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Overview

Organization for Computational Neurosciences (OCNS)

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- Leonid Rubchinsky, Indiana and Purdue University, USA
- Ben Torben-Nielsen, Blue Brain Project, EPFL, Switzerland
- Taro Toyozumi, RIKEN Brain Science Institute, Japan

2014 Local Organizers

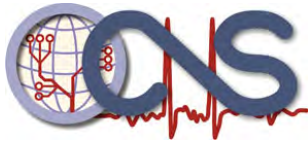
- Maurice Chacron, McGill University
- Yves de Koninck, Université Laval

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 philanthropic 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>.

CNS*2014 Sponsors

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



Organization for
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Neuroscience



CRC press



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Brain Corporation leverages neuroscience and machine learning towards a platform enabling robots who learn. Our software and algorithms add intelligence to robotics platforms allowing them to become teachable, much like animals.

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Seeking Experts in Sensorimotor Control and Software Development

A number of full-time positions at all levels are available immediately in theoretical and computational modeling of sensorimotor control at Brain Corporation, San Diego, CA.

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Requirements:

We are seeking candidates who are exceptional in one or more of the following areas:

- Feed-forward and recurrent neural networks, spike-timing plasticity and dynamics.
- Machine learning techniques, including convolutional networks and deep learning.
- Sensorimotor transformations, sensorimotor processing and reinforcement learning.

Plus have hacker level ability in regards to programming:

- Programming in two or more languages, including Python and C/C++/Objective-C.

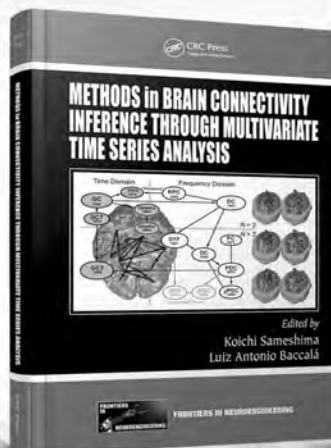
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The employee compensation package includes a stock option grant, matching 401k retirement contributions, medical, dental and vision insurance coverage, and annual performance bonuses. Employees have access to facilities on the Qualcomm campus and the office is located in proximity to UCSD and the Salk Institute in San Diego, California.

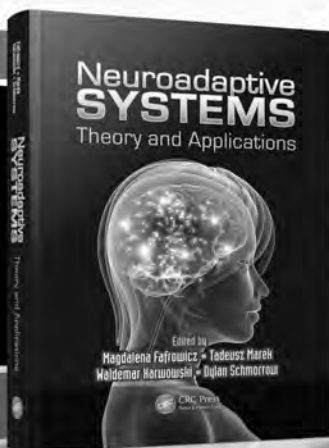
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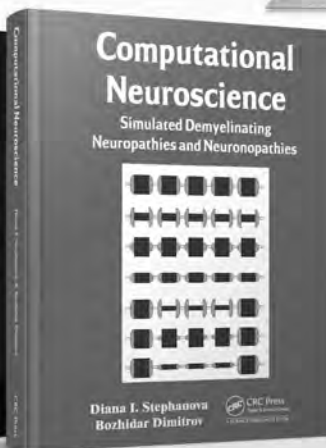
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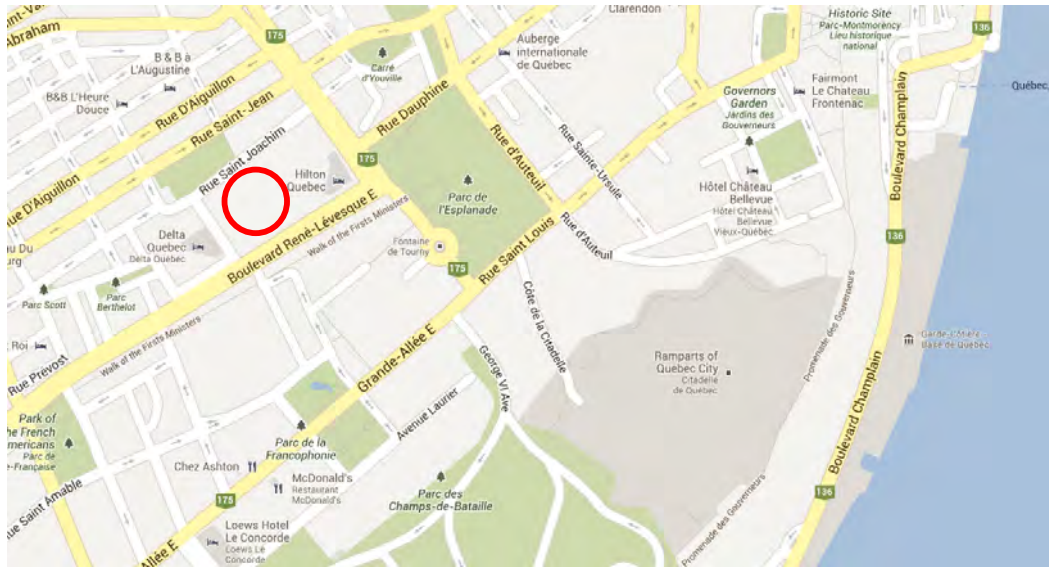
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Timetable

	Tutorials	Main Meeting			Workshops		
	Saturday July 26 th	Sunday July 27 th	Monday July 28 th	Tuesday July 29 th	Wednesday July 30 th	Thursday July 31 st	
8:00 to 9:00	Registration	Registration	Registration	Registration	Registration	Registration	
9:00 to 10:10	Tutorials (morning session)	Keynote 2: Christof Koch	Keynote 3: Henry Markram	Keynote 4: Frances Skinner	Workshop (morning session)	Workshop (morning session)	
10:10 to 10:40	Coffee break	Coffee break	Coffee break	Coffee break	Coffee break	Coffee break	
10:40 to 12:00	Tutorials (morning session)	Oral session 1	Oral sessions 4-5	Oral Session 7	Workshop (morning session)	Workshop (morning session)	
12:00 to 13:30	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch	
13:30 to 14:20	Tutorials (afternoon session)	Oral sessions 2-3	Oral session 6	OCNS member meeting	Workshop (afternoon session)	Workshop (afternoon session)	
14:20 to 14:50				Oral Session 8			
14:50 to 15:00	Tutorials (afternoon session)	Oral session 3	Oral session 6	Poster session 3	Workshop (afternoon session)	Workshop (afternoon session)	
15:00 to 15:20							Coffee break
15:20 to 15:30							
15:30 to 15:40							
15:40 to 16:00							
16:00 to 16:30							
17:00 to 17:15	Welcome and announcements	Poster session 1	Poster session 2	Walk to Banquet	Student/Post-doc career Workshop		
17:15 to 18:15	Keynote 1: Chris Eliasmith						
18:15 to 18:30	Welcome Reception						
18:30 to 19:00							
19:00 to 20:00							
20:00 to -			Party!	Banquet			

General Info

Map of the local area

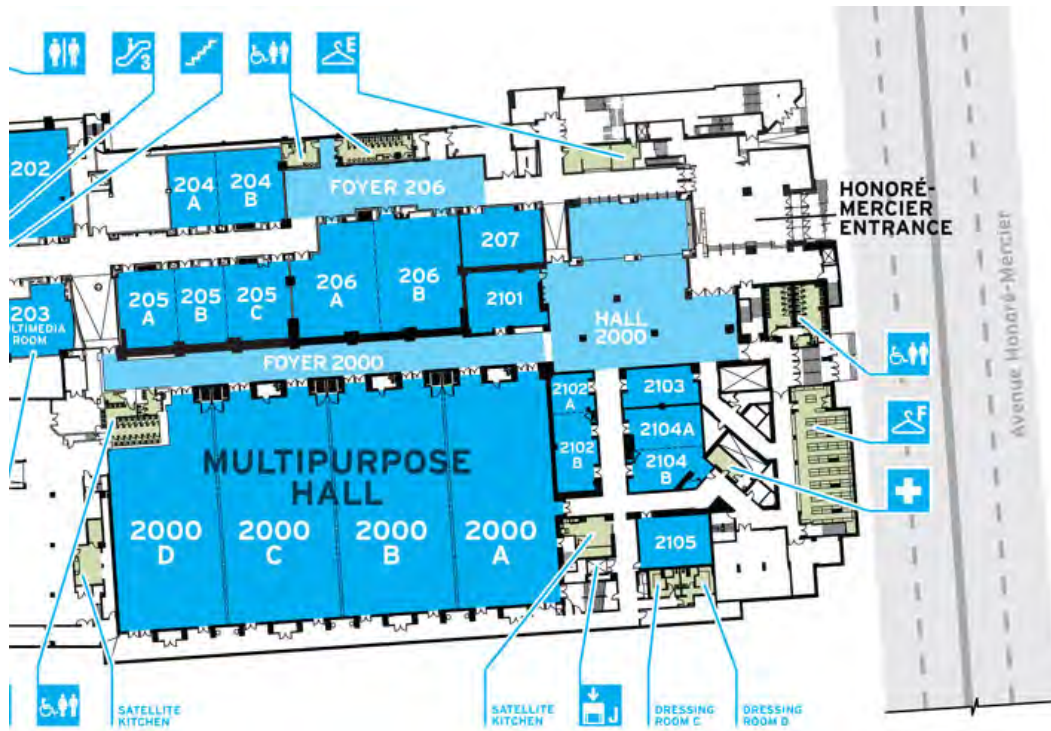


The main meeting, as well as all the tutorials and the workshops will all take place at the Québec City Convention center, 900, boul. René-Lévesque Est (see red circle on the map above). Located in the heart of Québec City, the Québec City Convention Centre is surrounded by a remarkable hotel and tourism environment and is steps away from the Parliament buildings, museums, nightclubs, and attractions like Old Québec and historic Battlefields Park. Hundreds of shops, boutiques and a variety of restaurants are only minutes away. With free high-speed wireless Internet access, the Convention Centre offers modern facilities with personalized service. Note that we have our own reserved entrance on "Avenue Honoré Mercier" in order to directly access the floorspace that is dedicated to CNS 2014.

Parking If coming by car to the convention center, please use P2 (marked by the red circle on the map below) as it has an underground tunnel that is directly linked to our entrance.



At the Meeting Venue



- Registration: Hall 2000
- Plenary Lectures: Room 2000A
- Workshops/tutorials: Rooms 207, 2101, 2102B, 2103, 2104A, 2104B, 2105
- Posters: Rooms 2000B/C

Money

- It is possible to withdraw Canadian dollars at almost any ATM.
- Visa and Mastercard are accepted in almost all stores.
- Banks are generally open from 9am to 5pm, or 6pm, from Monday to Saturday
- All prices **do not** include taxes (15 %).
- It is customary to give a tip (15 %) at restaurants

Language

The official language in Québec is French although although most shop owners, waiters, etc... will speak english as well.

Useful phone numbers

- Emergency (medical, police, fire, etc...): 911
- Québec city convention center: (418) 644-4000 or 1 (888) 679-4000
- Taxi Coop Québec (418) 525-5191
- Taxi Coop Sainte-Foy (418) 653-7777
- Taxi Laurier (418) 651-2727
- Taxi Québec (418) 525-8123

Restaurants: Inexpensive



13 results(s)

View Map



Korrigan Brasserie Artisanale

Geo. Area: Borough of La Cité - Limoilou / Downtown

Type: [Restaurants](#)

[Add to my favorites](#)

Located in the bustling Saint-Roch neighbourhood, Korrigan's good reputation is based on brewing high-quality beer the old-fashioned way. This bistro/brewery ...



Chez Ashton

Geo. Area: Borough of La Cité - Limoilou / Downtown

Type: [Snack Bars and Fast Food](#)

[Add to my favorites](#)

For 40 years, Chez Ashton has served food made from top quality ingredients. Specialties include roast beef sandwiches, hamburgers made from fresh ground ...



La Barberie, micro-brewery

Geo. Area: Borough of La Cité - Limoilou / Downtown

Type: [Bars and Discotheques](#)

[Add to my favorites](#)

Located in Québec City, this microbrewery has been creating first-rate beer since 1997. Wide selection of delicious and exclusive products for beer lovers ...

Buffet de l'Antiquaire

Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

Type: [Restaurants](#)

[Add to my favorites](#)

Tiny neighbourhood restaurant with a friendly family-type atmosphere. Authentic, traditional Québec cuisine found nowhere else. Fresh produce from the ...



Le Petit Cochon Dingue

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

♥ [Add to my favorites](#)

Traditional pastry shop whose desserts have helped establish the reputation of restaurants like Cochon Dingue, Lapin Sauté, Café du Monde and Paris Grill. ...



Restaurant Bistro Sous Le Fort

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

♥ [Add to my favorites](#)

Nestled in Quartier Petit-Champlain, this charming restaurant with courteous staff boasts a crackling fireplace in the winter, flowery terrace in the ...



Café-boulangerie Paillard

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

♥ [Add to my favorites](#)

An innovative European and North American inspired concept. Bread, pastries, sandwiches, soup and gelato. Café-Paillard serves delectable and organic ...



Restaurant L'Omelette

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

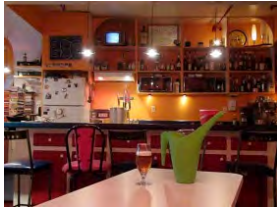
🍴 Type: [Restaurants](#)

♥ [Add to my favorites](#)

A wide selection of breakfasts, omelettes and French crêpes. Varied lunch and dinner menu: submarines, pizza, pasta, sandwiches, meat, fish and mussels. ...

La Cuisine

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown



Type: [Restaurants](#)

♥ [Add to my favorites](#)

Bistro/bar with a friendly, homelike atmosphere and colourful retro décor. Homemade food served both day and night. A modern twist to the traditional ...



Le Chic Shack

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

Type: [Restaurants](#)

♥ [Add to my favorites](#)

Burgers, salads, fries, homemade chips and milk shakes. Our burgers are served on buns baked by local baker Éric Borderon. Gluten-free bread available...



Pub Saint-Alexandre

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

Type: [Restaurants](#)

♥ [Add to my favorites](#)

English-style pub in the heart of Old Québec. Pub Saint-Alexandre offers 250 varieties of beer from all over the world, 22 draught beers and a wide variety ...



Le Cosmos Café

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

Type: [Restaurants](#)

♥ [Add to my favorites](#)

Restaurant specialty: a selection of 40 different breakfast dishes, served weekdays from 7 a.m. to 3 p.m., and from 8 a.m. on weekends. A wholesome menu ...

Restaurants: more expensive



17 results(s)



Feu Sacré Bistro Grill

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 0.37 km (from Downtown)

♥ [Add to my favorites](#)

Proud partners of the Nouvelle-France Hotels, the Feu Sacré restaurants propose a different dining concept and atmosphere—but the same culinary expertise—at ...



Restaurant Beffroi Steak House

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 0.39 km (from Downtown)

♥ [Add to my favorites](#)

Located in Old Québec, just a few steps away from the Saint-Jean Gate. The Restaurant Beffroi Steak House has mastered the art of grilling meat and fish ...



Voodoo Grill

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 0.42 km (from Downtown)

♥ [Add to my favorites](#)

Savour the excellent cuisine at Québec City's first real Supper Club. This trendy restaurant has over 150 kinds of tartare to choose from. After you eat, ...

La Grolla Restaurant Suisse

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 0.50 km (from Downtown)

♥ [Add to my favorites](#)

Charming Swiss café offering Swiss, Chinese and Bourguignon fondues, authentic raclette, seafood and our AAA filet mignon flambéed with cognac—an absolute ...



Le 47ième Parallèle

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 0.57 km (from Downtown)

♥ [Add to my favorites](#)

Near the Grand Théâtre de Québec and several major hotels. Distinctive international cuisine served amid stunning urban decor or on a magnificent terrace. ...



Conti Caffè

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

📏 Distance: 0.62 km (from Downtown)

♥ [Add to my favorites](#)

Colour, flavour, ambiance, freshness. These are the words that define the Conti Café, a delightful and brightly decorated restaurant where you can savour ...



Chez Boulay-bistro boréal

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

📏 Distance: 0.63 km (from Downtown)

♥ [Add to my favorites](#)

Seasonal Northern cuisine incorporating many locally-made or -grown foods at this bistro owned by chef Jean Luc Boulay and Arnaud Marchand. A variety ...



La Crémaillère

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

📏 Distance: 0.64 km (from Downtown)

♥ [Add to my favorites](#)

After the bustling summer months, time seems to slow down in Old Québec. As cold days are settling down in the city, the restaurant La Crémaillère is ...

Restaurant Le Continental

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

📏 Distance: 0.64 km (from Downtown)



♥ [Add to my favorites](#)

Québec's oldest gourmet restaurant. Located in a house built in 1845 by the Honourable Jean-Thomas Taschereau, justice of the Supreme Court of Canada. ...



Le Charles Baillairgé Resto-bar-lounge

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

📏 Distance: 0.67 km (from Downtown)

♥ [Add to my favorites](#)

Renowned for its gastronomic excellence, the Charles Baillairgé now has a new menu featuring contemporary international cuisine. The service at this Old ...



Restaurant-Pub D'Orsay

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

📏 Distance: 0.75 km (from Downtown)

♥ [Add to my favorites](#)

Treat yourself to a unique and magnificent setting in the heart of Vieux-Québec, facing City Hall and Basilique Notre-Dame de Québec. Without doubt, the ...



Charbon Steakhouse

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 0.94 km (from Downtown)

♥ [Add to my favorites](#)

AAA beef from Western Canada that is tenderized naturally. Fresh fish and exotic seafood grilled over wood charcoal the traditional way. You are sure ...

17 results(s)



Café Sirocco

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 1.02 km (from Downtown)

♥ [Add to my favorites](#)

Large selection of tartares. Tapas. Seafood. Entrées. Grilled meat. Fresh pasta. Martinis. Wine by the glass. A menu in the grand tradition of the best ...



Le Graffiti

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 1.04 km (from Downtown)

♥ [Add to my favorites](#)

The Graffiti has everything to please: a welcoming smile, relaxed, efficient service, warm décor, constantly changing menu, brilliant wine list. The restaurant ...



L'Échaudé

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

📏 Distance: 1.11 km (from Downtown)

♥ [Add to my favorites](#)

For 30 years, L'Échaudé has been the cornerstone of fine dining in the Old Port. The relaxed ambience, attentive staff and first-rate wine list complement ...

Le Clocher Penché Bistrot

📍 Geo. Area: Borough of La Cité - Limoilou / Downtown

🍴 Type: [Restaurants](#)

📏 Distance: 1.16 km (from Downtown)

♥ [Add to my favorites](#)



We strive to incorporate the passion of our agri-food suppliers and their dedication to their craft into every dish we prepare. The dining experience ...



Le Café du Monde

📍 Geo. Area: Borough of La Cité - Limoilou / Vieux-Québec

🍴 Type: [Restaurants](#)

📏 Distance: 1.36 km (from Downtown)

❤️ [Add to my favorites](#)

This authentic Parisian-style bistro offers the classics: mussels, steak and fries, duck confit and crème brûlée. Friendly and relaxed atmosphere. Extensive ...

Program

Tutorials

Tutorials		
Saturday July 26 th		
Tutorial	Morning	Afternoon
T1	Room 207	Room 207
T2	Room 2102B	Room 2102B
T3	Room 2104A	Room 2104A
T4	Room 2104B	Room 2104B
T5	Room 2103	Room 2103
T6	Room 2102B	Room 2102B
T7	Room 2101	Room 2101
T8	Room 2105	Room 2103
T9	Room 2105	Room 2105

T1 The Neural Engineering Framework (NEF): A General Purpose Method for Building Spiking Neuron Models

Room 207, 26 Jul 2014

Chris Eliasmith, University of Waterloo, CA, USA

Terrance Stewart, University of Waterloo, CA, USA

Moritz Deger, EPFL, Lausanne, Switzerland

T2 Themes in Computational Neuroendocrinology

Room 2102B, 26 Jul 2014

Joel Tabak, Florida State University, FL, USA

T3 Theory of correlation transfer and correlation structure in recurrent networks

Room 2104A, 26 Jul 2014

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

T4 Modeling and analysis of extracellular potentials

Room 2104B, 26 Jul 2014

Gaute Einevoll, Norwegian University of Life Sciences, Ås, Norway

Szymon Leski, Nencki Institute of Experimental Biology, Warsaw, Poland

Espen Hagen, Norwegian University of Life Sciences, Ås, Norway

- T5 NEURON Simulation Software**
Room 2103, 26 Jul 2014
Bill Lytton, SUNY Downstate Medical Center, USA
- T6 Constructing biologically realistic neuron and network models with GENESIS**
Room 2102B 26 Jul 2014
Hugo Cornelis, University of Texas Health Science Center at San Antonio, USA
- T7 Modeling of spiking neural networks with BRIAN**
Room 2101, 26 Jul 2014
Romain Brette, Institut de la Vision, Paris, France
Marcel Stimberg, Institut de la Vision, Paris, France
Pierre Yger, Institut de la Vision, Paris, France
Dan Goodman, Harvard Medical School, Boston, USA
- T8 Implementing neuron models for the NEST simulator**
Room 2103, 26 Jul 2014
Jochen M. Eppler, Research Center Jülich, Germany
Jannis Schücker, Research Center Jülich, Germany
- T9 Neuronal Model Parameter Search Techniques**
Room 2105, 26 Jul 2014
Cengiz Günay, Emory University, USA
Anca Doloç-Mihu, Emory University, Atlanta, USA
Vladislav Sekulic, University of Toronto, Canada
Tomasz G. Smolinski, Delaware State University, USA

Main Meeting

Saturday July 26

- 9:00 – 16:30 **Tutorials**
- 17:00 – 17:15 **Welcome and announcements**
- 17.15 – 18:15 K1 **Keynote 1:**
How to build large, multi-scale, functional brain models
Chris Eliasmith
- 18:15 – 20:00 **Welcome reception in "Hall 2000"**

Sunday July 27

9:00 – 9:10 **Announcements**

9:10 – 10:10 K2 **Keynote 2:**
*Exploring Cortex in a High-Throughput Manner by Building Brain
Observatories*
Christof Koch

10:10 – 10:40 **Break**

Oral session I: Large Networks

10:40 – 11:00 O1 *Simulating spiking neural networks on massively parallel graphical
processing units using a code generation approach with GeNN*
Esin Yavuz*, James Turner, and Thomas Nowotny

11:00 – 11:20 O2 *Patterns in network activity and information processing in a de-
tailed computer model of the cerebellar granular layer*
Shyam Kumar, Sungho Hong*, and Erik De Schutter

11:20 – 11:40 O3 *Mean Field Analysis Gives Accurate Predictions of the Behaviour
of Large Networks of Sparsely Coupled and Heterogeneous Neurons*
Wilten Nicola*, Felix Njap, Katie Ferguson, Frances Skinner, and Sue Ann
Campbell

11:40 – 12:00 O4 *Extracting novel information from neuroimaging data using neural
fields*
Dimitris Pinotsis*

12:00 – 13:30 **Break for lunch**

Oral session II: Single-cell modeling

13:30 – 13:50 O5 *Automatic fitness function selection for compartment model opti-
mization*
Timothy Rumbell*, Danel Draguljic, Jennifer Luebke, Patrick Hof, and
Christina M Weaver

13:50 – 14:30 F1 **Featured oral 1:**
*Optical coactivation in cortical cells: reprogramming the excitation-
inhibition balancing act to control neuronal gain in abstract and
detailed models*
Sarah Jarvis*, Konstantin Nikolic, and Simon R Schultz

Oral session III: Sensation and cognition

- 14:30 – 14:50 O6 *Predicting neural responses to natural sound in the auditory brainstem*
Dominika Lyzwa*, J Michael Herrmann
- 14:50 – 15:20 **Break**
- 15:00 – 15:20 O7 *Causal correlation paths across cortical areas in decision making*
Adrià Tauste Campo*, Marina Martinez-Garcia, Verónica Nácher, Gustavo Deco, and Ranulfo Romo
- 15:20 – 15:40 O8 *Trial-by-trial modeling of electrophysiological signals during inverse Bayesian inference*
Antonio Kolossa*, Bruno Kopp, and Tim Fingscheidt
- 16:00 – 19:00 **Poster session I: Posters P1 – P75, Sponsored by Brain Corporation**

Monday July 28

9:00 – 9:10 **Announcements**

9:10 – 10:10 K3 **Keynote 3:**
In silico Neuroscience: the next era
Henry Markram

10:10 – 10:40 **Break**

Oral session IV: Pattern generation

10:40 – 11:00 O9 *Parameter correlations maintaining bursting activity*
Anca Doloc-Mihu*, Ronald Calabrese

11:00 – 11:20 O10 *Organization of left-right coordination of neuronal activity in the mammalian spinal cord locomotor CPG: Insights from computational modeling*
Ilya Rybak*, Natalia Shevtsova, Adolfo Talpalar, Sergey Markin, Ronald Harris-Warrick, and Ole Kiehn

Oral session V: Synaptic plasticity

11:20 – 11:40 O11 *Determinants of gain modulation enabled by short-term depression at an inhibitory cerebellar synapse.*
Dimitris Bampasakis*, Reinoud Maex, Neil Davey, and Volker Steuber

11:40 – 12:00 O12 *Stable reinforcement learning via temporal competition between LTP and LTD traces*
Marco Huertas*, Sarah Schwettmann, Alfredo Kirkwood, and Harel Shouval

12:00 – 14:00 **Break for lunch**

Oral session VI: Network topology and dynamics

13:30 – 13:50 O13 *Neural graphs: Small-worlds, after all?*
Michelle Rudolph-Lilith*, Lyle Muller

13:50 – 14:30 F2 **Featured oral 2:**
Network community, clusters and hubs in cortical micro circuits.
Masanori Shimono*, John M Beggs

14:30 – 14:50 O14 *A k-population model to calculate the firing rate of neuronal networks with degree correlations*
Christian Schmeltzer*, Alexandre Kihara, Igor Sokolov, and Sten Rüdiger

- 14:50 – 15:20 **Break**
- 15:20 – 15:40 O15 *Criticality in cortical ensembles is supported by complex functional networks*
Paolo Massobrio*, Valentina Pasquale, and Sergio Martinoia
- 15:40 – 16:00 O16 *The interplay of intrinsic excitability and network topology in spatiotemporal pattern generation in neural networks.*
James Roach*, Leonard Sander, and Michal Zochowski
- 16:00 – 19:00 **Poster session II: Posters P76 – P150, Sponsored by Brain Corporation**
- 19:00 **CNS Party at "Chez Maurice"**

Tuesday July 29

9:00 – 9:10 **Announcements**

9:10 – 10:10 K4 **Keynote 4:**
Balancing and Tight Coupling: An approach to determine dynamic mechanisms of biological brain networks
Frances K Skinner

10:10 – 10:40 **Break**

Oral session VII: Pathological activity

10:40 – 11:20 F3 **Featured oral 3:**
Inhibitory single neuron control of seizures and epileptic traveling waves in humans
Omar Ahmed*, Mark Kramer, Wilson Truccolo, Jason Naftulin, Nicholas Potter, Emad Eskandar, Garth Cosgrove, Andy Blum, Leigh Hochberg, and Sydney Cash

11:20 – 11:40 O17 *Synchronization of the Parkinsonian Globus Pallidus by Gap Junctions*
Bettina Schwab*, Hil Meijer, Richard van Wezel, and Stephan van Gils

11:40 – 12:00 O18 *The dynamic separation of pallidal neurons into anti-phase oscillatory groups under Parkinsonian conditions in a computational model*
Robert Merrison-Hort*, Roman Borisyuk

12:00 – 13:30 **Break for lunch**

13:30 – 14:20 **OCNS Member Meeting**

Oral session VIII: Chaos

14:20 – 14:40 O19 *The sleeping brain regulates to the edge of chaos*
Moira Steyn-Ross*, Alistair Steyn-Ross, and Jamie Sleight

14:40 – 15:00 O20 *Chaos in heterogeneous neural networks*
Merav Stern*, Johnatan Aljadeff, and Tatyana Sharpee

15:00 – 15:30 **Break**

15:30 – 18:30 **Poster session III: Posters P151 – P225, Sponsored by Brain Corporation**

18:30 – 19:00 **Time to walk to banquet location (15 min walk)**

19:00 **Banquet at "La Chapelle du Musée"**

Wednesday July 30

9:00 – 19:00 **Workshops**

Thursday July 31

9:00 – 19:00 **Workshops**

Workshops

Workshops			
Workshop	Wednesday July 30 th	Thursday July 31 st	
W1	Room 207	Room 207	
W2	Room 2103	Room 2103	
W3	Room 2101	Room 2101	
W4	Room 2102B	[Cross-hatched pattern]	
W5	Room 2104B		
W6	Room 2105		
W7	Room 2104A		
W8	[Diagonal hatched pattern]		Room 2102B
W9			Room 2104B
W10			Room 2105
W11		Room 2104A	
W12	Room 2101 (6-8 PM)	[Diagonal hatched pattern]	

W1 Cortical Oscillations: Computational models and dynamic mechanisms

Room 207, W, Th

Horacio Rotstein, New Jersey Institute of Technology

Mark Kramer, Boston University

W2 Computational methods and modeling of Astrocyte physiology and Neuron-glia interactions

Room 2103, W, Th

Hugues Berry, INRIA

Maurizio De Pitta, University of Chicago

W3 Methods of Information Theory in Computational Neuroscience

Room 2101, W, Th

Michael C Gastpar, Laboratory for Information in Networked Systems, EPFL and UC Berkeley

Conor Houghton, Department of Mathematics, Trinity College Dublin

Simon R Schultz, Department of Bioengineering, Imperial College

Tatyana O Sharpee, The Computational Neurobiology Laboratory, Salk Institute

- W4 Running parallel simulations on HPC resources via the Neuroscience Gateway Portal**
Room 2102B, W
Amit Majumdar, UCSD
Ted Carnevale, Yale School of Medicine
- W5 Resonance and Entrainment: From Dynamic Systems Theory to Targeted Brain Stimulation**
Room 2104B, W
Flavio Frolich, University of North Carolina at Chapel Hill
- W6 Methods of System Identification for Studying Information Processing in Sensory Systems**
Room 2105, W
Aurel A Lazar, Department of Electrical Engineering, Columbia University
Mikko I Juusola,
- W7 Dynamics of Disease States**
Room 2104A, W
Jonathan Rubin, University of Pittsburgh
Stephan Van Gils, University of Twente
- W8 Sleep Rhythms and Memory Consolidation**
Room 2102B, Th
Maxim Bazhenov, UC Riverside
Igor Timofeev, Laval University
- W9 Basal Ganglia: Structure, dynamics and function**
Room 2104B, Th
Arvind Kumar, Bernstein Center Freiburg, University of Freiburg, Germany
Jeanette Hellgren Kotaleski, Royal Institute of Technology, Stockholm, Sweden
- W10 Large-scale brain structure and dynamics**
Room 2105, Th
Jorge F Mejias, NYU
Xiao-Jing Wang,

W11 Finite-size fluctuations in neural systems - from ion channels to networks

Room 2104A, Th

Richard Naud, University of Ottawa, Canada

Tilo Schwalger, EPFL, Switzerland

Moritz Deger, EPFL, Switzerland

W12 Student/Post-doc career Workshop

Room 2101, W (6-8 PM)

Jorge F Mejias, NJIT

Tutorials

Tutorials		
Saturday July 26 th		
Tutorial	Morning	Afternoon
T1	Room 207	Room 207
T2	/	Room 2102B
T3	Room 2104A	Room 2104A
T4	Room 2104B	Room 2104B
T5	Room 2103	/
T6	Room 2102B	/
T7	Room 2101	Room 2101
T8	/	Room 2103
T9	Room 2105	Room 2105

T1 The Neural Engineering Framework (NEF): A General Purpose Method for Building Spiking Neuron Models

Room 207, 26 Jul 2014

Chris , University of Waterloo, CA, USA

Terrance Stewart, University of Waterloo, CA, USA

Moritz Deger, EPFL, Lausanne, Switzerland

We have recently created the world's largest biologically plausible brain model that is capable of performing several perceptual, motor, and cognitive tasks (Eliasmith et al., 2012). This model uses 2.5 million spiking neurons, takes visual input from a 28x28 pixel visual field, and controls a physically modelled arm. It has been shown to match a wide variety of neurophysiological and behavioral measures from animals and humans performing the same tasks. This tutorial is meant to introduce the software toolkit (Nengo) and theoretical background (NEF) to allow other researchers to use the same methods for exploring a wide variety of brain functions. We will focus on the underlying theory of the Neural Engineering Framework (NEF; Eliasmith and Anderson, 2003), a general method for implementing large-scale, nonlinear dynamics using spiking neurons. Our emphasis will be on building such models using a GUI and scripting in our open-source toolkit Nengo (). We will help participants construct networks that perform linear and non-linear computations in high dimensional state spaces, including arbitrary attractor networks (point, line, cyclic, chaotic), controlled oscillators and filters, and winner-take-all networks. We will discuss both how the networks can be learned online with a spike-based learning rule, or more efficiently constructed. If time permits, the tutorial will introduce our Semantic Pointer Architecture (Eliasmith, 2013), encapsulated in a Python module for Nengo which can be used to rapidly implement large-scale cognitive models that include (basic) visual processing, motor control, working memory, associative memory, and cognitive control.

