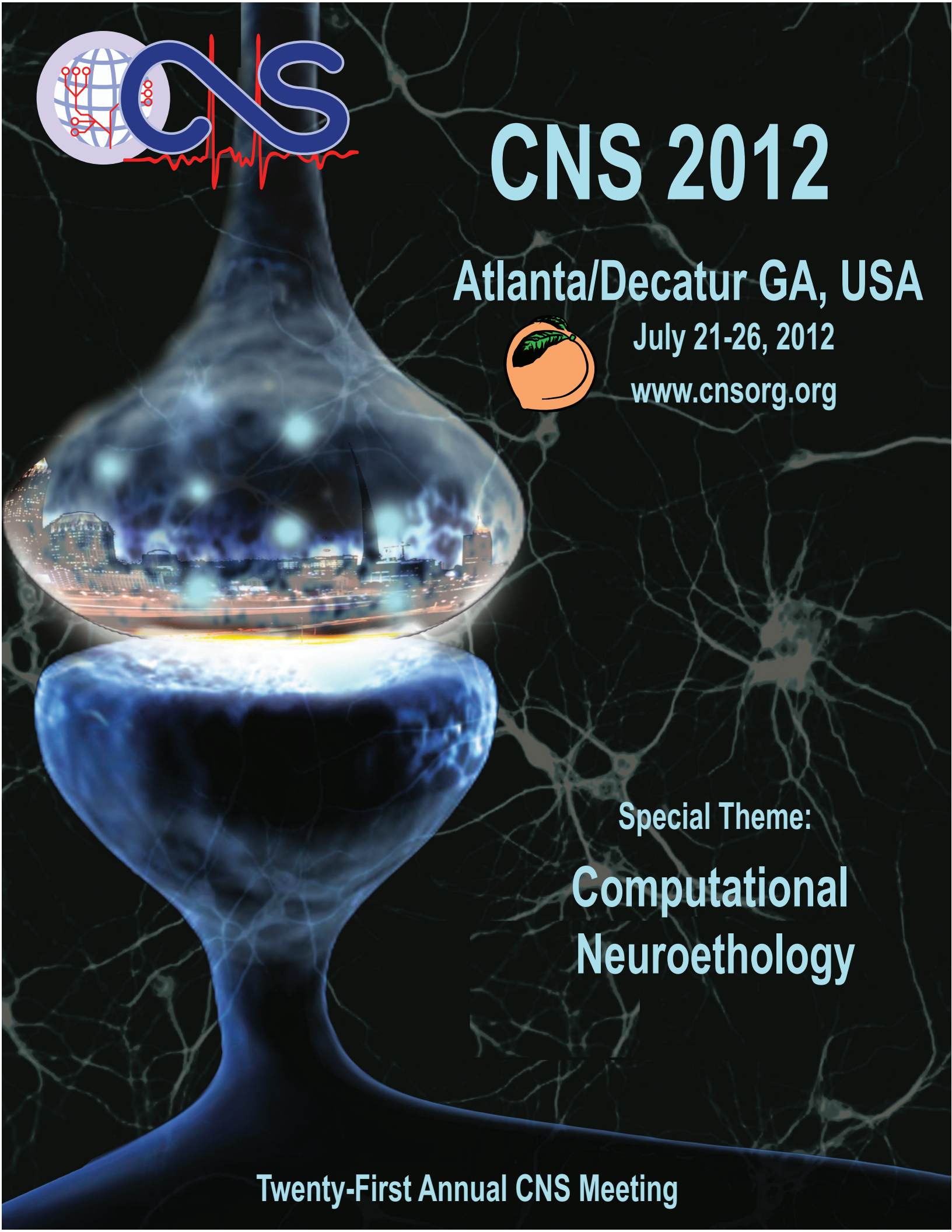
CNS 2012
Atlanta/Decatur GA, USA
July 21-26, 2012
www.cnsorg.org
Special Theme:
Computational Neuroethology
Twenty-First Annual CNS Meeting

Contents

Edited by
Anca Doloc-Mihu, Emory University, USA and **Udo Ernst**, University Bremen, Germany

We would like to thank the following persons for their invaluable contributions to this program book: OCNS Executive Board and Program Committee, as well as numerous CNS participants for the scientific content, Rachel Burger for the cover art, Cengiz Günay, David Nicholson, and Damon Lamb (Emory University, USA) for editing the cover art, Anca Doloc-Mihu (Emory University, USA) for providing maps and local information, and to the local volunteers from Emory University, Georgia State University, and Georgia Institute of Technology.

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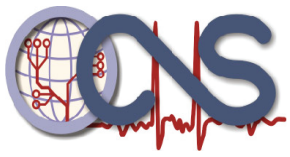
CNS*2012 Sponsors



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Computational & Life Sciences
The Computational and Life Sciences Strategic Initiative at Emory University

Center for BEHAVIORAL NEUROSCIENCE

Neuralynx
High Density Electrophysiology Recording Systems

Springer
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Center for Neuromics
College of Arts & Sciences

International IEEE EMBS Conference on
Neural Engineering
November 5-8, 2013 - San Diego -
- Just before the SfN Annual Meeting

FIU
FLORIDA INTERNATIONAL UNIVERSITY

NSF Neuroengineering-IGERT Program, University of Minnesota
http://www.igert-ne.umn.edu/

Local Info & Maps

About Downtown Decatur, GA

We invite you to discover Downtown Decatur (http://www.decaturga.com/), a city with a traditional small college-town atmosphere along with all the benefits of living in a major metropolitan area. Decatur is just minutes east of downtown Atlanta, minutes west of Stone Mountain, and the MARTA rail station is right under our downtown square, so getting here is a breeze.

Named after Stephen Decatur, a 19th century U.S. Naval hero, Decatur retains a strong connection to its history (http://www.decaturga.com/index.aspx?page=91), while preparing for the future with managed growth and responsible development. An extensive collection of art works (http://www.decaturga.com/index.aspx?page=94) are on display around the City. Learn more about Decatur's Points of Interest (http://www.decaturga.com/index.aspx?page=93) as well as community activities, business and City government.

Directions to Decatur & Agnes Scott College

Directions from Atlanta Airport (ATL) to Downtown Decatur via MARTA subway (here is a MARTA map https://ocns.memberclicks.net/assets/CNS_Meetings/CNS2012/martamap.pdf):

Take any train from Airport to Five Points station.

Change trains at Five Points station: take Blue Line train to Indian Creek. Get off at Decatur station.

Note: If you plan to use public transportation from the airport to Decatur, it is best to arrive at the ATL airport no later than 9pm for international flights and 10pm for within U.S. flights.

Walking directions from MARTA station to Agnes Scott (a short three-block walking distance ~16min):

Exit the terminal on the Church St. side and turn right on Church St.

Use the pedestrian tunnel to pass beneath the railroad tracks; you will merge at the E. College Avenue entrance to campus.

By Car from the North on I-85 (mileage approximate):

Take I-85 to the Clairmont Road exit.
Turn left onto Clairmont Road.
Turn right onto Commerce Drive. (4.9miles) (Disregard directional sign pointing left; continue right)
Turn left onto W. Trinity Place. (0.4 miles)
Turn right onto N. McDonough Street. (0.1 miles)
Follow N. McDonough Street over the railroad to Agnes Scott College. (0.3 miles)

By Car from the East on I-285 or I-20 (mileage approximate):

Take I-285 to Stone Mountain Freeway, Highway 78 exit.
Go west on Highway 78 (Street name changes to Scott Boulevard.)
Turn left onto Clairmont Road. (4.1 miles)
Turn right onto Commerce Drive. (0.7 miles) (Disregard directional sign pointing left; continue right)
Turn left onto W. Trinity Place. (0.4 miles)
Turn right onto N. McDonough Street. (0.1 miles)
Follow N. McDonough Street over the railroad to Agnes Scott College. (0.3 miles)

Restaurants Downtown Decatur

Decatur is a dining destination, attracting foodies from all across metro Atlanta to more than 80 mostly independent restaurants, cafés and pubs. Here is a list of all Decatur restaurants http://www.decaturga.com/index.aspx?page=577. However, we prepared for you **a list of our favorites** http://goo.gl/maps/hi7n. Enjoy!

Our favorite restaurants near Agnes Scott campus in Downtown Decatur

http://goo.gl/maps/hi7n

- Agnes Scott campus
- MARTA Decatur Station
- Warning! DO NOT CROSS HERE
- Pedestrian Tunnel
- Walking Path: MARTA to Agnes Scott (blue line)

Restaurants grouped by food type

- Deserts & Coffee
- European
- American
- Pubs & Sport Bars
- Mexican
- Asian

Asian		Price
Sushi Avenue	308-C W.Ponce*, 404-378-8448, http://www.sushiavenuedecatur.com	$$$-$$$$
Sushi Avenue on the Square	131 Sycamore St., 404-378-0228, http://www.sushiavenuedecatur.com	$$$-$$$$

Deserts & Coffee		Price
Cakes & Ale and The Bakery at C&A	151 Sycamore St., 404-377-7960, http://www.cakesandalerestaurant.com	$-$$$
The Chocolate Bar	201 W. Ponce*, 404-388-6591, http://www.thechocolatebardecatur.com	$$$
Java Monkey	425 Church St, 404-378-5002, http://www.javamonkeydecatur.com	$
The Yogurt Tap	419 Church St., 404-373-6090, http://www.theyogurttap.com	$
Swirlin' Twirlin'	335 W. Ponce*, 404-941-7859, http://www.SwirlinTwirlin.com	$

European		Price
Café Alsace	121 E. Ponce*, 404-373-5622, http://www.cafealsace.net	$$$$
Fellini's Pizza	333 Comerce Dr., 404-370-0551, http://www.fellinisatlanta.com	$
The Iberian Pig	121 Sycamore St., 404-371-8800, http://www.iberianpigatl.com	$$$$$
Mellow Mushroom	265 Ponce*, 404-370-0008, http://www.mellowmushroom.com	$$$
No. 246	129 E. Ponce*, 678-399-8246, http://www.no246.com	$$$$
Sapori di Napoli	314 Church St., 404-371-0001, http://www.saporidinapolipizzeria.com	$$-$$$

American		Price
Café Lily	308-B W. Ponce*, 404-371-9119, http://www.cafelily.com	$$$
Cakes & Ale	155 Sycamore Street, Decatur, GA 30030 404-377-7994, http://www.cakesandalerestaurant.com	$$$$$
Carpe Diem	105 Sycamore Pl., 404-687-9696, http://www.apresdiem.com	$$$
Eddie's Attic	515-B N. McDonough St., 404-377-4976, http://www.eddiesattic.com	$$
Farm Burger	410 W Ponce*, 404-378-5077, http://www.farmburger.net	$$
Jimmy John's	335 W. Ponce*, 404-474-6999, http://www.jimmyjohns.com	$$
Thumbs Up	174 W. Ponce*, 404-377-5623, http://www.thumbsupdiner.com	$-$$

Pubs & Sport Bars		Price
Brick Store Pub	125 E. Court Sq., 404-687-0990, http://www.brickstorepub.com	$$-$$$
Leon's Full Service	131 E Ponce*, 404-687-0500, http://www.leonsfullservice.com	$$$
The Marlay House	426 W. Ponce*, 404-270-9950, http://www.themarlayhouse.com	$$
Taco Mac	240 W. Ponce, 404-378-4140, http://www.tacomac.com	$-$$
Thinking Man Tavern	537 W. Howard Ave., 404-370-1717, http://www.thinkingmantavern.com	$
Harbor Bar	129 Church St, 404-371-0088, http://http://harbourdecatur.com/	$$-$$$
Twain's Brew Pub	211 E. Trinity Pl., 404-373-0063, http://www.twains.net	$$

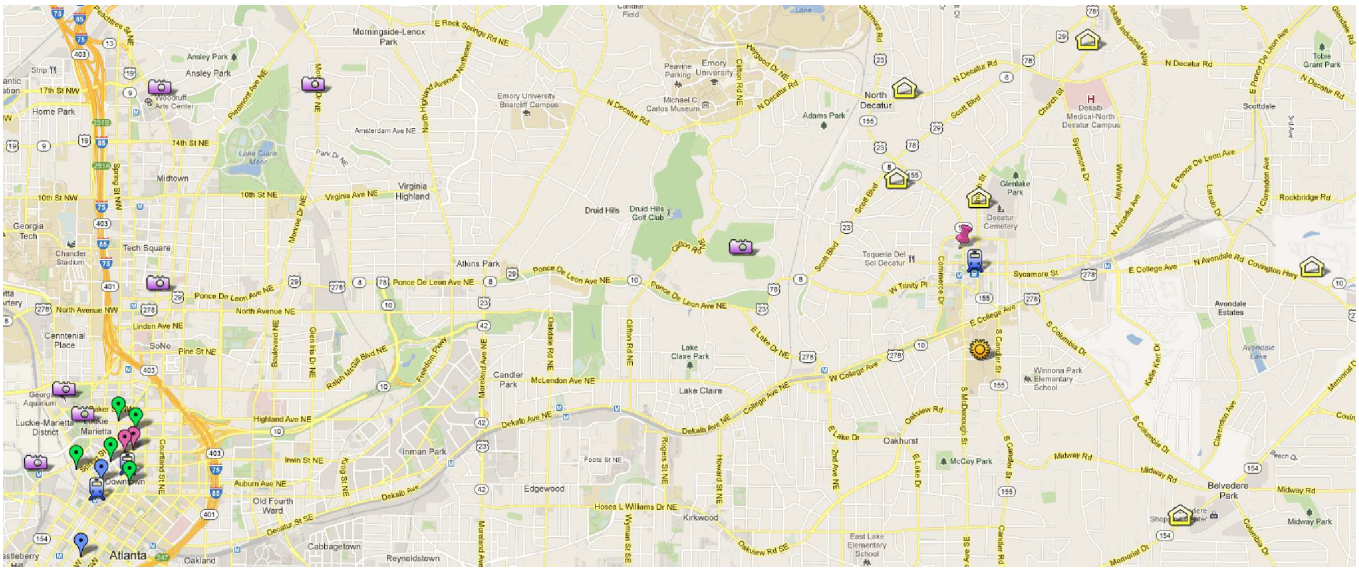
Mexican		Price
Mezcalito's	653 East Lake Dr., 404-687-0007, http://www.mezcalitoscantina.com	$$
Raging Burrito	141 Sycamore St., 404-377-3311, http://www.ragingburrito.com	$
Taqueria del Sol	359 W. Ponce*, 404-377-7668, http://www.taqueriadelsol.com	$

*Note: "Ponce" is Atlanta short-hand for "Ponce de Leon Avenue".

