Present address: School of Mathematical and Statistical Sciences, Arizona State University, Tempe, AZ, USA*

P4 **Robust estimation of millisecond timescale synchrony under nonstationary conditions and its physiological interpretation**

Jonathan Platkiewicz[1⋆], Kamran Diba[2], Pascale Quilichini[3,4], György Buzsaki[5], and Asohan Amarasingham[1]

[1]*Department of Mathematics, City College, City University of New York, New York, NY 10031, USA*
[2]*Department of Psychology, University of Wisconsin-Milwaukee, Wisconsin, WI 53201, USA*
[3]*Aix Marseille Université, Institut des Neurosciences des Systèmes, Marseille, France*
[4]*Inserm, UMR_S 1106, 27 Bd Jean Moulin, 13385 Marseille Cedex 5, France*
[5]*Neuroscience Institute, New York University, New York, NY 10016, USA*

P5 **Effects of a reduced efficacy of the KCC2 co-transporter in Temporal Lobe Epilepsy: single neuron and network study**

Anatoly Buchin[1,2⋆], Gilles Huberfeld[3,4], Richard Miles[5], Anton Chizhov[6], and Boris Gutkin[1,7]

[1]*École normale supérieure, Laboratoire des Neurosciences Cognitives, Group for Neural Theory (France, Paris)*
[2]*Peter the Great St.-Petersburg Polytechnic University (Russia, St.-Petersburg)*
[3]*Neurophysiology Department, Pitie-Salpetriere Hospital, UPMC (France, Paris)*
[4]*Epilepsie de l'Enfant et Plasticité Cérébrale, INSERM U1129 (France, Paris)*
[5]*Institut du Cerveau et de la Moelle Epiniere, Cortex et Epilepsie Group (France, Paris)*
[6]*Ioffe Physical Technical Institute, Computational Physics Laboratory (Russia, St.-Petersburg)*
[7]*Higher School of Economics (Russia, Moscow)*

P6 Role of topology in the spontaneous cortical activity

Silvia Scarpetta[1⋆], Antonio de Candia[2], and Ilenia Apicella[1]

[1]Department of Physics "E.R.Caianiello" & INFN, University of Salerno, Fisciano (SA) 84084, Italy
[2]Department of Physics, University of Napoli "Federico II", Napoli, Italy & INFN sezione di Napoli, Italy

P7 A network model of neural activity in essential tremor

Nada Yousif[1⋆], Michael Mace[2], Nicola Pavese[1], Roman Borisyuk[3], Dipankar Nandi[1], and Peter Bain[1]

[1]Division of Brain Sciences, Imperial College London, London, W6 8RF, UK
[2]Department of Bioengineering, Imperial College London, London, SW7 2AF, UK
[3]School of Computing and Mathematics, University of Plymouth, Plymouth, PL4 8AA, UK

P8 A biophysical neural network model for visual working memory that accounts for memory binding errors

Joao Barbosa⋆, Alberto Compte

Intitut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), 08036 Barcelona, Spain

P9 Postsynaptic mechanisms influencing the duration of depolarization discharges in hyperexcitable neuro-glial networks

Vasily Grigorovsky⋆, Berj Bardakjian

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P10 Different weightings of input components to hippocampal CA1 place cells in young and aged rats

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P11 Mechanisms of hippocampal sequence replay

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P12 Bayesian Supervised Learning and State Estimation in a Model of the Cerebellum

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P13 Investigating intrinsic and evoked activities in cultured neuronal networks by dimensional reduction techniques and high-density MEAs

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P14 Cell assembly dynamics of sparse inhibitory networks: a simple model for the activity of the Medium Spiny Neurons

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P15 Sharp wave-ripple complexes in a reduced model of the hippocampal CA3-CA1 network of the macaque monkey

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P16 Modelling the Mechanoreceptor's Dynamic Behaviour

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P17 The functional significance of fasciculation and repulsion in a computational model of axon growth

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P18 Are rich club regions masters or slaves of brain network dynamics?