Audience

All participants are encouraged to bring a laptop for installing and running Nengo (Linux, OS X, and Windows versions are provided), allowing for hands-on interactions with the models discussed.

References:

- [1] Eliasmith, C. (2013). How to build a brain: A neural architecture for biological cognition. New York, NY: Oxford University Press.
- [2] Eliasmith, C., & Anderson, C. (2003). Neural Engineering: Computation, Representation, and Dynamics in Neurobiological Systems. Cambridge: MIT Press.
- [3] Eliasmith, C., Stewart T.C., Choo X., Bekolay T., DeWolf T., Tang Y., & Rasmussen, D. (2012). A largescale model of the functioning brain. *Science*. 338(6111), 12021205.

T2 Themes in Computational Neuroendocrinology

Room 2102B, 26 Jul 2014

Joel Tabak, Florida State University, FL, USA

Computational neuroendocrinology regroups the various efforts, at different levels of organization, to better understand neuroendocrine regulations using computational models. Neuroendocrine systems are organized in 'endocrine axes'. Each axis includes neuronal populations in the hypothalamus, cells in the pituitary gland that releases one or multiple hormones, and the target organ of this particular set of hormones.

Computational models describe the activity of hypothalamic neurons and how these neuroendocrine cells regulate the activity of pituitary cells that secrete hormones such as growth hormone, prolactin, luteinizing hormone, etc. They may also describe how hormones released by target organs in response to pituitary hormones, such as steroids, feedback and affect hypothalamo-pituitary regulations. One recurring theme is to understand how these regulations can produce pulsatile patterns of hormone secretion, and how target cells interpret these pulsatile patterns. In this tutorial we will present examples of models that illustrate important themes in computational neuroendocrinology. These models range from the single cell level to the network level and, further, to the multi organ level. They will emphasize some key features of the neuroendocrine systems: endocrine cells have wide action potential and bursts that rely more on Ca^{2+} than Na^{+} voltage-dependent channels; the main transmitters of neuroendocrine regulations are not binding to receptor-channels but to G-protein coupled receptors that trigger second messenger cascades, leading to protein phosphorylation or gene expression; as a result neuroendocrine regulations do not operate at the millisecond time scale but at much slower time scales, from seconds to days.

T3 Theory of correlation transfer and correlation structure in recurrent networks

Room 2104A, 26 Jul 2014

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

In the first part, we will study correlations arising from pairs of neurons sharing common

fluctuations and/or inputs. Using integrate-and-fire neurons, we will show 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 inputs correlations will be discussed. We will show that the output correlations are generally weaker than the input correlations [Moreno-Bote and Parga, 2006], that the shape of the cross-correlation functions depends on the working regime of the neuron [Ostojic et al., 2009; Helias et al., 2013], and that the output correlations strongly depend on the output firing rate of the neurons [de la Rocha et al, 2007]. We will study generalizations of these results when the pair of neurons is reciprocally connected.

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 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] to the leaky integrate-and-fire model and present a unifying view of linearized theories of correlations [Helias et al, 2011].

At last, we will revisit the important question of how correlations affect information and vice-versa [Zohary et al, 1994] in neuronal circuits, showing novel results about information content in recurrent networks of integrate-and-fire neurons [Moreno-Bote and Pouget, Cosyne abstracts, 2011].

References:

- [1] Eliasmith, C. (2013). How to build a brain: A neural architecture for biological cognition. New York, NY: Oxford University Press.
- [2] Eliasmith, C., & Anderson, C. (2003). Neural Engineering: Computation, Representation, and Dynamics in Neurobiological Systems. Cambridge: MIT Press.
- [3] Eliasmith, C., Stewart T.C., Choo X., Bekolay T., DeWolf T., Tang Y., & Rasmussen, D. (2012). A largescale model of the functioning brain. *Science*. 338(6111), 12021205.
- [4] de la Rocha et al. (2007), Correlation between neural spike trains increases with firing rate, *Nature* 448:802-6
- [5] Ginzburg & Sompolinsky (1994), Theory of correlations in stochastic neural networks, *PRE* 50:3171-3190
- [6] Hawkes (1971), Point Spectra of Some Mutually Exciting Point Processes, *Journal of the Royal Statistical Society Series B* 33(3):438-443
- [7] Helias et al. (2011), Towards a unified theory of correlations in recurrent neural networks, *BMC Neuroscience* 12(Suppl 1):P73
- [8] Helias et al. (2013), Echoes in correlated neural systems, *New Journal of Physics* 15(2):023002
- [9] Moreno-Bote & Parga (2006), Auto- and crosscorrelograms for the spike response of leaky integrate-and-fire neurons with slow synapses, *PRL* 96:02810
- [10] Ostojic et al. (2009), How Connectivity, Background Activity, and Synaptic Properties Shape the Cross-Correlation between Spike Trains, *J Neurosci* 29(33):10234-10253

- [11] Renart et al. (2010), The Asynchronous State in Cortical Circuits, *Science* 327(5965):587-590
- [12] Shadlen & Newsome (1998), The variable discharge of cortical neurons: implications for connectivity, computation, and information coding, *J Neurosci* 18:3870-96
- [13] Tetzlaff et al. (2012), Decorrelation of neural-network activity by inhibitory feedback, *PLoS Comp Biol* 8(8):e1002596, doi:10.1371/journal.pcbi.1002596
- [14] Zohary et al. (1994), Correlated Neuronal Discharge Rate and Its Implications for Psychophysical Performance, *Nature* 370:140-14

T4 Modeling and analysis of extracellular potentials

Room 2104B, 26 Jul 2014

Gaute Einevoll, Norwegian University of Life Sciences, Ås, Norway

Szymon Leski, Nencki Institute of Experimental Biology, Warsaw, Poland

Espen Hagen, Norwegian University of Life Sciences, Ås, Norway

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 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
 - CSD 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 microcircuit model for a visual cortical column [22,23], will be presented.

References:

- [1] KH Pettersen et al, 'Extracellular spikes and CSD' in Handbook of Neural Activity Measurement, Cambridge (2012)
- [2] G Buzsaki et al, Nature Reviews Neuroscience 13:407 (2012)
- [3] GT Einevoll et al, Nature Reviews Neuroscience 14:770 (2013)
- [4] GT Einevoll et al, Current Opin Neurobiol 22:11 (2012)
- [5] G Holt, C Koch, J Comp Neurosci 6:169 (1999)
- [6] J Gold et al, J Neurophysiol 95:3113 (2006)
- [7] KH Pettersen and GT Einevoll, Biophys J 94:784 (2008)
- [8] KH Pettersen et al, J Comp Neurosci 24:291 (2008)
- [9] H Lindén et al, J Comp Neurosci 29: 423 (2010)
- [10] H Lindén et al, Neuron 72:859 (2011)
- [11] S Łęski et al, PLoS Comp Biol 9:e1003137 (2013)
- [12] KH Pettersen et al, J Neurosci Meth 154:116 (2006)
- [13] S Łęski et al, Neuroinform 5:207 (2007)
- [14] S Łęski et al, Neuroinform 9:401 (2011)
- [15] J Potworowski et al, Neural Comp 24:541 (2012)
- [16] GT Einevoll et al, J Neurophysiol 97:2174 (2007)
- [17] P Blomquist et al, PLoS Comp Biol 5:e1000328 (2009)

- [18] SL Gratiy et al, Front Neuroinf 5:32 (2011)
- [19] H Lindén et al, Front Neuroinf 7:41 (2014)
- [20] ML Hines et al, Front Neuroinf 3:1 (2009)
- [21] R Traub et al, J Neurophysiol 93:2194 (2005)
- [22] TC Potjans and M Diesmann, Cereb Cort 24:785 (2014)
- [23] E Hagen et al, BMC Neuroscience 14(Suppl 1):P119 (2013)

T5 NEURON Simulation Software

Room 2103, 26 Jul 2014

Bill Lytton, SUNY Downstate Medical Center, USA

This half-day tutorial will focus on several new features that have been added recently to the NEURON simulator environment, as well as highlighting older features that have had recent upgrades. Questions are encouraged during each talk and during time set aside at end of each talk.

Presentations will include the following:

1. Use of NEURON for multiscale modeling (Bill Lytton)
2. Use of the Python interpreter to work with hoc/nrniv objects (Sam Neymotin)
3. Reaction-diffusion (RxD) modeling techniques in NEURON (Robert McDougal)
4. ==== Coffee Break =====
5. Cell level modeling for synaptic distribution and current source density (Bill Lytton)
6. Design of large networks (Cliff Kerr)
7. NEURON interfacing: robots, sense inputs, mean fields models (Salvador Dura-Bernal)
8. Modelview to evaluate and use modelDB to build your own sim (Robert McDougal)
9. Discussion, questions, further examples..

T6 Constructing biologically realistic neuron and network models with GENESIS

Room 2102B, 26 Jul 2014

Hugo Cornelis, University of Texas Health Science Center at San Antonio, USA

This tutorial is aimed at people who are new to or have only elementary knowledge about the GENESIS-2 simulator, as well as those who have used GENESIS in the past and would like to

learn of new developments in cortical network modeling with GENESIS. After a quick overview of the GENESIS project [1], the tutorial demonstrates methods for single neuron modeling. It then continues with the use of the GENESIS neural simulator for the efficient modeling of large networks of biologically realistic neurons. The tutorial ends with a summary about the recent development of functionality for modeling spike-timing dependent plasticity in network models that include realistic neuronal morphology and axonal conduction delays for the delivery of action potentials.

The tutorial is a guide to the use of the CNS 2014 release of the Ultimate GENESIS Tutorial Distribution [2]. This is a newly updated version of a self-paced course on biologically realistic modeling in general, and creating simulations with GENESIS in particular. This package contains the full GENESIS 2.3 distribution, as well as recent patches that will be incorporated into the GENESIS 2.4 release later this year. It includes materials used by several recent international courses on neural modeling as well as new cortical network simulation examples with tutorial documentation. It comes with suggested exercises for independent study.

This tutorial should give you everything that you need to get started modeling with GENESIS, and to develop your own simulations starting from these examples.

References:

- [1] Bower JM, Beeman D (1998, 2003) *The Book of GENESIS: Exploring Realistic Neural Models with the General NEural SIMulation System*, second edn. Springer-Verlag, New York. (Free internet edition available at: <http://www.genesis-sim.org/GENESIS/bog/bog.html> [www.genesis-sim.org])
- [2] The "Ultimate GENESIS Tutorial Distribution" [genesis-sim.org](http://www.genesis-sim.org).

T7 Modeling of spiking neural networks with BRIAN

Room 2101, 26 Jul 2014

Romain Brette, Institut de la Vision, Paris, France

Marcel Stimberg, Institut de la Vision, Paris, France

Pierre Yger, Institut de la Vision, Paris, France

Dan Goodman, Harvard Medical School, Boston, USA

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.

In the morning, we will give an introduction to Brian and an overview of the existing Brian extensions (brian hears [4], model fitting toolbox [5], compartmental modelling). In the afternoon, more advanced topics (extending Brian; code generation[5], including the generation of "standalone code"; contributing to Brian) will be covered.

References:

- [1] <http://briansimulator.org>
- [2] Goodman & Brette (2009), The Brian simulator, *Front Neurosci*, doi:10.3389/neuro.01.026.2009.

- [3] Stimberg M, Goodman DFM, Benichoux V, and Brette R (2014). Equation-oriented specification of neural models for simulations. *Frontiers in Neuroinformatics* 8. doi:10.3389/fninf.2014.00000
- [4] Rossant et al. (2010), Automatic fitting of spiking neuron models to electrophysiological recordings, *Frontiers in Neuroinformatics*, doi:10.3389/neuro.11.002.2010
- [5] Goodman (2010), Code generation: a strategy for neural network simulators, *Neuroinformatics*, doi:10.1007/s12021-010-9082-x
- [6] Fontaine B, Goodman DFM, Benichoux V, Brette R (2011). Brian Hears: online auditory processing using vectorisation over channels. *Frontiers in Neuroinformatics* 5:9. doi:10.3389/fninf.2011.00009

T8 Implementing neuron models for the NEST simulator

Room 2103, 26 Jul 2014

Jochen M. Eppler, Research Center Jülich, Germany

Jannis Schücker, Research Center Jülich, Germany

The neural simulation tool NEST [1, www.nest-initiative.org] is a simulator for heterogeneous networks of point neurons or neurons with a small number of electrical compartments aiming at simulations of large neural systems. It is implemented in C++ and runs on a large range of architectures from single-processor desktop computers to large clusters and supercomputers with thousands of processor cores.

This tutorial is for researchers who are interested in the implementation of new neuron models for NEST in an efficient way. We will start with a more technical description of the scheduler, the parallelization facilities, and the neuron base class in NEST and continue with an in-depth discussion of the internals of an existing neuron model in NEST.

It is helpful if NEST or another simulator for spiking neuronal networks has been used previously and if the basic knowledge about neuronal modeling in general is present. Some programming background in C++ is beneficial but not required.

References:

- [1] BMarc-Oliver Gewaltig and Markus Diesmann (2007) NEST (Neural Simulation Tool), *Scholarpedia* 2 (4), p. 1430.

T9 Neuronal Model Parameter Search Techniques

Room 2105, 26 Jul 2014

Cengiz Günay, Emory University, USA

Anca Doloç-Mihu, Emory University, Atlanta, USA

Vladislav Sekulic, University of Toronto, Canada

Tomasz G. Smolinski, Delaware State University, USA

Parameter tuning of model neurons to mimic biologically realistic activity is a non-trivial

task. Multiple models may exhibit similar dynamics that match experimental data – i.e., there is no single ‘correct’ model. To address this issue, the ensemble modeling technique proposes to represent properties of living neurons with a set of neuronal models. Several approaches to ensemble modeling have been proposed over the years, but the two most prevalent parameter tuning methods are systematic ‘brute-force’ searches [1, 2] and various evolutionary algorithms-based techniques [3, 4, 5, 6]. Both approaches relay on traversing a very large parameter space (with thousands to millions of model instances), but utilize diametrically different ways to accomplish that. In both cases, however, entire collections of biologically realistic models are generated, whose neural activity characteristics can then be cataloged and studied using a database [1, 2]. The tutorial covers ‘tips and tricks,’ as well as various pitfalls in all stages of model construction, large-scale simulations on high performance computing clusters [S2], database construction and analysis of neural data, along with a discussion about the strengths and weaknesses of the two parameter search techniques. We will review software implementations for each technique: PANDORA Matlab Toolbox [7][S1] for the brute force method and NeRvolver (i.e., evolver of nerve cells) for evolutionary algorithms. PANDORA was used in recent projects for tuning models of rat globus pallidus neurons [2][M1], lobster pyloric network calcium sensors [8][M2], leech heart interneurons [9][M3,S3] and hippocampal O-LM interneurons (Skinner Lab, TWRI/UHN and Univ. Toronto). NeRvolver is a prototype of a computational intelligence-based system for automated construction, tuning, and analysis of neuronal models, which is currently under development in the Computational Intelligence and Bio (logical) informatics Laboratory at Delaware State University [10]. Through the utilization of computational intelligence methods (i.e., Multi-Objective Evolutionary Algorithms and Fuzzy Logic), the NeRvolver system generates classification rules describing biological phenomena discovered during the process of model creation or tuning. Thus in addition to producing neuronal models, NeRvolver provides “via such rules” insights into the functioning of the biological neurons being modeled. In the tutorial, we will present basic functionalities of the system and demonstrate how to analyze the results returned by the software. We will allocate enough time for Q&A and if participants bring a laptop pre-loaded with Matlab, they can follow some of our examples.

References:

- [1] Astrid A. Prinz, Cyrus P. Billimoria, and Eve Marder. Alternative to hand-tuning conductance-based models: Construction and analysis of databases of model neurons. *J Neurophysiol*, 90:3998–4015, 2003.
- [2] Cengiz Günay, Jeremy R. Edgerton, and Dieter Jaeger. Channel density distributions explain spiking variability in the globus pallidus: A combined physiology and computer simulation database approach. *J. Neurosci.*, 28(30):7476–91, July 2008.
- [3] Pablo Achard and Erik De Schutter. Complex parameter landscape for a complex neuron model. *PLoS Comput Biol*, 2(7):794–804, Jul 2006.
- [4] Tomasz G. Smolinski and Astrid A. Prinz. Computational intelligence in modeling of biological neurons: A case study of an invertebrate pacemaker neuron. In *Proceedings of the International Joint Conference on Neural Networks*, pages 2964–2970, Atlanta, GA, 2009.
- [5] Tomasz G. Smolinski and Astrid A. Prinz. Multi-objective evolutionary algorithms for model neuron parameter value selection matching biological behavior under different simulation scenarios. *BMC Neuroscience*, 10(Suppl 1):P260, 2009.
- [6] Damon G. Lamb and Ronald L. Calabrese. Correlated conductance parameters in leech heart motor neurons contribute to motor pattern formation. *PLoS One*, 8(11):e79267, 2013.

- [7] Cengiz Günay, Jeremy R. Edgerton, Su Li, Thomas Sangrey, Astrid A. Prinz, and Dieter Jaeger. Database analysis of simulated and recorded electrophysiological datasets with PANDORA's Toolbox. *Neuroinformatics*, 7(2):93–111, 2009.
- [8] Cengiz Günay and Astrid A. Prinz. Model calcium sensors for network homeostasis: Sensor and readout parameter analysis from a database of model neuronal networks. *J Neurosci*, 30:1686–1698, Feb 2010. NIHMS176368,PMC2851246.
- [9] Anca Doloc-Mihu and Ronald L. Calabrese. A database of computational models of a half-center oscillator for analyzing how neuronal parameters influence network activity. *J Biol Phys*, 37(3):263–283, Jun 2011.
- [10] Emlyne Forren, Myles Johnson-Gray, Parth Patel, and Tomasz G. Smolinski. Nervolver: a computational intelligence-based system for automated construction, tuning, and analysis of neuronal models. *BMC Neuroscience*, 13(Suppl 1):P36, 2012.

Model and Software Links:

- Rat globus pallidus neuron model
(<https://senselab.med.yale.edu/modeldb/ShowModel.asp?model=114639>)
- Lobster stomatogastric ganglion pyloric network model
(<http://senselab.med.yale.edu/ModelDB/showmodel.asp?model=144387>)
- Half-center oscillator database of leech heart interneuron model
(<http://senselab.med.yale.edu/ModelDB/ShowModel.asp?model=144518>)
- PANDORA Matlab Toolbox
(<http://software.incf.org/software/pandora>)
- Parallel parameter search scripts for simulating neuron models
(<https://github.com/cengique/param-search-neuro>)
- Half-Center Oscillator model database (HCO-db)
(http://www.biology.emory.edu/research/Calabrese/hco-db/hcoDB_Main.html)

Invited Presentations



Chris Eliasmith

Dept of Philosophy

Dept of Systems Design Engineering

*Canada Research Chair in Theoretical Neuroscience,
University of Waterloo, ON, Canada*

K1 – How to build large, multi-scale, functional brain models

Recently, several large-scale brain models have been presented, including those from the Human Brain Project and IBM's Synapse Project. However, these large, complex models do not exhibit interesting psychological (i.e., motor, perceptual, and cognitive) behaviors. Consequently, they are difficult to compare to much of what we know about the brain. In this talk, I describe the methods (e.g., the Neural Engineering Framework) and tools (e.g., Nengo (<http://nengo.ca>)) used to construct what is currently the largest *functional* brain simulation. This model is called the Semantic Pointer Architecture Unified Network (Spaun) and uses 2.4 million spiking neurons organized to respect known anatomical and physiological constraints. I demonstrate the variety of behaviors the model exhibits and show that it is similar in many respects to human and animal behaviour. I show how Spaun allows comparison of the model to data across scales and across measurement modalities (e.g. spike trains, reaction times, error rates). I argue that constructing such large-scale simulations that permit this broad range of comparison to data is critical for advancing our understanding of neural and cognitive function, and I suggest that it helps to unify our understanding of how the mind works.



Christof Koch

*Allen Institute for Brain Science,
Seattle, WA, USA*

K2 – Exploring Cortex in a High-Throughput Manner by Building Brain Observatories

The *Allen Institute for Brain Science* has, over the past ten years, produced a series of brain atlases (www.brain-map.org). These are large (3 TB, >1 million slides) public resources, integrating genome-wide gene expression, and neuroanatomical data across the entire brain for developing and adult humans, non-human primates and mice, complemented by high-resolution, cellular-based anatomical connectivity data in several thousand mice. It is the single largest integrated neuroscience database world-wide. Anybody can freely access this data without any restrictions.

We are embarked on an ambitious 10-year initiative to understand the structure and function of the neocortex and associated satellite structures in humans and mice. We are setting up high through-put pipelines to exhaustively characterize the morphology, electrophysiology and transcriptome of cell types as well as their synaptic interconnections in the human neocortex (via a combination of fetal, neurosurgical and post-mortem tissues & human stem cells differentiated into forebrain neurons) and in the laboratory mouse. We are building brain observatories to image the activities of neurons throughout the cortico-thalamic system in behaving mice, to record their electrical activities, and to analyze their connectivity at the ultra-structural level. We are constructing biophysically detailed as well as simplified computer simulations of these networks and of their information processing capabilities. In keeping with the Allen Institute for Brain Science's core value of open science, all data, knowledge and tools from this initiative will be shared with the broader scientific community.



Henry Markram
EPFL,
Lausanne, Switzerland

K3 – In silico Neuroscience: the next era

I claim that mapping the brain using only experimental approaches is intractable and provide a predictive, algorithmic reconstruction strategy that uses sparse data as a companion approach to make it tractable. A first draft detailed anatomical and physiological map of a prototypical neocortical microcircuit will be presented. The microcircuit is 0.28 mm^3 in volume and contains 31,000 neurons belonging to 55 morphological neuron types and 207 morpho-electrical sub-types distributed across 6 layers. The reconstruction predicts: the number of neurons of each type per layer (*the neurome*) with around 7.5 million intrinsic and 27 million extrinsic connections forming around 40 and 141 million synapses, respectively (*the connectome*); the detailed anatomy and physiology of 2,258 unique synaptic pathways between neurons of different morphological types, 31,628 unique pathways between neurons of different morpho-electrical types, 600 intra-laminar and 1,658 inter-laminar pathways; and the complete map of intrinsic synapses for all neurons (*their synaptomes*). *In silico* simulations of the reconstructed microcircuit provide novel, simple and standardized measures of microcircuit behaviour and the computational role of any component of the microcircuit. The study demonstrates that a dense structural and functional mapping of the brain is in principle tractable and officially marks the beginning of the era of *in silico* neuroscience.



Frances K Skinner

*Toronto Western Research Institute
University Health Network,
and University of Toronto,
Toronto, ON, Canada*

K4 – Balancing and Tight Coupling: An approach to determine dynamic mechanisms of biological brain networks

Without a doubt, it is an extremely challenging endeavour to understand how our brains work. Oscillatory output, as produced by brain networks, has been shown to be important for brain functioning. Due to the high degree of sophistication and technical expertise required in experimentation, modeling, computation and analyses, it is clear that to move forth in our understanding, open and interactive collaborations between several individuals and disciplines are required.

In this talk, I will discuss our developing approach to determine essential features and mechanisms for the generation of rhythmic, population output in microcircuits of the hippocampus. Due to its importance in learning and memory, as well as its association with pathological conditions, the hippocampus is a heavily studied brain structure. Furthermore, evidence is accumulating that pathological states are associated with particular changes in normal rhythmic activities. Through collaborative efforts, we have developed and are developing cellular and network models with tight experimental linkages. We are using them to identify critical cellular and synaptic aspects of dynamic mechanisms that can be examined in biological microcircuits. Overall, we aim to use our models to determine dynamic mechanisms used by biological microcircuits (from which one could consider building macrocircuits) and to use them to gain insight into disease mechanisms.

Contributed Talks

F1 **Optical coactivation in cortical cells: reprogramming the excitation-inhibition balancing act to control neuronal gain in abstract and detailed models**

Sarah Jarvis^{1*}, Konstantin Nikolic^{2,3}, and Simon R Schultz³

¹*Department of Bioengineering, Imperial College, London SW7 2AZ UK*

²*Institute of Biomedical Engineering, Department of Electrical and Electronic Engineering, Imperial College, London SW7 2AZ UK*

The interplay of excitatory and inhibitory activity in neuronal populations is finely regulated within cortical layers, with their imbalance being heavily implicated as the underlying cause for many neurological disorders, such as autism, schizophrenia and epilepsy. A key regulatory mechanism is gain modulation, in which the amplitude of response changes while the cell's selectivity remains unaffected. Previous work has addressed gain modulation by examining the interplay of excitatory and inhibitory input at the soma [1]. However, given the non-linear integration that occurs in dendritic arbors, it remains unclear how gain is modulated when the input is located at synaptic locations.

For investigating and manipulating this balance of activity throughout the entire neuronal morphology, optogenetics is a powerful tool due to the fine temporal and spatial precision it provides [2]. Furthermore, due to the development of excitatory opsins, such as Channelrhodopsin-2 (ChR2), that depolarize neuronal membrane and silencing opsins, including halorhodopsin (NpHR), that hyperpolarize the membrane, disjoint subdomains of the dendritic and soma morphology can be targeted. This capability has recently been furthered by the development of co-activated opsins, such as ChR2-NpHR [3], which allow independent excitation and inhibition within the same neural population due to the different preferential excitation wavelengths of each opsin ($\lambda = 490, 585\text{nm}$ for ChR2 and NpHR respectively). Together, these opsins provide a potential window through which to examine the interplay of competing excitatory and inhibitory inputs for differing spatial and temporal patterns of activation.

We demonstrated previously that gain modulation in a detailed model of a Layer 5 Pyramidal cell using co-activated opsins is possible but highly dependent on the dendritic subdomains targeted [4,5], with whole cell illumination necessary to illicit gain modulation. In contrast, partial illumination of only the apical dendrites and soma resulted in no gain modulation. This suggests a strong link between potential for gain modulation and neuron morphology. While this result helps to untangle the relative contribution of excitatory and inhibitory influences, and warns of inadvertent errors when shallow illumination occurs experimentally.

We investigate this relation by first testing optical activation in abstracted neuron morphologies that include models of ChR2 and NpHR. By creating a family of neural morphologies that extend a simple ball-and-stick neuron model, we investigate how uni-, bi- and multi-polar neurons vary gain modulation upon partial illumination. External driving input is provided as both current injection and as multiple synaptic-like events at locations on dendrites, rather than the soma, to mimic input conditions for both *in vitro* and *in vivo* experiments. Using these models, we identify optimal illumination strategies for each morphological class of neuron, and predict how robust neuronal response is upon partial illumination. Finally, we test detailed neuron morphologies, including stellate interneurons, to test the predictions made by our abstract models.

Our results highlight the role of dendritic subdomains and the localized contribution of excitatory and inhibitory activity in gain modulation. Importantly, our model allows us to predict experimental illumination strategies that are tailored to neuronal morphology and are robust to any limitations that can occur experimentally.

Acknowledgements

This work was supported by Wellcome Trust grant 097816/Z/11/A. SJJ is supported by Marie Curie Intra-European Fellowship within the 7th European Community Framework Programme.

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F2 Network community, clusters and hubs in cortical micro circuits

Masanori Shimono^{1,2*}, John M Beggs²

¹*JSPS Fellow,*

²*Department of Physics, University of Indiana, Bloomington, IN, 47405, USA*

Networks of cortical neurons are essentially non-random [1]. Although it is known that such networks show interesting structure at multiple temporal and spatial scales [2], almost no experimental work has been done to reveal how structures at these different scales relate to each other.

This study aimed to clarify important relations between non-randomness in groups of 3-6 neurons (clusters) and non-randomness in groups of 50-100 neurons (communities) through five steps. First, we recorded spontaneous activity of up to 500 neurons from rodent somatosensory cortex using a 512ch. multi-electrode system over one hour [3]. Second, we reconstructed effective connectivity using transfer entropy [4]. Third, we compared topologies of effective networks at the 3-6 neuron scale (clusters including motifs [Figure1-B]) with topologies of synaptic connections measured from 12 neuron simultaneous patch clamp experiments [5,6]. Fourth, we constructed community or modular structures representing non-randomness from larger groups of neurons [Figure1-D]. Fifth, we evaluated the extent to which structure at each of these scales was robust. We did this by swapping connections from high degree nodes (hubs) with those from low degree nodes (non-hubs).

We found three things. First, the degree-distribution followed a power-law. This demonstrated that hubs could not have been the result of random sampling from a Gaussian distribution. Second, effective networks consisting of hundreds of cortical neurons have distinctive non-random structures of connectivity at two different scales. Third, structure at the cluster level was relatively more fragile than structure at the community level. The difference between non-randomness evaluated by cluster and community will become the important first step to understand multiple different scales of cortical neuronal networks.

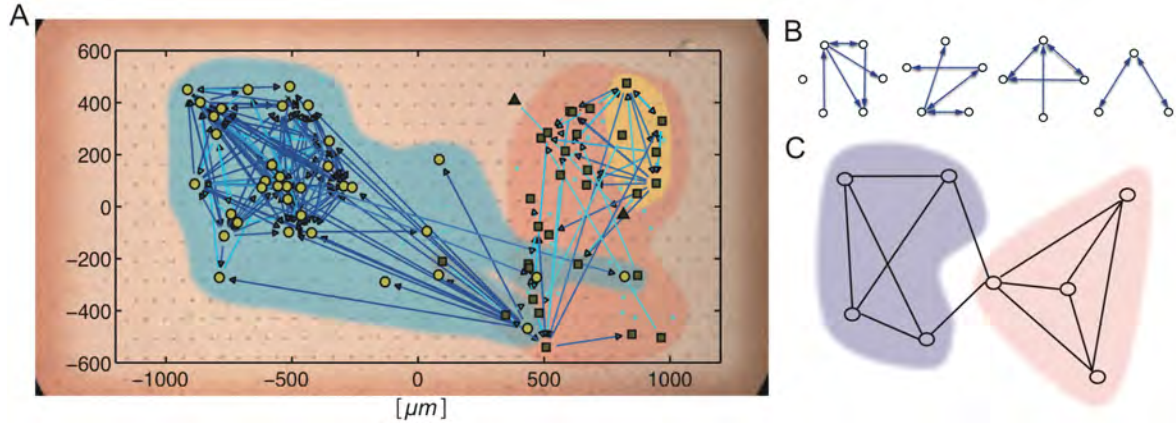


Figure 1: (A) An example of spatial distribution of neurons and effective connections. Different markers indicate different communities. The biggest two communities are covered by blue and red regions. Upper-right yellow region is an example cluster of 6 neurons. (B) Examples of clusters of 3-6 neurons. (C) An illustration of community structures. Connections are relatively denser among neurons within each community and sparser between neurons in different communities.

Acknowledgements

The authors are grateful to Olaf Sporns for important suggestions, to Rodrigo de Campos Perin in the Henry Markram team at EPFL for essential advices, and to Alan Litke, Fang-Chin Yeh, Shinya Ito, Pawel Hottowy and Deborah Gunning for their all supports to accomplish this study. This study was supported by a Grant-in-Aid for JSPS Fellows for Research Abroad.