Things to See in Atlanta

Atlanta (Georgia) is a Brave and Beautiful City, the Southeast's most alluring destination. Click here http://www.atlanta.net/ to see What to Do in Atlanta!

Recommended Hotels and Things to See in Downtown Atlanta http://goo.gl/maps/Pj48



Agnes Scott campus

MARTA Atlanta Downtown (Five Points) and Decatur Stations

CNS 2012 Hotel in Downtown Decatur - Courtyard by Marriott

Hotels near Downtown Decatur on direct MARTA bus line

Hotels in Atlanta on direct MARTA line (bus or subway) to Agnes Scott campus, grouped by price (see Google clickable map for details).

Our picks to see in Atlanta marked with on the map

Centennial Olympic Park
Georgia Aquarium
Inside CNN Studio Tour
Fernbank Museum of Natural History
Atlanta Botanical Garden
High Museum of Art
The Fox Theater
Atlanta Braves and Turner Field (not shown on map)
Stone Mountain Park (not shown on map)

Save 51% with Atlanta CityPASS (69 USD), which gives you admission to 5 must-see Atlanta attractions: Georgia Aquarium, World of Coca Cola, Inside CNN Studio Tour, High Museum of Art OR Fernbank Museum of Natural History, Zoo Atlanta OR Atlanta History Center. Here is more info about the CityPASS http://www.citypass.com/atlanta.

Link to our Facebook Page, where you can share your CNS pictures!
http://www.facebook.com/#!/CNS2012Atlanta

CNS*2012 Party

This year's exclusive CNS*2012 party will be held on Monday evening at Twain's Billiards and Brew Pub (http://www.twains.net/), a pub located a five-minute walk from the Agnes Scott campus. "Twain's" brews their own beer, and their beers have received several national accolades and have been sold in partnership with several American craft breweries. "Twain's" will be catering the event and you will also be served a welcome drink and snacks at arrival.

*Live Stand-Up Comedy at the CNS*2012 Party*

"Dr. Pete" is a Georgia Tech engineering professor by day, and stand-up comedian by night, Pete Ludovice is out to prove that nerds can be funny and not just funny looking. His research interests include the simulation of synthetic and biological macromolecules and the use of humorous improvisation to catalyze technical innovation.

"Feel the Power of the Dork Side" is the title of his one-man show that is currently touring the U.S. from Boston to San Francisco – it is a humorous look at science and technology and its practitioners, like your science class on nitrous oxide. Pete also has a weekly radio show and podcast titled "Inside the Black Box," whose motto is "Science, only funnier."

Pete Ludovice, pete@drpetecomedy.com
http://www.drpetecomedy.com

CNS*2012 Banquet

The banquet will take place in Evans Hall at Agnes Scott College on Tuesday, July 24th, from 7pm till midnight. The event will start with **dinner** (vegetarian and gluten free options available) followed by live music entertainment. Our Local Committee has selected for you the **Savoy Kings**, **one of the hottest swing bands in Atlanta** playing upbeat jazz perfect for dancing. As usual, **prizes** will be awarded to the best student posters during the banquet.




http://www.savoykings.com/

The Savoy Kings is one of the hottest live bands in the always hot Atlanta swing scene. Playing live, they have built a steady following for over three years and become the center of the massive Atlanta swing universe. Whether they're playing a Count Basie standard or a T-Bone Walker blues, it is all Swing.

Timetable

	Saturday 7/21	Sunday 7/22	Monday 7/23	Tuesday 7/24	Wednesday 7/25	Thursday 7/26
	Tutorials	**Main Meeting, Presser Hall – Gaines Auditorium**			**Workshops**	
9:00	**Tutorials morning session** **Bullock Hall** T1 – room 102 W T2 – room 112 W T3 – room 210 E T4 – room 209 W B T6 – basement G 9	Welcome & announcements	Announcements	Announcements	**W2**-Bullock, 210E **W4**-Evans, Bsmt AB **W5**-Bullock,209W AB **W6**-Presser, Gaines Audit. **W7**-Evans, Bsmt C **W9**-Bullock, G9 **W11**-Bullock, 102W	**W1**- Bullock, 209W AB **W3**- Bullock, 210E **W6**- Presser, Gaines Audit. **W8**- Evans, Bsmt C **W9**-Bullock, G9 **W10**- Evans, Bsmt AB
9:10		**Keynote 1: Barbara Webb**	**Keynote 2: Donald Edwards**	**Keynote 3: Malcolm Maciver**		
9:20						
9:30						
9:40						
9:50						
10:00						
10:10			**Coffee Break Rebekah Hall**	**Coffee Break Rebekah Hall**		
10:20		**Coffee Break Rebekah Hall**			**Coffee Break Bullock Hall Atrium**	**Coffee Break Bullock Hall Atrium**
10:30			**Oral O12**	**Featured oral F3**		
10:40		**Oral O1**				
10:50			**Oral O13**			
11:00		**Oral O2**				
11:10			**Oral O14**	**Oral O17**		
11:20		**Oral O3**			**W2, W4, W5, W6 W7, W9, W11** (same rooms)	**W1, W3, W6, W8, W9, W10** (same rooms)
11:30			**Lunch Break**	**OCNS Member meeting**		
11:40		**Oral O4**				
11:50						
12:00	**Lunch Break**	**Group picture, outside Evans**			**Lunch Break**	**Lunch Break**
12:10						
12:20		**Lunch Break**				
12:30			**NSF Session** Evans, Bsmt. AB			
12:40						
13:20			**Lunch Break**	**Lunch Break**		
13:30	**Tutorials afternoon session** **Bullock Hall** T1 – room 102 W T2 – room 112 W T3 – room 210 E T5 – room 209 W A T7 – room 209 W B				**W2, W4, W5, W6 W7, W9, W11** (same rooms)	**W1, W3, W6, W8, W9, W10** (same rooms)
13:40						
13:45			**Featured oral F2**			
13:50						
14:00		**Oral O5**		**Oral O18**		
14:10						
14:20		**Oral O6**		**Oral O19**		
14:25			**Oral O16**			
14:30						
14:40		**Oral O7**		**Oral O20**		
14:45			**Coffee Break Rebekah Hall**			
14:50						
15:00		**Oral O8**	**Poster session II** **Bullock Atrium** *Posters 64-126*	**Coffee Break Rebekah Hall**	**Coffee Break Bullock Hall Atrium**	
15:10						
15:20		**Oral O9**		**Poster session III** **Bullock Atrium** *Posters 127-189*		
15:30						
15:40		**Coffee Break Rebekah Hall**				
15:50					**W2, W4, W5, W6 W7, W9, W11** (same rooms)	
16:00						
16:10		**Oral O10**				
16:20						
16:30	**Break**	**Featured oral F1**				
16:40						
16:50						
17:00	**Welcome reception** **Evans Hall**					
17:10		**Oral O11**				
17:20						
17:30		**Break for dinner**				
17:40						
17:50						
18:00			**Break for dinner**		**W12 – Postdoc Career Bullock** G9	
18:10						
18:20						
18:30						
19:30		**Poster session I** **Bullock Atrium** *Posters 1-63*		**CNS 2012 Banquet** **Evans Hall**		
19:50						
20:00						
20:50						
21:00			**CNS 2012 Party at Twain's Pub**			
22:30						
24:00						

Tutorials

Time: **July 21st, 2012**.
Place: **Agnes Scott campus, Bullock** (Science Center) building.

No.	Name	Time	Room
T1	Modeling and interpretation of extracellular potentials	9:00-12:00 and 13:30-16:30	102 W
T2	Theory of correlation transfer and correlation structure in recurrent networks	9:00-12:00 and 13:30-16:30	112 W
T3	Parameter Search for Neural Spiking Activity: Creation and Analysis of Simulation Databases	9:00-12:00 and 13:30-16:30	210 E
T4	Complex networks and graph theoretical concepts	9:00-12:00	209 W B
T5	Workflows for reproducible research in computational neuroscience	13:30-16:30	209 W A
T6	The finer points of modeling (with NEURON)	9:00-12:00	G 9
T7	Real-time simulation of large-scale neural models using the NeoCortical Simulator (NCS)	13:30-16:30	209 W B

Workshops

Time: **Wednesday & Thursday, July 25-26th, 2012**.
Place: **Agnes Scott campus**, Buildings: **Presser, Evans, and Bullock**.

No.	Name	Day	Time	Building	Room
W1	Behavior Informatics	Thu	9-18:00	Bullock	209 W A+B
W2	Computational Neuroethology	Wed	9-18:00	Bullock	210 E
W3	Dynamic nature, olfactory system	Thu	9-18:00	Bullock	210 E
W4	Multi-Scale Modeling	Wed	9-18:00	Evans	Basement AB
W5	Methods of Systems Identification	Wed	9-18:00	Bullock	209 W A+B
W6	Methods of Information Theory	Wed & Thu	9-18:00	Presser	Gaines Auditorium
W7	Evolutionary algorithms	Wed	9-18:00	Evans	Basement C
W8	Principles of motor pattern generation	Thu	9-18:00	Evans	Basement C
W9	Dynamics of rhythm generation	Wed & Thu	9-18:00	Bullock	G9
W10	Neurological disease dynamics	Thu	9-18:00	Evans	Basement AB
W11	Neuromechanical modeling of posture and locomotion	Wed	9-18:00	Bullock	102 W
W12	Postdoc/early career strategy	Wed	18-20:00	Bullock	G9

Tutorials

Tutorials will take place prior to the main meeting in the morning and in the afternoon of **July 21st, 2012** at **Agnes Scott campus, Bullock** (Science Center) building.

T1 **Modeling and interpretation of extracellular potentials**

Room 102 W, 9:00-12:00 and 13:30-16:30

Gaute T. 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

T2 **Theory of correlation transfer and correlation structure in recurrent networks**

Room 112 W, 9:00-12:00 and 13:30-16:30

Ruben Moreno-Bote, Foundation Sant Joan de Déu, Barcelona, Spain
Moritz Helias, Research Center Jülich, Germany

T3 **Parameter Search for Neural Spiking Activity: Creation and Analysis of Simulation Databases**

Room 210 E, 9:00-12:00 and 13:30-16:30

Cengiz Gunay, Emory University, Atlanta, USA
Anca Doloc-Mihu, Emory University, Atlanta, USA
Vladislav Sekulic, University of Toronto, Canada

T4 **Complex networks and graph theoretical concepts**

Room 209 W B, 9:00-12:00

Duane Nykamp, University of Minnesota, USA

T5 **Workflows for reproducible research in computational neuroscience**

Room 209 W A, 13:30-16:30

Andrew P. Davison, UNIC, CNRS, Gif sur Yvette, France

T6 **The finer points of modeling (with NEURON)**

Room G9, 9:00-12:00

Ted Carnevale, Yale University School of Medicine, New Haven, USA
William W. Lytton, SUNY Downstate Medical Center, NY, USA

T7 Real-time simulation of large-scale neural models using the NeoCortical Simulator (NCS)

Room 209 W B, 13:30-16:30

Laurence C. Jayet Bray, Brain Computation Laboratory, Dept. of Computer Science & Engineering, University of Nevada, Reno, USA

Roger V. Hoang, Brain Computation Laboratory, Dept. of Computer Science & Engineering, University of Nevada, Reno, USA

Frederick C. Jr. Harris, Brain Computation Laboratory, Dept. of Computer Science & Engineering, University of Nevada, Reno, USA

Main Meeting

Main meeting will take place from **July 22 - 24th, 2012** at **Agnes Scott campus**. All sessions will be held in **Presser Hall, Gaines Auditorium**, unless specially stated.