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P19 Multi-compartmental modeling in Brian 2

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P20 Emergence of ITD tuning in the MSO with a realistic periphery model

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P21 Proteomics investigation identifies prominent changes in synapse-related proteins in a fragile X mouse model

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P22 Numerical Simulations in Two-Dimensional Neural Fields

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P23 The Neurodynamics of Epilepsy: A homotopy analysis between current-based and conductance-based synapses in a neural field model of epilepsy

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P24 Incremental stability of delayed neural fields: a unifying framework for endogenous and exogenous sources of pathological oscillations

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P25 A Spiking network model of Basal Ganglia to study the effect of Dopamine medication and STN-DBS during Probabilistic Learning task

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P26 Modulation of neural firing through intracellular ATP dynamics governed by energy feedback from the vascular system

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P27 Could the prior development of the retinotopic map account for the radial bias in the orientation map in V1?

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P28 An auto-encoder network realizes sparse features under the influence of desynchronized vascular dynamics

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P29 A model of learning temporal delays, representative of adaptive myelination

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P30 Identifying excitatory and inhibitory synapses in neuronal networks from dynamics using Transfer Entropy

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P31 Early Dysregulation of Trigeminal Motor Pool Excitability in a Mouse Model for Neurodegenerative Motoneuron Disease

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P32 Non-invasively recorded transient pathological high-frequency oscillations in the epileptic brain: a novel signature of seizure evolution

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P33 Neuromechanical bistability contributes to robust and flexible behavior in a model of motor pattern generation

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P34 Investigating the effects of beta-amyloid on hippocampal signalling in Alzheimer's disease

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P35 A novel method to find out sensory neuron tracts in the Drosophila brain

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P36 The contribution of subthreshold preference in inhibitory neurons to network response

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P37 Extending the tempotron with hierarchical dendrites allows faster learning

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P38 A phase-locked loop epilepsy network emulator for localizing, forecasting, and controlling ictal activity

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P39 Using phase response curves to predict synchronization times for neural circuits

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P40 An asymptotic approximation to the cable equation for arbitrary diameter taper

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P41 Model-based prediction of maximum pool size in the ribbon synapse

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P42 Coregulation of the Na/K pump and the h-current as a mechanism for robust neuromodulation

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P43 Robustness of spatial learning in flickering networks

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P44 A topological approach to synaptic connectivity and spatial memory

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P45 Towards "biophysical psychiatry": A modeling approach for studying effects of schizophrenia-linked genes on single-neuron excitability

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P46 Nonlinear system identification of receptive fields from spiking neuron data

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P47 An empirical model of Drosophila Photoreceptor-LMC network

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P48 Modeling of respiratory network: to sigh or not to sigh

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P49 A model postulating a pivotal role of the levator-depressor neuro-muscular systems in locomotion of the stick insect

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P50 Modelling searching movements of the front leg in the stick insect by means of a neuro-muscular model

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P51 Invariance to frequency and time dilation along the ascending ferret auditory system

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P52 Purkinje cells: the forest shapes the trees

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P53 The ionic mechanism of the Purkinje cell dendritic spikes generation and propagation: a model exploration

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P54 Implementation of Parallel Spatial Stochastic Reaction-Diffusion Simulation in STEPS

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P55 Accurate Approximation to Stochastic Reaction Diffusion on Unstructured Meshes in STEPS

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P56 ATP consumption in molecular signaling of CA1 Hippocampus neurons

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P57 Dynamic model of whole cortex reveals disassortative hub structure in the intracortical connectome

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P58 The Neurodynamical Basis of Multi-Item Working Memory Capacity: Sequential vs Simultaneous Stimulation Paradigms

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P59 Firing rate response of neocortical neurons in the fluctuation-driven regime

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P60 How neuronal correlations affect the LFP signal?