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F3 Inhibitory single neuron control of seizures and epileptic traveling waves in humans

Omar Ahmed^{1*}, Mark Kramer⁷, Wilson Truccolo⁵, Jason Naftulin¹, Nicholas Potter⁴, Emad Eskandar², Garth Cosgrove³, Andy Blum⁴, Leigh Hochberg^{1,6}, and Sydney Cash¹

¹*Dept. of Neurology, Harvard Medical School and Massachusetts General Hospital, Boston, MA 02114*

²*Dept. of Neurosurgery, Harvard Medical School and Massachusetts General Hospital, Boston, MA 02114*

³*Dept. of Neurosurgery, Rhode Island Hospital, Providence, RI 02903*

⁴*Dept. of Neurology, Rhode Island Hospital, Providence, RI 02903*

⁵*Dept. of Neuroscience, Brown University, Providence, RI 02912*

⁶*School of Engineering, Brown University, Providence, RI 02912*

⁷*Dept. of Mathematics & Statistics, Boston University, MA 02215*

Inhibitory neuronal activity is critical for the normal functioning of the brain, but is thought to go awry during neurological disorders such as epilepsy. Animal models have suggested both decreased and increased inhibition as possible initiators of epileptic activity, but it is not known if, or how, human inhibitory neurons shape seizures. Here, using large-scale recordings of neocortical single neurons in patients with secondarily generalized tonic-clonic seizures, we show that fast-spiking (FS) inhibitory activity first increases as a seizure spreads across the neocortex, impeding and altering the spatial flow of fast epileptic traveling waves. Unexpectedly, however, FS cells cease firing less than half-way through a seizure. We use biophysically-realistic computational models to show that this cessation is due to FS cells entering depolarization block as a result of extracellular potassium accumulation during the seizure and not because they are inhibited by other inhibitory subtypes. Strikingly, this absence of FS inhibitory activity is accompanied by dramatic increases in local seizure amplitude along with unobstructed traveling waves and is seen during all secondarily generalized seizures examined, independent of etiology or focus. FS cessation also leads to prominent spike-and-wave events, suggesting that FS cell dynamics control the transition between the tonic and clonic phases of these seizures. Thus, it may be possible to curtail human seizures by preventing inhibitory neurons from entering potassium-dependent depolarization block, a novel and potentially powerful therapeutic avenue in treating multiple kinds of epilepsies.

Acknowledgements

We would like to thank the patient volunteers. This work was supported by postdoctoral fellowships from the Epilepsy Foundation (222178), MGH (2012A052031) & NINDS (F32-NS083208) awarded to OJA and by R01-NS062092 (SSC), R01-NS072023 (MAK) and R01-NS079533 (WT).

O1 Simulating spiking neural networks on massively parallel graphical processing units using a code generation approach with GeNN

Esin Yavuz*, James Turner, and Thomas Nowotny

CCNR, School of Engineering and Informatics, University of Sussex, Falmer, Brighton, BN1 9QJ, UK

A major challenge in computational neuroscience is to achieve high performance for real-time simulations of full size brain networks. Recent advances in GPU technology provide massively parallel, low-cost and efficient hardware that is widely available on the computer market. However, the comparatively low-level programming that is necessary to create an efficient GPU-compatible implementation of neuronal network simulations can be challenging, even for otherwise experienced programmers. To resolve this problem a number of tools for simulating spiking neural networks (SNN) on GPUs have been developed [1,2], but using a particular simulator usually comes with restrictions to particular supported neuron models, synapse models or connectivity schemes. Besides being inconvenient, this can unduly influence the path of scientific enquiry.

Here we present GeNN (GPU enhance neuronal networks), which builds on NVIDIA's common unified device architecture (CUDA) to enable a more flexible framework. CUDA allows programmers to write C-like code and execute it on NVIDIA's massively parallel GPUs. However, in order to achieve good performance, it is critical but not trivial to make the right choices on how to parallelize a computational problem, organize its data in memory and optimize the memory access patterns. GeNN is based on the idea that much of this optimization can be cast into heuristics that allow the GeNN meta-compiler to generate optimized GPU code from a basic description of the neuronal network model in a minimal domain specific language of C function calls. For further simplification, this description may also be obtained by translating variables, dynamical equations and parameters from an external simulator into GeNN input files. We are developing this approach for the Brian 2 [3] and SpineCreator/SpineML [4] systems.

Using a code generation approach in GeNN has important advantages: 1. A large number of different neuron and synapse models can be provided without performance losses in the final simulation code. 2. The generated simulator code can be optimized for the available GPU hardware and for the specific model. 3. The framework is intrinsically extensible: New GPU optimization strategies, including strategies of other simulators, can be added in the generated code for situations where they are effective. The first release version of GeNN is available at <http://sourceforge.net/projects/genn>. It has been built and optimized for simulating neuronal networks with an anatomical structure (separate neuron populations with sparse or dense connection patterns with the possibility to use some common learning rules).

We have executed performance and scalability tests on an NVIDIA Tesla C2070 GPU with an Intel Xeon(R) E5-2609 CPU running Ubuntu 12.04 LTS. Our results show that as the network size increases, GPU simulations never fail to outperform CPU simulations. But we are also able to demonstrate the performance limits of using GPUs with GeNN under different scenarios of network connectivity, learning rules and simulation parameters, confirming the that GPU acceleration can differ largely depending on the particular details of the model of interest.

Acknowledgements

This project is supported by the EPSRC (Green Brain Project, grant number EP/J019690/1).

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O2 Patterns in network activity and information processing in a detailed computer model of the cerebellar granular layer

Shyam Kumar^{1,2}, Sungho Hong^{1*}, and Erik De Schutter^{1,2}

¹*Computational Neuroscience Unit, Okinawa Institute of Science and Technology, Onna-son, Okinawa 904-0895, Japan*

²*Department of Theoretical Neurobiology and Neuroengineering, University of Antwerp, Wilrijk, Belgium 2610*

In the cerebellar cortex, the granular layer is at the first stage of processing information from other brain regions delivered via mossy fibers. The major components of this neural network are numerous and tiny granule cells (GrC), which are the only excitatory neurons, and Golgi cells (GoC) that provide the only inhibitory inputs to GrCs. Despite such structural simplicity, many questions about their functions remain unanswered.

Here we investigate the signal transformation property of the granular layer neural network with our three-dimensional large-scale and detailed computer model, composed of the biophysically detailed 8 x 10⁵ GrC and 2000 GoC models with their physiological synaptic and electrical connectivity. With background and constant mossy fiber inputs, the model shows network-wide oscillations driven by the synchronized GoC firing, as in previous simulation and experimental studies [1-3]. Oscillation frequency was usually higher than the Golgi cell-firing rate, as some GoCs exhibited cycle skipping. With more physiological and diverse paradigms of mossy fiber stimulation, we could observe interesting patterns which hint that this oscillation and rate coding synergistically contribute to the network outputs. For example, when the mossy fiber stimulation is spatially limited, there is anti-correlation in the GrC spike count between the stimulated and unstimulated region, suggesting center-surround “receptive fields” [4], while the oscillation persists and opens time windows for well-timed spikes [5] (Fig. 1). Our results suggest how the complex dynamics of the granular layer network due to cellular and synaptic properties can contribute to its rich information processing.

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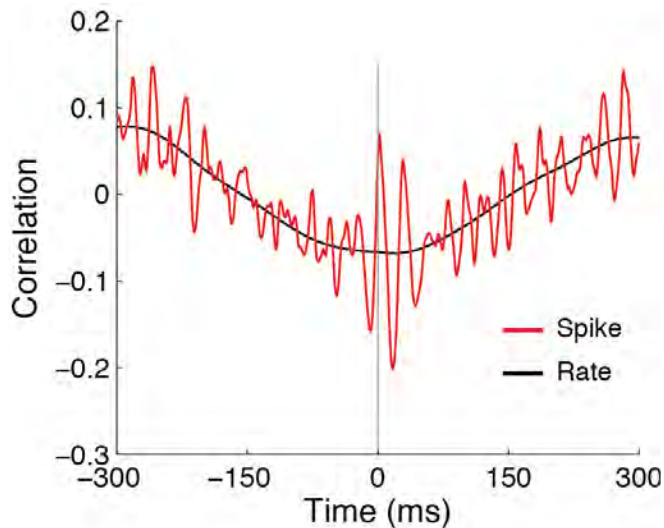


Figure 1: Cross-correlogram between the average GrC activity within the stimulated and unstimulated region (diameter of each region = $200 \mu\text{m}$, distance = $500 \mu\text{m}$). The stimulated region was given high frequency mossy fiber input of 20 Hz and 50 Hz, each lasting 300 ms. The spike trains were formed with a 1 ms bin, while the rate is evaluated with a 100 ms time window.

O3 Mean Field Analysis Gives Accurate Predictions of the Behaviour of Large Networks of Sparsely Coupled and Heterogeneous Neurons

Wilten Nicola^{1*}, Felix Njap^{1,2}, Katie Ferguson^{2,4}, Frances Skinner^{2,3,4}, and Sue Ann Campbell¹

¹*Department of Applied Mathematics & Center for Theoretical Neuroscience, University of Waterloo, Waterloo, ON, Canada*

²*Division of Fundamental Neurobiology, Toronto Western Research Institute, Toronto, ON, Canada*

³*Department of Medicine (Neurology), University of Toronto, Toronto, ON, Canada*

⁴*Department of Physiology, University of Toronto, Toronto, ON, Canada*

Large networks of integrate-and-fire (IF) model neurons are often used to simulate and study the behaviour of biologically realistic networks. However, to fully study the large network behaviour requires an exploration of large regions of a multidimensional parameter space. Such exploration is generally not feasible with large network models, due to the computational time required to simulate a network with biologically significant size. To circumvent these difficulties we use a mean-field approach, based on the work of [1].

We consider a sparsely coupled, excitatory network of 10,000 Izhikevich model neurons [2], with Destexhe-type synapses [3]. The cellular models were fit to hippocampal CA1 pyramidal neurons and have heterogeneous applied currents with a normal distribution. We derived a mean-field system for the network which consists of differential equations for the mean of the adaptation current and the synaptic conductance.

As CA1 is an area that displays prominent theta oscillations [4], we used the mean-field system to study how the frequency of bursting depends on various model parameters. Figure 1A

shows an example study. These studies were successful in guiding numerical simulations of the large network. When parameter values determined from the mean-field analysis are used in a large network simulation, bursting of the predicted frequency occurs (Figure 1B).

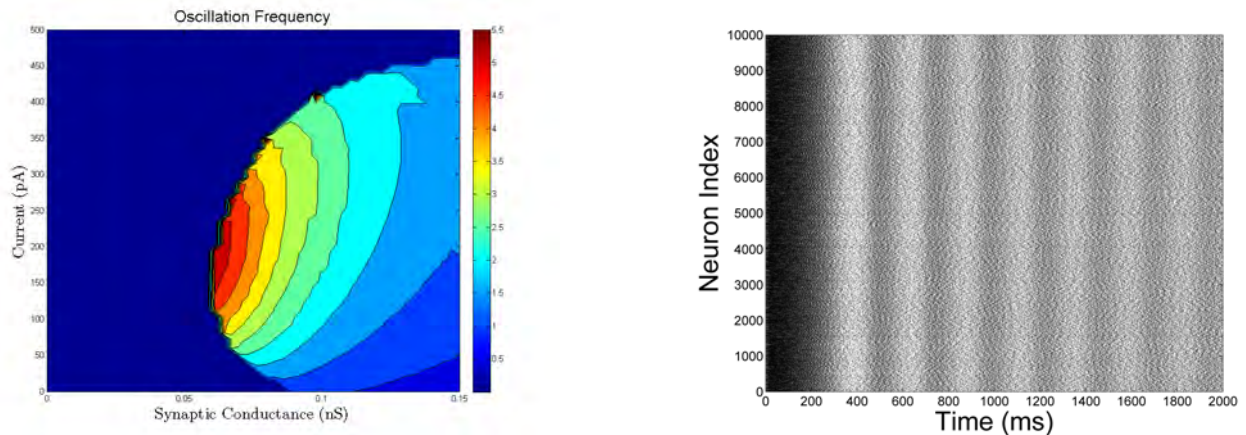


Figure 1: (Left) Mean-field prediction of the bursting frequency as a function of the unitary synaptic conductance and the mean applied current. (Right) Simulation of a network of 10,000 neurons with unitary conductance 0.058 nS and mean current 250 pA, showing an oscillation in the theta frequency range as predicted by the mean-field system of equations.

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O4 Extracting novel information from neuroimaging data using neural fields

Dimitris Pinotsis*

The Wellcome Trust Center for Neuroimaging, University College London, WC1N 3BG, UK

This talk will introduce new links between the theory of differential equations and the analysis of neuroimaging data. We will focus on a class of population models called neural fields: these are models of how the brain is wired [1] and how it responds in different experimental conditions, which embody topographic features of cortical sources [2]. We will demonstrate how neural fields can be used to interpret brain responses measured with electrophysiology [3]. The inversion of such models is based upon Bayesian techniques and provides estimates of biologically and functionally meaningful quantities among different experimental conditions.

Neural fields model current fluxes as continuous processes on the cortical sheet, using partial

differential equations (PDEs). The key advance that neural field models offer, over other population models (like neural masses), is that they embody spatial parameters (like the density and extent of lateral connections). This allows one to model responses not just in time but also over space. Conversely, these models are particularly useful for explaining observed cortical responses over different spatial scales; for example, with high-density recordings, at the epidural or intracortical level. However, the impact of spatially extensive dynamics is not restricted to expression over space but can also have profound effects on temporal (e.g., spectral) responses at one point (or averaged locally over the cortical surface)[4]. This means that neural field models may also play a key role in the modelling of non-invasive electrophysiological data that does not resolve spatial activity directly.

We will shed light on different uses of neural fields and put forward three reasons why these models can be useful in the analysis of neuroimaging data. Each of these motivations is demonstrated by analysing a particular dataset obtained using three different modalities: electrocorticography (ECoG), magnetoencephalography (MEG) and local field potential recordings (LFPs). We will argue that neural fields allow one to: (i) compare evidences for alternative hypotheses regarding the important neurobiological determinants of stimulus-specific response variability[5]; (ii) make inferences about between subject variability in cortical function and microstructure using non-invasive data [6] and (iii) obtain estimates of spatial parameters describing cortical sources in the absence of spatially resolved data [7].

Our analyses exploit dynamic causal modelling [8] and include model space explorations that embody different hypotheses about the generation of observed responses in relation to model evidence - obtained using Variational Bayes [9]. This model comparison uses a variational free-energy bound to furnish optimized models in a manner similar to fitting empirical spectra with AR and ARMA models, see e.g.[10]. The advantage this approach has over other optimization criteria is that it provides an optimal balance between model fit and complexity; yielding models that are both parsimonious and accurate. The analyses presented here showcase particular instances where neural field models serve as a mathematical microscope, allowing us to extract information that is hidden in electrophysiological data.

Acknowledgements

The Wellcome Trust funded this work. The author is grateful to Prof K Friston, FRS, for valuable support and enlightening discussions.

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O5 Automatic fitness function selection for compartment model optimization

Timothy Rumbell^{1*}, Danel Draguljic², Jennifer Luebke³, Patrick Hof¹, and Christina M Weaver²

¹*Fishberg Department of Neuroscience and Friedman Brain Institute, Icahn School of Medicine at Mount Sinai, New York, NY 10029*

²*Department of Mathematics, Franklin and Marshall College, Lancaster, PA, 17604*

³*Department of Anatomy and Neurobiology, Boston University School of Medicine, Boston, MA, 02118*

During normal aging, layer 3 pyramidal neurons of the rhesus monkey prefrontal cortex (PFC) exhibit significant morphological changes, as well as higher action potential firing rates in vitro [1]. Computational modeling of individual neurons can provide insight into the ionic mechanisms underlying the increased excitability, which are currently unknown. A unique database of electrophysiological recordings and morphologic reconstructions from the same neurons, gathered through whole-cell patch clamp recording, confocal microscopy and 3D digital tracing, constrains the models. Initial modeling of six young and six aged neurons demonstrated that morphological features alone do not account entirely for the electrophysiological changes with aging [2]. It is now necessary to explore the parameter space of passive cable properties and active membrane channel conductances and kinetics, to uncover parameter combinations that reproduce the firing patterns observed in neurons of each age group.

Differential Evolution (DE) is an evolutionary optimization method capable of identifying a population of candidate models throughout parameter space that closely match empirically observed firing patterns. The quality of fit achieved by an optimization is reliant on the ‘fitness functions’ used to measure the accuracy of the model. Previous neuronal compartment modeling studies using parameter optimization have introduced multiple types of fitness measurement [3], but have not described a general method to determine weights for each type. Here we introduce a novel method for automatically establishing weights of minimally correlated fitness functions, and apply it to optimization of models of young and aged PFC neurons. First, a Latin hypercube design (< 1000 points) provides a space-filling sampling of parameter space; the candidate fitness functions are then evaluated at each of these points. Second, clusters of fitness functions that are highly correlated across the hypercube are pruned to leave one representative member. Third, a principal component analysis of the remaining fitness functions across the hypercube identifies a set of fitness functions representing most of the variability in the parameter space, which are selected for use in the optimization. Fourth, weights for each selected fitness function are calculated based on the combination of coefficients for principal components and variance explained by those components. Finally, DE is conducted on the Neuroscience Gateway [4] using this automatically constructed optimization protocol.

We demonstrate the method with a compartment model comprising a simplified pyramidal neuron morphology and three ion channels, optimized to data from representative young and aged neurons. Compared to a manual approach involving iterative generation of fitness functions, our novel method produces better fitting models using a tenth of the computation time. Future

work will extend the automatic protocol generation to prioritize which parameters to optimize, a critical step as more ion channels are added to the model to improve fitness. This method will be used to generate morphologically detailed models of 20+ young and aged PFC neurons, predicting which ionic mechanisms underlie age-related physiological changes.

Acknowledgements

This project was supported by NIH grants AG00001, AG025062, and AG035071.

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O6 Predicting neural responses to natural sound in the auditory brainstem

Dominika Lyzwa^{1,2*}, J Michael Herrmann²

¹*Dept. of Nonlinear Dynamics, Max Planck Inst. for Dynamics and Self-Organization, Göttingen, 37077, Germany*

²*Institute of Perception, Action and Behaviour, University of Edinburgh, Edinburgh, EH 8 9AB, UK*

The inferior colliculus is the main processing station in the auditory midbrain and integrates projections from nearly all ascending brainstem nuclei. Apart from being a converging station, the central nucleus of the inferior colliculus (ICC) is essential for extracting time-varying spectrotemporal information [1] and therefore might be important for processing complex sounds such as speech and vocalizations. The ICC has been the target for a human auditory prosthesis [2], which might benefit from model predictions of the neural response in the ICC to incoming sound. Natural sounds such as speech and vocalizations, which display a wide spectrum of acoustic properties, such as harmonics, correlations, amplitude and frequency modulations and are very well suited to study the auditory system.

This study is based on several sets of multi-unit activity recorded simultaneously from 32 sites in the contralateral ICC of guinea pigs while acoustically presenting a diverse set of conspecific vocalizations to the right ear. Recordings were taken either along the tonotopic gradient using double-shank electrodes or within iso-frequency lamina using double-tetrode electrodes. We investigated predictive power of several models of temporal responses in the ICC to vocalizations and artificial sound. The tested models include 1) a modified version of the physiologically detailed Meddis Model [4], which was altered in order to match spiking threshold in the guinea pig ICC and to include adaptation effects and output the trial-averaged spiking responses, the peri-stimulus time histograms (PSTH), 2) a generalized linear model and 3) a filtering model

with a bandpass filter of 1/3 octaves around the best frequency, with subsequent normalization and rectification for each unit, followed by spatial filtering for nearby units. Predictive power was evaluated by means of the correlation value of the envelope of the PSTHs from the predicted and the experimentally obtained responses.

We find that our relatively simple, filtering approach yields surprisingly good overlap of predicted and measured responses for some multi-units, but has poor predictive power for other units. The models (1-2) yield overall better overlap for the whole set of vocalizations but do not perform optimally in predicting the temporal course of the response. Our findings indicate distributions of optimal predictive power in the inferior colliculus over a large best frequency range across and within isofrequency laminae.

Acknowledgements

This work was supported by the BMBF in the National Network for Computational Neuroscience, grant number #01GQ0811 to BFNT Göttingen. We would like to thank Thilo Rode, Tanja Hartmann, and Hugh H. Lim for the guinea pig recordings and vocalizations.

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O7 Causal correlation paths across cortical areas in decision making

Adrià Tauste Campo^{1*}, Marina Martinez-Garcia¹, Verónica Nácher², Gustavo Deco¹, and Ranulfo Romo²

¹*Department of Information and Communication Technologies, Universitat Pompeu Fabra, Barcelona, 08018 Spain*

²*Instituto de Fisiología Celular-Neurociencias, Universidad Nacional Autónoma de México, 04510 México D.F., México*

We study how neural spike activity encode, integrate and communicate information across different brain areas. An ideal paradigm to study this problem is the vibrotactile discrimination task designed by Romo et al. [1]. This is a complex process, which requires communicating information from the sensory areas that perceive the tactile stimuli to superior areas that integrate this sensory information and report the decision. Previous works on this task have characterized the role played by sensory and motor areas using the correlation between single-neuron rate responses and the task variables, namely the two stimulation frequencies and the decision [2]. In the present work, we investigate the causal correlations that arise between nearby and distant cells while the monkey is performing the task under fixed stimulation frequencies.

To this end, we use simultaneous multiple-cell recordings to estimate causal across five cortical areas (S1, S2, SMA, DPC and M1) over the time course of the discrimination task. Causal

correlations are estimated with a sequential universal estimator of the directed information based on the context-tree weighting algorithm [3][4]. Statistical tests on the estimates for four stimulation frequency pairs suggest that significant causal correlations ('causal paths') are highly distributed across the studied cortical areas and are equally present in feedforward and feedback interactions between sensory and motor areas. Furthermore, the percentage of incoming causal paths is steady during the time course of the task for destination areas S2, SMA, DPC and M1 while it decays during the stimulation periods for S1. The task-specificity of these results is assessed by a control task, where the monkey receives both stimuli but it is requested not to perform the task. Specifically, during the passive stimulation task there is an abrupt decrease in the number of causal correlations after the first stimulation, which is shown to be independent of the spike-train variability of each area.

Conclusions: Neuronal causal correlation paths that are specific to the discriminations task are ubiquitous, bidirectional and remain approximately constant along the task in both sensory and motor areas. These findings are robust to the stimulation pair under study and the spike-train variability of each area.

Acknowledgements

A. Tauste Campo agrees funding from the European Union under the 7th Framework Programme, grants FP7-PEOPLE-2013-IEF no. 329837.

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O8 Trial-by-trial modeling of electrophysiological signals during inverse Bayesian inference

Antonio Kolossa^{1*}, Bruno Kopp², and Tim Fingscheidt¹

¹*Institute for Communications Technology, Technische Universität Braunschweig, Braunschweig, 38106, Germany*

²*Department of Neurology, Hannover Medical School, Hannover, 30625, Germany*

Empirical support for the Bayesian brain hypothesis, although of major theoretical importance for cognitive neuroscience, is surprisingly scarce. The literature still lacks definitive functional neuroimaging evidence that neural activities code and compute Bayesian probabilities. Here, we introduce a new experimental design to relate electrophysiological measures to Bayesian inference. Specifically, an urns-and-balls paradigm was used to study neural underpinnings of probabilistic inverse inference. Event-related potentials (ERPs) were recorded from human participants who performed the urns-and-balls paradigm, and computational modeling was conducted on trial-by-trial electrophysiological signals. Five computational models were compared with respect to their capacity to predict electrophysiological measures. One Bayesian model (BAY) was compared with another Bayesian model which takes potential effects of non-linear probability weighting

into account (BAYS). A predictive surprise model (TOPS) of sequential probability revisions was derived from the Bayesian models. A comparison was made with two published models of surprise (DIF [1] and OST [2]).

Subsets of the trial-by-trial electrophysiological signals were differentially sensitive to model predictors: The anteriorly distributed N250 was best fit by the DIF model, the BAYS model provided the best fit to the anteriorly distributed P3a, whereas the posteriorly distributed P3b and Slow Wave were best fit by the TOPS model. Figure 1 shows the model fit in log-Bayes factor [3] as scalp maps for the BAYS and TOPS models for P3a and P3b time windows, respectively. Table 1 summarizes the model comparison by translating the log-Bayes factors to posterior model probabilities [4] for all models and all ERPs at the respective time windows and electrodes. These results show that dissociable cortical activities code and compute different aspects of Bayesian updating. However, these activities might be best described as being Bayes optimal, implying that they reflect Bayesian inference, modulated by non-linear probability weighting, as originally conjectured by prospect theory [5,6].

ERP waves and electrodes				
	N250	P3a	P3b	SW
Model	C4	FCz	Pz	O1
OST	0.02	<0.01	<0.01	<0.01
DIF	0.66	<0.01	<0.01	<0.01
TOP _S	0.28	<0.01	0.88	0.82
BAY	<0.01	<0.01	<0.01	<0.01
BAY _S	0.04	0.99	0.12	0.18

Table 1: Posterior model probabilities

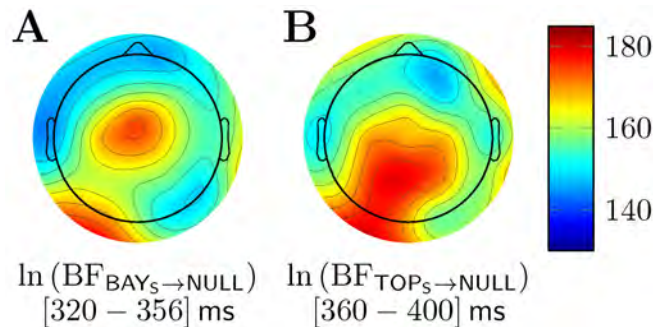


Figure 1: Scalp maps of averaged log-Bayes factors of models with non-linear probability weighting versus a null model. A. Bayesian surprise model (BAY_S). B. Predictive surprise model (TOP_S).

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O9 Parameter correlations maintaining bursting activity

Anca Doloc-Mihu*, Ronald Calabrese

Department of Biology, Emory University, Atlanta, GA, 30322, USA

In this study, we focused on the role of correlated conductances in the robust maintenance of functional bursting activity. Recent experimental and computational studies suggest that linearly correlated sets of parameters (intrinsic and synaptic properties of neurons) allow central pattern generating (CPG) neurons to produce and maintain their rhythmic activity regardless of changing internal and external conditions. However, the mechanisms that allow multiple parameters to interact, thereby producing and maintaining rhythmic network activity, are less clear.

For our study, we used our existing database (HCO-db) [1] of instances of a half center oscillator (HCO) model [2]. The HCO single-compartment conductance-based model [2] consists of two mutually inhibitory neurons and replicates the electrical activity of the oscillator interneurons of the leech heartbeat CPG under a variety of experimental conditions. From the database, we identified functional activity groups of isolated neuron and half-center oscillator (HCO) model instances and realistic subgroups of each such group that showed burst characteristics (principally period and spike frequency) similar to the animal. To find linear correlations among the conductance parameters maintaining functional leech bursting activity, we applied Principal Component Analysis (PCA) to each of these four groups. PCA identified a set of three maximal conductances (leak current, \bar{g}_{Leak} ; a persistent K current, \bar{g}_{K2} ; and a persistent Na⁺ current, \bar{g}_P) that correlate linearly for the two groups of regular and realistic isolated neuron instances (Figure 1 A). Our 3D visualizations of HCO instances (Figure 1 B) in the reduced space of \bar{g}_{Leak} , \bar{g}_{K2} , and \bar{g}_P suggested that there might be a non-linear relationships between parameters for these instances.

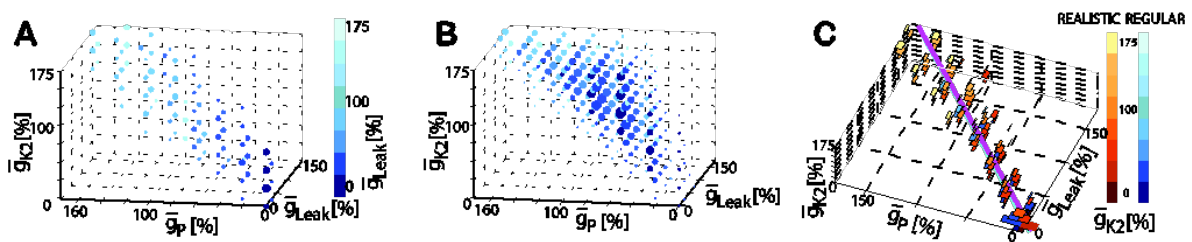


Figure 1: Plots of the groups of instances in the 3D space given by the \bar{g}_{Leak} , \bar{g}_{K2} , and \bar{g}_P maximal conductances. A. Realistic isolated neurons (83 points; 307 instances); B. Realistic HCOs (243 points; 99,066 instances); C. Realistic and regular/ not realistic isolated neurons (83 realistic vs. 91 regular points) and their ODR lines, magenta for realistic and cyan for regular.

A least square fit regression line (3D Orthogonal Distance Regression (ODR) line) to each group of isolated neurons (Figure 1 C) showed a tendency for the realistic instances to be at the high values on all axes and a tendency of the regular/not realistic instances to be at the low and middle values on all axes. From our analysis, it appears that none of the \bar{g}_{Leak} , \bar{g}_{K2} , or \bar{g}_P

parameters is sufficient by itself to produce regular and realistic isolated neuron instances, but they must work together (in linear combination) in almost equal amounts towards producing the respective instances. Experimental studies have shown that period is a key attribute influenced by modulatory inputs and temperature variations in heart interneurons. Thus, we explored the sensitivity of period to changes in maximal conductances of \bar{g}_{Leak} , \bar{g}_{K2} , and \bar{g}_P , and we found that for our realistic isolated neurons the effect of these parameters on period could not be assessed because when varied individually bursting activity was not maintained. Current studies are focused on determining which parameters can, when varied, smoothly control period, while maintaining bursting activity.

Acknowledgements

Work supported by the National Institute Health Grant NS085006 to R.L.Calabrese.

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O10 Organization of left-right coordination of neuronal activity in the mammalian spinal cord locomotor CPG: Insights from computational modeling

Ilya Rybak^{1*}, Natalia Shevtsova¹, Adolfo Talpalar², Sergey Markin¹, Ronald Harris-Warrick³, and Ole Kiehn²

¹*Department of Neurobiology and Anatomy, Drexel University College of Medicine, Philadelphia, PA 19129, USA*

²*Department of Neuroscience, Karolinska Institute, Stockholm, 17177, Sweden*

³*Department of Neurobiology and Behavior, Cornell University, Ithaca, NY 14853, USA*

Different gaits of locomotion in mammals are based on the appropriate coordination of neuronal activity in the spinal cord controlling movements of left and right limbs. This left-right coordination is provided in the spinal cord by the commissural interneurons (CINs) whose axons cross the midline and affect neural circuits on the contralateral side of the cord. Several types of CINs have been genetically identified, including the excitatory V3 CINs and the inhibitory ($V0_D$) and excitatory ($V0_V$) V0 CINs. Talpalar et al. [1] recently demonstrated that (a) ablation of both V0 CIN types leads to a left-right synchronized, “hopping” activity at all locomotor frequencies, whereas (b) selective ablation of the excitatory $V0_V$ CINs maintains alternation at low frequencies but switches to synchronized activity at high frequencies while (c) ablation of only the inhibitory $V0_D$ CINs leads to a lack of left–right alternation at low frequencies, but maintains alternation at high frequencies. The genetically identified, ipsilaterally projecting excitatory V2a interneurons are recruited with an increase in locomotor speed [2] and contribute to left-right alternation at high locomotor frequencies [3,4]. Our objective was to construct and analyze a computational model of the bilaterally interacting central pattern generators (CPGs) that could reproduce and explain these findings.

In our model, the CPG on each side of the cord consisted of flexor and extensor half-centers.

Each neural population, including the half-centers and CIN populations, consisted of 50-200 neurons modeled in the Hodgkin-Huxley style. The intrinsic bursting of CPG neurons was based on a persistent sodium current in these neurons.

During model construction, we assumed that left-right coordination of activity depends on the balance between the three CIN pathways providing interactions between the left and right CPGs: the V3-mediated pathway that supports left-right synchronization and the $V0_D$ - and $V0_V$ - mediated pathways that provide left-right alternation. The activity of each (left and right) inhibitory $V0_D$ population was driven by the ipsilateral flexor half-center. The recruitment of $V0_D$ neurons was progressively reduced with an increase in locomotor speed, because of the reduction of burst amplitude. The left and right $V0_V$ pathways could be organized in two ways: (1) the $V0_V$ activity on each side was driven by the ipsilateral flexor half-center and its action on contralateral circuits was mediated by an inhibitory population, or (2) the $V0_V$ activity was driven by the ipsilateral extensor half-center and it excited the contralateral circuits. In any case, the $V0_V$ activation was mediated by the ipsilateral V2a neurons progressively recruited with increasing locomotor speed.

The model demonstrates: (1) a left-right alternating pattern under control conditions; (2) a synchronized hopping pattern at any frequency after removing both the $V0$ populations; (3) a synchronized pattern at low frequencies with alternation at high frequencies after removing the $V0_D$ populations; (4) an alternating pattern at low frequencies with synchronized hopping at high frequencies after removing either $V0_V$ or V2a populations. The model closely reproduces and suggests an explanation for the experimental data of Talpalar et al. [1], Zhong et al. [2], and Crone et al. [3,4], proposes the organization of commissural interactions in the spinal cord defining the left-right alternation at different locomotor speeds, and generates predictions for future experimental investigations.