Saturday July 21

17:00 – 21:00 **Opening Reception (Evans Hall)**

Sunday July 22

9:00 – 9:20 **Welcome & Announcements**

9:20 – 10:20 K1 **Keynote Lecture:**
FRONTIERS LECTURE Neurons and behaviour: computational and robot models of insect sensorimotor control
Barbara Webb

10:20 – 10:50 **Break**

Oral Session I: Oscillations

10:50 – 11:10 O1 ***Experimentally constrained network model of hippocampal fast-firing parvalbumin-positive interneurons***
Katie Ferguson*, Carey Huh, Bénédicte Amilhon, Rosanah Murugesu, Sylvain Williams, and Frances Skinner

11:10 – 11:30 O2 ***A Dynamical Study of Pulse-Coupled Oscillators in the Brain***
Tanushree Luke*, Ernest Barreto, and Paul So

11:30 – 11:50 O3 ***Oxygen Dynamics during in Vitro Seizures***
Yina Wei*, Ghanim Ullah, Justin Ingram, and Steven Schiff

11:50 – 12:10 O4 ***Does CaMKII decode Ca2+ oscillations?***
Thiago M Pinto*, Maria Schilstra, and Volker Steuber

12:10 – 12:20 **Group picture**

12:10 – 14:00 **Break for Lunch**

Oral session II: Sensory systems

14:00 – 14:20 O5 ***Modeling the Dynamics of Neural Codes in the Olfaction of the Manduca-sexta Moth***
Eli Shlizerman*, Jeff Riffell, and J Nathan Kutz

14:20 – 14:40 O6 ***Estimating Receptive Fields and Spike-Processing Neural Circuits in Drosophila***
Aurel A. Lazar, Yevgeniy Slutskiy*

14:40 – 15:00 O7 ***Spatiotemporal pattern discrimination using predictive dynamic neural fields***
Jean-Charles Quinton*, Bernard Girau

15:00 – 15:20 O8 ***Predicting Eye Movements in a Contour Detection Task***
Udo Ernst*, Nathalie van Humbeeck, Nadine Schmitt, Frouke Hermens, and Johan Wagemans

15:20 – 15:40 O9 ***Neuronal Synchronization and Multiscale Information Representation***
Meyer Pesenson*

15:40 – 16:10 **Break**

Oral session III: Anesthesia and sleep

16:10 – 16:30 O10 ***fMRI correlates for low frequency local field potentials appear as a spatiotemporal dynamic under multiple anesthetic conditions***
Garth Thompson*, Wen-Ju Pan, Matthew Magnuson, Dieter Jaeger, and Shella Keilholz

16:30 – 17:10 F1 **Featured Oral:**
Chaotic dynamics underpins the slow oscillation of general anesthesia and nonREM sleep
Moira L. Steyn-Ross, D. Alistair Steyn-Ross*, and Jamie Sleigh

17:10 – 17:30 O11 ***Acetylcholine and synaptic homeostasis***
Christian Fink*, Victoria Booth, and Michal Zochowski

17:30 – 19:30 **Break for Dinner**

19:30 – 22:30 **Poster Session I: Posters 1 – 63**

Monday July 23

9:00 – 9:10 **Announcements**

9:10 – 10:10 K2 **Keynote Lecture:**
Neuromechanical simulation and hybrid systems: approaches to understanding the role of reafference in posture and locomotion
Donald Edwards

10:10 – 10:40 **Break**

Oral session IV: Navigation

10:40 – 11:00 O12 ***Goal-Related Navigation of a Neuromorphic Virtual Robot***
Laurence Jayet Bray*, Emily Barker, Gareth Ferneyhough, Roger Hoang, Bobby Bryant, Sergiu Dascalu, and Frederick C Harris

11:00 – 11:20 O13 ***A Neural Network Based Holistic Model of Ant Route Navigation***
Bart Baddeley, Paul Graham, Philip Husbands, and Andy Philippides*

Oral session V: Memory

11:20 – 11:40 O14 ***Balanced cortical microcircuitry for maintaining short-term memory***
Sukbin Lim*, Mark S. Goldman

11:40 – 13:45 **Break for Lunch**

12:30 – 13:30 M1 ***NSF: Theory and Applications***
Ken Whang*

Oral session VI: Leeches

13:45 – 14:25 F2 **Featured Oral:**
Utilizing multi-functional neuronal responses during different behaviors to uniquely identify all neurons in the leech ganglion.
Edward Frady*, William B. Kristan

14:25 – 14:45 O16 ***Individual differences in leech heart motor neuron models***
Damon Lamb*, Ronald L Calabrese

14:45 – 15:00 **Break**

15:00 – 18:00 **Poster session II: Posters 64 – 126**

18:00 – 20:00 **Break for Dinner**

20:00 – 24:00 **CNS 2012 Party at Twain's Pub**

Tuesday July 24

9:00 – 9:10 **Announcements**

9:10 – 10:10 K3 **Keynote Lecture:**
Energy-Information Trade-Offs Between Movement and Sensing
Malcolm Maciver

10:10 – 10:40 **Break**

Oral session VII: Electric fish

10:40 – 11:20 F3 **Featured Oral:**
Signal cancellation and contrast invariance in electrosensory systems
Jorge F Mejias*, Gary Marsat, Kieran Bol, Erik Harvey-Girard, Leonard Maler, and Andre Longtin

11:20 – 11:40 O17 ***Parallel coding of first and second order stimulus attributes***
Patrick Mcgillivray, Katrin Vonderschen, Eric Fortune, and Maurice J Chacron*

11:40 – 12:40 **OCNS Member Meeting**

12:40 – 14:00 **Break for Lunch**

Oral session VIII

14:00 – 14:20 O18 ***Short term synaptic depression with stochastic vesicle dynamics imposes a high-pass filter on presynaptic information***
Robert Rosenbaum*, Jonathan Rubin, and Brent Doiron

14:20 – 14:40 O19 ***Drug-dominated dopamine circuits spiral addicts down to a cognitive/behavioral conflict: A neurocomputational theory***
Mehdi Keramati, Boris Gutkin*

14:40 – 15:00 O20 ***The Open Source Brain Initiative: enabling collaborative modelling in computational neuroscience***
Padraig Gleeson*, Eugenio Piasini, Sharon Crook, Robert Cannon, Volker Steuber, Dieter Jaeger, Sergio Solinas, Egidio d'Angelo, and Angus Silver

15:00 – 15:30 **Break**

15:30 – 18:30 **Poster session III: Posters 127 – 189**

18:30 – 24:00 **CNS 2012 Banquet (ticket required) (Evans)**

Wed. July 25 & Thu. July 26: Workshops, 9:00 - 18:00

Workshops

Place: **Agnes Scott** campus, **Presser, Evans, and Bullock** buildings. Time: July 25-26, 2012.

W1 **Behavior Informatics: data bases, data mining and experiments in virtual worlds**

Bullock, Room 209 W A+B, Day(s): Thu, 9:00:18:00

Ansgar Koene, RIKEN BSI, Tokyo, Japan

W2 **Computational Neuroethological Approaches to Problems in Social Neuroscience**

Bullock, Room 210 E, Day(s): Wed, 9:00:18:00

Robert Liu, Emory University, Atlanta, GA, USA
Elizabeth Buffalo, Emory University, Atlanta, GA, USA

W3 **Examining the dynamic nature of neural representations with the olfactory system**

Bullock, Room 210 E, Day(s): Thu, 9:00:18:00

Christopher Buckley, RIKEN BSI, Tokyo, Japan
Taro Toyizumi, RIKEN BSI, Tokyo, Japan
Thomas Nowotny, University of Sussex, Sussex, UK

W4 **Multi-Scale Modeling in Computational Neuroscience II: Challenges and Opportunities**

Evans, Room Basement AB, Day(s): Wed, 9:00:18:00

James Bower, UTSA, San Antonio, Texas, USA
Ilya Rybak, Drexel University, Philadelphia, PA, USA

W5 **Methods of Systems Identification for Studying Information Processing in Sensory Systems**

Bullock, Room 209 W A+B, Day(s): Wed, 9:00:18:00

Aurel Lazar, Columbia University, New York, NY, USA
Mikko Juusola, University of Sheffield, Sheffield, UK

W6 **Methods of Information Theory in Computational Neuroscience**

Presser, Room Gaines Auditorium, Day(s): Wed & Thu, 9:00:18:00

Todd Coleman, UCSD, San Diego, CA, USA
Michael Gastpar, EPFL, Lausane, Switzerland
Conor Houghton, Trinity College, Dublin, Ireland
Aurel Lazar, Columbia University, New York, NY, USA
Simon Schultz, ICL, London, UK
Tatyana Sharpee, Salk Institute, San Diego, USA

W7 **Modern evolutionary algorithms in computational neuroscience : tools to parameterize, explore model properties & design model structures**

Evans, Room Basement C, Day(s): Wed, 9:00:18:00

Benoît Girard, UPMC/CNRS, Paris, France
Denis Sheynikhovich, UPMC/CNRS, Paris, France
Jean-Baptiste Mouret, UPMC/CNRS, Paris, France
Stephane Doncieux, UPMC/CNRS, Paris, France

W8 **Principles of motor pattern generation: experiments and modeling**

Evans, Room Basement C, Day(s): Thu, 9:00:18:00

Carmen Canavier, LSU HSC, New Orleans, LA, USA
Andrey Shilnikov, GSU, Atlanta, USA

W9 **Dynamics of rhythm generation**

Bullock, Room G9, Day(s): Wed & Thu, 9:00:18:00

Roman Borisyuk, University of Plymouth, Plymouth, UK
Ronald Calabrese, Emory University, Atlanta, GA
Alan Roberts, University of Bristol, Bristol, UK
Gennady Cymbalyuk, Georgia State University, Atlanta, GA

W10 **Disease dynamics: Computational modeling of neurological diseases**

Evans, Room Basement AB, Day(s): Thu, 9:00:18:00

Sharmila Venugopal, UCLA, Los Angeles, CA, USA
Ranu Jung, FIU, Miami, FL, USA

W11 **Neuromechanical modeling of posture and locomotion**

Bullock, Room 102 W, Day(s): Wed, 9:00:18:00

Boris I. Prilutsky, Georgia Institute of Technology, Atlanta, GA, USA
Alexander N. Klishko, Georgia Institute of Technology, Atlanta,GA, USA

W12 **Postdoc and Student Career Strategy Workshop**

Bullock, Room G9, Day(s): Wed, 18:00:20:00

Nathan W. Schultheiss, Boston University, Boston, MA, USA

Tutorials

T1 Modeling and interpretation of extracellular potentials

Room 102 W, 9:00-12:00 and 13:30-16:30

Gaute T. 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 workhorse in electrophysiology, the interpretation of such recordings is not trivial. 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. However, 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. 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 [1-3], MUA [4] and LFP, both from single neurons [5] and populations of neurons [4,6], and
- methods for
  - estimation of current source density [7] from LFP data, such as the iCSD [8-10] and kCSD methods [11], and
  - decomposition of recorded signals in cortex into contributions from various laminar populations, i.e., (i) laminar population analysis (LPA) [12] based on joint modeling of LFP and MUA, and (ii) a novel scheme using LFP and known constraints on the synaptic connections [13]

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

- LFPy (compneuro.umb.no/LFPy), a versatile tool based on Python and the simulation program NEURON [14] (www.neuron.yale.edu) for calculation of extracellular potentials around neurons, and

- tools for iCSD analysis, in particular,
  - CSDplotter (for linear multielectrodes [8]) (software.incf.org/software/csdplotter)
  - iCSD 2D (for 2D multishank electrodes [10]) (software.incf.org/software/icsd-2d)

References:
1. G Holt & C Koch (1999). J Comp Neurosci 6:169.
2. J Gold et al (2006). J Neurophysiol 95:3113.
3. KH Pettersen and GT Einevoll (2008). Biophys J 94:784.
4. KH Pettersen et al (2008). J Comp Neurosci 24:291.
5. H Lindén et al (2010). J Comp Neurosci 29: 423.
6. H Lindén et al (2011). Neuron 72:859.
7. C Nicholson and JA Freeman (1975). J Neurophsyiol 38:356.
8. KH Pettersen et al (2006). J Neurosci Meth 154:116.
9. S Łęski et al (2007). Neuroinform 5:207.
10. S Łęski et al (2011). Neuroinform 9:401.
11. J Potworowski et al (2012). Neural Comp 24:541.
12. GT Einevoll et al (2007). J Neurophysiol 97:2174.
13. SL Gratiy et al (2011). Front Neuroinf 5:32.
14. ML Hines et al (2009). Front Neuroinf 3:1.

T2 Theory of correlation transfer and correlation structure in recurrent networks

Room 112 W, 9:00-12:00 and 13:30-16:30

Ruben Moreno-Bote, Foundation Sant Joan de Déu, Barcelona, Spain
Moritz Helias, Research Center Jülich, Germany

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, 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 2010] and by negative feedback [Tetzlaff

2010]. 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 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. Ginzburg & Sompolinsky (1994). Theory of correlations in stochastic neural networks, PRE 50:3171-3190.
2. Renart et al. (2010). The Asynchronous State in Cortical Circuits, Science 327(5965):587-590.
3. Tetzlaff et al. (2010). Decorrelation of low-frequency neural activity by inhibitory feedback, BMC Neuroscience 11(Suppl 1):O11.
4. Hawkes (1971). Point Spectra of Some Mutually Exciting Point Processes, Journal of the Royal Statistical Society Series B 33(3):438-443.
5. Helias et al. (2011). Towards a unified theory of correlations in recurrent neural networks, BMC Neuroscience 12(Suppl 1):P73.
6. Shadlen & Newsome (1998). The variable discharge of cortical neurons: implications for connectivity, computation, and information coding, J Neurosci 18:3870-96.
7. Moreno-Bote & Parga (2006). Auto- and crosscorrelograms for the spike response of leaky integrate-and-fire neurons with slow synapses, PRL 96:028101.
8. de la Rocha et al. (2007). Correlation between neural spike trains increases with firing rate, Nature 448:802-6.
9. Zohary et al. (1994). Correlated Neuronal Discharge Rate and Its Implications for Psychophysical Performance, Nature 370:140-14, Complex networks and graph theoretical concepts.