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P61 Effective connectivity analysis explains metastable states of ongoing activity in cortically embedded systems of coupled synfire chains

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P62 Deterministic neural networks as sources of uncorrelated noise for probabilistic computations

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P63 Using Dynamic Time Warping for Quantifying Effects of Sinusoidal Oscillation Deviations during EEG Time Series Prediction and for Finding Interesting EEG and fMRI Changes

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P64 A full rat-scale model of the basal ganglia and thalamocortical network to reproduce Parkinsonian tremor

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P65 Should Hebbian learning be selective for negative excess kurtosis?

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P66 Decoding position from multiunit activity using a marked point process filter

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P67 Hybrid scheme for modeling local field potentials from point-neuron networks

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P68 Can ionic diffusion have an effect on extracellular potentials?

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P69 Attentional spreading over feature attributes and feature dimensions: Distributed top-down modulation or joint neural coding?

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P70 Probing information routing mechanisms by precisely-timed electrical stimulation pulses: a modelling study

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P71 Area Summation Is Related To Efficient Neural Representation

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P72 Enhanced novelty detection in auditory scenes through adaptation of inhibition

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P73 Mechanisms of cortical high-gamma activity (60-200 Hz) investigated with computational modeling

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P74 A reservoir network model for sensory-guided probabilistic decision making

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P75 Stimulus induced resonance in a neural mass model driven with a temporally correlated noise

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P76 Modeling thalamic dynamics with a network of integrate and fire neurons

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P77 Surprise minimization as a learning strategy in neural networks

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P78 A hierarchy of time scales supports unsupervised learning of behavioral sequences

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P79 Bridging spiking neuron models and mesoscopic population models – A general theory for neural population dynamics

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P80 Variable Bin Size Selection for Periestimulus Time Histograms (PSTH) with Minimum Mean Square Error Criteria

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P81 Neural model of biological motion recognition based on shading cues.

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P82 Visualizing, editing and simulating neuronal models with the Open Source Brain 3D explorer

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P83 Effects of spike-time dependent plasticity on deep brain stimulation of the basal ganglia for treatment of Parkinson's disease

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P84 Computational modelling predicts activity-dependent neuronal regulation by nitric oxide increases metabolic pathway activity

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P85 Computational neural modelling of auditory cortical receptive fields

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P86 The interaction between integration and segmentation neurons for motion perception

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P87 A simple effective model for STDP: from spike pairs and triplets to rate-encoding plasticity

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P88 Slow points and adiabatic fixed points in recurrent neural networks

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P89 Limit cycles with transient state dynamics in cyclic networks

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P90 Graph theoretical comparison of functional connectivity between cLTP treated and untreated microelectrode arrays

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P91 Simulation of AMPA and NMDA contribution to postsynaptic response

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P92 Detecting network states in white noise

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P93 Is it right to estimate inter-modular connectivity from local field potentials?

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P94 Analyzing adaptive modulation in spinal motor neurons using Multi-Objective Evolutionary Algorithms

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P95 Synaptic input patterns triggering local dendritic spikes in vivo

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P96 Functional consequences of non-equilibrium dynamics caused by antisymmetric and symmetric learning rules

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P97 Identifying and exploiting the anatomical origin of population rate oscillations in multi-layered spiking networks

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P98 Homeostatic intrinsic plasticity, neural heterogeneity and memory maintenance

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P99 Brain state dependent stimulus information in the auditory thalamocortical system

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P100 Different roles for ipsilateral positive feed back and commissural inhibitory networks in oculomotor velocity to position neural integration

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P101 A categorical approach to neurodynamical modelling of musical tonality

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P103 Balancing the critical period of spiking neurons with attractor-less STDP

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P104 Spiking neural network configuration designed for switching between basic forms of movement in a biped robot

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P105 Dynamics analysis of neural univariate time series by recurrence plots

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P106 Simulating electrode arrangements on microelectrode arrays