Acknowledgements

Supported by NIH grant R01NS7323, Swedish Research Council, and Torsten and Ragnar Söderbergs foundations.

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O11 Determinants of gain modulation enabled by short-term depression at an inhibitory cerebellar synapse.

Dimitris Bampasakis^{1*}, Reinoud Maex², Neil Davey¹, and Volker Steuber¹

¹*Science and Technology Research Institute, University of Hertfordshire, Hatfield AL10 9AB, UK*

²*Department of Cognitive Sciences, École Normale Supérieure, Paris 75005, France*

Neurons adapt rapidly the slope, also known as gain, of their input-output function to time-varying conditions. Gain modulation is a prominent mechanism in many brain processes, such as auditory processing and attention scaling of orientation tuning curves. It is known to amplify neuronal signals, prevent firing saturation, and play a key role in coordinate transformation [1].

Synaptic short-term depression (STD) at the excitatory synapse from mossy fibres (MFs) to granule cells in the cerebellum has previously been found to introduce a gain change, and enhance inhibition-mediated gain modulation [2]. Similar results were discovered for STD at the inhibitory synapse from Purkinje cells (PCs) to cerebellar nucleus (CN) neurons, where STD modulates gain and enhances excitation-mediated gain modulation [3]. In both cases – whether STD is applied at the excitatory or inhibitory synapse, respectively – the non-linearity introduced by STD in the relationship between input firing rate and average conductance, was found to underlie the effects of STD.

We use a multi-compartmental model of a cerebellar nucleus neuron [4] to understand how STD at an inhibitory synapse can add a multiplicative component in the transformation performed by excitatory input. To do so, we use input from PCs, applied at an inhibitory synapse with STD, and excitatory input from MFs, while changing the level of STD by manipulating the presynaptic release probability (R) [5]. We find that gain modulation resulting from the introduction of STD increases with the extent of depression. To further our understanding, we investigate the effects of STD using synchronous input, regular input, and their combination. We find that the multiplicative component introduced by STD remains, but varies in value for different input conditions. Moreover, we present a detailed analysis of how a non-linear mapping between input spike rate and synaptic conductance can result in multiplicative operations.

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O12 Stable reinforcement learning via temporal competition between LTP and LTD traces

Marco Huertas^{1*}, Sarah Schwettmann^{1,2}, Alfredo Kirkwood³, and Harel Shouval¹

¹University of Texas Medical School, Dep. Neurobiology and Anatomy, Houston, TX 77030, USA

²Rice University, Dep. Computational and Applied Mathematics, Houston, TX 77005, USA

³Johns Hopkins University, Mind/Brain Institute, Baltimore, MD, 21218, USA

Neuronal systems that are involved in reinforcement learning must solve the temporal credit assignment problem, i.e., how is a stimulus associated with a reward that is delayed in time? Theoretical studies [1,2,3] have postulated that neural activity underlying learning ‘tags’ synapses with an ‘eligibility trace’, and that the subsequent arrival of a reward converts the eligibility traces into actual modification of synaptic efficacies. While eligibility traces provide one simple solution to the temporal credit assignment problem, they alone do not constitute a stable learning rule because there is no other mechanism indicating when learning should cease. In order to attain stability, rules involving eligibility traces often assume that once the association is learned, further learning is prevented via an inhibition of the reward stimulus [1,3,4].

Although synaptic plasticity is responsible for reinforcement learning in the brain, theories of reinforcement learning are generally abstract and involve neither neurons nor synapses. Furthermore, biophysical theories of synaptic plasticity typically model unsupervised learning and ignore the contribution of reinforcement. Here we describe a biophysically based theory of reinforcement-modulated synaptic plasticity and postulate the existence of two eligibility traces with different temporal profiles: one corresponding to the induction of LTP, and the other to the induction of LTD. The traces have different kinetics and their difference in magnitude at the time of reward determines if synaptic modification will correspond to LTP or LTD. Due to the difference in their decay rates, the LTP and LTD traces can exhibit temporal competition at the reward time and thus provides a mechanism for stable reinforcement learning without the need to inhibit reward. We test this novel reinforcement-learning rule on an experimentally motivated model of a recurrent cortical network [5], and compare the model results to experimental results at both the cellular and circuit levels. We further suggest that these eligibility traces are implemented via kinases and phosphatases, thus accounting for results at both the cellular and system levels.

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O13 Neural graphs: Small-worlds, after all?

Michelle Rudolph-Lilith*, Lyle Muller

Unité des Neurosciences, Information et Complexité (UNIC), CNRS UPR-3293, Gif-sur-Yvette, 91198, France

In recent years, small-world graphs have gained considerable interest as models of real-world systems, which often display features residing between regularity and randomness. The most notable of these models is the Watts-Strogatz graph [1], though alternatives have been proposed [2]. The unifying characteristics of these models are that any two nodes are joined with a small number of links between them (i.e. short path length), while at the same time connected node pairs exhibit an abundance of triangular relations resulting in a high degree of local redundancy (i.e. high clustering).

Theoretical investigations of small-world graph models have generally applied asymptotic evaluations in the limit of large system size [3] or the continuum approximation [4] to the algorithmic definition of the graph, in the absence of an analytic representation. In this study, we introduce a generative model of directed small-world graphs, a canonical model of Watts-Strogatz digraphs, and propose an approach that yields the graph's defining adjacency matrix in algebraic terms, with the goal to provide mathematically rigorous access to the study of finite-size small-world graphs [5]. The proposed approach makes use of random annihilation operators whose algebraic properties can be utilized to assess algebraically well-defined graph-theoretic measures in an analytically exact framework, valid nonasymptotically for all graph sizes. We demonstrated the application of our approach by calculating, for the first time, the asymmetry index and total clustering coefficient of small worlds in an exact fashion.

We then utilize the exact nonasymptotic expression for the clustering coefficient in order to assess the small-worldness of structural brain networks in an analytic setting. Using the number of nodes and edges of the given brain networks to construct the equivalent small-world network, we observe that a significant edge rewiring of at least 20% up to 60% is required to produce the small-worldness indices observed in these networks. Importantly, the maximum of the small-worldness index however occurs in all cases at one order of magnitude lower than the required rewiring found. This result suggests that neural graphs reside far away from the small-world regime of the Watts-Strogatz model.

Acknowledgements

The authors wish to thank OD Little for comments. This work was supported by CNRS, the European Community (BrainScales Project No. FP7-269921), and École des Neurosciences de Paris Ile-de-France.

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O14 A k-population model to calculate the firing rate of neuronal networks with degree correlations

Christian Schmeltzer^{1*}, Alexandre Kihara², Igor Sokolov¹, and Sten Rüdiger¹

¹*Institut für Theoretische Physik, Humboldt Universität, Berlin, Germany, 12489, USA*

²*Universidade Federal do ABC, Santo André, Brazil*

Revealing the interplay of structure and function of the brain is one of the most intriguing topics in neuroscience. The theory of complex networks is a promising approach to this aim, where one assumes that high cognitive processes arise as emergent properties of a network, in which many inane neurons are connected by a complex topology [1]. In this regard, we analyze analytically the emerging responses of networks with increasingly complex connectivity. We present a mathematical theory to calculate the firing rate of a network of leaky integrate-and-fire neurons, taking into account network features such as degree distributions and degree correlations (Fig. 1). Heterogeneous connectivity and degree correlations have been shown to heavily influence network function and dynamics [2, 3]. Our method is to divide the neuronal network in k-populations according to the number k of afferent synaptic links that connect to the neuron. Then, the steady state firing rates for these coupled populations can be calculated self-consistently. One of our main findings is that the population heterogeneity yields substantial deviations from mean-field calculations, where one ignores the network properties [4]. Importantly, our analysis shows that networks with assortative degree correlations lead to firing patterns even for sub-threshold inputs, where an uncorrelated network would not fire and thus, to a much larger sensitivity to low stimuli (Fig. 2). Using information theory we further find an optimum in assortativity, with larger levels reducing again sensitivity for signal ensembles.

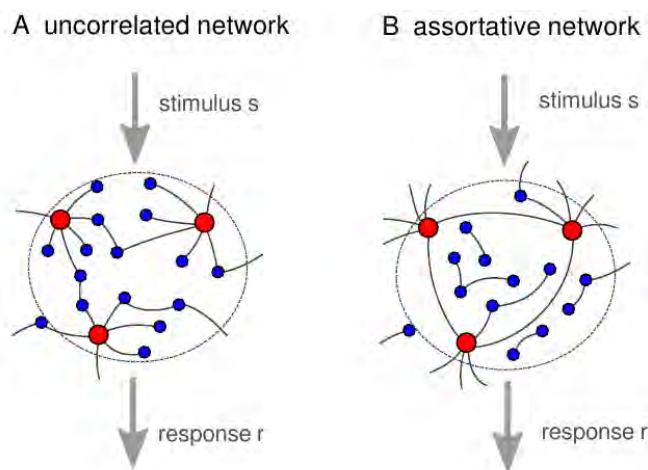


Figure 1: Schematic of the complex neural network. In the uncorrelated network (A), highly connected neurons (red dots) and poorly connected neurons (blue dots) are joined randomly. In the network with assortative degree correlations (B), neurons with similar connectivity are joined preferably. The network firing rate r is the response to a Poissonian external input current with rate s , which is injected into each neuron.

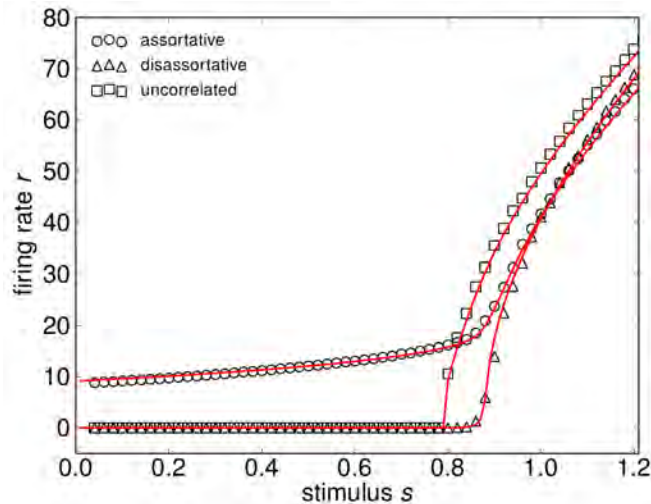


Figure 2: Firing rate of a heterogeneous network of integrate and fire neurons with in-degree correlations. Simulation results (dots) and theoretical predictions (lines). The assortative network shows sustained activity for very small stimuli.

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O15 Criticality in cortical ensembles is supported by complex functional networks

Paolo Massobrio^{1*}, Valentina Pasquale², and Sergio Martinoia¹

¹*Department of Informatics, Bioengineering, Robotics and Systems Engineering (DIBRIS), University of Genova, Genova, 16145, ITALY*

²*Department of Neuroscience and Brain Technologies - NTECH, Istituto Italiano di Tecnologia (IIT), 16163, Genova, ITALY*

Complex network topologies represent the necessary substrate to support complex brain function. It is widely recognized that the topological features of cortical networks are tightly linked to aspects of brain function by supporting which electrophysiological patterns can and cannot occur.

In this work, we investigated the interplay between network topology and spontaneous dynamics within the framework of neuronal avalanches and self-organized criticality (SOC) [1]. The main goal of this study is to sustain the hypothesis that the emergence of critical states, which in their turn would optimize functional properties in the cortex, is supported by specific complex network topologies. Experimental evidences showed that dissociated cortical assemblies coupled to Micro-Electrode Arrays (MEAs) can exhibit scale-free distributions of neuronal avalanches [2],

a hallmark of SOC, thus demonstrating that they preserve self-organization properties featured by *in vivo*-formed cell assemblies [3]. However, the determinants of the emergence of different dynamical states (critical, subcritical or supercritical) remain unclear. Here, we adopted a reverse-engineering approach, by making use of an *in silico* neuronal network model reproducing the spiking and bursting activity of biological networks to explore the relationship between connectivity and dynamics. In our computational network model, connectivity is known *a priori* and thus it is possible to establish interdependencies between the avalanche distributions and the actual connectivity. Network topologies were designed following the canonical architectures of scale-free, random, and small-world graphs [4]. We simulated the spontaneous activity, by sweeping the most common parameters used to characterize these graphs, such as clustering coefficient, connection density, synaptic weight distributions, etc. [5]. From the simulations, we found that: (i) random networks only showed super-critical dynamics in a physiologically relevant domain of activity parameters (e.g. firing rate); (ii) scale-free and small-world architectures may account for the variability observed in the experimental data and the transition from subcriticality to criticality is ruled by the degree of 'small-worldness'; (iii) excitation and inhibition should be appropriately balanced to allow for criticality [6].

Acknowledgements

The research leading to these results has received funding from the European Union's Seventh Framework Programme (ICT-FET FP7/2007-2013, FET Young Explorers scheme) under grant agreement no 284772 (BrainBow).

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O16 The interplay of intrinsic excitability and network topology in spatiotemporal pattern generation in neural networks.

James Roach^{1*}, Leonard Sander², and Michal Zochowski²

¹*Neuroscience Graduate Program, University of Michigan, Ann Arbor, MI 48109, USA*

²*Department of Physics, University of Michigan, Ann Arbor, MI 48109, USA*

It is clear that spatiotemporal patterning in brain networks is a complex outcome of network physical connectivity and dynamical properties of interacting neurons, however characterization

of this interaction remains elusive. These dynamical properties of the cells are affected/controlled by various neuromodulators secreted by the brain at various cognitive cycles or as a part of the response to the incoming stimuli. During sleep the brain cycles through distinct spatiotemporal patterns of neural activity. Acetylcholine (ACh) is a major regulatory factor of sleep states and plays an important role in the transition from slow wave sleep to waking or rapid eye movement sleep. Slow wave sleep is a slow oscillation in firing rate that travels through the cortical network and occurs when ACh levels are low [1]. At the cellular level, ACh causes changes in neural excitability by shifting the neural phase response curve (PRC) from type 2 to type 1 (Figure 1A)[2]. Previous modeling studies show that the shift of the PRC leads to a change from synchronous (type 2 PRC) to asynchronous (type 1 PRC), network dynamics while during low ACh levels networks display a high level of synchrony and network wide bursts[3]. As of yet the effects of intermediate cholinergic modulation have not been investigated. In this study, we use a Hodgkin-Huxley type model neuron which allows us to simulate different ACh levels and control a continuous transition from a type 1 to type 2 PRC [4]. We show that the PRC type of neurons drives different spatial patterns of activity within networks, with activity being highly localized for type 1 PRC neurons (Figure 1B) then quickly transitioning to wave dynamics as neurons are shifted to a type 2 PRC (Figure 1B). In networks composed of type 1 neurons, the region where activity is localized is defined by heterogeneities in network structure, with as little as a 1% increase in synaptic strength being sufficient to define the location of high activity. Additionally, the highly active zone is the origin of traveling waves in type 2 networks. When in the wave regime, decreasing cholinergic modulation of the PRC increases the speed that waves travel across the network. In summary, the precise character of frequency dynamics is governed by the interplay between network structure and the intrinsic excitability of component neurons. Expanding upon our results, we argue (1) that the intrinsic excitability of neurons shapes how activity spreads through a network and (2) that the focal point of traveling waves during slow wave sleep is a region selected for by synaptic potentiation.

Acknowledgements

This material is based upon work supported by the NSF GRFP under Grant No. DGE 1256260 (JPR), NSF CMMI 1029388 (MRZ), and NSF PoLS 1058034 (MRZ & LMS).

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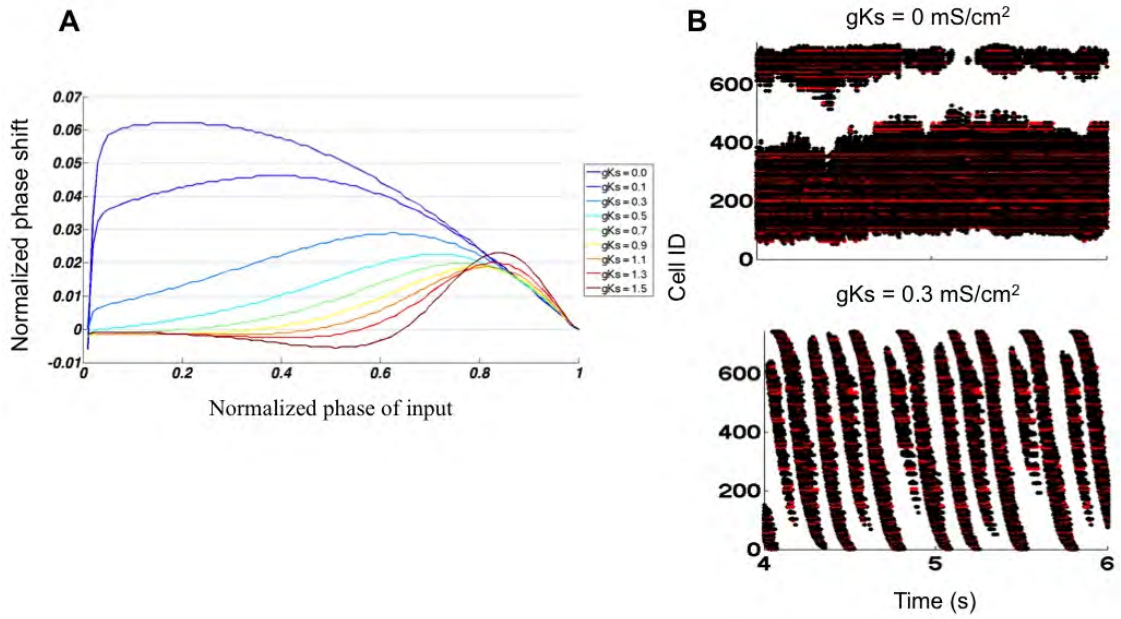


Figure 1: PRC induced changes in network dynamics. A: Increasing g_{Ks} shifts the PRC of the model neuron from type 1 at low values to type 2 at high values. B: Raster plots showing characteristic dynamics for networks at two different PRC types. Cells are sorted by distance from the origin in xy space and black dots represent excitatory action potentials and red dots indicate inhibitory action potentials.

O17 Synchronization of the Parkinsonian Globus Pallidus by Gap Junctions

Bettina Schwab^{1,2*}, Hil Meijer¹, Richard van Wezel^{2,3}, and Stephan van Gils¹

¹*Applied Analysis, MIRA Institute of Biomedical Technology and Technical Medicine, University of Twente, 7500AE Enschede, The Netherlands*

²*Biomedical Signals and Systems, MIRA Institute of Biomedical Technology and Technical Medicine, University of Twente, 7500AE Enschede, The Netherlands*

³*Biophysics, Donders Institute for Brain, Cognition and Behavior, Radboud University, Nijmegen, The Netherlands*

The mechanisms for the emergence and transmission of synchronized oscillations in Parkinson's disease (PD) still remain debated. In a previous publication [1], we argue that the external globus pallidus (GPe) has a crucial role in desynchronising and synchronizing the basal ganglia. While neural activity of the healthy GPe shows almost no correlations between pairs of neurons, prominent synchronization in the β frequency band arises after dopamine depletion.

Intrinsic factors of the GPe, in particular its internal connections, could be take major roles in this synchronisation process.

We introduce pallidal gap junctional coupling as a possible mechanism for synchronization of the GPe after dopamine depletion. In a confocal imaging study, we show the presence of the neural gap junction protein Cx36 in the human GPe, including a possible remodeling process in PD patients. Dopamine has been shown to down-regulate the conductance of gap junctions in different regions of the brain [2,3], making dopamine depletion a possible candidate for increased influence of gap junctional coupling in PD.

To see what effect electrical coupling in the GPe could have, we incorporate gap junctions in a small conductance-based model of the basal ganglia. In both GPe and GPi, gap junctional coupling has clear effects on synchrony. Especially numerous coupling with sufficient strength in the GPe is able to synchronize the whole basal ganglia. Next, we focus on dynamics inside the GPe. Phase-response curve analysis is used to describe the susceptibility of GPe neurons to synchronize with input, depending on electrical coupling to other GPe neurons. Additionally, we simulate the effect of gap junctions on synchrony in a larger network of the GPe, including biologically realistic cell models and inhibitory synaptic coupling.

Conclusions: We hypothesize that strong gap junctional coupling in the GPe disturbs the self-desynchronization in this nucleus and leads to long-range synchronization. Pallidal gap junctions, which are potentially modulated by dopamine, could be a powerful trigger of synchrony in Parkinson's disease. We stress that also gap junctions in other nuclei such as the striatum may play important roles.

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O18 The dynamic separation of pallidal neurons into anti-phase oscillatory groups under Parkinsonian conditions in a computational model

Robert Merrison-Hort*, Roman Borisyuk

School of Computing & Mathematics, Plymouth University, Plymouth, Devon, PL4 8AA, UK

Neurons in the globus pallidus (GP) of urethane anesthetized rats typically display one of four spiking patterns: tonic, non-modulated, firing (the NM group); firing that occurs preferentially during either the active or inactive phases of slow cortical oscillations (TA or TI group, respectively); or silence/quiescence (QU group). In healthy animals the vast majority of neurons are in the non-modulated group. However, under conditions of experimentally-induced Parkinsonism there is a dramatic increase in the number of neurons whose firing patterns show modulation by the slow cortical rhythm, either in-phase or anti-phase [1]. The mechanism that underlies the increased tendency for GP neurons to become entrained by cortical rhythms is unclear, but it

may contribute to some of the motor symptoms of Parkinson's disease.

There are two main pathways from the cortex to the GP: via the inhibitory striatum and via the excitatory subthalamic nucleus (STN), but it is not known how these inputs sculpt the pathological pallidal firing patterns. To study this we developed a neural network model of single compartment conductance-based (Hodgkin-Huxley) pallidal neurons, based on a previous multi-compartment model [2]. The GP neurons received rhythmic input from STN neurons and reciprocal inhibition from each other. Under 'healthy' conditions, almost all model GP neurons showed tonic firing that was not significantly modulated by the rhythmic STN input (Figure 1A,B). We attempted to model 'Parkinsonian' conditions by increasing the intensity of STN neuron firing and the strength of STN-GP and GP-GP synapses. Under these conditions, two groups of anti-phase oscillatory GP neurons emerged (Figure 1C,D). Our model also includes downregulation of Hyperpolarization activated Cyclic Nucleotide-gated (HCN) channels in response to bursting, since this may contribute to emergence of Parkinsonian activity [3]. We found that this provides better agreement with experimental data but that it is not essential in order for the two groups to appear.

Our results [4] support the hypothesis that oscillatory entrainment occurs primarily via the subthalamic pathway. We find that as a result of the interplay between excitatory input from the STN and mutual inhibition between GP neurons, the network shows a self-organizing dynamical behavior where two groups of neurons (TI and TA) emerge out of a homogeneous population.

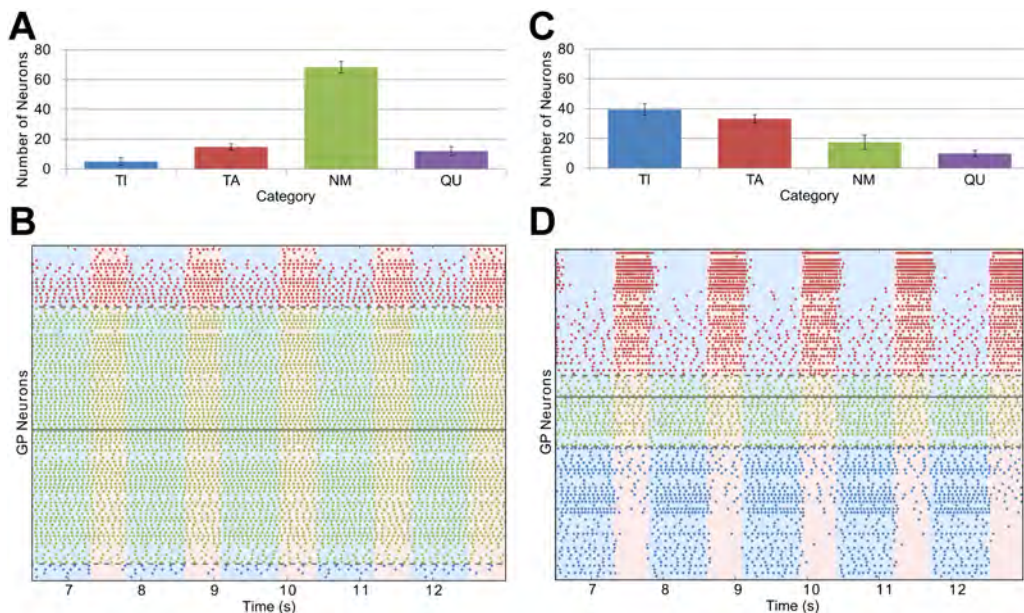


Figure 1: Proportions of neurons in each category (A,C) and spiking activity sorted by classification confidence (B,D) for healthy (left) and Parkinsonian (right) parameters.

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O19 The sleeping brain regulates to the edge of chaos

Moira Steyn-Ross^{1*}, Alistair Steyn-Ross¹, and Jamie Sleight²

¹*School of Engineering, University of Waikato, Hamilton 3240, New Zealand*

²*Waikato Clinical School, University of Auckland, Hamilton 3240, New Zealand*

One of the most intriguing ideas in complexity theory is the notion that some systems can organize dynamically to a point critically poised between order and disorder, hovering at the so-called 'edge of chaos'. It has been proposed that the computational performance of neural networks is optimized when close to the order–disorder phase transition. In this presentation we explore the novel hypothesis that the human brain may be operating at the edge of chaos during slow-wave sleep (SWS), the deepest phase of NREM (non-rapid-eye-movement) sleep.

We build on an existing continuum model of the cortex [1] to incorporate known changes in specific neurotransmitter concentrations—GABA increase with simultaneous acetylcholine (ACh) decrease—during descent from wake into natural SWS [2]. The GABA boost is modeled as an anesthetic-like prolongation of the inhibitory postsynaptic potential (IPSP) paired with a restriction of gap-junction connectivity, while ACh suppression reduces resting cell voltage but enhances excitatory synaptic efficiency. Our model is able to produce a plausible sequence of time-series for EEG progression through the stages of NREM sleep (see Fig. 1).

These sleep-induced neurotransmitter changes can have profound effects on cortical stability: alterations in inhibitory gap-junction connectivity controls a pattern-forming Turing instability, and manipulations of IPSP duration can lead to Hopf temporal oscillations which, in a pathological limit, can lead to whole-of-cortex seizure. We argue that normal brain function requires a balance between Turing and Hopf instabilities, and that descent into deep sleep entails a rebalancing in favor the Hopf instability. Model simulations predict that the spatiotemporal patterns for NREM sleep stages-1 to -4 are chaotic, showing exponential trajectory divergence from closely similar starting conditions. In contrast, the seizure state is highly ordered and non-chaotic. Since most sleepers do not proceed to seizure, we posit the existence of a protective mechanism that regulates the naturally sleeping brain so that it remains close to—but does not cross—the disorder/order boundary during deepest sleep.

There is clinical evidence that high cortical activity is associated with closure of gap-junctions [3]. This has motivated a learning rule that regulates the gap-junction conductivity based on the spatial covariance of inhibitory firing-rate activity across the two-dimensional cortical grid. We find that this rule enables the cortex to regulate its slow-wave dynamics from chaotic to marginally-ordered, and that regulation failure typically leads to seizure onset.

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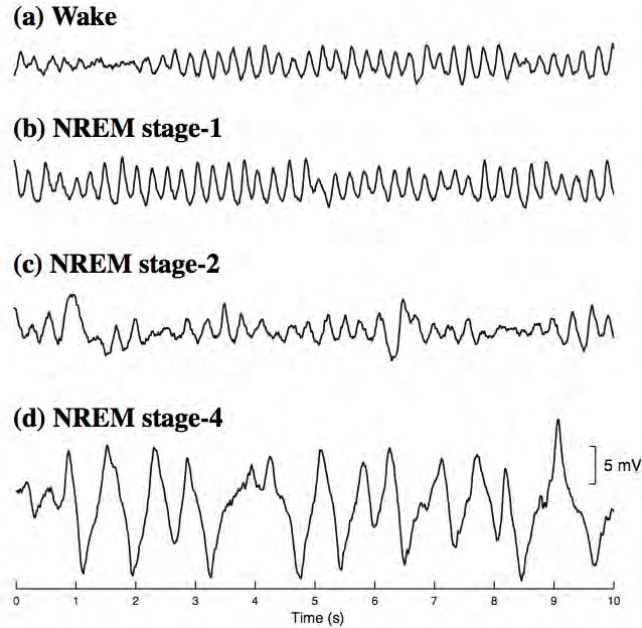


Figure 1: Model-generated sleep electrocorticograms for descent from wake to deep NREM. Model predictions are the excitatory soma voltage recorded at one point on a 120 x 120 cortical grid after filtering with a 0.5-Hz high-pass filter.

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O20 Chaos in heterogeneous neural networks

Merav Stern*, Johnatan Aljadeff, and Tatyana Sharpee

I. The critical transition point

There is accumulating evidence that biological neural networks possess optimal computational capacity when they are at or near a critical point in which the network transitions to a chaotic regime. We derive a formula for the critical point of a general heterogeneous neural network. This formula relates the structure of the network to its critical point. The heterogeneity of the network may describe the spatial structure, a multiplicity of cell types or any selective connectivity rules.

To define the network we divide the N neurons into D groups such that $\sum_{d=1,\dots,D} N_d = N$. The synaptic weight between neurons i, j (the connectivity matrix element J_{ij}) is drawn from a centered distribution with standard deviation summarized in a $D \times D$ rule matrix $N^{-1/2}G_{c(i)d(j)}$ (insets to A, $c(i)$ is the type index of neuron i). The network obeys the standard rate dynamics $(d/dt)x_i = -x_i + \sum_{j=1,\dots,N} J_{ij} \tanh x_j$.

The global behavior of the network changes from a single fixed point to chaos when $r = 1$, r being the radius of the circle that bounds the spectrum of the connectivity matrix (panel A). We derived a formula, in terms of the matrix G and the vector N_d , for r that can also be thought of as an effective gain [1]: it is the square root of the maximal eigenvalue of a $D \times D$ matrix M

whose c, d element is $M_{cd} = N^{-1}N_c(G_{cd})^2$.

We use our understanding of the general heterogeneous dynamical system to a network with a large fraction of cells in the subcritical regime, and a small fraction of supercritical neurons. This can be thought of as a model of a network where adult neurogenesis occurs, where a small fraction of hyperexcitable neurons are continuously integrated. Using a supervised learning algorithm (FORCE, [2]) we show that r is as a good coordinate to describe the network’s “learnability” (panels B,C). Learning is optimal for values of r similar to those found in a homogenous network. Our results suggest that the new neurons can allow the network to be poised at criticality with no global changes to connectivity, and that their specific roles are context dependent, in contrast to previous hypotheses.

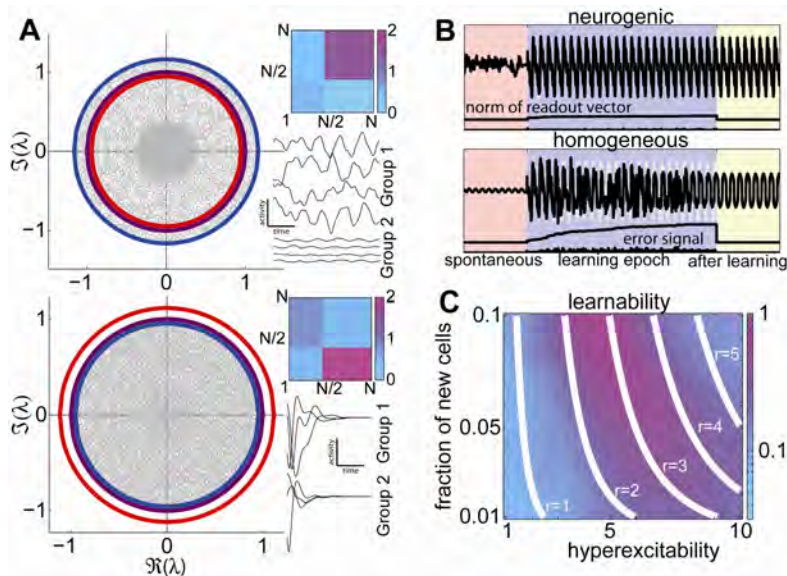


Figure 1: (A) Example spectra of connectivity matrices (gray) with $r > 1$ (top, indicated in blue and purple respectively) and $r < 1$ (bottom). The average synaptic gain (red) does not give the correct boundary of the spectrum and would predict opposite behavior. The matrix $G_{c(i)d(j)}$ is indicated by the color plots (top), and activity of representative neurons from the two groups (bottom) of each example. (B) The activity of a readout unit during spontaneous activity, a FORCE learning epoch, and post learning for neurogenic and homogeneous subcritical networks. The neurogenic network quickly matches the target signal (gray) and robustly reproduces it. (C) The learnability of an ensemble of neurogenic networks as a function of the hyperexcitability and new neuron fraction coincides with contour lines of r (white).