T3 Parameter Search for Neural Spiking Activity: Creation and Analysis of Simulation Databases

Room 210 E, 9:00-12:00 and 13:30-16:30

Cengiz Gunay, Emory University, Atlanta, USA
Anca Doloc-Mihu, Emory University, Atlanta, USA
Vladislav Sekulic, University of Toronto, Canada

Parameter tuning of model neurons to achieve biologically realistic spiking patterns is a non-trivial task, for which several methods have been proposed. One method is to perform a systematic search through a very large parameter space (with thousands to millions of model instances), and then categorize spiking neural activity characteristics in a database [1-8]. This technique is of key importance because of the existence of multiple parameter sets that give similar dynamics, both experimentally and in silico – i.e. there is no single "correct" model. In this tutorial, we will teach some of the implementations of this

method (e.g., the PANDORA Matlab Toolbox [6,11]) used in recent projects for tuning models of rat globus pallidus neurons [5,9], lobster pyloric network calcium sensors [7,10], leech heart interneurons [8,12] and hippocampal O-LM interneurons (Skinner Lab, TWRI/UHN and Univ. Toronto).
The tutorial will be composed of three parts that will include the following topics:

1. Running simulations for systematic parameter search
    - Model complexity versus simulation time trade off (single compartment versus full morphology; how many channels to include?)
    - Working with Hodgkin-Huxley type ion channels and morphological reconstructions (e.g., determining dendritic distributions of Ih channels in hippocampal O-LM interneurons)
    - Determining ranges for channel, synapse, and morphology parameters
    - Setting up simulations and storage to accommodate a large number of output files
    - Examples using GENESIS, NEURON and custom C/C++ simulators
    - Control of simulations on high-performance clusters
    - Troubleshooting common pitfalls
2. Extracting of activity characteristics and constructing of databases
    - Measuring spike shape, firing rate and bursting properties
    - Analyzing large number of simulation output files
    - Standardizing feature extraction and error handling
    - Examples using Matlab, Java and shell scripting languages
3. Analysis of information in databases
    - Calculating histograms, correlations, etc.
    - Ranking simulations based on similarity to recordings
    - Multivariate parameter analysis
    - Data mining methods
    - Visualization (e.g., dimensional stacking)
    - Higher order methods (e.g., factor and principal component analyses)

Each of these parts will have time allocated for Q&A and interaction with the audience. If participants bring a laptop pre-loaded with Matlab, they can follow some of our examples.

References:
1. Prinz AA, Billimoria CP, and Marder E (2003). Alternative to hand-tuning conductance-based models: Construction and analysis of databases of model neurons. J Neurophysiol, 90:3998–4015.
2. Prinz AA, Bucher D, and Marder E (2004). Similar network activity from disparate circuit parameters. Nat. Neurosci. 7(12): 1345-1352.

3. Calin-Jageman RJ, Tunstall MJ, Mensh BD, Katz PS, Frost WN (2007) Parameter space analysis suggests multi-site plasticity contributes to motor pattern initiation in Tritonia. J Neurophysiol 98:2382–2398.
4. Lytton WW, Omurtag A (2007). Tonic-clonic transitions in computer simulation. J Clin Neurophysiol. 24(2):175-81.
5. Günay C, Edgerton JR, and Jaeger D (2008). Channel density distributions explain spiking variability in the globus pallidus: A combined physiology and computer simulation database approach. J. Neurosci., 28(30): 7476–91.
6. Günay C, Edgerton JR, Li S, Sangrey T, Prinz AA, and Jaeger D (2009). Database analysis of simulated and recorded electrophysiological datasets with PANDORA's Toolbox. Neuroinformatics, 7(2):93-111.
7. Günay C, and Prinz AA (2010). Model calcium sensors for network homeostasis: Sensor and readout parameter analysis from a database of model neuronal networks. J Neurosci, 30:1686– 1698.
8. Doloc-Mihu A, and Calabrese RL (2011). 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.

Model and Software Links:
9. Rat globus pallidus neuron model (https://senselab.med.yale.edu/modeldb/ShowModel.asp?model=114639)
10. Lobster stomatogastric ganglion pyloric network model
(http://senselab.med.yale.edu/ModelDB/showmodel.asp?model=144387)
11. PANDORA Matlab Toolbox (http://software.incf.org/software/pandora)
12. Half-Center Oscillator model database
(http://senselab.med.yale.edu/ModelDB/ShowModel.asp?model=144518)

T4 Complex networks and graph theoretical concepts

Room 209 W B, 9:00-12:00

Duane Nykamp, University of Minnesota, USA

Increasing evidence suggests a structure in the brain's networks that isn't well described by standard random graph models. Such findings open up the debate whether or not the networks in the brain are "small world" or "scale-free," contain central well-connected "hubs," are highly "clustered" or "modular." But, how does ones interpret the significance of this supposedly "non-random" structure? Can we determine how such network features influence the dynamics of neuronal networks? In this tutorial, we will introduce basic graph theoretical concepts and their application to complex networks. We will examine experimental findings about network structure in the brain and discuss the potential of the graph theoretical framework on shedding light on the function of neural circuits.

T5 Workflows for reproducible research in computational neuroscience

Room 209 W A, 13:30-16:30

Andrew P. Davison, UNIC, CNRS, Gif sur Yvette, France

Reliably repeating previous experiments, one of the cornerstones of the scientific method, ought to be easy in computational neuroscience, given that computers are deterministic, not suffering from the problems of inter-subject and trial-to-trial variability that make reproduction of biological experiments so challenging. In general, however, it is not at all easy, especially when running someone else's code, or when months or years have elapsed since the original experiment.

The failure to routinely achieve replicability in computational neuroscience (probably in computational science in general, see Donoho et al., 2009 [1]) has important implications for both the credibility of the field and for its rate of progress (since reuse of existing code is fundamental to good software engineering). For individual researchers, as the example of ModelDB has shown, sharing reliable code enhances reputation and leads to increased impact.

In this tutorial we will identify the reasons for the difficulties often encountered in reproducing computational experiments, and some best practices for making our work more reliable and more easily reproducible by ourselves and others (without adding a huge burden to either our day-to-day research or the publication process).

We will then cover a number of tools that can facilitate a reproducible workflow and allow tracking the provenance of results from a published article back through intermediate analysis stages to the original models and simulations. The tools that will be covered include Git [2], Mercurial [3], Sumatra [4] and VisTrails [5].

References:
1. Donoho et al. (2009). 15 Years of Reproducible Research in Computational Harmonic Analysis, Computing in Science and Engineering 11: 8-18. doi:10.1109/MCSE.2009.15.
2. http://git-scm.com/
3. http://mercurial.selenic.com/
4. http://neuralensemble.org/sumatra
5. http://www.vistrails.org/

T6 The finer points of modeling (with NEURON)

Room G9, 9:00-12:00

Ted Carnevale, Yale University School of Medicine, New Haven, USA
William W. Lytton, SUNY Downstate Medical Center, NY, USA

This tutorial will focus on practical aspects of constructing and using models of cells and networks that will help modelers improve their productivity and the quality of their models. We will cover topics that include efficient strategies for specifying model properties, tactics and tools for debugging, and what we judge to be important, if sometimes overlooked, aspects of hoc, Python, and NMODL. This is not an "introductory" course–attendees are assumed to be familiar with using hoc or Python to develop

NEURON models of cells or networks. Applicants with a strong interest in specific questions or topics are encouraged to email suggestions to ted dot carnevale at yale dot edu before June 16, 2012.

T7 Real-time simulation of large-scale neural models using the NeoCortical Simulator (NCS)

Room 209 W B, 13:30-16:30

Laurence C. Jayet Bray, Brain Computation Laboratory, Dept. of Computer Science & Engineering, University of Nevada, Reno, USA

Roger V. Hoang, Brain Computation Laboratory, Dept. of Computer Science & Engineering, University of Nevada, Reno, USA

Frederick C. Jr. Harris, Brain Computation Laboratory, Dept. of Computer Science & Engineering, University of Nevada, Reno, USA

This tutorial will mostly concentrate on how to design large-scale models using the NeoCortical Simulator (NCS), and to run simulations in real-time.

This is an introductory course for attendees who wish to learn a new simulation program, which emphasizes the construction, the simulation, and the analysis of current brain models. Additional information will be given on distribution capabilities, levels of abstraction, software and hardware platforms, possible real-time virtual robotic applications, and how does NCS differ from other simulation programs.

Current research has demonstrated a recent software optimization and hardware improvements, which have helped increase simulation speed and ameliorate the robustness of complex brain models.
NCS requires NO computer programming experience.
Applicants with further questions are welcome to contact Laurence at ljayet at cse dot unr dot edu.

Keynote Lectures

Barbara Webb

Institute for Perception, Action and Behaviour, School of Informatics, University of Edinburgh, UK

E-mail: bwebb@inf.ed.ac.uk

K1 – FRONTIERS LECTURE Neurons and behaviour: computational and robot models of insect sensorimotor control

Neural circuits evolved to control behaviour, so behaviour should always be the context when we try to understand what a neural circuit computes. One way to maintain this focus is to embody computational models of neural processing in robots that sense and act. Another is to investigate circuits for which it is plausible to connect the properties of individual neurons to the production of adaptive behaviour. The work I will describe combines these approaches by building models of insect sensorimotor behaviours and testing them in robotic implementations. In many cases, these supposedly simple and specialised circuits have properties that provide wider insights into higher cognition. I will discuss recent work on navigation and learning in insects to illustrate these issues.

Donald Edwards

Neuroscience Institute, Georgia State University, Atlanta, USA

E-mail: dedwards@gsu.edu

K2 – Neuromechanical simulation and hybrid systems: approaches to understanding the role of reafference in posture and locomotion

A major challenge in studying the neural control of posture and locomotion in legged, multi-jointed animals is to reconcile the synaptic and cellular responses obtained from anesthetized, restrained, and dissected preparations with the EMG and limb movement data obtained from freely behaving animals. This challenge is compounded by the importance of reafference in modulating locomotor output, especially in legged animals, for which step-wise variations in the terrain can demand cycle-to-cycle changes in the output. We are meeting this challenge in our study of crayfish locomotion with three approaches: in vivo motion analysis and EMG recordings, neuromechanical simulation with AnimatLab, and a hybrid system consisting of an in vitro preparation and neuromechanical simulation that closes the sensorimotor feedback loop. Motion analysis and EMG recordings reveal the motor patterns that underlie reflex responses to perturbation while the animal stands and walks. How those motor patterns are produced and lead to the recorded leg and body movements are what needs to be explained by neuromechanical simulation and in vitro experiments. We have used the neuromechanical simulator AnimatLab (www.AnimatLab.com) to reconstruct the body, including the exoskeleton, joints, muscles, and sensors, and the relevant neural circuits in a neuromechanical model of the crayfish situated in a virtual physical world. The model can then be tested to determine whether experimentally identified neural mechanisms of control are sufficient to account for the EMG and movement patterns recorded in vivo. The hybrid system allows a dissected ventral nerve cord and single leg preparation to be connected to a neuromechanical model of the leg, muscles, and sensors to create an in vitro preparation in which the sensorimotor feedback loops can be closed. Recorded spikes from identified levator and depressor motor neurons excite model levator and depressor muscles to evoke leg movements in real time. Movements of the leg then stretch and release a model stretch receptor; the calculated length changes then mechanically drive the real stretch receptor to create the same length changes in real time. The spiking responses of the live stretch receptor afferents then project back to the ventral nerve cord to complete the sensorimotor feedback loop. This talk will describe each of these approaches, and relate how closing the feedback loop enables reflex reversal to increase the walking locomotor frequency by three-fold.

Malcolm Maciver

Dept. of Biomedical & Mechanical Engineering, Northwestern University, Evaston, USA

E-mail: maciver@northwestern.edu

K3 – Energy-Information Trade-Offs Between Movement and Sensing

Weakly electric fish are a popular model system within sensory neurobiology. More recently, our group and others have been working on their biomechanics, and we have found many ways in which the sensory biology of electric fish is interwoven with their biomechanics. In my talk, I will show how the fish's omnidirectional sensing is complemented with a biomechanical plant that is nonholonomic but nonetheless able to reach all parts of the omnidirectional sensory space within 3-4 sensorimotor delay time cycles of the organism. A robotic weakly electric fish and fully resolved 3D Navier Stokes simulations are elucidating the mechanistic principles underlying this remarkable small-time maneuverability. An energetics approach shows that when sensory performance and locomotory performance are in conflict, they are co-optimized. Quantifying the sensory volumes and time-limited movement spaces of aquatic animals has highlighted the vastly different sensory biophysics of water versus air. This difference may have been important for the evolution of neural mechanisms of planning (one form of consciousness) in vertebrates through greatly increasing the ratio of the active sensory space to the time-limited movement space of early tetrapods.