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P107 Modeling the interplay between Structural Plasticity and Spike-timing-dependent Plasticity

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P108 Understanding short-timescale neuronal firing sequences via bias matrices

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P109 Effects of multimodal distribution of delays in brain network dynamics

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P110 Using the Connectome to predict epileptic seizure propagation in the human brain

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P111 Adaptive control of ventilation using electrical stimulation in a biomechanical model

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P112 Predictable Implications of Random Photon Absorption for Photoreceptors' Gain Control

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P113 An increase in the extracellular potassium concentration can cause seizures

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P114 Discrete cortical representations and their stability in the presence of synaptic turnover

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P115 Influence of recurrent interactions on texture processing in networks with different visual map organizations

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P116 A Newton-based shooting method to find synaptic threshold in active cables

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P117 Fully-automated multi-objective optimization for fitting a neuronal model with real morphology

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P118 Granule cell excitability mediates gamma and beta oscillations in a model of the dendrodendritic microcircuit

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P119 Distinct and competing interneuron populations can generate fast and slow gamma in oscillatory models of CA1

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P120 Modeling spontaneous activity across an excitable epithelium: Support for a coordination scenario of early neural evolution

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P121 Mechanisms of spikelet generation in cortical pyramidal neurons

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P122 Spiking network modeling of neuronal dynamics in individual rats

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P123 T-type calcium channels trigger a hyperpolarization induced afterdepolarization in substantia nigra dopamine neurons

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P124 Assessing Performance of Directed Functional Connectivity Measures in the Presence of Common Source

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P125 How slow K+ currents impact on spike generation mechanism?

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P126 Excitatory to inhibitory connectivity shaped by synaptic and homeostatic plasticity

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P127 Phase lead/lag due to degree inhomogeneity in complex oscillator network with application to brain networks

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P128 Dynamics on global brain networks at the neuronal resolution

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P129 Subthreshold resonance in biophysically-based models of low- and high-input conductance motoneurons

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P130 A super-resolution approach for receptive fields estimation of neuronal ensembles

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P131 Nonparametric estimation of characteristics of the interspike interval distribution

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P132 The role of mechanosensory T cells for stimulus encoding in the local bend network of the leech

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P133 Time-resolved and parameter-free measures of spike train synchrony: Properties and applications

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P134 Detecting parallel bursts in in silico generated parallel spike train data

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P135 Minimal set of nodes to control the dynamics of biological neuronal networks

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P137 A role of local VTA GABAergic neurons in mediating dopamine neuron response to nicotine

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P138 Qualitatively different scenarios for co-activation of NMDA, AMPA and GABA receptor currents on dopaminergic neuron

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P139 Prefrontal-hippocampal theta coherence, sharp wave ripples, and bursts of cortical unit activity underlie choices and encoding in the radial arm maze

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P140 Mode-Locking Behavior of Izhikevich Neurons under Periodic External Forcing

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P141 The OpenWorm Project: currently available resources and future plans

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P142 Encoding of information using neural fingerprints

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P143 Reconstructing dynamical models from optogenetic data

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P144 Computational Model of Medial Temporal Lobe Epilepsy

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P145 PyMICE - a Python(TM) library for analysis of mice behaviour

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P146 A Biologically Based Neural Network Model for Decision Making

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P147 Numerical characterization of noisy fluctuations in two different types of stochastic differential equation models of neural signaling

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P148 Extending computational models of astrocyte-neuron interactions with biochemical mechanisms on the postsynaptic terminal

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P149 Information-theoretic analysis of a dynamic release site using a two-channel model of depression

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P150 Interplay of Intrinsic and Network Heterogeneity in Strongly Recurrent Spiking Networks

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P151 Parallelizing large networks using NEURON-Python

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P152 Motor cortex neurons: from experiment to model via evolutionary algorithms