II. Multiple activity modes

We study the activity of a recurrent neural network consisting of multiple cell groups through the structure of its correlations by showing how the rules that govern the strengths of connections between the different cell groups shape the average autocorrelation found in each group. We derive an analytical expression for the number of independent autocorrelation modes the network can concurrently sustain. Each mode corresponds to a non-zero component of the network’s autocorrelation, when it is projected on a specific set of basis vectors. In a companion abstract we derive a formula for the first mode, and hence the entire network, to become active. When the network is just above the critical point where it becomes active all groups of cells have the same autocorrelation function up to a constant multiplicative factor. We derive here a formula for this multiplicative factor which is in fact the ratio of the average firing rate of each group. As the effective synaptic gain grows a second activity mode appears, the autocorrelation functions

of each group have different shapes, and the network becomes doubly chaotic. We generalize this result to understand how many modes of activity can be found in a heterogeneous network based on its connectivity structure. Finally, we use our theory to understand the dynamics of a clustered network where cells from the same group are strongly connected compared to cells from different groups. We show how this structure can lead to a one or more activity modes and interesting switching effects in the identity of the dominant cluster.

To model the heterogeneous network we include N neurons that are divided into D groups. The synaptic weight between neurons i, j is drawn from a centered distribution with standard deviations summarized in a $D \times D$ rule matrix $N^{-1/2}G_{c(i)d(j)}$ where $c(i)$ is the group neuron i belongs to. The network obeys the standard rate dynamics $(d/dt)x_i = -x_i + \sum_{j=1, \dots, N} J_{ij} \tanh x_j$. The global behavior of the network changes according to the real part of the eigenvalues of a $D \times D$ matrix M whose c, d element is $M_{cd} = N^{-1}N_c(G_{cd})^2$. When M 's largest eigenvalue, Λ_1 , become larger than 1 the network become chaotic. The ratios of the components of the leading eigenvector V_1 are the ratios of the autocorrelations functions of the different groups (panel A). When Λ_2 becomes larger than 1 the network is doubly chaotic (panel B). In general, the autocorrelation vector has a non-zero projection only on eigenvectors of M with eigenvalues greater than 1 (panel C) and hence the number of active modes in the network is equal to the number of eigenvalues of M that are larger than 1.

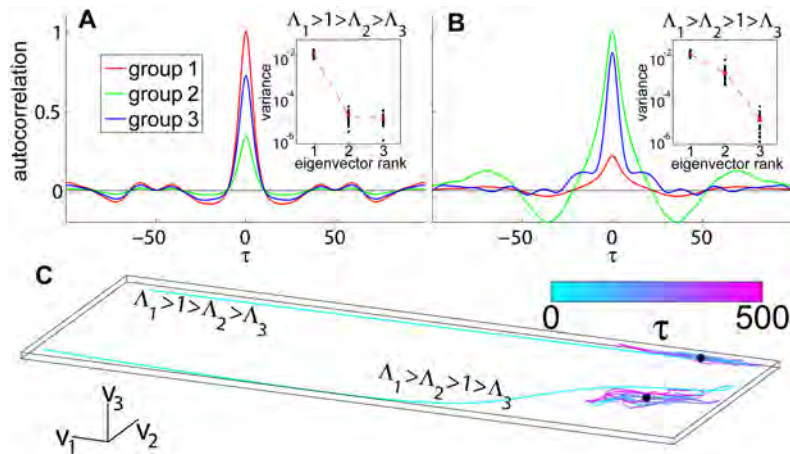


Figure 2: (A) For an example network with 1200 neurons divided to 3 equally sized groups we plot the autocorrelation function averaged over neurons belonging to the same group. G was chosen such that one eigenvalue of M is greater than 1. Independent of the time lag τ the autocorrelations maintain a constant ratio that is equal to the ratio of the components of the eigenvector of M corresponding to the leading eigenvalue. Inset: for 20 example networks we computed the variance of the autocorrelation vector along the three eigenvectors of M and found that the variation in autocorrelation along the leading eigenvector is three orders of magnitude larger than along the other two directions. (B) In this example network M has two eigenvalues greater than 1. The autocorrelations are no longer a constant ratio of each other, indicating that the network maintains two modes of autocorrelation concurrently. Inset: when averaged over 20 networks we see that the variation along the two eigenvectors with eigenvalues greater than 1 is significantly larger than along the third eigenvector. (C) For the two examples networks shown in (A,B) we plotted the trajectory of the autocorrelation vector as a function of the time lag τ , and show that they are confined to in the subspace spanned by the eigenvectors, V_1 and V_2 which has eigenvalues greater than 1.

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Workshops

Workshops			
Workshop	Wednesday July 30 th	Thursday July 31 st	
W1	Room 207	Room 207	
W2	Room 2103	Room 2103	
W3	Room 2101	Room 2101	
W4	Room 2102B	[Cross-hatched pattern]	
W5	Room 2104B		
W6	Room 2105		
W7	Room 2104A		
W8	[Diagonal hatched pattern]		Room 2102B
W9			Room 2104B
W10			Room 2105
W11		Room 2104A	
W12	Room 2101 (6-8 PM)	[Diagonal hatched pattern]	

W1 Cortical Oscillations: Computational models and dynamic mechanisms

Room 207, W, Th

Horacio Rotstein, New Jersey Institute of Technology

Mark Kramer, Boston University

Oscillatory activity at various frequency ranges have been observed in various areas of the brain and are believed to be important for cognitive functions such as learning, memory, navigation and attention. Disruption of rhythmic oscillations has been implicated in diseases of the nervous system including epilepsy and schizophrenia. Neuronal oscillations have been studied at the single cell level, as the result of the interaction of a neuron's intrinsic properties, at the network level, 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 both the underlying mechanisms that govern the generation of these rhythms at the various levels of organization mentioned above and their functional implications for cognition.

Speakers:

- Alla Borisyuk (University of Utah, UT, USA)
- Christoph Borgers (Tufts University, MA, USA)

- Victoria Booth (University of Michigan, MI, USA)
- Mark Cunningham (University of Newcastle upon Tyne, UK)
- Carina Curto (University of Nebraska-Lincoln, NE, USA)
- Vassilis Cutsuridis (Foundation for Research and Technology - Hellas, Greece)
- Flavio Frohlich (University of North Carolina, NC, USA)
- David Hansel (University Paris Descartes, France)
- Michael Hasselmo (Boston University, MA, USA)
- Mark Kramer (Boston University, MA, USA)
- Paola Malerba (University of California at Riverside, USA)
- Adrien Peyrache (NYU, NY, USA)
- Horacio G. Rotstein (NJIT, NJ, USA)
- Jonathan E. Rubin (University of Pittsburgh, PA, USA)
- Frances Skinner (University of Toronto, ON, Canada)
- Jiannis Taxidis (UCLA, CA, USA)
- Roger Traub (IBM, NY, USA)
- John A. White (University of Utah, UT, USA)

W2 Computational methods and modeling of Astrocyte physiology and Neuron-glia interactions

Room 2103, W, Th

Hugues Berry, INRIA

Maurizio De Pitta, University of Chicago

In recent years, the simultaneous recognition that astrocytes sense neighboring neuronal activity and release neuroactive agents (or 'gliotransmitters') has been instrumental in the uncovering of the many possible roles played by these cells in the regulation of synaptic transmission and neuronal activity. These findings suggest that information travels and is processed not just in the neuronal circuitry but in an expanded neuron-glia network. However, much remains elusive about the role of astrocyte signaling in brain information processing. Besides the lack of conclusive experimental evidence, this is partly due to a substantial lack of a theoretical framework to address modeling and characterization of the many possible astrocyte functions. Computational modeling is challenged by the fact that many details remain hitherto unknown and conventional approaches to describe neuronal and synaptic function may be unsuitable to explain experimental observations when astrocytic signaling is taken into account. Progress in astrocyte modeling is

also in part hampered by the lack of workshop or annual event specifically dedicated to computational approaches of neuron-glia interactions. This workshop aims at filling this gap, by bringing together a panel of scientists that work on modeling of neuron-glia interactions. This aims at providing both the expert from the field and the OCNS community in general, a comprehensive picture of the current state of the art, the open questions and the theoretical challenges put forth by the modeling of astrocyte physiology.

Speakers:

- Mahmood Amiri, Italian Institute of Technology, Genoa, Italy
- Hugues Berry, INRIA, Lyon, France
- Maurizio De Pittà, University of Chicago, IL, USA
- David Holcman, Ecole Normale Supérieure, Paris, France
- Viktor B. Kazantsev, N. I. Lobachevsky State University of Nizhni Novgorod, Russia
- Konstantin Mergenthaler, Technische Universität Berlin, Germany
- Suhita Nadkarni, Indian Institute of Science Education and Research at Pune, India
- Annalisa Scimemi, State University of New York at Albany, NY, USA
- Minchul Kang, Saint Thomas University at Miami, FL, USA

W3 Methods of Information Theory in Computational Neuroscience

Room 2101, W, Th

Michael C Gastpar, Laboratory for Information in Networked Systems, EPFL and UC Berkeley

Conor Houghton, Department of Mathematics, Trinity College Dublin

Simon R Schultz, Department of Bioengineering, Imperial College

Tatyana O Sharpee, The Computational Neurobiology Laboratory, Salk Institute

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.

References:

- C.E. Shannon, A Mathematical Theory of Communication, Bell System Technical Journal, vol. 27, pp. 379-423 and 623-656, 1948.
- Milenkovic, O., Alterovitz, G., Battail, G., Coleman, T. P., et al., Eds., Special Issue on Molecular Biology and Neuroscience, IEEE Transactions on Information Theory, Volume 56, Number 2, February, 2010.
- Dimitrov, A.G., Lazar, A.A. and Victor, J.D., Information Theory in Neuroscience, Journal of Computational Neuroscience, Vol. 30, No. 1, February 2011, pp. 1-5, Special Issue on Methods of Information Theory.

Speakers:

- Andre Longtin, Physics Department, University of Ottawa
- Maurice Chacron, Faculty of Medicine, McGill University
- Mike DeWeese, Physics Department and the Helen Wills Neuroscience Institute, UC Berkeley
- Lav R. Varshney, Department of Electrical and Computer Engineering, UIUC
- Peter Grassberger, Complexity Science Group, University of Calgary
- Byron Yu, Electrical & Computer Engineering and Biomedical Engineering, Carnegie Mellon University
- Jean Lienard, Oregon Hearing Research Center, Oregon Health & Science University
- Vijay Balasubramanian, Department of Physics and Astronomy, University of Pennsylvania
- Kechen Zhang, Department of Biomedical Engineering, Johns Hopkins University
- Chris DiMattina, Department of Psychology, Florida Gulf Coast University

W4 Running parallel simulations on HPC resources via the Neuroscience Gateway Portal

Room 2102B, W

Amit Majumdar, UCSD

Ted Carnevale, Yale School of Medicine

This workshop presents the the Neuroscience Gateway Portal at CNS 2014, Quebec City, Canada. Access to HPC resources is growing ever more important as advances in experimental and theoretical neuroscience drive the formulation of increasingly complex models and simulation projects that impose computational burdens exceeding the capabilities of locally available hardware. The NSG is designed to eliminate most administrative and technical barriers to using HPC resources. It offers free access to these resources through a streamlined application process. Its web-based interface simplifies the tasks of uploading models, specifying job parameters, monitoring job status, and storing and retrieving output data. Simulators currently installed include

NEURON, GENESIS3, MOOSE, NEST, PyNN, and Brian. This workshop will combine didactic presentations by NSG's developers, discussions with developers of simulators for spiking neural networks, and hands on instruction in how to use the portal (participants are invited to bring laptop computers for this). Registration information to follow soon.

Speakers:

- Vadim Astakhov, UCSD
- Anita Bandrowski, UCSD
- Ted Carnevale , Yale School of Medicine, (Co-PI)
- Michael Hines, Yale School of Medicine
- Amit Majumdar, UCSD, (PI)
- Maryann Martone, UCSD, (Co-PI)
- Subhashini Sivagnanam, UCSD
- Kenneth Yoshimoto, UCSD

W5 Resonance and Entrainment: From Dynamic Systems Theory to Targeted Brain Stimulation

Room 2104B, W

Flavio Frolich, University of North Carolina at Chapel Hill

Oscillations are a prevalent feature of neuronal activity and are of fundamental importance for orchestrating behavior. Despite the ubiquitous observations about rhythmic synchronization of neuronal activity, the causal role of oscillatory activity in the nervous system has remained a matter of debate. Neuroscience is currently undergoing a major transformation due to the advent of tools such as optogenetics and transcranial alternating current stimulation to probe for the causal role of oscillatory activity by selective enhancement or suppression of specific oscillatory activity patterns. These new experimental perturbations have emerged as a unique opportunity to establish the causal role of brain oscillations. However, progress has been hampered by a lack of understanding how external perturbations modulate ongoing endogenous oscillatory activity in the brain. Mathematical frameworks from dynamic system theory such as resonance and phase response curves can provide important cues for experimental design and explanation of experimental data but has yet had only limited impact on the broader network neuroscience community. Advancing our understanding of targeted modulation of neuronal oscillations will not only provide new approaches to test for the causal role of activity patterns but will hopefully also offer novel brain stimulation approaches for the treatment of neurological and psychiatric illnesses that have associated with impaired oscillation structure of brain activity. This workshop aims to bring together researchers from a several different areas of research that share a common interest in the mechanisms of entrainment of neuronal oscillation. In particular, the workshop will aim to bridge mathematical and computational neuroscience, systems neuroscience, and translational neuroscience. This workshop will have been a success if it establishes an active

dialogue about how we can interact with oscillations in the nervous system to probe for their functional and behavioral roles and if novel interdisciplinary collaborations originate from this workshop.

Speakers:

- Farzan Nadim (NJIT): Resonance and synaptic dynamics (tentatively confirmed)
- Yosef Yarom (Hebrew University): Subthreshold oscillations and resonance (confirmed)
- Horacio Rotstein (NJIT): Biophysical and dynamic mechanisms of resonance in neurons and networks (confirmed)
- Michael Hasselmo (BU): Theta resonance and grid cells (tentatively confirmed)
- Vikaas Sohal (UCSF): Optogenetic control of cortical oscillations (confirmed)
- Tommaso Fellin (U of Genova): Layer-specific control of cortical oscillations (confirmed)
- Michael Halassa (NYU): State-dependent organization of thalamic reticular microcircuits (confirmed)
- Steven Schiff (PennState): Neural Control Engineering for modulating cortical oscillations (confirmed)
- Charles Schroeder (Columbia): Endogenous modulation of cortical oscillations (confirmed)
- Flavio Frohlich (UNC): Brain Stimulation by Network Resonance (confirmed)

W6 Methods of System Identification for Studying Information Processing in Sensory Systems

Room 2105, W

Aurel A Lazar, Department of Electrical Engineering, Columbia University

Mikko I Juusola,

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.

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.

References:

- Vasilis Z. Marmarelis (2004). Nonlinear Dynamic Modeling of Physiological Systems. Wiley-IEEE Press, Hoboken, NJ, 2004.
- Wu, M., David, S., & Gallant, J. (2006). Complete Functional Characterization of Sensory Neurons by System Identification. Annual Review of Neuroscience, 29, 477–505.
- Ljung, L. (2010). Perspectives on System Identification, Annual Reviews in Control, 34 (2010), 1-12.

Speakers:

- Thomas R. Clandinin, Department of Neurobiology, Stanford University.
- Claude Desplan, Department of Biology, NYU.
- Mark A. Frye, Department of Integrative Biology and Physiology, UCLA.
- Mikko I. Juusola, Department of Biomedical Science, University of Sheffield.
- Arvind Kumar, Bernstein Center Freiburg, University of Freiburg.
- Aurel A. Lazar, Department of Electrical Engineering, Columbia University.
- Stefan Mihalas, Allen Institute for Brain Science.
- Tatyana O. Sharpee, The Computational Neurobiology Laboratory, Salk Institute.
- Glenn C. Turner, Cold Spring Harbor Laboratory.

W7 Dynamics of Disease States

Room 2104A, W

Jonathan Rubin, University of Pittsburgh

Stephan Van Gils, University of Twente

Changes at molecular, cellular and network levels may lead to a variety of disorders involving pathological brain states. Examples range from schizophrenia and spreading depression to disorders with significant motor pathologies, such as Parkinson's disease and epilepsy. Computational modeling based on experimental data offers means to gain insight about the underlying mechanisms and generate novel predictions. In particular, this approach provides the opportunity to explore ideas and tease apart factors that are inaccessible in wet lab experiments and even allows for the simulation of therapeutic approaches, including closed-loop or individualized therapies. In this workshop we will discuss how new insights at the molecular, cellular and network levels can be incorporated into computational modeling of pathological brain states. We aim to stimulate discussion that promotes advances in the use of computation in the development of pharmaceutical, surgical or electrical interventions.

Speakers:

- Viktor Jirsa
- Wim van Drongelen
- Marc Goodfellow
- Robert Rosenbaum
- Bettina Schwab
- Ingo Bojak
- Steven Schiff
- Markus Dahlem
- Wytse Wadman
- Shane Lee
- Sid Visser
- Theoden Netoff

W8 Sleep Rhythms and Memory Consolidation

Room 2102B, Th

Maxim Bazhenov, UC Riverside

Igor Timofeev, Laval University

During slow-wave sleep the cortex is decoupled from external inputs and can be devoted to consolidating previously acquired labile memories into stable memories. Recently, memory replay has been demonstrated during sleep and associated with characteristic oscillations giving rise to the hypothesis that these may form the critical neural substrate of memory consolidation. However, these studies have mainly focused on the rat hippocampus; while replay has also been demonstrated in other structures and species, evidence remains sparse, especially concerning the specific interactions between thalamic, hippocampal and cortical networks which subserve sleep-dependent consolidation of memory. At the same time recent technological developments make now possible active interaction with brain structures, thus opening a possibility of controlling and enhancing consolidation processes. In this workshop, we will discuss new findings from animal, human and computation works that explain fundamental mechanisms of sleep rhythm generation and sleep rhythms contribute to memory consolidation. The goal of this workshop is to bring together experimental and computational neuroscientists to discuss fundamental principles of the network dynamics of the brain that are involved in the processes of memory and learning.

Speakers:

- Maxim Bazhenov (UC Riverside)
- Jean-Marc Fellous (Univ Arizona)

- Eric Halgren (UC San Diego)
- Andre Longtin (Univ Ottawa)
- Sara Mednick (UC Riverside)
- Hong-Viet Ngo (Univ Tubingen)
- Alex Roxin (Campus de Bellaterra, Barcelona)
- Tim Rogers (Univ Wisconsin-Madison)
- Igor Timofeev (Laval Univ)

W9 Basal Ganglia: Structure, dynamics and function

Room 2104B, Th

Arvind Kumar, Bernstein Center Freiburg, University of Freiburg, Germany

Jeanette Hellgren Kotaleski, Royal Institute of Technology, Stockholm, Sweden

The basal ganglia (BG) are involved in a wide range of motor and cognitive processes, and accordingly, their dysfunction can lead to several neurological diseases. To understand the computational role of BG in these various functions and dysfunction several bottom-up and top-down models have been proposed. Bottom-up computational approaches have addressed the dynamical properties and interaction of the neural activity in the BG nuclei, while top-down approaches rather have described BG function inspired by machine learning algorithms.

Recent advances in experimental methods have allowed for the characterization of BG activity and cortico-basal ganglia interactions with great detail both in normal and pathological conditions and have challenged the classical feedforward view of the basal ganglia network.

In this workshop, we bring together both experimentalists and theoreticians to review the recent advances in understanding of BG function. Specifically, we will discuss how computational models of BG have advanced to integrate the new data on BG network structure and neuronal activity and thus, to understand how the relationship between BG dynamics and function/dysfunction of BG. Finally, we will discuss how top-down functional models could be linked to the bottom-up dynamical models and provide new predictions and explanations of the experimental data in terms of neuronal and network properties.

Speakers:

- Joshua Berke (Michigan State University, Ann Arbor, MI, USA)
- Avrama Blackwell (George Mason University, Fairfax, VA, USA)
- Michael Frank (Brown University, Providence, RI, USA)
- Aryn Gittis (Carnegie Mellon University, Pittsburg, PA, USA)
- Jesse Goldberg (Cornell University, NY, USA)
- Frank Hamker (Chemnitz University of Technology, Chemnitz, Germany)

- Ahmed Moustafa (University of Western Sydney, Australia)
- Jyotika Bahuguna (University of Freiburg, Germany)
- Mikael Lindhal (Royal Institute of Technology, Stockholm, Sweden)

W10 Large-scale brain structure and dynamics

Room 2105, Th

Jorge F Mejias, NYU

Xiao-Jing Wang,

Tackling core Neuroscience problems such as memory, perception or attention has been possible, up to now, by focusing on small brain areas where simplified dynamics and connectivity patterns could be assumed, or by using full brain imaging techniques which provide little information about the dynamics of local microcircuits. With the development of novel imaging and neuroanatomical techniques in the recent years, a more systematic study of the brain at a full-size scale is getting close to reality. However, our current understanding of the dynamics of large-scale brain networks, constituted by a complex composite of different neural micro- and mesocircuits, is very limited and requires a significant theoretical and computational effort.

In this one-day workshop, recent theoretical and experimental advances on the structure and dynamics of large-scale neural circuits will be presented. Our main goal will be to foster interactions between experimental, computational and theoretical neuroscientists interested in the brain as a large-scale networked system.

Speakers:

- Chris Eliasmith (U. Waterloo)
- Henry Kennedy (INSERM, Lyon)
- Stefan Mihalas (Allen Institute, Seattle)
- Randy McIntosh (Baycrest Center, Toronto)
- Adrian Ponce (UPF, Barcelona)
- Rishidev Chaudhuri (NYU)
- Francis Song (NYU)

W11 Finite-size fluctuations in neural systems - from ion channels to networks

Room 2104A, Th

Richard Naud, University of Ottawa, Canada

Tilo Schwalger, EPFL, Switzerland

Moritz Deger, EPFL, Switzerland

Fluctuations of neural dynamics generated in systems of small (finite) size are ubiquitous on

many levels of the nervous system and have important functional implications. For instance, ionic currents are noisy if mediated by a small population of ion channels (channel noise), synapses become unreliable when they contain a finite number of vesicles and neurotransmitters, and the population activity of neural circuits fluctuates due to a finite number of neurons. Such finite-size effects contribute to neural variability, which fundamentally limits neural signal processing. On the other hand, in nonlinear systems, intrinsic noise may lead to drastic effects: In particular, finite-size fluctuations may enable transitions between different neural states, decorrelate neural activity and even improve information processing. Accounting for finite-size fluctuations is a challenging theoretical problem that requires advanced methods for treating non-equilibrium statistical systems. Recently, there has been much analytical progress characterizing finite-size effects: from understanding information transmission through stochastic synapses, to elucidating spike initiation in the face of channel noise, and all the way to capturing the statistics of the fluctuations of entire neural networks. In this workshop, we bring together researchers on finite-size effects on various scales of description throughout the neurosciences. Although individual system properties and function might differ, common mathematical approaches and computational roles will be discussed to further our understanding of the puzzling roles of stochasticity in neural dynamics and computation.

Speakers:

- Taro Toyozumi (group leader, RIKEN Brain Institute, Japan)
- John A. White (Professor, University of Utah)
- Maurizio Mattia (researcher, Istituto Superiore di SanitÃ , Rome, Italy)
- Gregory Dumont (postdoctoral fellow, group of AndrÃ© Longtin, University of Ottawa, Canada)
- Brent Doiron (associate Professor, University of Pittsburgh, USA)
- Alex Bird (doctoral student with Magnus Richardson, University of Warwick, UK)
- Tilo Schwalger (postdoctoral fellow, group of Wulfram Gerstner, EPFL, Switzerland)

W12 Student/Post-doc career Workshop

Room 2101, W 6-8 PM

Jorge F Mejias, NJIT

This workshop, aimed at graduate students and post-doctoral fellows, will discuss career options.

Posters

Posters

Sunday Posters Posters P1 – P75

- P1 Computational Multifactoriality in a Detailed Neural Network Model Resembling Center-Surround Suppression Deficits in Schizophrenia**
Christoph Metzner^{1,2*}, Achim Schweikard¹, and Bartosz Zuurawski³
¹*Institute for Robotics and Cognitive Systems, University of Luebeck, 23538 Luebeck, Germany*
²*Graduate School for Computing in Medicine and Life Sciences, University of Luebeck, 23538 Luebeck, Germany*
³*Department of Psychiatry, University of Luebeck, Schleswig-Holstein, 23538 Luebeck, Germany*
- P2 Measuring predictability of autonomous network transitions into bursting dynamics**
Sima Mofakham^{1*}, Michal Zochowski^{1,2}
¹*Department of Biophysics, University of Michigan, Ann Arbor, MI, USA*
²*Department of Physics, University of Michigan, Ann Arbor, MI, USA*
- P3 Interaction of neuronal resonance properties and network connectivity in pattern formation and separation**
Elizabeth Shtrahman^{1*}, Michal Zochowski²
¹*Applied Physics Program, University of Michigan, Ann Arbor, MI 48109, USA*
²*Department of Physics, University of Michigan, Ann Arbor, MI 48109, USA*
- P4 Large-scale spiking circuit simulation of spatio-temporal dynamics in superior colliculus**
Richard Veale^{1,2*}, Tadashi Isa^{2,3}, and Masatoshi Yoshida^{2,3}
¹*Cognitive Science Program, Indiana University, Bloomington, IN, USA*
²*Dept. of Developmental Physiology, NIPS, Okazaki, Japan*
³*Dept. of Physiological Sciences, Graduate University for Advanced Studies (SOKENDAI), Hayama, Japan*
- P5 Functional Identification of an Antennal Lobe DM4 Projection Neuron of the Fruit Fly**
Aurel A. Lazar^{*}, Chung-Heng Yeh
Department of Electrical Engineering, Columbia University, New York, NY 10027, USA

- P6 Neural Pathway Prediction based on Multi-neuron Spike Train Data**
Yi Zeng^{1*}, Tielin Zhang^{1,2}, and Bo Xu¹
¹*Institute of Automation, Chinese Academy of Sciences, Beijing, China*
²*University of Chinese Academy of Sciences, Beijing, China*
- P7 Neural Spike Prediction based on Spreading Activation**
Tielin Zhang^{1,2*}, Yi Zeng¹, and Bo Xu¹
¹*Institute of Automation, Chinese Academy of Sciences, Beijing, China*
²*University of Chinese Academy of Sciences, Beijing, China*
- P8 “Adaptive learning” as a mechanistic candidate for reaching optimal task-set representations flexibly**
Salva Ardid*, Matthew Balcarras, and Thilo Womelsdorf
Department of Biology and Center for Vision Research, York University, Toronto, Ontario, Canada, M3J 1P3
- P9 Reservoir of neurons with adaptive time constants: a hybrid model for robust motor-sensory temporal processing**
Sakyasingha Dasgupta^{1,3*}, Poramate Manoonpong^{2,3}, and Florentin Wörgötter^{1,3}
¹*III. Institute for Physics – Biophysics, Georg-August University, Göttingen, Germany*
²*Bernstein Center for Computational Neuroscience, Göttingen, Germany*
³*Mærsk Mc-Kinney Møller Institute, University of Southern Denmark, Odense, Denmark*
- P10 The Association between cell assemblies and transient dynamics**
Christian Tetzlaff^{1,2*}, Sakyasingha Dasgupta^{1,2}, and Florentin Wörgötter^{1,2}
¹*III. Institute for Physics - Biophysics, Georg-August University, Göttingen, Germany*
²*Bernstein Center for Computational Neuroscience, Göttingen, Germany*
- P11 Noise-induced speed up in repetitively firing neurons occurs far from spike threshold**
Todd Troyer^{1*}, David Barraza¹, Michael Farries², and Charles Wilson¹
¹*Biology Department, University of Texas, San Antonio, TX 78249, USA*
²*Dept. of Psychology, University of Michigan, Ann Arbor, MI, 48109, USA*
- P12 Variability in respiratory rhythm generation: in vitro and in silico models**
Chris Fietkiewicz^{1*}, Christopher Wilson²
¹*Dept. of Elec. Eng. and Comp. Sci., Case Western Reserve University, Cleveland, OH, 44107, USA*
²*Division of Physiology, School of Medicine, Loma Linda University, Loma Linda, CA, 92350, USA*

- P13 The effects of interactions between intrinsic properties and network parameters on bilateral phasing in a reduced leech heartbeat system**
Adam Weaver*
Department of Biology, Saint Michael's College, Colchester, VT 05439, USA
- P14 The “tweaking principle” for task switching**
Salva Ardid^{1,2*}, Xiao-Jing Wang^{1,3}
¹*Department of Neurobiology and Kavli Institute for Neuroscience, Yale University, New Haven, Connecticut 06510, USA*
²*Department of Biology and Center for Vision Research, York University, Toronto, Ontario, Canada, M3J 1P3*
³*Center for Neural Science, New York University, New York, New York 10003, USA*
- P15 Non-selective excitatory feedback and precise spike timing produce selective relative inhibition**
Biao Han^{1,2*}, Rufin Vanrullen^{1,2}
¹*Center de Recherche Cerveau et Cognition, Université de Toulouse, Toulouse, 31062, France*
²*CNRS, UMR 5549, Faculté de Médecine de Purpan, CHU Purpan, Toulouse Cedex, 31052, France*
- P16 Two-dimensional patterns in neural fields subject to finite transmission speed**
Eric Nichols^{1*}, Kevin Green^{1,2}, Axel Hutt¹, and Lennaert van Veen²
¹*INRIA Nancy, Team NeuroSys, 615 rue du Jardin Botanique, 54600 Villers-lès-Nancy, France*
²*Faculty of Science, University of Ontario Institute of Technology, 2000 Simcoe Street North, Oshawa, L1H 7K4 Ontario, Canada*
- P17 Homeostatic structural plasticity – a key to neuronal network formation and repair**
Markus Butz^{1*}, Arjen van Ooyen²
¹*Simulation Lab Neuroscience, IAS, Jülich Aachen Research Alliance, Forschungszentrum Jülich*
²*Computational Neuroscience Group, Neuroscience Campus Amsterdam, VU Universiteit Amsterdam*
- P18 Connectivity from spike trains of neocortex neuron populations**
Mark Hereld^{1,2*}, Jyothsna Suresh³, Mihailo Radojicic³, Lorenzo Pesce^{2,3}, and Wim van Drongelen^{1,3}
¹*Computation Institute, The University of Chicago and Argonne National Laboratory, IL, USA*
²*Mathematics and Computer Science, Argonne National Laboratory, Argonne, IL, USA*
³*Department of Pediatrics, The University of Chicago, Chicago, IL, USA*

- P19 Boundary Effects Across Filter Spatial Scales**
 Calden Wloka^{1,2*}, Neil Bruce³, and John Tsotsos^{1,2}
¹*Electrical Engineering and Computer Science, York University, Toronto, ON, Canada, M3J 1P3*
²*Center for Vision Research, York University, Toronto, ON, Canada, M3J 1P3*
³*Department of Computer Science, University of Manitoba, Winnipeg, MB, Canada, R3T 2N2*
- P20 Modelling of Neocortical Neural Dynamics during Human Focal Seizures**
 Ernest Ho^{1,2*}, Wilson Truccolo^{1,2}
¹*Department of Neuroscience, Brown University, Providence, RI, 02912, USA.*
²*Center for Neurorestoration and Neurotechnology, Department of Veterans Affairs, Providence, RI, USA.*
- P21 A digital hardware design for real-time simulation of large neural-system models in physical settings**
 Murphy Berzish^{*}, Bryan P Tripp
Center for Theoretical Neuroscience, University of Waterloo, Waterloo, Ontario, Canada, N2L 3G1
- P22 Fast approximate models of large networks**
 Bryan P Tripp^{*}
Center for Theoretical Neuroscience, University of Waterloo, Waterloo, ON, Canada, N2L 3G1
- P23 Optimal activity, avalanches and criticality in a model of the Primary Visual Area**
 Germano S Bortolotto¹, Jheniffer J Gonsalves¹, Mauricio Girardi-Schappo^{1*}, Thiago P Da Silva², Manasses P Nóbrega², Leonel T Pinto², and Marcelo H R Henrique Tragtenberg¹
¹*Department of Physics, Federal University of Santa Catarina, 88040-900, Florianópolis, SC, Brazil*
²*Department of Chemical Engineering and Food Engineering, Federal University of Santa Catarina, 88040-900, Florianópolis, SC, Brazil*
- P24 A map-based logistic neuron model: an efficient way to obtain many different neural behaviors**
 Rafael V Stenzinger, Jheniffer J Gonsalves, Mauricio Girardi-Schappo^{*}, and Marcelo H R Henrique Tragtenberg
Physics Department, Federal University of Santa Catarina, Florianópolis, SC – 88040-900, Brazil