Featured & Oral Talks

F1 Chaotic dynamics underpins the slow oscillation of general anesthesia and nonREM sleep

Moira L. Steyn-Ross[1], D. Alistair Steyn-Ross[1⋆], and Jamie Sleigh[2]

[1] *School of Engineering, University of Waikato, Hamilton 3240, New Zealand*
[2] *Waikato Clinical School, University of Auckland, Hamilton 3240, New Zealand*

Electrical recordings of brain activity show that entry into anesthetic unconsciousness is signposted by the emergence of large, slow oscillations of electrical activity (~1 Hz) that appear very similar to the slow waves observed in natural sleep. In this phase, populations of cortical neurons periodically switch between hyperpolarized inactivity ("down" state), and wake-like depolarized activation ("up" state) [1]. The origin of the slow oscillation has not yet been unambiguously established, and remains an area of intense research and debate [2,3]. Here we suggest a novel mechanism in which the up- and down-states are generated spontaneously by emergent chaotic waves of spatiotemporal activity that sweep the cortex. We present a mean-field model of the cortex in which populations of neurons are densely interlinked by both chemical synapses - including idealized long-range spatially heterogeneous connections - and by direct electrical connections forming a continuous network of interneuronal gap junctions. Anesthetic effect is modeled as a moderate reduction in inhibitory diffusion, paired with an increase in inhibitory postsynaptic potential. We explore model dynamics in the vicinity of a general-anesthetic induced transition from wake to coma. In this region the system is poised at a codimension-2 point where competing Turing (spatial) and Hopf (temporal) instabilities co-exist. We argue that normal functioning of the resting "default-wake" brain requires a delicate balance between these instabilities. Reduction of gap-junction diffusivity disturbs the balance in favor of the Hopf instability, eventually predicting global seizure in the limit of severe imbalance. Our cortical model predicts that introduction of anesthetic to the awake brain will force a subtle rebalancing of dynamic pressures resulting in a coma state that is characterized by emergent low-frequency oscillations whose dynamics is chaotic in time and space: see Fig. 1. We quantify cortical dynamics in terms of a phase coherence measure and demonstrate that the model-predicted turbulent slow-wave state is characterized by low phase coherence. This prediction is supported by clinical studies of phase synchronization changes in EEG during induction of propofol anesthesia [4].

Conclusion

A spontaneous, spatiotemporally chaotic state - generated by nonlinear Turing–Hopf interaction - is the underlying mechanism for the slow oscillation observed in general anesthesia. A similar transition to low-frequency chaos may also occur in natural nonREM sleep, and if so, may have significant implications for synaptic downscaling and memory processing hypothesized to occur during deep sleep.

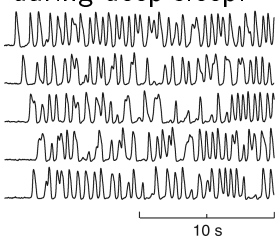


Figure 1: Spontaneous slow-wave oscillations in cortical firing-rate patterns during 20 s of simulated anesthesia. Traces were recorded from five equally-spaced points lying along the midline of the 25-x25-cm simulated cortical grid. Time-series are chaotic in space and time.

References
1. Steriade M, Nuñez A, and Amzica F (1993). A novel slow (<1 Hz) oscillation of neocortical neurons in vivo: depolarizing and hyperpolarizing components. J Neurosci, 13:3252 – 3265.
2. Timofeev I, Grenier F, Bazhenov M, Sejnowski TJ, and Steriade M (2000). Origin of slow cortical oscillations in deafferented cortical slabs. Cereb Cortex, 10(12):1185 – 1199.
3. Sanchez-Vives MV and McCormick DA (2000). Cellular and network mechanisms of rhythmic recurrent activity in neocortex. Nat Neurosci, 3(10):1027 – 1034.
4. Koskinen M, Seppänen T, Tuukkanen J, Yli-Hankala A, and Jäntti V (2001). Propofol anesthesia induces phase synchronization changes in EEG. Clin Neurophysiol, 112(2):386 – 392.

F2 Utilizing multi-functional neuronal responses during different behaviors to uniquely identify all neurons in the leech ganglion.

Edward Frady[1⋆], William B. Kristan[2]

[1] *Department of Neurosciences, UC San Diego, La Jolla, CA 92093, USA*
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In the leech, we can observe several behaviors – swimming, crawling, shortening, and local-bending – while imaging neuronal activity with voltage-sensitive dyes (VSD) [1]. To understand the underlying neural mechanisms of these behavioral pattern generators, we must understand the functional properties of the neurons and the connectivity between neurons, which requires collecting and synthesizing data across several animals. However, this synthesis is not trivial because more than 70% of the neurons in the leech ganglia have not been identified. We have created a system which utilizes the functional responses of each neuron during these behaviors, as well as during unconventional stimulations, to match homologous pairs of neurons across different animals. Neurons in the leech are almost all multi-functional, in that they are involved in many different behaviors. For a particular neuron, we can use these multi-functional responses as a tag indicating the neuron's identity.

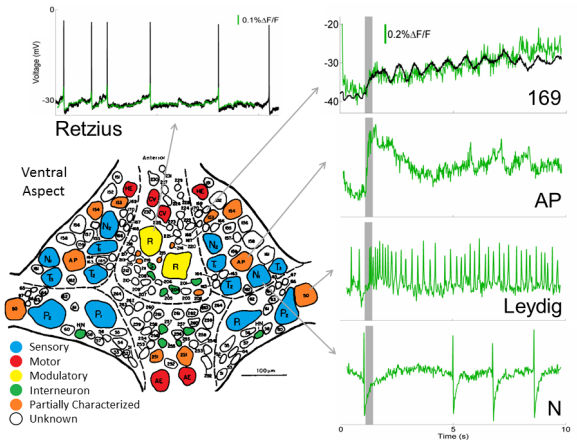

Figure 1: The ventral aspect of the leech ganglion is shown with many of the known cells highlighted in color. About two-thirds are unknown, indicated in white. Several VSD recordings of cells are shown in green. Simultaneous intracellular recordings are shown in black for the Retzius cell and cell 169.

For instance, a neuron in the central packet on the ventral surface can be identified from its neighbors because it oscillates in phase with the dorsal contraction during swimming, and out of phase with the dorsal contraction during crawling. We have developed an algorithm that efficiently combines all of

these functional properties as well as each neuron's morphological properties (soma size and position) to match homologous cells across animals. We have recently developed a new VSD [2] that has given us unprecedented signal-to-noise ratio and temporal resolution of neural activity. This allows us to use cues such as action-potential shape and response timing as further indicators of neuronal identity. Figure 1 shows VSD recordings of several neurons that can now be identified based on their action potentials – such as the Retzius, Leydig, and N cell, or based on response to stimulations – such as the rapid response of the AP cell or the oscillations of cell 169 during swimming. We have used these functional features to identify almost every neuron in ganglion 10 by matching homologs across many different animals. Each animal reveals a different subset of all neurons in the circuit, which are all combined to identify virtually every cell in the leech ganglion. We have used several statistical functional connectivity techniques to estimate the connection profile of these neurons. With these connectivity predictions, we can probe identified cells with micro-electrodes to validate connections and build up the leech connectome with the knowledge of all of the neurons in the circuit and their functional properties during each behavior.

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F3 Signal cancellation and contrast invariance in electrosensory systems

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When processing sensory input, it is of vital importance for the neural systems to be able to discriminate a novel stimulus from the background of redundant, unimportant signals. Neural mechanisms responsible for prediction and cancellation of redundant information could be an efficient way to achieve such discrimination. While the concrete mechanisms that the brain employs for this task are presently unknown, a network able to perform this cancellation is thought to exist in the electrosensory lateral line lobe (ELL) of weakly electric fish [1]. This fish emits a high-frequency (600-1000 Hz) sinusoidal electric organ discharge (EOD) into its environment to sense its surroundings and communicate to conspecifics. Small objects such as prey create spatially localized amplitude modulations (AMs) of the EOD, whereas tail bending or communication signals induce spatially global AMs [2]. These AMs are detected by electroreceptors that densely cover the body of the fish, and provide feedforward input to pyramidal cells in the ELL. It is known that a subpopulation of such pyramidal cells, the superficial pyramidal (SP) cells, remove low-frequency predictable global signals (i.e. tail bending) from their input to maximize detection of novel local stimuli (i.e. prey) [1]. This is presumably achieved using a feedback pathway involving the granule cell layer (a cerebellar-like structure known as EGp). These granule cells connect to SP cells via parallel fibers (PFs)

which may be acting as delay lines segregated into frequency channels to destructively interfere with the global stimulus. Recent in vitro studies found a novel burst timing-dependent learning rule which would be able to shape this feedback [3].

Following a previous work [4], we study the cancellation of low-frequency simple redundant signals, i.e. sine waves, in the ELL of the weakly electric fish. The study combines in vitro data, in vivo electrophysiology recordings from neurons in the ELL and numerical modeling to address this issue. More precisely, we model the neural network responsible for signal cancellation in the ELL of the fish, and compare our predictions with electrophysiology data recorded in vivo [4]. In the model, we assume the presence of: 1) stimulus-driven feedback to the SP neurons, 2) a large variety of temporal delays in the PFs transmitting such feedback, and 3) burst-induced long-term plasticity. We show that the modeled network is able to efficiently cancel global redundant signals by shaping the feedback as a negative image of the global signal arriving to the SP cells. Such negative image is generated via the burst-induced anti-Hebbian learning rule in the PF-SP cell synapses, while the full period of the signal is covered by the incoming feedback due to the wide range of PF delays present in the network. The cancellation is found to be in agreement with in vivo recordings, and it is strong for signals with frequencies up to 16 Hz, enabling a clearer background above which to detect relevant non-repetitive stimuli such as prey signals (and thus to better capture the prey). Due to the importance of the phase-relationship between the feedback and the stimulus, the mechanism is found to be frequency-specific, suggesting the presence of multiple frequency channels as observed in vivo [4]. Interestingly, our model predicts that the cancellation is maintained for signals with different AM strengths (i.e. contrasts). Such contrast-invariance is highly desirable since natural signals would display different contrasts depending, for instance, on the distance between the fish and the origin of the EOD perturbation.

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O1 Experimentally constrained network model of hippocampal fast-firing parvalbumin-positive interneurons

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The fast-firing properties of parvalbumin-positive (PV+) interneurons, and their extensive connections with neighbouring excitatory neurons, provide them with enormous potential to influence network rhythms and hence behaviour. Thus, it is not entirely surprising that these cells have been implicated in playing a role in a variety of pathologies (e.g. epilepsy [1]). Mathematical modeling allows one to explore the contribution of this population in a simplified setting, and make predictions to guide further experimental work. However, direct links between existing fast-spiking interneuron models and empirically determined cellular intrinsic and network characteristics are unclear, and therefore model predictions can be difficult to interpret in a biological setting. Therefore, we have created a network model of PV+ interneurons that is tied to experimental work on multiple levels, and we use this model to investigate the potential of this population to realize coherent oscillations.

Our PV+ interneurons are represented with an Izhikevich-type model [2], and involve parameter values that are designed to approximate the cell's intrinsic properties. To determine these parameters, spike characteristics and passive properties were extracted from whole-cell patch clamp recordings of PV+ interneurons in the CA1 region of an intact hippocampal preparation in vitro. Our network model is composed of these individual PV+ cell models, and the network size, architecture, and synaptic properties are chosen to be consistent with those found in the literature. Recordings during emergent network oscillations [3] provided us with information about realistic firing rates and synaptic activity of PV+ interneurons. These firing rates, used in combination with the cell's intrinsic frequency-current profile, provided physiological constraints on the amount of synaptic current the PV+ cells receive during these spontaneous network oscillations. Under voltage clamp, excitatory post-synaptic current peaks are used in our model as an upper bound on the range of synaptic input. We used this network model to determine whether coherent rhythms could be produced within experimental constraints.

Our model produced intrinsic properties and spiking behaviors which approximated the experimentally determined membrane capacitance, resting membrane potential, threshold potential, spike width, spike peak potential, peak after-hyperpolarizing potential, and amount of adaptation. Model parameters were determined such that the slope of the model's frequency-current profile and the model rheobase current were within the range of our experimental data. As such, we have produced a network model of PV+

interneurons that has direct links to cellular characteristics with model parameters that have clear biological interpretations. In addition, network simulations of our PV+ interneuron model produced coherent gamma output. Since the firing properties and network architecture of PV+ interneurons puts them in an ideal position to influence network activity, this cell type will likely remain a focus of experimentalists and modelers alike. A model such as ours, with clear links to biology, may be used as a platform to investigate the role of these fast-firing PV+ interneurons in network oscillations and behaviour.