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P153 Large-scale M1 microcircuit model with plastic input connections from biological PMd neurons used for prosthetic arm control

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P154 A lower bound on the number of mechanisms for discriminating fourth and higher order spatial correlations

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P155 Spatiotemporal brain network analysis of healthy humans based on magnetoencephalography and functional MRI in the resting state

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P156 Pattern Recognition of Hodgkin-Huxley Equations by Auto-regressive Laguerre Volterra Network

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P157 Neural coding of monaural and binaural intensity at low stimulus frequencies

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P158 Voltage sensitive currents and information processing by single neurons

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P159 A neural model of the optomotor system accounts for ordered responses to decreasing stimulus spatial frequencies

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P160 Orientation selectivity in a model of primary visual cortex with and without orientation map

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P161 When function mirrors structure: how slow waves are shaped by cortical layers

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P162 A novel method for approximating equilibrium single-channel Ca2+ domains

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P163 How central inputs and force and velocity feedbacks determine motoneurons activity during voluntary hand movements

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P164 Induction of Long-Term Potentiation and Depression in individual synapses of CA1 pyramidal neurons

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P165 Cell-type specific connectivity accounts for diverse in vivo functional roles of inhibitory neurons in V1

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P166 Reconstructing the directionality of coupling between cortical populations with negative phase lag

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P167 On the basic mechanisms of anticipated synchronization in neuronal circuits

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P168 Information transfer by local field potentials in the hippocampal formation

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P169 ROS-MUSIC Toolchain for Spiking Neural Network Simulations in a Robotic Environment

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P170 SPIKE-Synchronization: A parameter-free and time-resolved coincidence detector with an intuitive multivariate extension

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P171 Novel perspective on field recordings in zebrafish models of epilepsy

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P172 Shaping Pathological Cortical Dynamics with High-Frequency Neurostimulation

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P173 Synaptic inputs are tuned to match intrinsic properties to maintain phase in oscillatory neural networks

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P174 Dynamical Sensory Representations establish a Rapid Odor Code in a Spiking Model of the Insect Olfactory System

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P175 Application of generalized linear models to investigate functional synaptic coupling and synchrony in an animal model of schizophrenia

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P176 The Role of Horizontal Connections for the Modulation of Border-Ownership Selective Neurons in Visual Cortex

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P177 A computational model of cell culture dynamics: the role of connectivity and synaptic receptors in the appearance of synchronized bursting events

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P178 PyRhO: A virtual optogenetics laboratory

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P179 An Efficient and Accurate Solver for Large, Sparse Neural Networks

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P180 Noise signature on interval timing

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P181 Spiking neural network model of reinforcement learning in the honeybee implemented on the GPU

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P182 Estimating Numerical Error in Neural Network Simulations on Graphics Processing Units

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P183 Low-dimensional spike rate dynamics of coupled adaptive model neurons

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P184 pypet: A Python Toolkit for Simulations and Numerical Experiments

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P185 Extending integrate-and fire model neurons to account for the effects of weak electric fields in the presence of dendrites

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P186 Decoding of naturalistic textures from spike patterns of neuromorphic artificial mechanoreceptors

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P187 Hierarchical organization of multiscale communities in brain networks is non-tree structured

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P188 Sourcing brain histone modification data and development of algorithm for identification of hypersensitive sites

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P189 Vibrational resonance in feed-forward-loop neuronal network motifs

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P190 Neural representation in F5: cross-decoding from observation to execution

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P191 Auditory Noise Influences Human Visual Perception of Ambiguous Information: Multi-modal integration during Bistable Perception

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P192 Local interaction in Rretinal ganglion ccell mosaics can seed generate a consistent spatial periodicity of in cortical orientation functional maps

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P193 How bifurcations affect functional connectivity in finite-size neural networks

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P194 Canonical correlations reveal co-variability between spike trains and local field potentials in area MT

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P195 Local structure supports learning of deterministic behavior in recurrent neural networks