- P25 Evolutionary algorithm search for connectivity patterns conducive to bursting in respiratory neural networks**
 Daniel Robb^{1*}, Maya Shende¹, Peter F Griffin¹, and Natalia Toporikova²
¹*Department of Mathematics, Computer Science and Physics, Roanoke College, Salem, VA 24153, USA*
²*Department of Biology, Washington and Lee University, Lexington, VA 24450, USA*
- P26 Asynchronous Coding in Neuronal Networks**
 Eric Kuebler*, Jean-Philippe Thivierge
School of Psychology, University of Ottawa, Ottawa, Ontario, Canada, K1N 6R5
- P27 Decision-making in a population of spiking neurons shaped by dynamics of intrinsic noise**
 Lydia Richardson^{1*}, Jean-Philippe Thivierge²
¹*Department of Biomedical Sciences, University of Ottawa, Ottawa, Ontario, Canada, K1N 9A8*
²*Department of Psychology, University of Ottawa, Ottawa, Ontario, Canada, K1N 9A8*
- P28 A reverse-engineering approach to building our understanding of nervous systems**
 Herve Thevenon*
Imezio Ltd, Wellington, New Zealand
- P29 Parallel Spike Trains Analysis using Positive Definite Kernels**
 Taro Tezuka*
Faculty of Library, Information, and Media Science, University of Tsukuba, Tsukuba, 305-0821, Japan
- P30 Self-organized cell assembly formation**
 Timo Nachstedt^{1,2*}, Florentin Wörgötter^{1,2}, and Christian Tetzlaff^{1,2}
¹*Third Institute of Physics, Georg-August-Universität, Göttingen, 37077, Germany*
²*Bernstein Center for Computational Neuroscience, Göttingen, 37077, Germany*
- P31 Development of avalanches and efficient communication in neuronal networks**
 Jean-Philippe Thivierge^{1*}, Joseph Tauskela²
¹*School of Psychology and Center for Neural Dynamics, University of Ottawa, Ottawa, Ontario K1N 6N5, Canada*
²*Human Health Therapeutics, National Research Council, Ottawa, Ontario K1A 0R6, Canada*

- P32 Modeling emotion-creativity interaction following brief training**
Xiaiqan Ding¹, Rongxiang Tang², Changhao Jiang³, and Yi-Yuan Tang^{4*}
¹*Department of Physics, Dalian University of Technology, Dalian 116024, China*
²*Department of Psychology, University of Texas at Austin, Austin, TX78705, USA*
³*Capital University of Physical Education and Sports, Beijing 100191, China*
⁴*Department of Psychology, Texas Tech University, Lubbock, TX79409, USA*
- P33 Brief meditation increases fiber wiring between striatum and corona radiata**
Yi-Yuan Tang^{1*}, Huiyan Shao¹, and Rongxiang Tang²
¹*Department of Psychology, Texas Tech University, Lubbock, TX 79409, USA*
²*Department of Psychology, University of Texas at Austin, Austin, TX 78705, USA*
- P34 Optical imaging of prefrontal cortex hemodynamic response in executive function induced by increased cardiovascular activity**
Nicoladie D Tam*
Department of Biological Sciences, University of North Texas, Denton, TX 76203, USA
- P35 Computational optimization problems in social interaction and empathic social emotion**
Nicoladie D Tam*
Department of Biological Sciences, University of North Texas, Denton, TX 76203, USA
- P36 The spatial structure of correlations in natural scenes shapes neural coding in mouse primary visual cortex**
Rajeev V Rikhye*, Mriganka Sur
Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology, Cambridge, MA 02139, USA
- P37 Some computational and comparative views derived from the quantitative analysis of the cerebellar structure**
Fahad Sultan*
Department of Cognitive Neurology, University Tübingen, Tübingen, 72076, Germany
- P38 Computational Modeling of the Development of Detailed Facial Representations along Ventral Pathway**
Akihiro Eguchi*, Simon Stringer
Oxford Center for Theoretical Neuroscience and Artificial Intelligence, University of Oxford, OX1 3UD, UK

- P39 Combining machine learning and simulations of a morphologically realistic model to study modulation of neuronal activity in cerebellar nuclei during absence epilepsy**
Parimala Alva^{1*}, Lieke Kros², Oscar H J Eelkman Rooda², Chris I De Zeeuw³, Rod Adams¹, Neil Davey¹, Freek E Hoebeek², and Volker Steuber¹
¹*Science and Technology Research Institute, University of Hertfordshire, Hatfield AL10 9AB, UK*
²*Department of Neuroscience, Erasmus Medical Center, Rotterdam, The Netherlands*
³*Netherlands Institute for Neuroscience, Royal Dutch Academy for Arts and Sciences, Amsterdam, Netherlands*
- P40 Information theoretical analysis of differences in information transmission in cerebellar Purkinje cells across species**
Kirsty Kidd*, James Bower, Daniel Polani, Neil Davey, and Volker Steuber
Science and Technology Research Institute, University of Hertfordshire, Hatfield, Hertfordshire, AL10 9AB, UK
- P41 A system for automated analysis of conductance correlations involved in recovery of electrical activity after neuromodulator deprivation in stomatogastric neuron models**
Atish Malik¹, Astrid A Prinz², and Tomasz G Smolinski^{1*}
¹*Department of Computer and Information Sciences, Delaware State University, Dover, DE 19901, USA*
²*Department of Biology, Emory University, Atlanta, GA 30322, USA*
- P42 Network models provide insight into how oriens-lacunosum-moleculare (OLM) and bistratified cell (BSC) interactions influence local CA1 theta rhythms**
Katie Ferguson^{1,2}, Carey Huh³, Bénédicte Amilhon³, Sylvain Williams³, and Frances Skinner^{1,4,2*}
¹*Toronto Western Research Institute, University Health Network, Toronto, Ontario, M5T 2S8, Canada*
²*Physiology, University of Toronto, Toronto, Ontario, M5S 1A1, Canada*
³*Psychiatry, Douglas Mental Health University Institute, McGill University, Montreal, Quebec, H4G 1X6, Canada*
⁴*Medicine (Neurology), University of Toronto, Toronto, Ontario, M5S 1A1, Canada*

P43 Non-uniform dendritic distributions of Ih channels in experimentally-derived multi-compartment models of oriens-lacunosum/moleculare hippocampal interneurons

Vladislav Sekulic^{1,2*}, Tse-Chiang Chen^{4,1}, John Lawrence^{5,6}, and Frances Skinner^{1,3,2}

¹*Toronto Western Research Institute, University Health Network, Toronto, Ontario, M5T 2S8, Canada*

²*Department of Physiology, University of Toronto, Ontario, M5S 2J7, Canada*

³*Department of Medicine (Neurology), University of Toronto, Ontario, M5S 2J7, Canada*

⁴*Department of Medical Biophysics, University of Toronto, Ontario, M5S 2J7, Canada*

⁵*Department of Biomedical and Pharmaceutical Sciences, University of Montana, Missoula, Montana, 59812, USA*

⁶*NIH COBRE Center for Structural and Functional Neuroscience, University of Montana, Missoula, Montana, 59812, USA*

P44 Modeling interneuron-specific (IS) interneurons in hippocampus

Alexandre Guet-McCreight^{1,2*}, Olivier Camiré³, Lisa Topolnik³, and Frances Skinner^{1,4,2}

¹*Toronto Western Research Institute, University Health Network, Toronto, Ontario, M5T 2S8, Canada*

²*Physiology, University of Toronto, Toronto, Ontario, M5S 1A1, Canada*

³*Biochemistry, Microbiology and Bioinformatics, Université Laval, Québec City, Québec, Canada, G1J 2G3*

⁴*Medicine (Neurology), University of Toronto, Toronto, Ontario, M5S 1A1, Canada*

P45 Automated code generation from LEMS, the general purpose model specification language underpinning NeuroML2

Boris Marin^{1,2*}, Pdraig Gleeson¹, Matteo Cantarelli^{1,3}, Robert Cannon⁴, and Angus Silver¹

¹*Department of Neuroscience, Physiology and Physiology, University College London, London, UK*

²*CAPES Foundation, Ministry of Education of Brazil, Brasilia, DF, Brazil*

³*Metacell LLC, San Diego, CA*

⁴*Textensor Limited, Edinburgh, UK*

P46 Neural Representation of Interval Timing Using Electroconvulsive Therapy

Jonathan Flynn^{1*}, Nitin Tandon², and Harel Shouval¹

¹*Department of Neurobiology and Anatomy, UTHSC at Houston, TX 77030, USA*

²*Department of Neurosurgery, UTHSC at Houston, TX 77030, USA*

- P47 Noise- and stimulus-dependence of the optimal encoding nonlinearities in a simple ON/OFF retinal circuit model**
 Braden Brinkman^{1,2*}, Alison Weber^{1,2,3}, Fred Rieke^{2,3,4}, and Eric Shea-Brown^{1,2,3}
¹*Department of Applied Mathematics, University of Washington, Seattle, WA 98195, USA*
²*Department of Physiology and Biophysics, University of Washington, Seattle, WA 98195, USA*
³*Program in Neurobiology and Behavior, University of Washington, Seattle, WA 98195, USA*
⁴*Howard Hughes Medical Institute, University of Washington, Seattle, WA 98195, USA*
- P48 Structured chaos shapes joint spike-response noise entropy in temporally driven balanced networks**
 Guillaume Lajoie^{1,3*}, Jean-Philippe Thivierge², and Eric Shea-Brown³
¹*Dept. of Nonlinear Dynamics, Max Planck Institute for Dynamics and Self-Organization, Göttingen, 37018, Germany*
²*Dept. of Psychology, University of Ottawa, Ottawa, Ontario, Canada, K1N 6N5*
³*Dept. of Applied Mathematics, University of Washington, Seattle, Washington, 98195, USA*
- P49 When does recurrent connectivity improve neural population coding?**
 Joel Zylberberg^{1*}, Eric Shea-Brown^{1,2}
¹*Department of Applied Mathematics, University of Washington, Seattle, WA 98195, USA*
²*Department of Physiology and Biophysics, University of Washington, Seattle, WA 98195, USA*
- P50 Spiking neural network model of cortical auditory source segregation**
 Lakshmi Krishnan^{1*}, Michael Campos², and Shihab Shamma^{1,3}
¹*Department of Electrical and Computer Engineering, University of Maryland, College Park, MD 20783, USA*
²*Qualcomm Research, San Diego, CA 92121, USA*
³*Department Etude Cognitive, Ecole Normale Suprieure, Paris 75005, France*
- P51 Quadratic programming by spiking neuronal networks**
 Ruben Moreno-Bote^{1,2*}, Philipp Schustek¹
¹*Research Unit, Parc Sanitari Sant Joan de Deu and Universitat de Barcelona, Esplugues de Llobregat, Barcelona, Spain, 08950*
²*Centro de Investigación Biomédica en Red de Salud Mental (CIBERSAM), Esplugues de Llobregat, Barcelona, Spain, 08950*
- P52 Auditory stimulation modulates amygdala network dynamics**
 Francois Windels*, Peter Stratton, and Pankaj Sah
Queensland Brain Institute, The University of Queensland, Brisbane, Queensland

- P53 Mathematical modeling and analysis of spinal circuits involved in locomotor pattern generation and frequency-dependent left-right coordination**
 Yaroslav Molkov¹, Bartholomew Bacak², and Ilya Rybak^{2*}
¹*Department of Mathematical Sciences, Indiana University – Purdue University Indianapolis, IN 46202, USA*
²*Department of Neurobiology and Anatomy, Drexel University College of Medicine, Philadelphia, PA 19129, USA*
- P54 Understanding and Distinguishing Three Time Scale Oscillations**
 Yangyang Wang^{1*}, Pingyu Nan², Vivien Kirk², and Jonathan Rubin¹
¹*Department of Mathematics, University of Pittsburgh, Pittsburgh, PA 15260, USA*
²*Department of Mathematics, University of Auckland, Auckland, 1142, New Zealand*
- P55 Multiple rhythms from one network: phase plane and stochastic analyses of rhythmic activity in turtle motor circuits**
 Abigail Snyder*, Jonathan Rubin
Department of Mathematics, University of Pittsburgh, Pittsburgh, PA 15213, USA
- P56 Measuring Synchronous Bursting and Spiking under Varying Second Order Network Connectivity Statistics**
 David Burstein*, Jonathan Rubin
Department of Mathematics, University of Pittsburgh, Pittsburgh, PA 15217, USA
- P57 The response of the subthalamo-pallidal networks of the Basal Ganglia to oscillatory cortical input in Parkinson’s disease**
 Sungwoo Ahn¹, S. Elizabeth Zauber², Robert Worth^{1,3}, and Leonid Rubchinsky^{1,4*}
¹*Department of Mathematical Sciences, Indiana University Purdue University Indianapolis, IN 46032, USA*
²*Department of Neurology, Indiana University School of Medicine, Indianapolis, IN 46202, USA*
³*Department of Neurological Surgery, Indiana University School of Medicine, Indianapolis, IN 46202, USA*
⁴*Stark Neurosciences Research Institute, Indiana University School of Medicine, Indianapolis, IN 46032, USA*
- P58 β CaMKII regulates bidirectional long-term plasticity in cerebellar Purkinje cells by a CaMKII/PP2B switch mechanism**
 Thiago M Pinto^{1,2*}, Maria Schilstra², Volker Steuber², and Antonio C Roque¹
¹*Departamento de Física, FFCLRP, Universidade de São Paulo, Ribeirão Preto, SP, 14040-901, Brazil*
²*Science and Technology Research Institute, University of Hertfordshire, Hatfield, Herts, AL10 9AB, UK*

- P59 Electrical coupling in the retina ganglion cell layer increases the dynamic range**
Cesar Celis, Rodrigo Publio, and Antonio C Roque*
Department of Physics, FFCLRP, University of Sao Paulo, Ribeirao Preto, Sao Paulo, Brazil
- P60 Differential effects of stimulus strength and volitional control in bistable perception**
James Rankin^{1*}, John Rinzel^{1,2}
¹*Center for Neural Science, New York University, 4 Washington Place, 10003 New York, NY*
²*Courant Institute of Mathematical Sciences, New York University, 251 Mercer St, 10012 New York, NY*
- P61 Long-term plasticity determines the postsynaptic response to correlated afferents with multivesicular short-term synaptic depression**
Alexander Bird^{1,2,3*}, Magnus Richardson¹
¹*Warwick Systems Biology Center, University of Warwick, Coventry CV4 7AL, UK*
²*Warwick Systems Biology DTC, University of Warwick, Coventry CV4 7AL, UK*
³*School of Life Sciences, University of Warwick, Coventry CV4 7AL, UK*
- P62 Network dynamics contribute to a gamma rhythm highly robust to synaptic variation**
Steven Hauser¹, Mark Reimers^{2*}
¹*University of Virginia, Charlottesville, VA 22903, USA*
²*Virginia Institute of Psychiatric and Behavioral Genetics, Virginia Commonwealth University, Richmond, VA 23298, USA*
- P63 Muscarinic metabotropic receptor M4 modulates the Hippocampal CA1 LTP possibly through local GABAergic interneurons**
Querusche Zanona^{1*}, Flávia Boos¹, Ana Paula Crestani¹, Johanna Duran¹, Maria Elisa Calcagnotto^{1,2}, and Jorge Quillfeldt^{1,3}
¹*Neuroscience Graduate Program, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, 90040-060, Brazil*
²*Biochemistry Department, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, 90035-003, Brazil*
³*Biophysics Department, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, 90540-000, Brazil*

- P64 Estimation of artificial neuron parameters that obtain a required distribution of coupled system periods in a hybrid network**
 Ryan Hooper^{1*}, Ruben Tikidzhi-Khamburyan², Carmen Canavier^{2,3}, and Astrid A Prinz⁴
¹*Dept. of Biomedical Engineering, Georgia Tech./Emory Univ., Atlanta, GA 30332*
²*Dept. of Cell Biol. and Anat., Louisiana State Univ. Hlth. Sci. Ctr., New Orleans, LA 70112*
³*Neurosci. Ctr. for Excellence, Louisiana State Univ. Hlth. Sci. Ctr., New Orleans, LA 70112*
⁴*Dept. of Biology, Emory Univ., Atlanta, GA 30322*
- P65 Estimation of spike initiation zone and synaptic input parameters of a Drosophila motoneuron using a morphologically reconstructed model**
 Cengiz Gunay^{1,2*}, Astrid A Prinz¹
¹*Dept. Biology, Emory University, Atlanta, Georgia 30322, USA*
²*Dept. Math and Computer Sci., Emory University, Atlanta, Georgia 30322, USA*
- P66 Auditory Object Feature Maps with a Hierarchical Network of Independent Components?**
 Jean Rouat*, Simon Brodeur, and Eric Plourde
NECOTIS, Département génie électrique, génie informatique, Université de Sherbrooke, Québec, Canada, J1K 2R1
- P67 The effect of trained parameters in Bayesian neural encoding models for the auditory system**
 Eric Plourde*
Department of Electrical and Computer Engineering, Université de Sherbrooke, Sherbrooke, Québec, J1K 2R1, Canada
- P68 A simple model for eletrocommunication – “Refractoriness Avoidance Response”?**
 Rafael Tuma Guariento^{1*}, Thiago S Mosqueiro¹, Angel A Caputi², and Reynaldo D Pinto¹
¹*Instituto de Física de São Carlos, Universidade de São Paulo, São Carlos, SP 13566-590, BR*
²*Department of Integrative and Computational Neurosciences, Instituto de Investigaciones Biológicas Clemente Estable, Montevideo, Uruguay*
- P69 Afferent-hair cell connectivity as a possible source of spike train irregularity in turtle vestibular bouton afferents**
 Bill Holmes*, Janice Huwe, Michael Rowe, and Ellengene Peterson
Department of Biological Sciences, Neuroscience Program, Ohio University, Athens, OH 45701, USA

- P70 Neural dynamics of perceptual detection under temporal uncertainty**
 Federico Carnevale^{1*}, Omri Barak², Victor De Lafuente³, Ranulfo Romo^{4,5}, and Néstor Parga¹
¹*Departamento de Física Teórica, Universidad Autónoma de Madrid, Cantoblanco 28049, Madrid, Spain*
²*Faculty of Medicine, Technion - Israel Institute of Technology, Haifa 32000, Israel*
³*Instituto de Neurobiología, Universidad Nacional Autónoma de México, Querétaro 76230, México*
⁴*El Colegio Nacional, 06020 México DF, México*
⁵*Instituto de Fisiología Celular, Universidad Nacional Autónoma de México, 04510 México DF, México*
- P71 The dopamine signal in decision-making tasks with stimulus and timing uncertainty**
 Stefania Sarno^{1*}, Victor De Lafuente², Ranulfo Romo^{3,4}, and Néstor Parga¹
¹*Departamento de Física Teórica, Universidad Autónoma de Madrid, Cantoblanco 28049, Madrid, Spain*
²*Instituto de Neurobiología, Universidad Nacional Autónoma de México, Querétaro 76230, México*
³*El Colegio Nacional, 06020 México DF, México*
⁴*Instituto de Fisiología Celular, Universidad Nacional Autónoma de México, 04510 México DF, México*
- P72 How Noise Correlations Impact the Amount of Information in Superior Colliculus: the Analysis of a Population with Shared Receptive Fields**
 Saba Farbodkia*, Kelly Shen, Gregory Day, and Martin Paré
Center for Neuroscience Studies, Queen's University, Kingston, Ontario, K7L 3N6, Canada
- P73 A dynamical systems model of the effect of Locus Coeruleus firing on single trial cortical state dynamics**
 Houman Safaai^{1*}, Ricardo Neves², Oxana Eschenko², Nikos Logothetis², and Stefano Panzeri¹
¹*Center for Neuroscience and Cognitive Systems, Istituto Italiano di Tecnologia, Corso Bettini 31, 38068 Rovereto, Italy*
²*Department of Physiology of Cognitive Processes, Max Planck Institute for Biological Cybernetics, 72076 Tübingen, Germany*
- P74 What is the effect of noise on the interval timing neural network?**
 Sorinel A Oprisan^{1*}, Derek Novo¹, and Catalin V Buhusi²
¹*Department of Physics and Astronomy, College of Charleston, Charleston, SC 29424, USA*
²*Department of Psychology, Utah State University, Logan, UT 84322, USA*

P75 Are phase resetting curves tunable?

Sorinel A Oprisan*, Davy Vanderweyen, and Derek Tuck

Department of Physics and Astronomy, College of Charleston, Charleston, SC 29424, USA

Monday Posters
Posters P76 – P150

- P76 Dynamics of a network of excitatory and inhibitory neurons induced by depolarization block**
Christopher Kim, Duane Nykamp*
School of Mathematics, University of Minnesota, Minneapolis, MN, 55455, USA
- P77 Classifying chemical sensor data using GPU-accelerated bio-mimetic neuronal networks based on the insect olfactory system**
Alan Diamond¹, Michael Schmuker², Amalia Z. Berna³, Stephen Trowell³, and Thomas Nowotny^{1*}
¹*School of Engineering and Informatics, University of Sussex, Falmer Brighton, BN1 9QJ, UK*
²*Neuroinformatics & Theoretical Neuroscience, Inst. for Biology, Freie Universität Berlin, 14195 Berlin, Germany*
³*CSIRO Ecosystem Sciences and Food Futures Flagship, GPO Box 1700 Canberra, ACT 2601, Australia*
- P78 Increased striatal inhibition in the basal ganglia leads to phase-synchronized firing in a model of the globus pallidus externus-subthalamic nucleus network**
DRAWN Kanishka C Basnayake^{1*}, Taishin Nomura^{1,2}
¹*Division of Biophysical Engineering, School of Engineering Science, Osaka University, 560-8531, Japan.*
²*Department of Bioengineering, Graduate School of Engineering Science, Osaka University, 560-8531, Japan.*
- P79 Using multi-objective evolutionary algorithms to predict the parameters that determine membrane resonance in a biophysical model of bursting neurons**
David Fox^{1*}, Hua-An Tseng¹, Horacio G. Rotstein², and Farzan Nadim^{1,2}
¹*Department of Biological Sciences, NJIT-Rutgers University, Newark, NJ 07102, USA*
²*Department of Mathematical Sciences, NJIT, Newark, NJ 07102, USA*
- P80 Stochastic Modulation of Oscillatory Neural Activity**
Jérémie Lefebvre^{1*}, Axel Hutt², Kevin Whittingstall³, and Micah M. Murray¹
¹*Laboratory for Investigative Neurophysiology, Center Hospitalier Universitaire Vaudois, Lausanne, 1011, Switzerland*
²*INRIA CR Nancy - Grand Est, Team NEUROSYS, Villers-les-Nancy, 54600, France, EU*
³*Department of Diagnostic Radiology, University of Sherbrooke, Sherbrooke, Québec, Canada, J1K 2R1*

- P81 EEG study of the cortical representation and classification of the emotional connotations in words**
Yuqiao Gu^{1*}, Massimo Poesio^{1,2}, and Brian Murphy³
¹*CLIC, CIMeC - Center for Mind/Brain Sciences, Università degli Studi di Trento, Rovereto (TN), I – 38068, Italy*
²*School of Computer Science and Electronic Engineering, University of Essex, Colchester, CO7 9QZ, UK*
³*Knowledge & Data Engineering (EEECS) Queen’s University Belfast, UK*
- P82 A dynamic model for delta rhythm fit to high-frequency cortical activity data shows discrete functional connectivity in mouse cortex**
Mark Reimers^{1*}, Majid Mohajerani^{2,3}, and Timothy Murphy³
¹*Department of Psychiatry, Virginia Commonwealth University, Richmond, VA 23221, USA.*
²*Canadian Center for Behavioral Neuroscience, University of Lethbridge, Lethbridge, AB, Canada.*
³*Department of Psychiatry, University of BC, Vancouver, BC, Canada.*
- P83 Modeling task-specific manifestations of serotonin in Basal Ganglia using risk-based decision making**
B Pragathi Balasubramani¹, Srinivasa Chakravarthy^{1*}, Balaraman Ravindran², and Ahmed A Moustafa³
¹*Dept. of Biotechnology, Indian Institute of Technology Madras, Chennai 600036, Tamil Nadu, India*
²*Dept. of Computer Science, Indian Institute of Technology Madras, Chennai 600036, Tamil Nadu, India*
³*School of Social Sciences and Psychology, University of Western Sydney, Penrith NSW 2751, Australia*
- P84 The representation of semantic similarities between object concepts in the brain: a hypergraph-based model**
Skiker Kaoutar^{1*}, Mounir Maouene²
¹*LIST Laboratory, FST, Abdelmalek Essaadi’s University, Tangier, Morocco*
²*Department of computer science, ENSAT, Abdelmalek Essaadi’s University, Tangier, Morocco*

- P85 Temporal sequence learning via adaptation in biologically plausible Spiking Neural Networks**
 Renato Duarte^{1,2*}, Peggy Series², and Abigail Morrison^{1,3,4}
¹*Bernstein Center Freiburg, Albert-Ludwig University of Freiburg, Freiburg im Breisgau, 79104, Germany*
²*Institute for Adaptive and Neural Computation, School of Informatics, University of Edinburgh, Edinburgh, EH8 9AB, UK*
³*Institute of Neuroscience and Medicine (INM-6), Computational and Systems Neuroscience, Jülich Research Center, Jülich, 52425, Germany*
⁴*Institute of Cognitive Neuroscience, Faculty of Psychology, Ruhr-University Bochum, Bochum, 44801, Germany*
- P86 Calcium current improves coincidence detection of the LIF model**
 Yansong Chua^{1,2*}, Moritz Helias¹, and Abigail Morrison^{1,2,3}
¹*Institute of Neuroscience and Medicine (INM-6) and Institute for Advanced Simulation (IAS-6), Juelich Forschungszentrum, Juelich, Germany*
²*Bernstein Center Freiburg, Albert-Ludwigs University, Freiburg im Breisgau, Germany*
³*Institute for Cognitive Neuroscience, Faculty of Psychology, Ruhr University of Bochum, Bochum, Germany*
- P87 Ephaptic Synchronization as a Mechanism for Selective Amplification of Stimuli**
 Aman Chawla*, Salvatore Morgera
Department of Electrical Engineering, University of South Florida, Tampa, FL33620, USA
- P88 A theory of decision-making using diffusion-to-bound models: choice, reaction-time and confidence**
 Philipp Schustek¹, Ruben Moreno-Bote^{1,2*}
¹*Research Unit, Parc Sanitari Sant Joan de Deu and Universitat de Barcelona, Esplugues de Llobregat, Barcelona, Spain, 08950*
²*Centro de Investigación Biomédica en Red de Salud Mental (CIBERSAM), Esplugues de Llobregat, Barcelona, Spain, 08950*
- P89 Bursting neurons in the hippocampal formation convey information about LFP features**
 Maria Constantinou^{1*}, Daniel Elijah¹, Daniel Squirrell¹, Inés Samengo², John Gigg¹, and Marcelo Montemurro¹
¹*Faculty of Life Sciences, University of Manchester, Manchester, M13 9PT, UK*
²*Centro Atómico Bariloche and Instituto Balseiro, San Carlos de Bariloche, 8400, Argentina*

- P90 Scaling of spike-timing based neuron model for mammalian olfaction with network size**
Bolun Chen¹, Jan Engelbrecht^{1*}, and Renato Mirollo²
¹*Physics Department, Boston College, Chestnut Hill, MA 02467, USA*
²*Mathematics Department, Boston College, Chestnut Hill, MA 02467, USA*
- P91 Splay states in networks of identical integrate-and-fire neurons**
Jan Engelbrecht^{1*}, Bolun Chen¹, and Renato Mirollo²
¹*Physics Department, Boston College, Chestnut Hill, MA 02467, USA*
²*Mathematics Department, Boston College, Chestnut Hill, MA 02467, USA*
- P92 The computational properties of a simplified cortical column model**
Nicholas Cain*, Ram Iyer, Christof Koch, and Stefan Mihalas
Allen Institute for Brain Science, Seattle, WA 98103, USA
- P93 Entorhinal cortex stellate cell synchronization**
Patrick Crotty*, Betty Anderson, Mary Rose Devine, and Anna Miettinen
Department of Physics and Astronomy, Colgate University, NY 13346, USA
- P94 Bifurcation analysis of anti-phase oscillations and synchrony in the tadpole central pattern generator**
Roman Borisyuk, Robert Merrison-Hort*
School of Computing & Mathematics, Plymouth University, Plymouth, Devon, PL4 8AA, UK
- P95 A linear-nonlinear model accurately predicts cortical responses to simultaneous electrical stimulation with a retinal implant**
Kerry Halupka^{1,2}, Mohit Shivdasani⁴, Shaun Cloerty^{5,6}, David Grayden^{1,2,3,4}, Anthony N Burkitt^{1,2,3,4}, and Hamish Meffin^{3,1,2*}
¹*Neural Engineering Laboratory, Dept. of Electrical & Electronic Engineering, University of Melbourne, Parkville, VIC 3010*
²*Center for Neural Engineering Laboratory, University of Melbourne, Parkville, VIC 3010*
³*National ICT Australia, Victoria Research Lab, University of Melbourne, Parkville, VIC 3010*
⁴*Bionics Institute, 384-388 Albert St, East Melbourne, VIC 3002*
⁵*National Vision Research Institute, Australian College of Optometry, Carlton, VIC 3053.*
⁶*Dept. Optometry and Vision Sciences, University of Melbourne, Parkville, VIC 3010.*

P96 Spike history model for neural control

Tatiana Kameneva^{1,2,3*}, Miganoosh Abramian⁴, David Grayden^{1,2,3,5}, Anthony N Burkitt^{1,2,3,5}, and Hamish Meffin^{1,2,3}

¹*NeuroEngineering Laboratory, Department of Electrical Electronic Engineering, The University of Melbourne, Australia*

²*Center for Neural Engineering, The University of Melbourne, Australia*

³*National ICT Australia, Victoria Research Lab, Australia*

⁴*Graduate School of Biomedical Engineering, The University of New South Wales, Australia*

⁵*Bionics Institute, East Melbourne, Australia*

P97 A computational model on the goldfish Mauthner cell

Tuomo Mäki-Marttunen^{1,2*}, Violeta Medan^{1,3}

¹*Departamento de Fisiología, Biología Molecular y Celular, Universidad de Buenos Aires, Argentina*

²*Department of Signal Processing, Tampere University of Technology, Finland*

³*Instituto de Fisiología, Biología Molecular y Neurociencias, Universidad de Buenos Aires - CONICET, Argentina*

P98 Modeling astrocyte-neuron interactions in a tripartite synapse

Marja-Leena Linne^{1*}, Riikka Havela¹, Ausra Saudargiene², and Liam McDaid³

¹*Computational Neuroscience Research Group, Department of Signal Processing, Tampere University of Technology, Tampere, Finland*

²*Department of Informatics, Vytautas Magnus University, Kaunas, Lithuania*

³*School of Computing and Intelligent Systems, University of Ulster, Northern Ireland*

P99 Partial correlation analysis for functional connectivity studies in cortical networks

Daniele Poli, Vito Paolo Pastore, Sergio Martinoia, and Paolo Massobrio*

Department of Informatics, Bioengineering, Robotics and Systems Engineering (DIBRIS), University of Genova, Genova, 16145, ITALY