Acknowledgements
This work was supported by the Canadian Institutes of Health Research and Natural Sciences and Engineering Research Council of Canada

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O2 A Dynamical Study of Pulse-Coupled Oscillators in the Brain

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In 1967, Winfree[1] proposed a novel mathematical approach to describe phenomena of collective synchrony in nature (i.e. flashing of fireflies, clapping in a theatre, alpha rhythms, etc.) using a large coupled network of phase oscillators with a diversity of natural frequencies. By analyzing this large heterogeneous network from a "mean field" approach, the spontaneous synchrony can be understood as a critical phase transition similar to most statistical mechanical systems. In this work, we employ this approach to model the phase transitions and bifurcation structures of a large network of pulse-coupled theta neurons [2] by appropriate choice of Winfree's "response" and "influence" functions, the latter of which is parameterized by a "sharpness" parameter n [1]. As this parameter increases, the influence function approximates the behavior of a pulse-coupled synapse. Assuming a Lorentzian distribution of natural frequencies of width D and mean value w_0, taking the thermodynamic limit, and employing the Ott-Antonsen reduction method [3], the collective dynamics of the pulse-coupled network can be analytically reduced to a single low-dimensional dynamical equation for the mean field parameter $z(t)$.

We analyze the bifurcation diagrams for different values of the sharpness parameter n. We find that more complex behavior is apparent with increasing sharpness of the influence function, and that equilibria outside the physically relevant region (within the unit circle) affect the transient dynamics of $z(t)$ inside the circle. Further, we find that some level of coherence always exists in the network for non-zero coupling, in contrast to other mean field coupled phase oscillator networks [1,4]. Most interestingly, heterogeneity is observed to suppress complexity in the collective/macroscopic behavior. As the network becomes more

homogeneous ($\Delta \to 0$), more complex dynamic including aperiodic and multistability emerge from the macroscopic mean field.

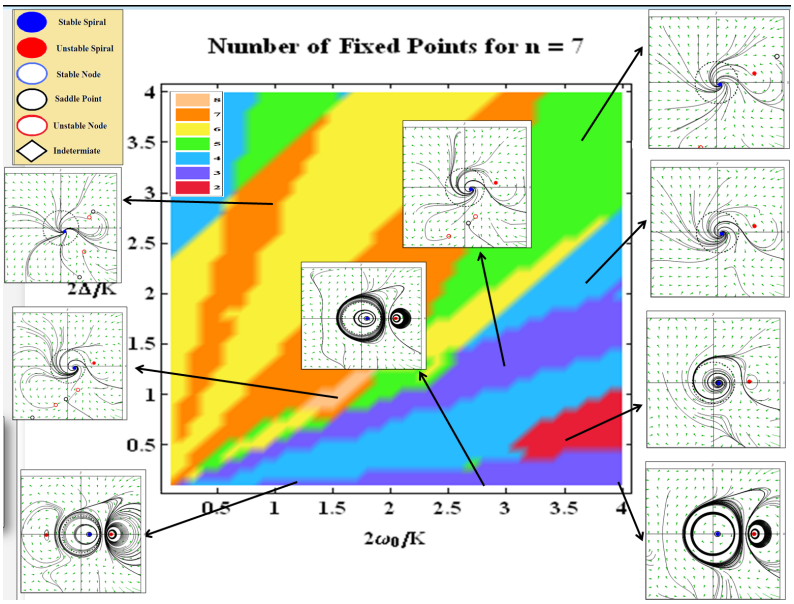

Figure 1: A sample bifurcation diagram showing the complex structure of fixed points at various locations in parameter space, for a sharpness parameter of ($n = 7$). Several representative phase portraits from several distinct region of parameter space are included.

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O3 Oxygen Dynamics during in Vitro Seizures

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Oxygen is an essential element for brain activity. The brain is a metabolic engine that requires 20% of the body's metabolic energy, despite being only 2% of the human body mass [1]. Two thirds of brain's metabolic energy is dedicated to supporting neural spiking activity. Much of the O2 dependent ATP metabolism in single neurons is used by energetic Na/K-ATPase pumps that transport $3Na^+$ outwards with $2K^+$ inward against their concentration gradients for each ATP hydrolyzed [2,3]. However, understanding the relationship between seizures and real-time oxygen dynamics has been restricted by current technical limitations. Computational models can offer insight to help understand the measurements from experiments.

We have performed experiments relating seizure activity at the cellular level with simultaneous real-time O2 microdomain measurements. In this paper, we build a mathematical neuron model that extends the Hodgkin-Huxley formalism containing leak currents for sodium, potassium and chloride ions, transient sodium currents, and delayed rectifier potassium currents. This neuron was embedded within an extracellular space and a simplified glia-endothelium system. The Na^+ and K^+ ion concentrations as well as extracellular oxygen density were continuously estimated. Hypoxia was modeled by reducing both neuron and glial Na/K-ATPase pump activities.

During seizure events, the extracellular K^+ and intracellular Na^+ were increased, which further activated the Na/K-ATPase pump activity. Energy (ATP) and O2 demand were simultaneously increased. Therefore, local available [O2] decreased substantially during seizure events, and the apparent O2 debt substantially outlasts the intense electrical activity of a seizure, as shown in Figure 1. This result is consistent with experimental data [4]. We also observed that hypoxia alone can induce seizure like events, which occurs only in a narrow range of bath oxygen pressure, reflecting experimental observations. Lastly, we reproduced the interplay between excitatory and inhibitory neurons seen in experiments. Our model accounts for the different $[O_2]$ levels that we have observed during seizures in pyramidal cell layers vs. inhibitory (oriens lacunosum moleculare) cell layers. Our work suggests the critical importance of modeling extracellular ion concentration and oxygen dynamics to properly understand the underlying mechanisms behind seizure and related phenomena.

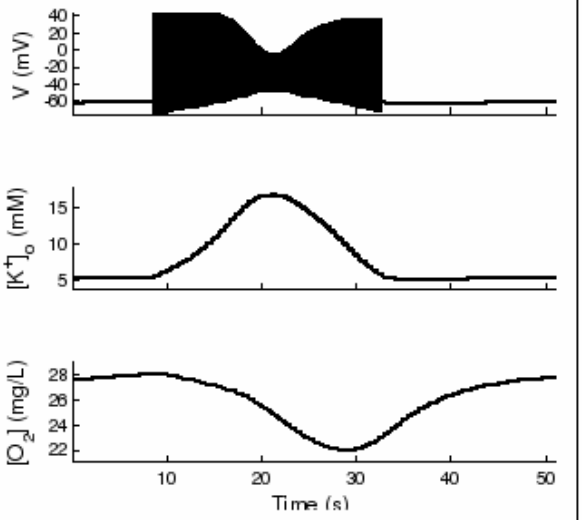

Figure 1. Membrane potentials (top trace), extracellular potassium concentration (middle trace), and oxygen density (bottom trace) from a single model neuron during a seizure event. The time course of $[O_2]$ debt is qualitatively similar to that observed experimentally.

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O4 Does CaMKII decode Ca2+ oscillations?

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Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII), which is present in high concentrations in the brain, contributes to many forms of synaptic plasticity. The induction of synaptic plasticity by CaMKII involves an intracellular signalling cascade that links neuronal Ca^{2+} signals with the phosphorylation of neurotransmitter receptors; an important step in this biochemical cascade is the autophosphorylation of CaMKII after binding of Ca^{2+}/calmodulin (Ca_4-CaM).

The dependence of this autophosphorylation reaction on the temporal structure of Ca_4-CaM signals has been investigated in previous experiments [1] and computer simulations [2]. These experimental and theoretical studies have indicated that the autophosphorylation of CaMKII is sensitive to the frequency of repetitive Ca^{2+} pulses, and it has been concluded that CaMKII can decode oscillatory Ca^{2+} signals [1,2].

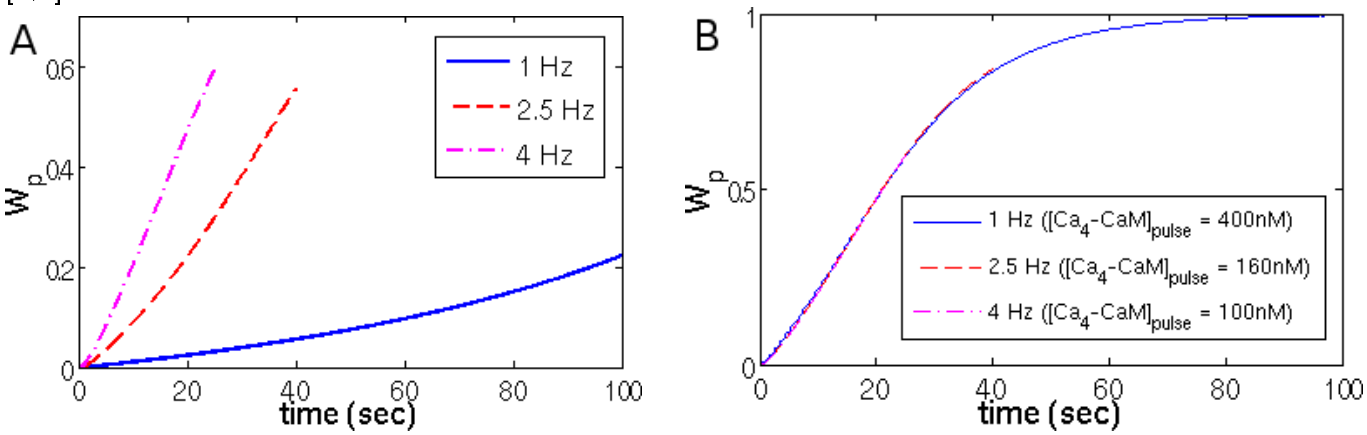

Figure 1: CaMKII phosphorylation and its dependence on the effective Ca_4-CaM concentration. (A) Temporal evolution of the phosphorylated form of CaMKII (Wp) in response to one hundred 200 ms square pulses of Ca_4-CaM (100 nM) at frequencies of 1 Hz (solid blue), 2.5 Hz (dashed red) and 4 Hz (dashed-dotted magenta) in our simplified model. (B) Wp in response to one hundred 200 ms square pulses of Ca_4-CaM at 1, 2.5 and 4 Hz, but with scaled pulse amplitudes so that the effective concentration of Ca_4-CaM is 80 nM. The amplitudes of Ca_4-CaM pulses are 400 nM at 1 Hz (solid blue), 160 nM at 2.5 Hz (dashed red) and 100 nM at 4 Hz (dashed-dotted magenta).

Here, we apply a simplified version of the commonly used CaMKII activation model by Dupont and collaborators [2] to investigate the mechanism that underlies the dependence of the overall autophosphorylation kinetics on the frequency of Ca^{2+} oscillations. In the simulations by Dupont et al., CaMKII was subjected to different average, or 'effective', Ca_4-CaM concentrations, which in turn affected the average concentration of the CaMKII subunits, and the autophosphorylation kinetics.

We first replicate the simulation results presented in [2] with our simplified model (Figure 1A). To identify the mechanism that underlies the observed frequency dependence, we then rescale the Ca_4-CaM concentrations to an equal effective concentration, and compare the phosphorylation kinetics (Figure 1B). We demonstrate that in our model the overall phosphorylation rate under sustained application of Ca_4-CaM pulses depends on the average ('effective') concentration of Ca_4-CaM in the system, rather than on the pulse frequency itself. Moreover, we show that the application of a constant level of Ca_4-CaM with the same mean concentration as in the pulsed protocol results in the same level of CaMKII phosphorylation.

Our simulation results indicate that the notion of CaMKII as a decoder of Ca^{2+} oscillations is misleading and suggest experimental tests with rescaled Ca_4-CaM concentrations.

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O5 Modeling the Dynamics of Neural Codes in the Olfaction of the Manduca-sexta Moth

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The Anntenal Lobe (AL) is the olfactory processing unit in insects, composed of projection neurons (PNs) and local neurons (LNs) [1]. It has been demonstrated that the AL reformats the sensory input information that it receives into spatiotemporal firing patterns exhibited by the PNs [2,3]. In many insects the LNs are mainly inhibitory which suggests that the inhibition is responsible for shaping the input into a robust pattern [4]. The robustness of the pattern is expressed as follows: over several applications of the same odor the projection of the data recorded from the PNs onto a few dominant firing patterns results in a robust low dimensional trajectory. This trajectory appears to be similar to a trajectory that converges to a stable unique fixed point [5].

In this work, we resolve several open questions raised by [5,6]. Specifically, we study how interactions between the LNs and PNs permit creation of robust and spatiotemporal codes. We further propose a simple model that mimics the dynamics of the AL and the dynamics of mixtures of odors. Our work is a combination of theoretical analysis and experimental studies.

Theoretically we consider a network of excitatory-inhibitory firing units that has a similar structure to the AL. By proposing to project the dynamical equations of the network onto given orthogonal spatial patterns, we derive the conditions on the interactions between the inhibitory and excitatory population such that the network will support a unique stable fixed point. The analysis is based on requiring that the inhibitory neurons will suppress inputs that are not associated to specific odors (noise and unknown patterns) but being neutral to the given spatial patterns. These conditions allow us to prescribe the connections between PNs and LNs and do not require specific symmetries (in contrast to Hopfield networks).

Experimentally we record the dynamics of PNs in the AL of the Manduca Sexta moth both for inputs that are single odor or mixtures of two odors. Extracting the spatial patterns (first PCA mode) obtained from experiments with single odor inputs we calibrate the network. We are able to establish similarity between the model dynamics and experimental projections and thus validate our theoretical construction. Once the model is calibrated we test it against experiments with inputs that are different ratios of two odors revealing similar dynamics. To test the importance of inhibition, conjectured to be responsible for

existence of a stable fixed point, we repeat the experiments in which the inhibition is blocked by a drug. Results demonstrate that the robustness of the dynamics is destroyed.