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P196 Approximate nonlinear filtering with a recurrent neural network

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P197 The role of microcircuits in the pre-frontal cortex in detecting and encoding temporally patterned information

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P198 Multiplexed coding through synchronous and asynchronous spiking

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P199 Partial Information Decomposition as a Unified Approach to the Characterization and Design of Neural Goal Functions

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P200 Cooperation/supervision of a habit by a cognitive strategy in a goal-directed navigational paradigm

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P201 A Minimum-Error, Energy-Constrained Neural Encoder Predicts an Instantaneous Spike-Rate code

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P202 Regular and irregular stimuli result in changes in mice eye movement and cerebellar nuclei neuron model behavior

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P203 Contributions from active dendritic conductances to the Local Field Potential

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P204 Cross-Cultural Differences in Visual Attention: A computational modelling study

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P205 Synaptic transmission of spike trains with arbitrary interspike intervals

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P206 Coarse-grained description of the spatio-temporal dynamics of network activity from experimentally verified single-neuron models and connectivity

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P207 On-line identification of the end of motor imageries based on the alpha rebound detection

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P208 Optimal signal detection with neuronal diversity: balancing the gullible and the prudent neurons

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P209 A cortical multi-layered model and the properties of its internally-generated activity

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P210 Effect of synaptic plasticity on functional connectivity and global activity of a neocortical network model

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P211 Dynamics of competition between coupled spiking networks in the balanced state

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P212 The formation of habits in the neocortex under the implicit supervision of the basal ganglia

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P213 A realistic model of pitch explains the N100m morphology evoked by dyads

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P214 The Delayed Response Network: Towards a single layer universal neural network approximator and delay-based learning

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P215 Extracellular potassium concentration defines neuronal bursting properties

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P216 The suppression curve as a new representation of the premature EEG maturation

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P217 Multi-scale detection of rate and variance changes in neuronal spike trains

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P218 Joint pausiness in parallel spike trains

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P219 Temperature-induced changes of spike timing precision and network synchronisation

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P220 Dendritic distribution of synaptic input creates a trade-off between input selectivity and flexibility

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P221 Self-organization of information processing in developing neuronal networks

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P222 Markov Stability partitioning shows spectrally dependent community structure amongst thalamocortical neural ensembles

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P223 A robust model of sensory tuning using dendritic non-linearities

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P224 An Information Theoretic measure of cross-frequency coupling

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P225 Looking at the Role of Direct and Indirect Pathways in Basal Ganglia Networks at Different Levels

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P226 – Withdrawn –

P227 Curvature of dendritic nonlinearities modulates higher-order spiking correlations

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P228 Large-scale Quantitative Analysis of Neurons via Morphological Structures by Fast Automatically Structural Tracing Algorithm (FAST)

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P229 Symmetries constrain the transition to heterogeneous chaos in balanced networks

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P230 Predicting surgical outcome in intractable epilepsy using a computational model of seizure initiation

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P231 EEG slow-wave mediates the fragmentation and coupling of cortical networks in propofol-induced general anesthesia

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P232 Thalamo-cortical mechanisms of the observed specific changes in frontal and occipital EEG rhythms during propofol-induced sedation

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P233 Description and removal of background activity in EEG power spectra under general anesthesia using the Lorentzian curve

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P234 Functional requirements for homeostatic inhibitory plasticity in recurrent networks

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P235 Structural plasticity and associative memory in balanced neural networks with spike-time dependent inhibitory plasticity.