P100 Global network community and non-uniform cell density in the macaque brain

Masanori Shimono^{1,2*}

¹*JSPS Fellow,*

²*Department of Physics, University of Indiana, Bloomington, IN, 47405, USA*

- P101 The Neuroscience Gateway Portal: High Performance Computing Made Easy**
 Ted Carnevale^{1*}, Amit Majumdar², Subha Sivagnanam², Kenneth Yoshimoto², Vadim Astakhov³, Anita Bandrowski³, and Maryann Martone⁴
¹*Neurobiology Department, Yale University Medical School, New Haven, CT 06520, USA*
²*San Diego Supercomputer Center, UC San Diego, La Jolla, CA 92093, USA*
³*Center for Research in Biological Systems, UC San Diego, La Jolla, CA 92093, USA*
⁴*Neuroscience Department, UC San Diego, La Jolla, CA 92093, USA*
- P102 Probability-Based Nonlinear Modeling of Neural Dynamical Systems with Point-Process Inputs and Outputs**
 Roman Sandler^{1*}, Dong Song¹, Robert Hampson³, Sam Deadwyler³, Theodore Berger¹, and Vasilis Marmarelis¹
¹*Department of Biomedical Engineering, University of Southern California, Los Angeles, CA 90089, USA*
²*Department of Physiology, Wake Forest School of Medicine, Winston-Salem, NC 27157, USA*
- P103 Temperature-robust neural activity using feedback control of ion channel expression**
 Timothy O’Leary*, Eve Marder
Volen Center for Complex Systems, Brandeis University, Waltham, MA 02454, USA
- P104 Enhanced Attention Precedes Self-initiated Locomotion in an Electric Fish**
 James Jun^{1,2,3*}, Andre Longtin^{1,2,3}, and Leonard Maler^{2,3}
¹*Department of Physics, University of Ottawa, Ottawa, ON, Canada, K1N 6N5*
²*Department of Cellular and Molecular Medicine, University of Ottawa, Ottawa, ON, Canada, K1H 8M5*
³*Center for Neural Dynamics of Physics, University of Ottawa, Ottawa, ON, Canada, K1H 8M5*
- P105 Brain dynamic functional connectivity in patients with disorders of consciousness**
 Veronica Mäki-Marttunen^{1,2*}
¹*Department of Neuroimaging, FLENI, Buenos Aires 1428, Argentina*
²*CONICET, Buenos Aires, Argentina*
- P106 Modulation of virtual arm trajectories via microstimulation in a spiking model of sensorimotor cortex**
 Salvador Dura-Bernal^{1*}, Kan Li², Austin Brockmeier², Cliff C Kerr¹, Samuel Neymotin¹, Jose Principe², Joseph Francis¹, and William W Lytton¹
¹*Department of Physiology and Pharmacology, SUNY Downstate, Brooklyn, NY 11203, USA*
²*Department of Electrical and Computer Engineering, University of Florida, Gainesville, FL 32611, USA*

P107 Network-level effects of optogenetic stimulation in a computer model of macaque primary motor cortex

Cliff C Kerr^{1,2*}, Daniel O'Shea³, Werapong Goo⁴, Salvador Dura-Bernal¹, Joseph Francis¹, Ilka Diester⁵, Paul Kalanithi⁶, Karl Deisseroth⁴, Krishna Shenoy⁴, and William W Lytton¹

¹*Department of Physiology and Pharmacology, SUNY Downstate Medical Center, Brooklyn, NY, USA*

²*Complex Systems Group, School of Physics, University of Sydney, Sydney, NSW, Australia*

³*Neurosciences Program, Stanford University, Stanford, CA, USA*

⁴*Department of Bioengineering, Stanford University, Stanford, CA, USA*

⁵*Ernst Strüngmann Institute, Frankfurt, Hessen, Germany*

⁶*Department of Neurosurgery, Stanford University, Stanford, CA, USA*

P108 Calcium regulation of HCN supports persistent activity associated with working memory: a multiscale model of prefrontal cortex

Samuel Neymotin^{1*}, Robert A McDougal², Michael Hines², and William W Lytton^{1,3}

¹*Department of Physiology & Pharmacology, SUNY Downstate, Brooklyn, NY, 11203, USA*

²*Department of Neurobiology, Yale Medical School, New Haven, CT, 06510, USA*

³*Department of Neurology, Kings County Hospital Center, Brooklyn, NY, 11203, USA*

P109 A method for multi-simulator reaction-diffusion with NEURON

Robert A McDougal^{1*}, Michael Hines¹, and William W Lytton^{2,3}

¹*Neurobiology, Yale University, New Haven, CT 06520, USA*

²*Physiology & Pharmacology, SUNY Downstate Medical Center, Brooklyn, NY 11203, USA*

³*Kings County Hospital, Brooklyn, NY 11203, USA*

P110 Modeling mGluR1 mediated synaptic depression in cerebellar Purkinje cells

Yizhen Su¹, Huo Lu^{2*}

¹*Doctor of Osteopathic Medicine – GA-Philadelphia College of Osteopathic Medicine, Suwanee, GA 30024, USA*

²*Department of Biomedical Sciences, Philadelphia College of Osteopathic Medicine, Suwanee, GA 30024, USA*

P111 Gain control via feedforward inhibition in noisy and delayed neural circuits

Jorge F Mejias^{1,2*}, Alexandre Payeur², Erik Selin², Leonard Maler^{3,4}, and Andre Longtin^{2,4}

¹*Center for Neural Science, New York University, New York, NY, 10012, USA.*

²*Department of Physics, University of Ottawa, Ottawa, ON, K1N6N5, Canada*

³*Department of Cellular and Molecular Medicine, University of Ottawa, Ottawa, ON, K1H8M5, Canada*

⁴*Center for Neural Dynamics, University of Ottawa, Ottawa, ON, K1N6N5, Canada*

P112 A phenomenological model for self-initiated movement in electric fish

Alexandre Melanson^{1,2*}, Jorge F Mejias³, James Jun^{1,2,4}, Leonard Maler⁴, and Andre Longtin^{1,2,4}

¹*Department of Physics, University of Ottawa, Ottawa, Ontario, Canada, K1N 6N5*

²*Center for Neural Dynamics, University of Ottawa, Ottawa, Ontario, Canada, K1N 6N5*

³*Center for Neural Science, New York University, New York, NY 10012, USA*

⁴*Department of Cellular and Molecular Medicine, University of Ottawa, Ottawa, Ontario, Canada, K1H 8M5*

P113 Modeling Sound Pulse Counting in Inferior Colliculus

Richard Naud^{1*}, Dave Houtman¹, Gary J. Rose², and Andre Longtin¹

¹*Department Physics, University of Ottawa, Ottawa, K1N 6N5, Canada*

²*Department Biology, University of Utah, Salt Lake City, UT, 84112, USA*

P114 Brain rhythms from delayed interaction of fluctuations

Alexandre Payeur^{1*}, Leonard Maler², and Andre Longtin^{1,2}

¹*Department of Physics, University of Ottawa, Ottawa, Canada, K1N 6N5*

²*Department of Cell and Molecular Medicine, University of Ottawa, Canada, K1H 8M5*

P115 Finite size effect induces stochastic gamma oscillation in inhibitory network with conduction delay

Gregory Dumont^{1,2,3*}, Georg Northoff^{2,3}, and Andre Longtin^{1,3}

¹*Physics Department, University of Ottawa, Canada*

²*Mind, Brain Imaging and Neuroethics, Royal Ottawa Healthcare, Institute of Mental Health Research, Ottawa, Canada*

³*Center for Neural Dynamics, University of Ottawa*

P116 Mechanism-based modeling of time-varying magnetic fields effects on cortical activity

Julien Modolo^{1,2,3*}, Alex W Thomas^{1,2,3}, and Alexandre Legros^{1,2,3,4}

¹*Human Threshold Research Group, Lawson Health Research Institute, London, ON, N6A4V2, Canada*

²*Department of Medical Biophysics, Western University, London, ON, Canada*

³*Department of Medical Imaging, Western University, London, ON, Canada*

⁴*School of Kinesiology, Western University, London, ON, Canada*

P117 WITHDRAWN: Mean field modeling of Basal ganglia – Functional consequences of network heterogeneity

Jyotika Bahuguna^{1,2*}, Mikael Lindahl^{2,3}, Jeanette Hellgren-Kotaleski^{2,3}, and Arvind Kumar¹

¹*Bernstein Center Freiburg, Faculty of Biology, University of Freiburg, Germany*

²*Computational Biology, School of Computer Science and Communication, KTH, Stockholm, Sweden*

³*Department Neuroscience, Karolinska Institute, Stockholm, Sweden.*

P118 Multimodal brain-computer interface communication in disorders of consciousness

Sebastian Halder*, Ivo Käthner, and Andrea Kübler

Institute of Psychology, University of Würzburg, 97070 Würzburg, Germany

P119 Fluctuation scaling in neural spike trains

Shinsuke Koyama^{1,2,3*}

¹*Department of Statistical Modeling, The Institute of Statistical Mathematics, Tokyo, Japan*

²*ERATO Sato Live Bio-Forecasting Project, Japan Science and Technology Agency, Kyoto, Japan*

³*Advanced Telecommunications Research Institute International (ATR), Kyoto, Japan*

P120 Rapid neural coding in the mouse retina with the first wave of spikes

Geoffrey Portelli^{1*}, John Barrett², Evelyne Sernagor², Timothée Masquelier^{3,4}, and Pierre Kornprobst¹

¹*Neuromathcomp, INRIA, Sophia Antipolis, 06902, France*

²*Institute of Neuroscience, Medical School, Newcastle University, Newcastle UK*

³*Institut de la Vision, UPMC Université Paris 06, Paris, 75012, France*

⁴*CNRS, UMR 7210, Paris, 75012, France*

P121 Microsaccades enable efficient synchrony-based visual feature learning and detection

Timothée Masquelier^{1,2*}, Geoffrey Portelli³, and Pierre Kornprobst³

¹*Institut de la Vision, UPMC Université Paris 06, Paris, 75012, France*

²*CNRS, UMR 7210, Paris, 75012, France*

³*Neuromathcomp Project Team, Inria Sophia Antipolis Méditerranée, 06902, France*

P122 Principles of high-fidelity, high-density 3-D neural recording

Caroline Moore-Kochlacs^{1,2*}, Jorg Scholvin⁴, Justin Kinney^{2,4}, Jacob Bernstein⁴, Young-Gyu Yoon⁵, Scott Arfin⁴, Nancy Kopell^{1,3}, and Ed S Boyden^{2,4,6,7}

¹*Graduate Program for Neuroscience, Boston University, Boston, MA 02215*

²*McGovern Institute, Massachusetts Institute of Technology, MA 02139*

³*Department of Mathematics and Statistics, Boston University, Boston, MA 02215*

⁴*Media Lab, Massachusetts Institute of Technology, MA 02139*

⁵*Electrical Engineering and Computer Science, Massachusetts Institute of Technology, MA 02139*

⁶*Brain and Cognitive Science, Massachusetts Institute of Technology, MA 02139*

⁷*Biological Engineering, Massachusetts Institute of Technology, MA 02139*

P123 Subthreshold resonance in biophysically-based models of low- and high-input WITH conductance motoneurons

DRAWN Vitor Chaud^{1,2*}, Andre Kohn¹

¹*Biomedical Engineering Laboratory, Dept. of Telecommunication and Control Engineering, Universidade de São Paulo, São Paulo, SP, Brasil*

²*Dept. of Electrical Engineering, Universidade Federal do Triângulo Mineiro, UFTM, Uberaba, MG, Brasil*

P124 Descriptive model for the prediction of motion direction from spike trains of ON-OFF directional selective retinal ganglion cells

Aurel Martiniuc^{1*}, Victor Bocos-Bintintan², Florian Roehrbein¹, and Alois Knoll¹

¹*Department of Computer Science, Technical University Munich, Garching, 85748, Germany*

²*Faculty of Environmental Science & Engineering, Babes-Bolyai University, Cluj-Napoca, 400429, Romania*

P125 Correlation between spike statistics and T-type calcium channel activation in simulated subthalamic nucleus neurons

Katsunori Kitano*

Department of Human and Computer Intelligence, Ritsumeikan University, Shiga 5258577, Japan

P126 Using fMRI to Characterize How Cortex Represents Limb Motions

Samir Menon^{1*}, Jack Zhu¹, Paul I Quigley¹, Franco Pestilli², Kwabena Boahen³, and Oussama Khatib¹

¹*Department of Computer Science, Stanford University, Stanford, CA, 94305, USA*

²*Department of Psychology, Stanford University, Stanford, CA, 94305, USA*

³*Department of Bioengineering, Stanford University, Stanford, CA, 94305, USA*

P127 Phase precession and the grid-to-place transformation

WITH Jorge Jaramillo^{1,2*}, Richard Kempner^{1,2}
DRAWN

¹*Department of Biology, Institute for Theoretical Biology, Humboldt-Universität zu Berlin, Berlin 10115, Germany*

²*Bernstein Center for Computational Neuroscience Berlin, Berlin 10115, Germany*

P128 Memory association dynamics on neural network with dynamic synapses

Yuichi Katori*

Institute of Industrial Science, The University of Tokyo,

P129 The emergence of cohorts of co-active neurons in random recurrent networks provides a mechanism for orientation and direction selectivity

Dmitry Tsigankov*, Matthias Kaschube

Frankfurt Institute for Advanced Studies, Frankfurt, Germany

P130 Modelling spatially realistic local field potentials in spiking neural networks using the VERTEX simulation tool

Richard Tomsett^{1,2*}, Matt Ainsworth³, Alexander Thiele⁴, Mehdi Sanayei⁴, Xing Chen⁴, Alwin Gieselmann⁴, Miles Whittington³, Mark Cunningham⁴, and Marcus Kaiser^{1,4}

¹*School of Computing Science, Newcastle University, NE1 7RU, UK*

²*Institute of Ageing and Health, Newcastle University, NE4 5PL, UK*

³*Hull York Medical School, University of York, YO10 5DD, UK*

⁴*Institute of Neuroscience, Newcastle University, NE2 4HH, UK*

P131 Modelling local field potential features during network gamma oscillations

Richard Tomsett^{1,2*}, Matt Ainsworth³, Alexander Thiele⁴, Mehdi Sanayei⁴, Xing Chen⁴, Alwin Gieselmann⁴, Miles Whittington³, Mark Cunningham⁴, and Marcus Kaiser^{1,4}

¹*School of Computing Science, Newcastle University, NE1 7RU, UK*

²*Institute of Ageing and Health, Newcastle University, NE4 5PL, UK*

³*Hull York Medical School, University of York, YO10 5DD, UK*

⁴*Institute of Neuroscience, Newcastle University, NE2 4HH, UK*

P132 Deciphering the Axonal Transport Kinetics of Neurofilaments using the Fluorescence Photo-activation Pulse-Escape Method

Yinyun Li^{1,2*}, Anthony Brown³, and Peter Jung¹

¹*Department of Physics and Astronomy, Ohio University, Athens, OH 45701, USA*

²*III Institute of Physics-Biophysics, Georg-August-University Goettingen, Goettingen, 37077, Germany*

³*Department of Neuroscience, Ohio State University, Columbus, OH 43210, USA*

- P133 Computational model of human ventilation for electrical stimulation following cervical spinal cord injury**
Brian Hillen*, Ranu Jung
Department of Biomedical Engineering, Florida International University, Miami, Florida, 33174, USA
- P134 Stimulation-induced ectopicity and propagation windows in model damaged axons**
Mathieu Lachance^{1,2*}, Andre Longtin², Catherine E Morris³, Na Yu², and Béla Joos²
¹*Département de physique, Cégep de l'Outaouais – campus Félix-Leclerc, Gatineau, Québec J8T 7T7*
²*Ottawa-Carleton Physics Institute, University of Ottawa, Ottawa, Ontario, Canada K1N 6N5*
³*Neurosciences, Ottawa Hospital Research Institute, Ottawa, Ontario, Canada K1H 8M5*
- P135 Action potential initiation in damaged axon initial segment**
Louis Jacques^{1*}, Catherine E Morris², Andre Longtin¹, and Béla Joos¹
¹*Department of Physics, University of Ottawa, Ottawa, Ontario, Canada, K1N 6N5*
²*Neurosciences, Ottawa Hospital Research Institute, Ottawa, Ontario, Canada, K1H 8M5*
- P136 Fast rhythm cycles as atomic fragments of cortical processing and learning**
Jenia Jitsev*
Functional Neural Circuits Group, Institute of Neuroscience and Medicine (INM-6) & Institute of Advanced Simulation (IAS-6), Forschungszentrum Juelich, 52425 Juelich, Germany
- P137 Computational modeling of Temporal and Sequential Dynamics of Foraging Decisions**
Kanghoon Jung^{1,2}, Hye-Rann Jang¹, Jerald Kralik², and Jaeseung Jeong^{1*}
¹*Department of Bio and Brain Engineering, Korea Advanced Institute of Science and Technology (KAIST), Daejeon 305-701, Korea*
²*Department of Psychological and Brain Sciences, Dartmouth College, Hanover, NH 03755, USA*
- P138 Modeling the effects of neuronal morphology on dendritic chloride diffusion and GABAergic inhibition**
Namrata Mohapatra^{1*}, Fidel Santamaria², and Peter Jedlicka¹
¹*Institute of Clinical Neuroanatomy, Neuroscience Center, Goethe-University, Frankfurt, Germany*
²*Biology Department and Neurosciences Institute, The University of Texas at San Antonio, San Antonio, USA*

- P139 Bursting suppression in propofol-induced general anesthesia as bi-stability in a non-linear neural mass model**
 Pedro Ernesto Garcia Rodriguez*, Axel Hutt
INRIA CR Nancy-Grand Est, Equipe NeuroSys, France
- P140 Comparing calcium influx with high-frequency stimulation and burst stimulation LTP protocols**
 Ximing Li, Bill Holmes*
Department of Biological Sciences, Neurosciences Program, Ohio University, Athens, OH 45701, USA
- P141 Simulating Stimulus- and TMS-Induced Interference in Short-Term Memory Using a Model of Prefrontal Cortex**
 Tyler Bancroft*, William Hockley, Philip Servos, and Jeremy Hogeveen
Department of Psychology, Wilfrid Laurier University, Waterloo, Ontario, Canada, N2L 3C5
- P142 Spontaneous firing activity in climbing fiber is critical for a realistic bi-hemispherical cerebellar neuronal network during robot control**
 Ruben Pinzon Morales*, Yutaka Hirata
Department of Computer Science, Chubu University Graduate School of Engineering, Kasugai, 487-8501, Japan
- P143 The number of granular cells in a cerebellar neuronal network model engaged during robot control increases with the complexity of the motor task**
 Ruben Pinzon Morales*, Yutaka Hirata
Department of Computer Science, Chubu University Graduate School of Engineering, Kasugai, 487-8501, Japan
- P144 Integrating Systems Biology Markup Language (SBML) with NEURON**
 Anna Bulanova^{1*}, Robert A McDougal¹, Samuel Neymotin², Victor Mutai¹, William W Lytton^{2,3}, and Michael Hines¹
¹*Neurobiology, Yale University, New Haven, CT 06520, USA*
²*Physiology & Pharmacology, SUNY Downstate Medical Center, Brooklyn, NY 11203, USA*
³*Neurology, Kings County Hospital, Brooklyn, NY 11203, USA*
- P145 Dynamics of dichoptic masking in the primary visual cortex**
 Eva Chadnova^{1,2*}, Alexandre Reynaud¹, Simon Clavagnier¹, Daniel H. Baker³, Sylvain Baillet², and Robert F. Hess¹
¹*McGill Vision Research Unit, McGill University, Montreal, Quebec, H3A2T5, Canada*
²*McConnell Brain Imaging Center, Montreal Neurological Institute, McGill University, Montreal, Quebec, H3A2B4, Canada*
³*Department of Psychology, University of York, Heslington, York, YO10 5DD, UK*

P146 The transfer function of the LIF model: from white to filtered noise

Jannis Schuecker^{1*}, Markus Diesmann^{1,2,3}, and Moritz Helias^{1,2}

¹*Institute of Neuroscience and Medicine (INM-6) and Institute for Advanced Simulation (IAS-6), Jülich Research Center and JARA, Jülich, Germany*

²*RIKEN Brain Science Institute, Wako, Saitama, Japan*

³*Medical Faculty, RWTH Aachen University, Germany*

P147 Kisspeptin mediation of estradiol-induced secretion of Luteinizing Hormone and Prolactin

Natalia Toporikova^{1*}, Philip Dishuck¹, Joel Tabak², and Cleyde Helena²

¹*Biology Department, Washington and Lee University, Lexington, VA, 24450, USA*

²*Program in Neuroscience, Florida State University, Tallahassee, FL, 32306, USA*

P148 SpineML and Brian 2.0 interfaces for using GPU enhanced Neuronal Networks (GeNN)

Thomas Nowotny^{1*}, Alexander J Cope², Esin Yavuz¹, Marcel Stimberg³, Dan F M Goodman⁴, James Marshall², and Kevin Gurney⁵

¹*CCNR, School of Engineering and Informatics, University of Sussex, Falmer, Brighton BN1 9QJ, UK*

²*Department of Computer Science, University of Sheffield, Sheffield S1 4DP, UK*

³*Institut d'Etudes de la Cognition, Ecole Normale Supérieure, Paris, France*

⁴*Harvard Medical School, Harvard University, Boston, MA 02115, USA*

⁵*Department of Psychology, University of Sheffield, Sheffield S10 2TP, UK*

P149 A large-scale physiological model of Inferior Olive neurons reveals climbing fiber intra-burst frequency depends on Olivocerebellar axon morphology

James Kozloski^{1*}, John Wagner², Herald Memelli³, and Viatcheslav Gurev¹

¹*Computational Biology Center, IBM T.J. Watson Research Center*

²*IBM Research Collaboratory for Life Sciences-Melbourne, Carlton, Australia*

³*State University of New York at Stony Brook, NY, USA*

P150 Effects of short-term synaptic plasticity mechanisms on the dynamics of the network conductances

Catalina Vich Llompert^{1*}, Paolo Massobrio², and Antoni Guillamon³

¹*Department of Mathematics and Computer Science, Escola Politècnica Superior, Universitat de les Illes Balears, Mallorca, Palma, 07122*

²*Department of Informatics, Bioengineering, Robotics, System Engineering (DIBRIS), University of Genova, Genova, Italy*

³*Department of Applied Mathematics I, EPSEB, Universitat Politècnica de Catalunya, Barcelona*

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P151 Dissecting estimation of conductances in subthreshold regimes

Catalina Vich Llompert^{1*}, Antoni Guillamon²

¹*Dept. of Mathematics and Computer Science, Universitat de les Illes Balears, Palma, 07122, Spain.*

²*Dept. of Applied Mathematics I, EPSEB, Universitat Politècnica de Catalunya, 08028, Barcelona.*

P152 Seizure Dynamics: A Computational Model based Approach Demonstrating Variability in Seizure Mechanisms

Richard Balson^{1,2,3,4}, Dean Freestone^{1,2,3}, Mark Cook^{2,3}, Anthony N Burkitt^{1,2,4*}, and David Grayden^{1,2,4}

¹*NeuroEngineering Laboratory, Dept. of Electrical & Electronic Engineering, University of Melbourne, Parkville, Victoria, 3010, Australia*

²*Center for Neural Engineering, University of Melbourne, Carlton, Victoria, 3010, Australia*

³*Department of Medicine St. Vincent's Hospital Melbourne, University of Melbourne, Fitzroy, Victoria, 3065*

⁴*The Bionics Institute, East Melbourne, Victoria, Australia, 3002*

P153 A computational model of the stellate cell microcircuit in the auditory brainstem

Timothy Esler^{1,2}, David Grayden^{1,2*}

¹*NeuroEngineering Laboratory, Dept. of Electrical & Electronic Engineering, University of Melbourne, Victoria 3010, Australia*

²*Center for Neural Engineering, University of Melbourne, Victoria 3010, Australia*

P154 Nitric oxide activity-dependent regulator compensates synaptic depression and enhances metabolic efficiency in the auditory brainstem

Christophe Michel¹, Matthias Hennig², and Bruce Graham^{1*}

¹*Computing Science and Mathematics, University of Stirling, Stirling, Scotland, FK9 4LA, UK*

²*School of Informatics, University of Edinburgh, Edinburgh, Scotland, EH8 9AB, UK*

P155 Neural frequency distributions may generate a new phase transition in models for synchronization

Marcelo H R Henrique Tragtenberg*, Caio Tiedt, and Mauricio Girardi-Schappo

Department of Physics, Federal University of Santa Catarina, 88040-900, Florianópolis, SC, Brazil

P156 Coordination of adaptive working memory and reinforcement learning systems explaining choice and reaction time in a human experiment.

Guillaume Viejo^{1,2*}, Mehdi Khamassi^{1,2}, Andrea Brovelli³, and Benoît Girard^{1,2}

¹*Sorbonne Universités, UPMC, Univ Paris 06, UMR 7222, ISIR, F-75005, Paris, France*

²*CNRS, UMR 7222, ISIR, F-75005, Paris, France*

³*Institut de Neurosciences de la Timone (INT), UMR 7289, CNRS - Aix Marseille Université, Marseille*

P157 Stabilizing working memory in spiking networks with biologically plausible synaptic dynamics

Alexander Seeholzer*, Moritz Deger, and Wulfram Gerstner

School of Life Sciences, Brain Mind Institute and School of Computer and Communication Sciences, École polytechnique fédérale de Lausanne, 1015 Lausanne EPFL, Switzerland

P158 The role of interconnected hub neurons in cortical dynamics

Hesam Setareh*, Moritz Deger, and Wulfram Gerstner

School of Life Sciences, Brain Mind Institute and School of Computer and Communication Sciences, École polytechnique fédérale de Lausanne, 1015 Lausanne EPFL, Switzerland

P159 Neuromodulation by surprise: A biologically plausible model of the learning rate dynamics

Mohammadjavad Faraji*, Kerstin Preuschoff, and Wulfram Gerstner

School of Life Sciences, Brain Mind Institute and School of Computer and Communication Sciences, Ecole Polytechnique Federal de Lausanne, Lausanne, Switzerland

P160 Hebbian-inspired rewiring of a random network replicates pattern of selectivity seen in PFC

Grace Lindsay^{1*}, Mattia Rigotti¹, Melissa R. Warden², Earl K. Miller³, and Stefano Fusi¹

¹*Department of Neuroscience, Columbia University, New York, NY 10026, USA*

²*Department of Bioengineering, Stanford University, Stanford, CA 94305, USA*

³*The Picower Institute for Learning and Memory & Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology, Cambridge, MA 02139, USA*

P161 Optimization of Membrane Excitability for Predictive Homeostasis of Spike Generation

Jaekyung Kim*, Christopher Fiorillo

Bio and Brain Engineering, KAIST (Korea Advanced Institute of Science and Technology), Daejeon 305-701 Korea

P162 Nonlinear Variability Measures for Respiratory Rhythm Generation

Sameer Alsharif, Chris Fietkiewicz*

Dept. of Elec. Eng. and Comp. Sci., Case Western Reserve University, Cleveland, OH, 44107, USA

P163 A connectionist model of context-based memory reconsolidation in the hippocampus: the role of sleep

Justin Lines^{1*}, Kelsey Nation², and Jean-Marc Fellous^{1,2,3}

¹*Department of Psychology, University of Arizona, Tucson, Arizona 85721, USA*

²*Neuroscience Graduate Interdisciplinary Program, University of Arizona, Tucson, Arizona 85719, USA*

³*Program in Applied Mathematics, University of Arizona, Tucson, Arizona 85721, USA*

P164 Neurodynamical model for visual action recognition

Martin Giese*, Leonid Fedorov

Section Computational Sensomotorics, HIH / CIN, University Clinic Tübingen, Germany

P165 Multistable network dynamics through lateral inhibition: an efficient mechanism for selective information routing.

Daniel Harnack*, Klaus Pawelzik, and Udo A Ernst

Institut für Theoretische Physik, Universität Bremen, Bremen, Germany

P166 A spiking-neuron model of memory encoding and replay in hippocampus

Oliver Trujillo*, Chris Eliasmith

Center for Theoretical Neuroscience, University of Waterloo, Waterloo, Ontario, Canada N2L 3G1

P167 A point process approach to identifying and tracking transitions in neural spiking dynamics in the subthalamic nucleus of Parkinson's patients

Xinyi Deng^{1*}, Emad Eskandar^{2,3}, and Uri Eden¹

¹*Department of Mathematics and Statistics, Boston University, Boston, Massachusetts, 02215, USA*

²*Department of Neurosurgery, Massachusetts General Hospital, Boston, Massachusetts, 02114, USA*

³*Harvard Medical School, Boston, Massachusetts, 02115, USA*

P168 Local circuit model of the subthalamo-pallidal network for the generation of parkinsonian oscillations.