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O6 Estimating Receptive Fields and Spike-Processing Neural Circuits in Drosophila

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One of the long-term goals of sensory neuroscience is the development of sound experimental and theoretical methods for understanding the functional organization of sensory systems. In this regard, Drosophila melanogaster is the model organism of choice: it boasts a relatively small brain, its sensory systems have been anatomically well characterized and it offers an extensive genetic toolbox for visualizing and altering its neural circuits. Despite these advantages however, comprehensive models of sensory processing in Drosophila are sparse, in particular due to the lack of methods for estimating spike-processing neural circuits in higher brain centers. The majority of existing neural circuit models and methods for their identification assume rate-based systems (see [1] for a review), and take both the input (stimuli) and the output (response rates) to be in the continuous domain. In a practical setting, however, outputs of most neurons in a sensory system are sequences of all-or-none action potentials. Furthermore, input signals are continuous only for those neurons that are located at the sensory periphery. In contrast, input signals for neurons upstream of sensory neurons are spatiotemporal spike trains. Hence, there is a need to develop a generic framework for estimation of both receptive fields in the periphery and of spatiotemporal spike processing upstream.

Here we propose a novel theoretical approach for estimating receptive fields in circuit models that incorporate biophysical spike-generating mechanisms (e.g., the Hodgkin-Huxley neuron) and admit both continuous sensory signals and multidimensional spike trains as input stimuli. We thus explicitly take into account the highly nonlinear nature of spike generation that has been shown to result in significant interactions between various stimulus features [2], [3] and to fundamentally affect the estimation of receptive fields [4]. Furthermore, and in contrast to many existing methods [1], our approach estimates receptive fields directly from spike times produced by a neuron, thereby obviating the need to repeat ex-

periments in order to compute the neuron's instantaneous rate of response (e.g., PSTH). The employed test signals belong to spaces of bandlimited functions and bridge the gap between identification using synthetic and naturalistic stimuli. This makes our methodology particularly attractive in those sensory modalities (most notably olfaction [5]), where it is difficult to produce stimuli that are white and/or have particular distribution/ attributes [1]-[4]. First, we work out in detail algorithms for identifying temporal, spatial and spatiotemporal receptive fields in the sensory periphery. We show that our methodology is readily generalizable to multiple receptive fields as well as to higher dimensions, allowing one to consider more complex receptive fields, if needed. Second, we demonstrate how to identify the processing of multiple spiking inputs converging onto the dendritic tree of a spiking neuron. Third, we show that the presented methodology allows one to model integration of sensory modalities in higher brain centers. Finally, we test the proposed approach using in-vivo data recorded from the olfactory system of Drosophila.

Acknowledgements
The work presented here was supported by NIH under the grant number R01DC008701-05.

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O7 Spatiotemporal pattern discrimination using predictive dynamic neural fields

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Prediction and competition mechanisms are here combined into a neuro-inspired computational model in order to enhance robustness for spatiotemporal tracking and pattern recognition tasks. The research presented in this abstract extends the initial experimental results and mathematical accuracy proof obtained with a single predictor [1] to a set of predictors. This distributed model is grounded on the Continuum Neural Field Theory (CNFT) that uses global inhibition and local excitation to implement competition [2]. External stimulations and internal predictions bias the dynamics of the field so as to constraint the selection and tracking of a target. Conflicting signals are indirectly used to filter out noise and inhibit predictors that are not adapted to the current situation (see Figure 1). The topology of the neural fields grants generalization capabilities to the system, and flexibility is thus further increased as interpolation occurs between predictors.

Results

The performance and emergent attentional properties of the model were ascertained on a 2D visual tracking application with ambiguous and noisy signals. Results are synthesized in Table 1, averaged over 60 simulations. A percentage lower than 100% means the performance has improved with an adequate predictor. A value below 20% generally means the original CNFT equation lost the target, in contrast with the extended version.

Table 1: Predictive/reactive tracking error ratio.

Scenario	Ratio
Competition between distant identical stimuli	114%
Moving target with 30 random distracters	67%
Moving target with Gaussian noise ($\sigma = 0.5$)	51%
Obstacle on trajectory (fixed distracter)	10%
Full occlusion of the target after convergence	17%

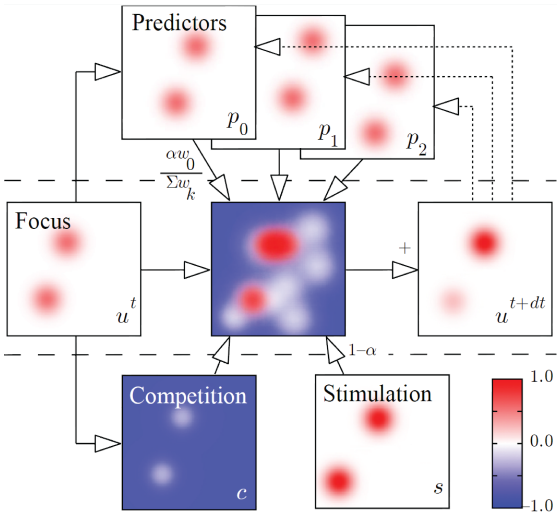

Figure 1. Graphical representation of the extended CNFT equation. The bubble on the focus field will preferentially move where the stimulation and focus activities are strongly correlated, but convergence is here biased by predictors that adequately anticipates the dynamics (p0).

Conclusions

While the predictors improve tracking performance when they adequately anticipate the dynamics, their inadequacy simply leads to a fall back on the original CNFT dynamics. This allows the system to perform correctly while learning the predictors, but also to discriminate between trajectories, as the relative level of assimilation of the dynamics is updated in real-time.

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O8 Predicting Eye Movements in a Contour Detection Task

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An important task for the visual system is grouping local image elements into meaningful objects. One

fundamental process for performing this task is contour integration, in which collinearly aligned local edges are merged into global contours. Models for contour integration often use iterative algorithms to explain how this cognitive process is performed in the brain. By employing an association field (AF) which quantifies how strongly two oriented edge elements are linked to be part of a contour, such a model integrates edge elements in a recurrent manner. This process generates saliency maps for contours of increasing lengths as time proceeds. Recently, we developed a probabilistic model of contour integration which explains human contour detection behavior to a previously unprecedented degree [1]. Given this performance, we wondered whether the model might also explain the spatiotemporal dynamics of contour integration. Measuring eye movements can be a useful method to test the corresponding model predictions, hypothesizing that subsequent fixations of subjects preferentially visit 'hotspots' of neural activity which dynamically emerge during the integration process. Here we compare model simulations with data from a recent experiment [3], in which eye movements were measured while observers were instructed to search for a 7-element contour embedded in a background of randomly oriented Gabor elements [2]. The experiment consisted of two tasks: for the first task observers were asked to indicate whether a global contour was on the left or right hemifield (left-right task), while the second task required observers to indicate presence or absence of a contour (present-absent task). The parameters of the model were first optimized for the left-right task, requiring it to reproduce both human performance and decisions as best as possible.

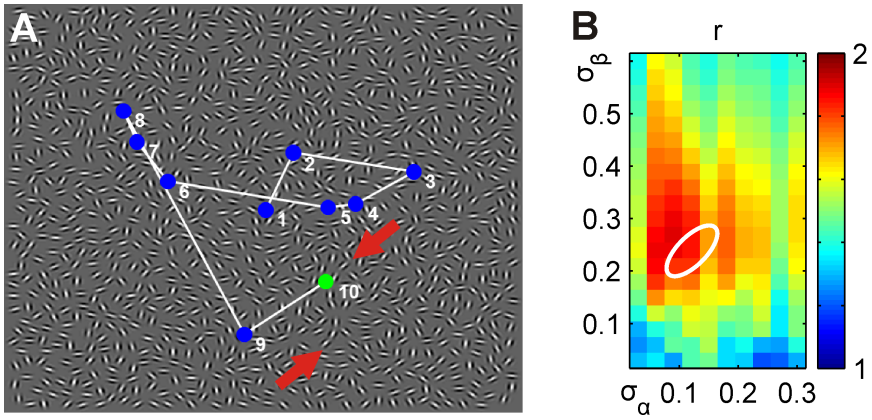

Figure 1. A. Sample stimulus containing a contour (red arrows), with overlaid saccade trajectory of one subject. B. Factor r by which model activity in the second task is higher at saccade target locations than at other locations, in dependence on two parameters defining the shape of its association field. The white ellipse denotes the parameter regime for which the model optimally fitted human contour detection behavior in the first task.

The optimal model was then used to predict potential locations for saccade targets which we compared to fixation trajectories of observers for stimuli from the second task in which no contour was present. For edge elements near saccade targets, the model predicts a probability to belong to a contour which is two times higher than for other edge elements. Thus, the statistical analysis shows that fixations are indeed not random, but are likely to occur on locations judged salient by the model. This result confirms both the validity of our model and the hypothesis that saccades on random Gabor fields preferentially visit locations with edge configurations similar to contours.

Acknowledgements
This work was supported by a PhD and a Postdoctoral fellowship of the Research Foundation Flanders (FWO-Vlaanderen), awarded to NVH and FH, respectively, and a Methusalem grant from the Flemish Government (METH/08/02), awarded to JW. We wish to thank Roger Watt for providing computer code for stimulus generation.

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O9 Neuronal Synchronization and Multiscale Information Representation

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Many important processes in neurobiology as well as neuronal engineering applications rely upon multiresolution representation and analysis of external information. There are various approaches which attempt to explain how human perception systems perform multiscale representation and sparse coding. The model proposed here is based on a new approach to multiresolution of input signals and reveals synchronization as a general mechanism for multiscale representation common to various sensory systems. The proposed mechanism is nonlinear and adaptive in the sense that it does not rely on convolution with a preconceived basis. For the visual system this approach is a major departure from the current linear paradigm, which holds that the structure of the receptive fields and their variations are responsible for performing multiscale analysis. While there are some well-known, important roles played by entrainment in neuronal systems, our model reveals a new function of dynamic coordination in the brain - multiscale encoding, thus demonstrating that synchronization plays a greater role in perception in general and in vision in particular, than was previously thought.

Acknowledgements
This work was supported by the grant from NGA, NURI HM1582-08-1-0019, and the grant AFOSR, MURI Award FA9550-09-1-0643.

O10 fMRI correlates for low frequency local field potentials appear as a spatiotemporal dynamic under multiple anesthetic conditions

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In the previous decade, interest in the "functional connectivity" of the brain has greatly increased, but the nature of the signal underlying derived predictive metrics remains poorly understood. A typical study uses functional magnetic resonance imaging (fMRI) and calculates regions of correlated low-frequency activity or "functional networks" when no task is being performed, the "resting state". However, unlike traditional block/event based fMRI, the spontaneous fluctuations that determine such networks may not relate to a standard "hemodynamic response" to neural activity and may be task and brain region dependent. Ten rats were anesthetized with either isoflurane (iso) or dexmedetomidine (med). Each rat had simultaneous

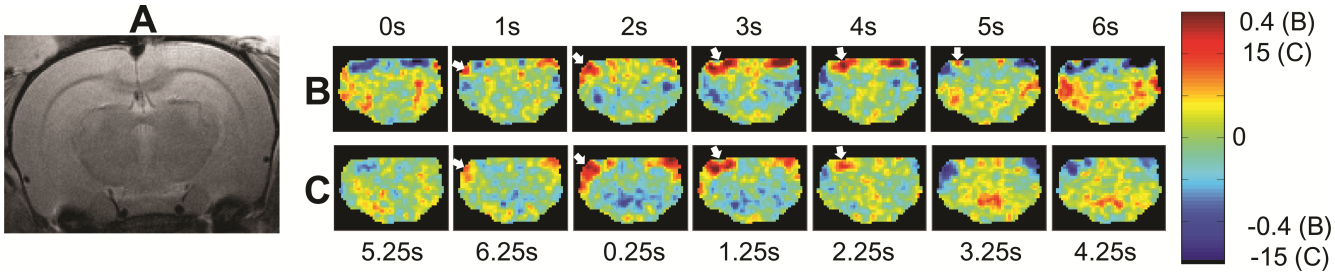

Figure 1: A. A coronal image of a rat's brain in the same plane as the fMRI images used in this study. B. (med) r_t between LFP and fMRI at each voxel, times listed are the time shift of LFP prior to fMRI. C. (med) fMRI pattern from Majeed et al. algorithm , times listed are arbitrary, so they are shifted to match (B).

local field potentials (LFP) recorded from implanted electrodes in bilateral primary somatosensory cortex (SI) simultaneously with single-slice fMRI of SI. After preprocessing, signals were filtered to regions of significant spectral coherence (0.04-0.18Hz iso, 0.05-0.3Hz med). Pearson correlation (r_t) was calculated between LFP signals at time shifts -10s to 10s relative to fMRI, at every fMRI voxel (Figure 1B). Instead of a simple hemodynamic response, the LFP correlates appeared both to have a component of spatial propagation (Figure 1B, white arrows), and alternation between positive and negative correlation. This was observed using both anesthesias and suggests that LFPs in coherent frequencies do not simply reflect local activation, but may instead be part of a large scale dynamic process. Using an fMRI-based algorithm validated in both anesthetized rats and awake humans , a spatiotemporal dynamic was produced that was highly similar to r_t (Figure 1C). Spatial correlation (r_s) between the two types of pattern reached a maximum at approximately the same shift between patterns in all rats, mean $r_s = 0.25$ (med) and mean $r_s = 0.23$ (iso), with mean $r_s > 0.10$ indicating significance at $p < 0.05$ when using boot-strapping and correcting for multiple comparisons. These results suggest that the neural basis of functional networks may be more complex than a simple hemodynamic response and possibly contains contributions from large-scale neuromodulatory processes.