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P236 Using transfer entropy to study synaptic integration in Purkinje cells

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P237 Animat control by spiking neural networks evolved with a genetic algorithm

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P238 Evolving small spiking neural networks to work as state machines for temporal pattern recognition

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P239 Identifying and tracking simulated synaptic inputs from neuronal firing: insights from in vitro experiments

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P240 Neural representation of a spatial odor memory in the honeybee mushroom body

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P241 The effect of synchronized pauses on the coding strategies of cerebellar nuclear neurons: A modeling study

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P242 Modeling of seizure transitions with ion concentration dynamics

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P243 Lateral Inhibition as the Organizer of the Bottom-Up Attentional Modulation in the Primary Visual Cortex

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P244 Computational interactions between decision and emotion

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P245 Differential temporal activation of oxy- and deoxy-hemodynamic signals in optical imaging using functional near-infrared spectroscopy (fNIRS)

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P246 Mapping the smoking addiction using dynamic causal modelling at rest

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P247 Brief mindfulness training alters causal brain connections in mTBI

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P249 Surround suppression and normalization in a model of coupled balanced cortical networks with short-term synaptic plasticity

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P250 Effect of power-law ionic conductances in the Hodgkin and Huxley model

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P251 Estimation of the synaptic conductance in a McKean-model neuron

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P252 Induction and consolidation of calcium-based homo- and heterosyanptic potentiation and depression

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P253 Interaction between memories in an abstract mathematical model based on the Hebbian cell assembly hypothesis

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P254 Towards a biological plausible model of the interaction of long-term memory and working memory

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P255 Sparse Coding and Dictionary Learning for Spike Trains to Find Spatio-temporal Patterns

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P256 Extreme sensitivity of reservoir computing to small network disruptions

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P257 Role of Na+ and Ca2+ currents in computational model of in-vitro sigh generation

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P258 Fast and accurate representations of stochastic ion channel fluctuations

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P259 Mechanisms for Synchronized Burst Firing in Pyramidal Cells using Oscillatory Inhibition: A Model for Attentional Control

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P260 Alpha phase modulates the effectiveness and directionality of cortical communication

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P261 Analysis of replacing DNase-seq data with histone marks in computational dimer prediction

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P262 Fitness and neural complexity of animats exposed to environmental change

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P263 Stochastic mean-field formulation of the dynamics of diluted neural networks

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P264 Two different mechanisms alternate during cortical synchronized states

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P265 Self-organization of complex cortex-like wiring in a spiking neural network model

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P266 Key features of neural variability emerge from self-organized sequence learning in a deterministic neural network

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P267 Modulation of hippocampal gamma oscillations by acetylcholine: insights from mathematical and in vitro optogenetic models

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P268 A Spatiotemporal Model of Spine Calcium Dynamics in the Hippocampus

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P269 Transient Synchrony in Delayed Coupled Neuronal Networks

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P270 Stabilizing synchrony with heterogeneity

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P271 Novel modes in a Wilson-Cowan network

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P272 Proof of concept: A spatial modular small-world self-organises by adaptive rewiring.

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P273 Lateral connections synchronize population activity in a spiking neural network model of mid-brain superior colliculus

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P274 Channel-specific input/output transformations arising from the interaction between dynamic synapses and subthreshold oscillations

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P275 Regularization of a half-center oscillator network by closed-loop control

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P276 Two-channel models of medial and superior olive based on psychoacoustics

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P277 A Self-Organizing Neural Network for Neuromuscular Control

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P278 Developing and validating an isotrigon texture discrimination task using Amazon Mechanical Turk

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P279 Neural model for multi-stability in visual action recognition

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P280 Optimization of input parameters to a CN neuron model to simulate its activity during and between epileptic absence seizures

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P281 A spiking neuron network model for the delayed motion direction discrimination task

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P282 Prediction of ion channel parameter differences between groups of young and aged pyramidal neurons using multi-stage compartmental model optimization

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P283 What Must Come Down Goes Up - the Effect of Noise on Weights in Spike-Timing-Dependent Plasticity

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P284 A Hebbian Cell Assembly based neural field model for the remote associate task and creative search

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P285 Recurrent Networks Expect

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P286 A novel method for spatial source localization using ECoG and SEEG recordings in human epilepsy patients

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P287 Stimulus discrimination and association with Hebbian cell assemblies