Osamu Shouno^{1,2*}, Kenji Doya¹

¹*Okinawa Institute of Science and Technology Graduate University, Tancha, Onna-son, Okinawa 904-0495, Japan*

²*Honda Research Institute Japan Co., Ltd., Honcho, Wako, Saitama 351-0188, Japan*

- P169 Model of Dynamics of Intracellular Chloride Based on Fluorescent Imaging**
Shiva Ghaani Farashahi¹, Jean Lienard¹, Susan Ingram², and Alexander Dimitrov^{1*}
¹*Department of Mathematics, Washington State University, Vancouver, WA, USA*
²*Department of Neurological Surgery, Oregon Health & Science University, Portland, OR, WSU*
- P170 Characterization of local invariances in the ascending ferret auditory system**
Jean Lienard¹, Stephen David², and Alexander Dimitrov^{1*}
¹*Department of Mathematics, Washington State University, Vancouver, WA, USA*
²*Oregon Health and Science University, Portland, OR, WSU*
- P171 Model exchange with the NeuroML Model Database**
Sharon Crook^{1,2*}, Suzanne Dietrich³
¹*School of Mathematical and Statistical Sciences, Arizona State University, Tempe, AZ 85287, USA*
²*School of Life Sciences, Arizona State University, Tempe, AZ 85287, USA*
³*School of Mathematical and Natural Sciences, Arizona State University, Phoenix, Arizona 85609 USA*
- P172 Hierarchical flow of sensory information in rat somatosensory cortex**
Houman Safaai^{1,2*}, Yanfang Zuo¹, Miguel Maravall³, Stefano Panzeri², and Mathew Diamond¹
¹*Tactile Perception and Learning Laboratory, International School for Advanced Studies (SISSA), 34136 Trieste, Italy*
²*Center for Neuroscience and Cognitive Systems, Istituto Italiano di Tecnologia, Corso Bettini 31, 38068 Rovereto, Italy*
³*Instituto de Neurociencias de Alicante, Consejo Superior de Investigaciones Científicas-Universidad Miguel Hernández, 03550 Sant Joan d'Alacant, Spain*
- P173 Estimating the transfer function of cortical neurons : from simple models to in vitro experiments**
Yann Zerlaut*, Gilles Ouanounou, Bartosz Telenczuk, Charlotte Deleuze, Thierry Bal, and Alain Destexhe
Unité de Neurosciences, Information and Complexité, CNRS UPR 3293, Gif-sur-Yvette 91198, France
- P174 Measurement of propagating waves from local field potentials and unit activity in the cortex of human and monkey.**
Lyle Muller^{1*}, Giacomo Benvenuti², Frédéric Chavane², and Alain Destexhe¹
¹*Unité des Neurosciences, Information et Complexité (UNIC), CNRS Gif-sur-Yvette, 91198, FR*
²*Institut de Neurosciences de la Timone (INT), Marseille, 13005, FR*

- P175 Role of external stimulation in shaping evoked activity in a macroscopic model of cortex**
 Matthieu Gilson*, Adrian Ponce-Alvarez, and Gustavo Deco
Dept. de Tecnologies de la Informació i les Comunicacions, Universitat Pompeu Fabra, Barcelona 08018, SPAIN
- P176 A NineML-based domain-specific language for computational exploration of connectivity in the cerebellar granular layer**
 Ivan Raikov^{1,2*}, Shyam Kumar S^{1,2}, Benjamin Torben-Nielsen, and Erik De Schutter^{1,2}
¹*Computational Neuroscience Unit, Okinawa Institute of Science and Technology, Onna-son, Okinawa, Japan*
²*University of Antwerp, Antwerp, Belgium*
- P177 Accurate approximation and MPI parallelization of spatial stochastic reaction-diffusion in STEPS**
 Iain Hepburn^{1,2*}, Weiliang Chen², and Erik De Schutter^{1,2}
¹*Theoretical Neurobiology, University of Antwerp, 2610 Antwerpen, Belgium*
²*Computational Neuroscience Unit, Okinawa Institute of Science and Technology, Okinawa, 904-0411, Japan*
- P178 Reduction of multi-compartmental biophysical models by incremental, automated retuning of their parameters and synaptic weights**
 Thomas Close^{1*}, Benjamin Torben-Nielsen¹, and Erik De Schutter^{1,2}
¹. *Computational Neuroscience Unit, Okinawa Institute of Science and Technology, Okinawa, Japan*
². *University of Antwerp, Antwerp, Belgium*
- P179 ATP consumption in molecular reactions of neuronal signaling**
 Nikon Rasumov*, Erik De Schutter
Okinawa Institute of Science and Technology, 1919-1 Tancha Onna-son, Okinawa, 904-0495 Japan
- P180 Microscopic cues shape neuronal morphology and microcircuits**
 Benjamin Torben-Nielsen*, Erik De Schutter
Computational Neuroscience Unit, Okinawa Institute of Science and Technology Graduate University, 1919-1 Tancha, Onna-son, Kunigami-gun, Okinawa, Japan 904-0495
- P181 Multifunctional central pattern generator controlling walking and paw shaking**
 Brian Bondy¹, Alexander Klishko², Boris Prilutsky², and Gennady Cymbalyuk^{1*}
¹*Neuroscience Institute, Georgia State University, Atlanta, Georgia, 30302, USA*
²*School of Applied Physiology, Georgia Institute of Technology, Atlanta, Georgia, 30332, USA*

- P182 Cellular mechanisms generating bursting activity in neuronal networks**
Jingjing Cannon, William Barnett, and Gennady Cymbalyuk*
Neuroscience Institute, Georgia State University, Atlanta, GA, 30302, USA
- P183 Bifurcation control of gait transition in insect locomotion**
William Barnett*, Gennady Cymbalyuk
Neuroscience Institute, Georgia State University, Atlanta, Georgia, 30302, USA
- P184 Theoretical understanding of three-dimensional, head-free gaze-shift**
Mehdi Daemi^{1,2*}, Douglas Crawford^{1,2}
¹*Department of Biology, York University, Toronto, ON, Canada*
²*Center for Vision Research, York University, Toronto, ON, Canada*
- P185 Neural coding strategies for extracting motion estimates from electrosensory contrast**
Stephen E Clarke^{1*}, Richard Naud², Andre Longtin², and Leonard Maler¹
¹*Department of Cellular and Molecular Medicine, University of Ottawa, Ottawa, Ontario, Canada, K1H 8M5*
²*Department of Physics, University of Ottawa, Ottawa, Ontario, Canada, K1N 6N5*
- P186 The formation of multi-synaptic connections by the interaction of synaptic and structural plasticity and their functional consequences**
Michael Fauth*, Florentin Wörgötter, and Christian Tetzlaff
Third Physics Institute, Bernstein Center for Computational Neuroscience, University Göttingen, 37077, GERMANY
- P187 A hierarchical model of vision (HMAX) can also recognize speech**
Matthew Roos*, Michael Wolmetz, and Mark Chevillet
Johns Hopkins University-Applied Physics Lab, Laurel, MD 20723, USA
- P188 The transformation of grid to place cells is robust to noise in the grid pattern**
Amir Hossein Azizi*, Sen Cheng
Department of Psychology, Ruhr-University Bochum, Bochum, NRW, 44801, Germany
- P189 An activity-dependent computational model of development of the retinotopic map along the dorsoventral axis in the primary visual cortex**
Ryan Philips, Srinivasa Chakravarthy*
Department of Biotechnology, Indian Institute of Technology Madras, Chennai 600036, Tamil Nadu, India

P190 Neural Correlations in the Electrosensory Lateral Line Lobe of the Weakly Electric fish, *Apteronotus leptorhynchus*: Analysis of Multi-Channel Recordings

Teerawat Monnor¹, Michael G. Metzen¹, and Maurice J Chacron^{1,2*}

¹*Department of Physiology, McGill University, Montreal, QC, H3G 1Y6, Canada*

²*Department of Physics, McGill University, Montreal, QC, H3G 1Y6, Canada*

P191 Changes in stimulus envelope reveal two classes of peripheral electrosensory neurons

Michael G. Metzen¹, Maurice J Chacron^{1,2*}

¹*Department of Physiology, McGill University, Montreal, QC, Canada*

²*Department of Physics, McGill University, Montreal, QC, Canada*

P192 Differential neural responses to naturally occurring envelopes in the electrosensory system

Chengjie Huang^{1*}, Maurice J Chacron^{1,2}

¹*Department of Physiology, McGill University, Montreal, Quebec H3G 1Y6, Canada*

²*Department of Physics, McGill University, Montreal, Quebec H3G 1Y6, Canada*

P193 Parallel pathways at the auditory periphery

Marcos Cantu^{1,2*}

¹*Center for Computational Neuroscience and Neural Technology (CompNet), Boston University, Boston, MA, USA*

²*Graduate Program for Neuroscience (GPN), Boston University, Boston, MA, USA*

P194 Simulating structural plasticity of large scale networks in NEST

Mikael Naveau*, Markus Butz

Simulation Lab Neuroscience - Bernstein Facility for Simulation and Database Technology, Institute for Advanced Simulation, Jülich Aachen Research Alliance, Forschungszentrum Jülich, 52425 Jülich, Germany

P195 Interspike intervals as a discrete time series with history and randomness

Sharon Norman^{1*}, Rob Butera^{1,2}

¹*School of Electrical and Computer Engineering, Georgia Institute of Technology, Atlanta, GA 30332, USA*

²*Department of Biomedical Engineering, Georgia Institute of Technology, Atlanta, GA 30332, USA*

P196 Integration of functional cerebral networks and genetic expression: the dual intertwined rings architecture of the cerebral cortex for real-time vs multi-temporal information processing

Claudia Cioli^{1*}, Salma Mesmoudi², Derek Beaton³, David Rudrauf¹, Hervé Abdi³, and Yves Burnod¹

¹*Laboratoire d'Imagerie Biomédicale UPMC - INSERM U1146 - CNRS UMR 7173, Paris, F-75634, France*

²*Université Paris 1 Panthéon-Sorbonne, Equipement d'Excellence MATRICE, Paris, F-75231*

³*School of Behavioral and Brain Sciences, The University of Texas at Dallas, Dallas, TX 75080-3021, USA*

P197 Goal-directed control with cortical units that are gated by both top-down feedback and oscillatory coherence

Robert Kerr^{1,2,3}, David Grayden^{1,2,3,4}, Doreen Thomas⁵, Matthieu Gilson^{1,2,6,7}, and Anthony N Burkitt^{1,2,3,4*}

¹*NeuroEngineering Laboratory, Dept. of Electrical & Electronic Engineering, University of Melbourne, Australia*

²*Center for Neural Engineering, University of Melbourne, Australia*

³*NICTA, Victoria Research Lab, University of Melbourne, Australia*

⁴*Bionics Institute, East Melbourne, Australia*

⁵*Department of Mechanical Engineering, University of Melbourne, Australia*

⁶*Laboratory for Neural Circuit Theory, RIKEN Brain Science Institute, Saitama, Japan*

⁷*Computational Neuroscience Group, University Pompeu Fabra, Barcelona, Spain*

P198 Hebbian learning in the MSO: emergence of interaural tuning

Pierre Yger^{1,2,3,4*}, Victor Benichoux^{1,2,3,4}, Marcel Stimberg^{1,2,3,4}, and Romain Brette^{1,2,3,4}

¹*Institut d'Etudes de la Cognition, Ecole Normale Supérieure, Paris, France*

²*Sorbonne Universités, UPMC Univ. Paris 06, UMR S 968, Institut de la Vision, Paris, F-75012, France*

³*INSERM, U968, Paris, F-75012, France*

⁴*CNRS, UMR 7210, Paris, F-75012, France*

P199 Brian 2: neural simulations on a variety of computational hardware

Dan F M Goodman^{1,2}, Marcel Stimberg^{3,4,5,6*}, Pierre Yger^{3,4,5,6}, and Romain Brette^{3,4,5,6}

¹*Department of Otology and Laryngology, Harvard Medical School, Boston, Massachusetts, 02114, USA*

²*Eaton-Peabody Laboratories, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts, 02114, USA*

³*Institut d'Etudes de la Cognition, Ecole Normale Supérieure, Paris, France*

⁴*Sorbonne Universités, UPMC Univ. Paris 06, UMR_S 968, Institut de la Vision, Paris, F-75012, France*

⁵*INSERM, U968, Paris, F-75012, France*

⁶*CNRS, UMR_7210, Paris, F-75012, France*

P200 Spiking models of interaural level difference encoding - beyond the rate subtraction code

Martin Spencer^{1,2,3,4*}, Bertrand Fontaine⁵, and Romain Brette^{1,2,3,4}

¹*Institut d'études de la cognition, École Normale Supérieure, Paris, 75005, France*

²*Sorbonne Universités, UPMC Univ. Paris 06, UMR_S 968, Institut de la Vision, Paris, F-75012, France*

³*INSERM, U968, Paris, F-75012, France*

⁴*CNRS, UMR_7210, Paris, F-75012, France*

⁵*Laboratory of Auditory Neurophysiology, University of Leuven, Leuven, Belgium*

P201 SPIKY: A graphical user interface for tracking spike train similarity

Thomas Kreuz*, Nebojsa Bozanic

Institute for Complex Systems, CNR, Sesto Fiorentino, Italy

P202 Modulation of neuronal entrainability by epilepsy-associated currents and noise: a spectral approach

Alla Borisyuk*

Department of Mathematics, University of Utah, Salt Lake City, UT 84112, USA

P203 Neural dynamics of the speed-accuracy trade-off

Dominic Standage^{1*}, Da-Hui Wang², and Gunnar Blohm¹

¹*Department of Biomedical and Molecular Sciences, Queen's University, Kingston, Canada*

²*Department of Systems Science, Beijing Normal University, Beijing, China*

P204 Molecular dependence of hippocampal bidirectional plasticity

Joanna Jedrzejewska-Szmek*, Andrew Chay, and Kim Avrama Blackwell

The Krasnow Institute for Advanced Study, George Mason University, Fairfax, VA 22030, USA

P205 The topology of astrocyte networks controls the propagation of intercellular calcium waves

Jules Lallouette^{1,2*}, Maurizio De Pittà^{1,2,3}, Eshel Ben-Jacob^{3,4}, and Hugues Berry^{1,2}

¹*EPI Beagle, INRIA Rhône-Alpes, Villeurbanne, France*

²*LIRIS, Université de Lyon, UMR5205 CNRS-INSA, Villeurbanne, France*

³*School of Physics and Astronomy, Tel Aviv University, Ramat Aviv, Israel*

⁴*Center for Theoretical Biological Physics, Rice University, Houston, TX, USA*

P206 Astrocytic Theory of Working Memory

Maurizio De Pittà^{1,2,3*}, Eshel Ben-Jacob^{4,5}, and Hugues Berry^{1,2}

¹*EPI Beagle, INRIA Rhône-Alpes, Villeurbanne, France*

²*LIRIS, Université de Lyon, UMR5205 CNRS-INSA, Villeurbanne, France*

³*Department of Statistics, The University of Chicago, 5734 S. University Ave., Chicago, IL, USA*

⁴*School of Physics and Astronomy, Tel Aviv University, Ramat Aviv, Israel*

⁵*Center for Theoretical Biological Physics, Rice University, Houston, TX, USA*

P207 Graph theoretic characterization of in vitro neuronal network development

Uzair Khawaja¹, Tyler Stone¹, Lisa Morkowchuk¹, Thomas R Kiehl², and Charles Bergeron^{1*}

¹*Analytics Lab, Albany College of Pharmacy and Health Sciences, Albany, New York, 12208, USA*

²*Neural Stem Cell Institute, Rensselaer, New York, 12144, USA*

P208 Best practices for avoiding dominant experimental bias in analysis of multi-electrode array signals

Tyler Stone¹, Uzair Khawaja¹, Nardeen Perko¹, Thomas R Kiehl^{2*}, and Charles Bergeron¹

¹*Analytics Lab, Albany College of Pharmacy and Health Sciences, Albany, New York, 12208, USA*

²*Neural Stem Cell Institute, Rensselaer, New York, 12144, USA*

P209 Visualizing spike activity during neuronal network development

Nicholas Vachon¹, Thomas R Kiehl², and Charles Bergeron^{1*}

¹*Analytics Lab, Albany College of Pharmacy and Health Sciences, Albany, New York, 12208, USA*

²*Neural Stem Cell Institute, Rensselaer, New York, 12144, USA*

P210 Chronic Upper Airway Obstruction Induces Abnormal Sleep/Wake Dynamics in Juvenile Rats

Gideon Gradwohl^{1,3*}, Nilly Berduga-Boura^{1,2}, Yael Segev², and Ariel Tarasiuk¹

¹*Sleep-Wake Disorders Unit, Soroka University Medical Center and Department of Physiology, Faculty of Health Sciences, Ben-Gurion University of the Negev, 84110, Israel*

²*Shraga Segal Department of Microbiology and Immunology, Faculty of Health Sciences, Ben-Gurion University of the Negev, 84110, Israel*

³*Unit of Biomedical Engineering, Department of Physics, Lev Academic Center, Jerusalem, 9116001, Israel*

- P211 Reproduction of EEG power spectrum over frontal region during the propofol-induced general anesthesia**
Meysam Hashemi^{1*}, Axel Hutt¹, Jamie Sleight², and Peter Beim Graben³
¹*INRIA CR Nancy - Grand Est, Villers-les-Nancy, France*
²*Department of Anaesthetics, Waikato Hospital, Hamilton, New Zealand*
³*Department of German Language and Linguistic, Humboldt-Universität zu Berlin, Germany*
- P212 Multiplex networks of cortical and hippocampal neurons revealed at different timescales**
Nicholas Timme^{1*}, Shinya Ito², Maxym Myroshnychenko¹, Fang-Chin Yeh¹, Emma Holski³, Alan Litke², and John Beggs¹
¹*Department of Physics, Indiana University, Bloomington, IN 47405, USA*
²*Santa Cruz Institute for Particle Physics, University of California at Santa Cruz, Santa Cruz, CA 95064 USA*
³*Microbiology & Environmental Toxicology Department, University of California at Santa Cruz, Santa Cruz, CA 95064 USA*
- P213 Patterns of information flow in local cortical networks**
Sunny Nigam^{1*}, Olaf Sporns², Masanori Shimono^{1,3}, and John Beggs¹
¹*Department of Physics, Indiana University, Bloomington, IN 47405, USA*
²*Department of Psychological and Brain Sciences, Indiana University, Bloomington, IN 47405, USA*
³*JSPS fellow*
- P214 Microscale impedance measurements suggest that ionic diffusion is implicated in generating extracellular potentials**
Claude Bedard^{1*}, Jean-Marie Gomès¹, Matthew Nelson², Pierre Pouget², Silvana Valtcheva³, Laurent Venance³, Yves Gioanni³, Thierry Bal¹, and Alain Destexhe¹
¹*UNIC, CNRS, Gif sur Yvette, France*
²*ICM, Paris, France*
³*CIRB, Collège de France, Paris, France*
- P215 Cable equation formalism for neuronal magnetic fields**
Alain Destexhe, Francesca Barbieri, and Claude Bedard*
. UNIC, CNRS, Gif sur Yvette, France
- P216 Neuronal Plasticity during Sleep Slow Wave Oscillations**
Yina Wei, Giri Krishnan*, and Maxim Bazhenov
Department of Cell Biology and Neuroscience, University of California Riverside, CA 92521 USA

- P217 Ion concentration dynamics leads to the very slow spontaneous neuronal oscillations**
Giri Krishnan*, Oscar Gonzalez, and Maxim Bazhenov
Department of Cell Biology and Neuroscience, University of California Riverside, CA 92521 USA
- P218 Hippocampal replay and cortical slow oscillations: a computational study**
Paola Malerba*, Giri Krishnan, and Maxim Bazhenov
Cell Biology and Neuroscience, University of California Riverside, Riverside, CA 92521, USA
- P219 Studying the effects of thalamic interneurons in a thalamocortical neural mass model**
Thomas Bond¹, Simon Durrant², Louise O'Hare², Daniel Turner¹, and Basabdatta Sen-Bhattacharya^{1*}
¹*School of Engineering, University of Lincoln, Lincoln, Lincolnshire LN6 7TS, UK*
²*School of Psychology, University of Lincoln, Lincoln, Lincolnshire LN6 7TS, UK*
- P220 Neuron population activity in the medial prefrontal cortex suggests superimposed codes for situation and situation value**
Nathan Insel^{1,2*}, Carol Barnes²
¹. *Department of Biology, University of Toronto Scarborough, Toronto, M1C 1A4, Canada*
². *Evelyn F. McKnight Brain Institute and ARL Division of Neural Systems, Memory & Aging, University of Arizona, Tucson AZ, 85724, USA*
- P221 Effects of astrocytic mechanisms on neuronal hyperexcitability**
Vasily Grigorovsky*, Berj Bardakjian
Institute of Biomaterials and Biomedical Engineering, University of Toronto, Toronto, Ontario, M5S 3G9, Canada
- P222 Local and long-range phase-amplitude coupling in a cortical spiking network model**
Peter Donhauser*, Sylvain Baillet
McConnell Brain Imaging Center, Montreal Neurological Institute, McGill University, Montreal, Canada
- P223 Multilayer Perceptrons, Hopfield's Associative Memories, and Restricted Boltzmann Machines**
Shin Asakawa*
Center for Information Sciences, Tokyo Woman's Christian University, 2-6-1 Zempukuji, Suginami-ku, Tokyo 167-8585, Japan

P224 The SpineML toolchain: enabling computational neuroscience through flexible tools for creating, sharing, and simulating neural models

Alexander J Cope^{1*}, Paul Richmond¹, and Dave Allerton²

¹*Department of Computer Science, University of Sheffield, Sheffield, South Yorkshire, S10 2TN, UK*

²*Department of ACSE, University of Sheffield, Sheffield, South Yorkshire, S10 2TN, UK*

P225 Sequential patterns of spikes and scale-invariance in modular networks

Timothee Leleu*, Kazuyuki Aihara

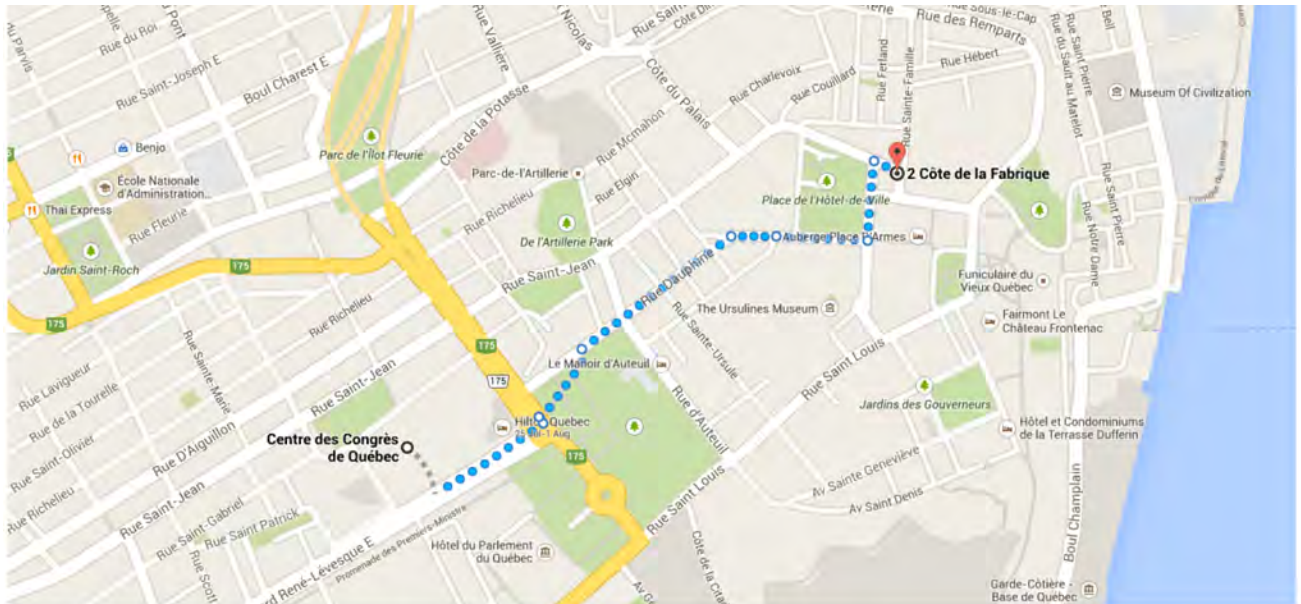
Institute of Industrial Science, The University of Tokyo, 4-6-1 Komaba, Meguro-ku, Tokyo 153-8505, Japan

Fun and Recreation

Banquet

The party will be held at the "Chapelle du musée" on Tuesday July 29th at 19:00, which is located at 2, Cote de la fabrique, Québec (QC), and is within a 12 minute walk from the convention center (see below for directions).





○ Centre des Congrès de Québec

Use caution - may involve errors or sections not suited for walking

1000 Boulevard René-Lévesque Est, Québec, QC G1R 2B5

- ↑

1. Head northeast on Boulevard René-Lévesque E toward Autoroute Dufferin-Montmorency/Av Honoré Mercier/Rte 175 S

200 m
- ↶

2. Turn left onto Autoroute Dufferin-Montmorency/Av Honoré Mercier N

11 m
- ↷

3. Turn right toward Rue Dauphine

130 m
- ↷

4. Turn right onto Rue Dauphine

280 m
- ↷

5. Slight right onto Rue Cook

68 m
- ↑

6. Continue onto Rue Sainte-Anne

140 m
- ↶

7. Turn left onto Rue des Jardins

120 m
- ↷

8. Turn right onto Côte de la Fabrique

40 m

i Destination will be on the left

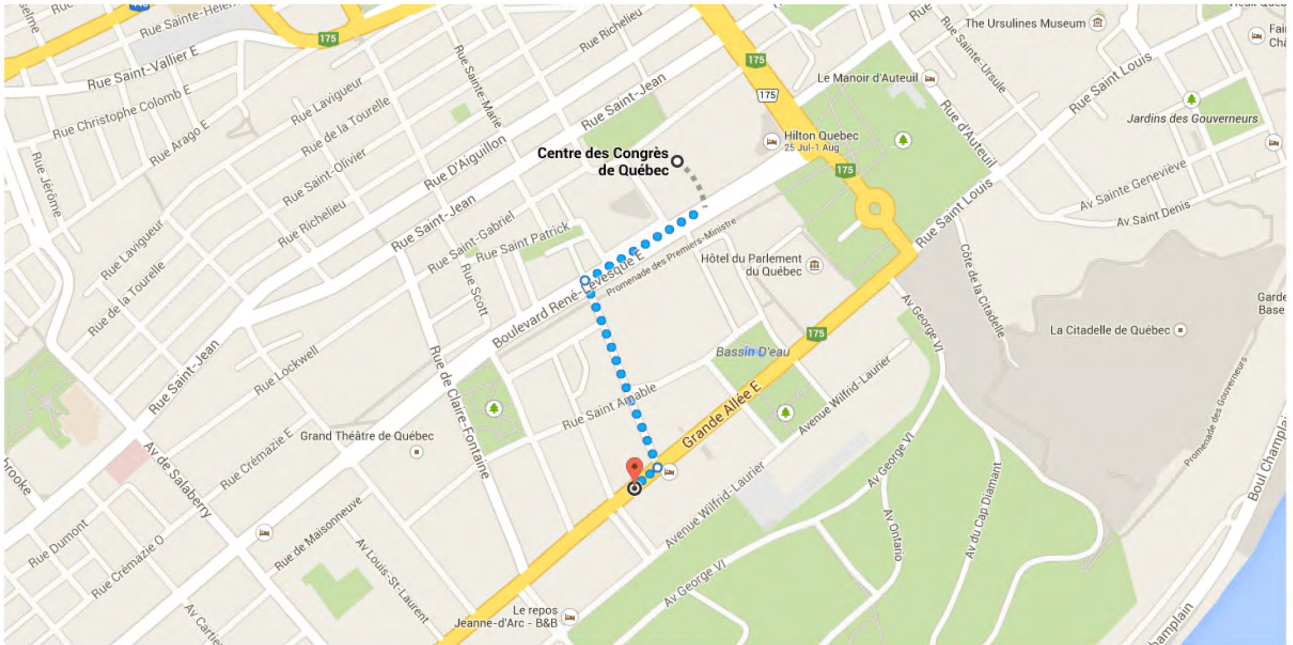
◎ 2 Côte de la Fabrique

Québec, QC G1R 3V6

Party

The party will be held at the bar "Chez Maurice" on Monday July 28th at 19:00, which is located at 575, Grande-Allée Est, Québec (QC), and is within a 10 minute walk from the convention center (see below for directions).

Directions from Centre des Congrès de Québec to 575 Grande Allée Est, Québec, QC G1R



○ Centre des Congrès de Québec

1000 Boulevard René-Lévesque Est, Québec,
QC G1R 2B5

Use caution - may involve errors or sections not suited for walking

1. Head **southwest** on **Boulevard René-Lévesque E** toward **Rue Louis Alexandre Taschereau**
↑ 220 m
2. Turn **left** onto **Rue de la Chevrotière**
↶ 300 m
3. Turn **right** onto **Grande Allée E**
↷ 48 m

📍 575 Grande Allée Est, Québec, QC G1R

Local and Touristic Information

What to do in and around Québec City

1

Fortifications of Québec

Why go to China when you can walk a great wall right here! Québec is **the only fortified city in North America north of Mexico**, with close to 4.6 km of [walls](#) and imposing gates to explore.

Discover beautiful cityscapes and see how Québec's defenses developed under the French and English regimes. Cannons, loopholes, a star-shaped Citadel, Artillery Park, and fortresses are all part of this outstanding tour!



Credit: OTQ

2

Old Québec

Visitors to [Old Québec](#) soon see why UNESCO designated it a **world heritage treasure**! You'll love [Château Frontenac](#) (the world's most photographed hotel), the centuries-old architecture, and the historic sites. The friendly atmosphere and affable locals add to the European charm. You'll find horse-drawn carriages, street



Credit: Olivier Lavigne-Ortiz

entertainers, singers, and artists, particularly at Old Québec's own open-air art gallery, Rue du Trésor. High atop Cape Diamond, stroll along the Dufferin Terrace overlooking the St. Lawrence River and the surrounding area. Or come watch the ice making its way down the river in winter.

3

Petit-Champlain District & Place-Royale

How about a trip back in time at [Place Royale](#), where Samuel de Champlain founded his first “abitation” in 1608? And why not do some window shopping in the nearby [Petit-Champlain District](#) while you're at it!

As you wander past period buildings along cobblestone streets, enjoy the area's **boutiques**, **art galleries**, and **restaurants**. There's magic in the air, particularly over the Christmas holiday season. The oldest neighborhood in North America is also home to [Musée de la civilisation](#), a bridge between the past and future with its modern design and fascinating exhibitions.



Credit: Stéphane Audet

4

St. Lawrence River & Vieux-Port de Québec

The **St. Lawrence River**—a massive presence cutting clean across the Québec area—cannot be overlooked. Gateway to America, it has been a part of the city’s economic landscape for over 400 years. Harbour and trade activities and the ever-growing number of cruise ships docking in



Credit: Office du tourisme de

the [Vieux-Port de Québec](#) testify to its importance. A public market, park, bike path and shows also bring the Vieux Port to life and help make this river-washed place truly idyllic.

Near the bridges that span the River, the [Aquarium du Québec](#) not only provides an outstanding view of the majestic waterway, but also presents the marine mammals and species that inhabit it. Close by, the [Promenade Samuel-De Champlain](#) is also worth a gander: the River flowing at your feet is simply spectacular!

5

Sainte-Anne-de-Beaupré Shrine

For many, the [Sainte-Anne-de-Beaupré Shrine](#) has been a “place of miracles” for the past 350 years. It's well worth a visit, whatever your beliefs.

The shrine, **North America's oldest pilgrimage site**, attracts some one million visitors a year. Marvel at the fabulous neo-Roman style basilica with its golden statue of Saint Anne. Admire the hundreds of stained glass windows, the nave, and the valuable works of art. Come recharge your batteries at this beautiful place of worship in the splendid countryside of the [Côte-de-Beaupré](#) region.



6

Montmorency Falls Park

This natural phenomenon is definitely not to be missed! **At 83 m high** (30 m higher than Niagara Falls) Montmorency Falls can be seen from all the way across the St. Lawrence River in Lévis! But the best views are from [Parc de la Chute-Montmorency](#), where you can feel



the full force—and spray—of the falls for yourself.

Take a gondola ride or walk the trails to the very top of the falls. In winter the spray freezes at the foot of the falls to form a huge “sugar loaf,” another intriguing Québec City attraction.

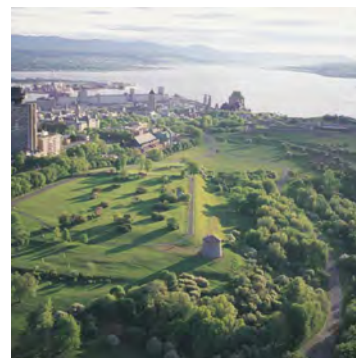
Credit: Office du tourisme de Québec / Jean-Guy Lavoie

7

Plains of Abraham

The scene of the 1759 battle between generals Wolfe and Montcalm, the [Plains of Abraham](#) are the heart and lungs of Québec City. Discover **one of the world's largest and finest urban parks**.

The Plains are perfect for all kinds of activities (walking, cycling, picnicking, cross-country skiing, and more) or simply meeting up with friends. It was here that hundreds of thousands of fans rocked to **Paul McCartney** and **Céline Dion** as part of Québec City's 400th anniversary celebrations, and it is here that Québec's national holiday is celebrated every June 24. [Musée national des beaux-arts du Québec](#)—renowned for its exhibits and collection of Québec art—is only a short walk away.



Credit: Luc-Antoine Couturier

8

Wendake

Enter the fascinating world of the Huron-Wendat **First Nation** and embark on a history tour that will take you back to the 17th century. The ancestral site of [Wendake](#) is a window onto aboriginal culture! Dance shows and legends will whisk you deep into this people's imagination, while [Musée huron-wendat](#) and various craft stores invite you to learn more about the nation's culture and traditional



Credit: Jean Louis Regis

know-how.

A short walk through scenic natural surroundings leads visitors to Kabir Kouba Falls by Rivière Saint-Charles, and sampling the game featured in traditional Huron cuisine is the perfect way to round out your voyage of discovery.

9

Parliament Hill

The province's [Parliament Hill](#) has never been so popular! Québec's **National Assembly** convenes here in the [Parliament Building](#), a marvelous architectural treasure!

The gorgeous [Fontaine de Tourny](#) was awarded a gold medal at the Paris World Fair in 1855. Today, it stands as a legacy of Québec City's 400th anniversary celebrations, turning heads with its 43 jets, water-themed sculptures, and beautiful nighttime lighting. Nearby [Observatoire de la Capitale](#) also turns visitors giddy with excitement with its tremendous views of Québec City and area from a height of 221 m!



Credit: Office du tourisme de Québec / Guy Lessard

10

Île d'Orléans

Imagine how beautiful Québec's countryside must have been in the 19th century. In fact, it looked very much like irresistible [Île d'Orléans](#) with its historic farms, churches, and **heritage homes**.

Visiting the many artisans and farm stalls lining the route is half the fun as you make your way from one quaint village to the next. In season, enjoy the **island's famous strawberries and apples**, as well as freshly baked bread, wines, ice ciders, blackcurrant liqueurs, and other local



Credit: Sébastien Larose

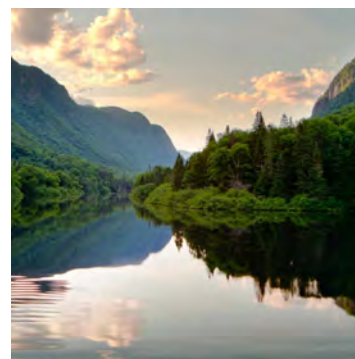
delicacies.

11

Parc national de la Jacques-Cartier

The spectacular Rivière Jacques-Cartier flows through a deep valley surrounded by steep wall soaring **up to 550 m in height!** [Parc national de la Jacques-Cartier](#) is protected by [Sépaq](#) (Québec's largest parks and outdoor recreation network) so that you can enjoy all your favorite outdoor pursuits.

With hiking, canoeing, kayaking, fishing, and camping all available in breathtaking surroundings, it's no wonder that the [Jacques-Cartier region](#) is known locally as Québec City's “green crescent.” And the pace doesn't slow in winter, so strap on your snowshoes, skies, or winter boots and get ready to explore trail after trail of winter fun!



Credit: Luc Rousseau

12

Chemin du Roy

[Chemin du Roy](#)—Canada's oldest highway—has linked Québec City and Montréal since 1737. This scenic route traverses beautiful pastoral landscapes as it winds its way along the St. Lawrence River.

Visitors heading toward [Portneuf](#) from Québec's City Hall pass through some of the province's prettiest villages, featuring manors, mills, museums, heritage homes, and other priceless heritage treasures. Nearly all of the road is part of Québec's “[Route verte](#)” bike path network, enabling cyclists to safely enjoy the scenery from the comfort of



Credit: Yves Tessier

Appendix

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