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P288 Anticipative Tracking in Two-Dimensional Continuous Attractor Neural Networks

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P290 The effect of Delta9-tetrahydrocannabinol, Cannabidiol, Menthol and Propofol on 5-Hydroxytryptamine type 3 Receptors—A Computational Approach

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P291 More flexibility for code generation with GeNN v2.1

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P292 Mathematical modelling of ICAN-mediated persistent firing in hippocampal neurons

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P293 Spatiotemporal dynamics in spiking simulations of superior colliculus fit via MCMC suggest disinhibition responsible for superlinear summation

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P294 Natural environment promotes deeper brain functional connectivity than built environment

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P295 Axon Initial Segment potassium Channel Density in Cortical Neurons

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P296 Key factors dominating the neural coding preference to 1/f signal

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P297 Learning experience facilitates sparse coding of new odors in a large-scale olfactory bulb model

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P298 Axon zippering in neuronal cell culture and its biophysical modeling

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P299 Computational estimation of calcium fluxes in isolated magnocellular neurons

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P300 Functional identification of complex cells from spike times and the decoding of visual stimuli

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P301 Retina of the fruit fly eyes: A detailed simulation model

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P302 Network heterogeneity and seizure generation

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P303 The role of adaptation current in synchronously firing inhibitory neural networks with various topologies

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P304 Modeling the formation and dynamics of cortical waves induced by cholinergic modulation.

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P305 Modelling impairment of evoked gamma range oscillations in schizophrenia

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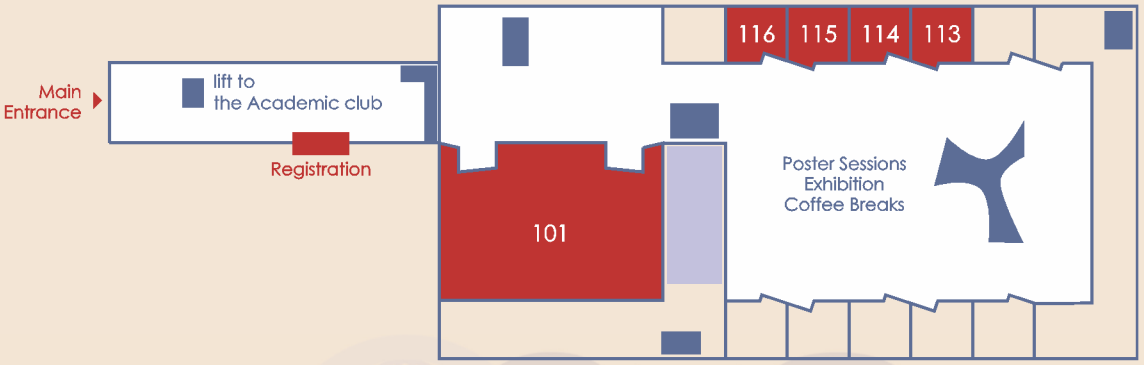

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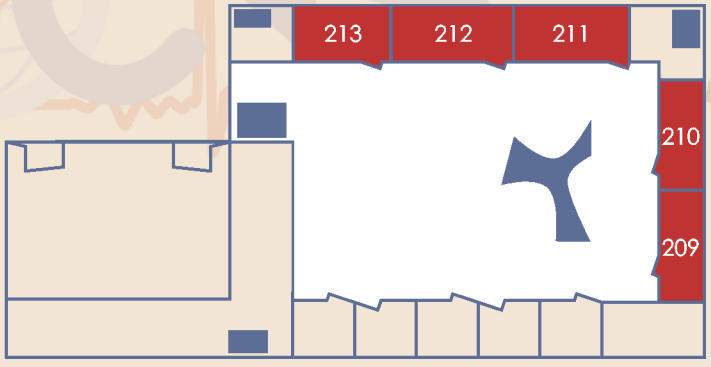

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