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O11 Acetylcholine and synaptic homeostasis

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The synaptic renormalization hypothesis posits that a primary function of sleep is to maintain synaptic homeostasis [1]. According to this theory, the flood of sensory signals processed by the brain during waking results in global potentiation of cortical synapses, a process which consumes energy and space and therefore cannot continue unabated. Sleep is therefore a period of global synaptic downscaling that maintains homeostasis, thereby conserving energy and cortical space. Specifically, it is slow-wave activity (SWA) during NREM sleep that is thought to induce this depotentiation. While evidence in support of both global potentiation of synapses during waking [2] and SWA-mediated downscaling of synapses during sleep [3] continues to mount, there is still much uncertainty about the biophysical mechanisms which may contribute to either synaptic upscaling or downscaling [4].

Waking and sleep states are promoted by the activity of brainstem and hypothalamic neuronal nuclei that express key neurotransmitters in thalamic and cortical brain regions [5]. Waking is characterized by high levels of noradrenaline, serotonin, histamine and acetylcholine, while all these neurotransmitters are at low levels during NREM sleep. We propose that the influence of acetylcholine (ACh) may provide a mechanism for both upscaling and downscaling of cortical synapses. Specifically, experimental studies have shown that ACh modulation switches the phase response curves of cortical pyramidal cells from Type II to Type I. Our computational studies of cortical networks show that the presence of ACh induces cellular and network dynamics which lead to net synaptic potentiation under a standard STDP rule, while the absence of ACh alters dynamics in such a way that the same STDP rule leads to net depotentiation (see Fig. 1). Thus the well-established prevalence of ACh in cortical circuits during waking may lead to global synaptic potentiation, while the absence of ACh during NREM sleep may lead to global depotentation. Counter-intuitively, the global potentiation induced by the presence of ACh in our simulated networks is due to asynchronous activity. This is due to the fact that in the asynchronous state, there exists important statistical structure to the network dynamics, so that post-synaptic neurons are more likely to fire immediately after (rather than before) a pre-synaptic action potential, thus leading to net potentiation of the network due to STDP.

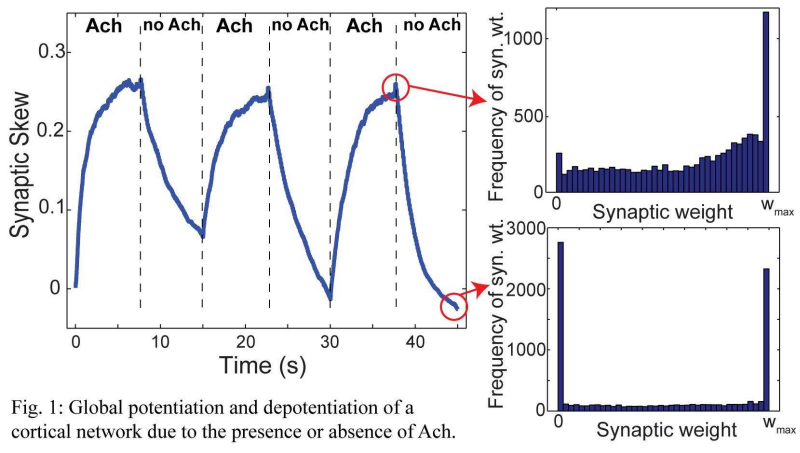

Fig. 1: Global potentiation and depotentiation of a cortical network due to the presence or absence of Ach.

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O12 Goal-Related Navigation of a Neuromorphic Virtual Robot

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The field of biologically inspired technology has evolved to the emergence of robots that operate autonomously. Some studies have focused on developing social robots that interact with humans by following social behaviors where other research have centered their efforts on mobile robots with the ability to navigate in their well-known environment. These general-purpose autonomous robots can perform a variety of functions independently, from recognizing people or objects to navigating in a familiar room. As of yet, no humanoid robot has been capable of traveling through a new suburban environment to reproduce goal-related learning and navigational activities.

Based on experimental findings, we propose a computational model that is composed of critical interacting brain regions and utilizes fundamental learning mechanisms. It is incorporated in a sophisticated robotic system where a virtual robot navigates through a new environment, learns and recognizes visual landmarks, and consequently makes correct turning decisions to reach a reward.

The detailed brain architecture included visual, entorhinal, prefrontal and premotor cortices, as well as the hippocampus. Our microcircuitry replicated some fundamental mammalian dynamics, which were integrated in a robotic loop. This virtual robotic system was designed around a number of components unique to our NeoCortical simulator (NCS) and our Virtual NeuroRobotic (VNR) paradigm. The neural simulation was executed on a remote computing cluster and was networked to the other system components (NCSTools, Webots, Gabor filter) using our Brain Communication Server (BCS), a server developed specifically for integration with NCS.

The virtual humanoid was able to navigate through a new virtual environment and reach a reward after a sequence of turning actions. Along the way, it encountered familiar and non-familiar external cues to provide guidance and follow the correct direction. This is the first bio-inspired robot that showed

high functionality during navigation while utilizing spiking cortical neurons in a real-time simulation. More importantly, it could take us a step closer to understanding memory impairments in Alzheimer's patients.

O13 A Neural Network Based Holistic Model of Ant Route Navigation

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The impressive ability of social insects to learn long foraging routes guided by visual information [1] provides proof that robust spatial behaviour can be produced with limited neural resources [2–3]. As such, social insects have become an important model system for understanding the minimal cognitive requirements for navigation [1]. This is a goal shared by biomimetic engineers and those studying animal cognition using a bottom-up approach to the understanding of natural intelligence [4]. Models of visual navigation that have been successful in replicating place homing are dominated by snapshot-type models where a single view of the world as memorized from the goal location is compared to the current view in order to drive a search for the goal [5, for review, see 6]. Snapshot approaches only allow for navigation in the immediate vicinity of the goal however, and do not achieve robust route navigation over longer distances [7].

Here we present a parsimonious model of visually guided route learning that addresses this issue [8]. We test this proposed route navigation strategy in simulation, by learning a series of routes through visually cluttered environments consisting of objects that are only distinguishable as silhouettes against the sky. Our navigation algorithm consists of two phases. The ant first traverses the route using a combination of path integration and obstacle avoidance during which the views used to learn the route are experienced. Subsequently, the ant navigates by visually scanning the world – a behaviour observed in ants in the field – and moving in the direction which is deemed most familiar. As proof of concept, we first determine view familiarity by exhaustive comparison with the set of views experienced during training. In subsequent experiments we train an artificial neural network to perform familiarity discrimination using the training views via the InfoMax algorithm [9].

By utilising the interaction of sensori-motor constraints and observed innate behaviours we show that it is possible to produce robust behaviour using a learnt holistic representation of a route. Furthermore, we show that the model captures the known properties of route navigation in desert ants. These include the ability to learn a route after a single training run and the ability to learn multiple idiosyncratic routes to a single goal. Importantly, navigation is independent of odometric or compass information, does not specify when or what to learn nor separate the routes into sequences of waypoints, so providing proof of concept that route navigation can be achieved without these elements. The algorithm also exhibits both place-search and route navigation with the same mechanism. As such, we believe the model represents the only detailed and complete model of insect route guidance to date.

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O14 Balanced cortical microcircuitry for maintaining short-term memory

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Persistent patterns of neural activity that last long after the offset of a stimulus are thought to be the neural substrate for short-term memory. Because the observed decay of persistent activity in memory circuits is much slower than the typical decay time constants associated with synaptic or intrinsic neuronal dynamics, it has been suggested that network interactions must be used to prolong the duration of persistent activity. Most often, these network interactions have been assumed to mediate positive feedback between neurons that supports a long-lasting reverberation of activity. However, most positive feedback models do not naturally fit the architecture of working memory-storing structures in neocortex that have been suggested to exhibit a close balance between excitation and inhibition. Furthermore, positive feedback models of analog memory storage are highly non-robust against commonly studied perturbations in network connectivity.

Here, we suggest a complementary mechanism for generating persistent activity based on the principle of corrective negative-feedback: an error-correcting signal of the form of a time-derivative of activity reduces memory slip when it occurs. Using analytic calculations, we show that neocortical circuit models with the

observed balance in strength, but with different kinetics, between excitatory and inhibitory synaptic inputs, produce a negative-derivative feedback signal that counteracts drifts in persistent activity. The networks maintain a continuum of stable firing rates even in the presence of intrinsic input-output nonlinearity, while still remaining responsive to external memory inputs. More generally, the networks act as temporal integrators of their inputs, for example converting step-like input into linearly ramping activity.

Memory networks operating in this balanced regime are robust against many commonly studied perturbations to synaptic weights that grossly disrupt the performance of persistent activity circuits based on positive feedback. Specifically, in response to uniform changes in synaptic excitation, synaptic inhibition, intrinsic neuronal gains or loss of a fraction of excitatory or inhibitory neurons, there is minimal decay or instability in persistent firing. Furthermore, spiking network models implementing derivative feedback generate persistent firing with Poisson-like statistics, as has been observed experimentally. This observed highly irregular activity occurs across a graded range of firing rates, and arises because the close balance between excitation and inhibition results in spikes being triggered primarily by fluctuations in, rather than means levels of, synaptic inputs.

To generate experimental predictions that distinguish among different mechanisms for short-term memory, we compared the correlation structure of excitatory and inhibitory inputs in the negative-derivative feedback models to that of typical analog memory models based on positive feedback. Negative-derivative feedback models exhibit a strong positive correlation between inhibitory and excitatory synaptic inputs, as suggested by recent experiments. By contrast, similarly responding neurons in positive feedback models either exhibited anti-correlations or weak correlations between their excitatory and inhibitory inputs.

Altogether, this work suggests a new paradigm for short-term memory storage based upon a balanced network with cortical-like architecture. Stabilization of responses through negative feedback is a common principle in engineering control systems. Our work suggests that a similar principle might be inherent to the circuitry of working memory systems.

O15 Stretching of memory in strategic decision making

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– WITHDRAWN –

O16 Individual differences in leech heart motor neuron models

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Motor neurons are frequently overlooked as critical contributors to the programming of motor output except where they directly play a role in central pattern generators (CPGs). Leech heart motor neurons

have been shown to contribute significant phase shifts to the rhythmic motor patterns they produce , although the most important factor in pattern formation is the interaction of CPG output and synaptic weights from the CPG onto the motor neurons. We seek to address the question of which neural parameters, in particular active conductances, are important for functional pattern formation, and how they influence it. We are well positioned to address this question, as we have a unique dataset comprising the complete leech heart motor neuron input pattern, output pattern, and synaptic weights for multiple ganglia in many individual animals. We have begun to exploit these data to develop more realistic Hodgkin-Huxley style biophysical models of the leech heart motor neurons by constraining models optimized by a multi-objective evolutionary algorithm to fit this animal-specific data. In particular, we extended the single compartment Garcia model to a multi-compartmental model and added a slow calcium and a calcium sensitive potassium channel, currents known to be present in the living motor neurons. Our model's dimensions are derived from reconstructions of fluorophore filled leech heart motor neurons in segments 8-12. Membrane currents were distributed according to experimental data and the result of hand tuning; e.g., the fast sodium current is only present in the axonal compartment. This base model was then parameterized such that the maximal conductances of the active currents present in each compartment, as well as the electrical coupling conductance between each pair of motor neurons, were allowed to vary as free parameters in our evolutionary algorithm. We then utilized our input-output data by delivering a particular input pattern (specific animal and motor neuron pair) to a model motor neuron and then comparing the resulting output with that recorded in the same animal. In this manner, we constrained our definition of an 'acceptable' model by the input/output pattern recorded in individual animals – such a model must transform the input into the correct output within some reasonable error bound. To generate acceptable models we use a Multi Objective Evolutionary Algorithm (MOEA) to create a population of models from which a subset achieves 'acceptable' status. In this framework, multiple independent fitness functions are evaluated separately – the phase, duty cycle, spike height, slow-wave height, and within-burst inter-spike interval (ISI) for the two phases produced. The targets for these parameters are drawn from the same individual animal as the input/output data, and error bounds are based on the within-animal variability, although slightly widened for parameters such as ISI, slow-wave and spike height, as they vary greatly between animals (derived primarily from within-animal experimental variability). Acceptable model simulation traces were all but indistinguishable from intracellular recordings from the living system, with the exception of measurement noise, even though they are constrained by a small number of functional parameters. In order to identify key parameters and relationships between synaptic weights and membrane conductances which are constrained by the functional requirements of the system, we will conduct a sensitivity analysis of the acceptable models and inspect the dimensional stacking of acceptable and failed models. The identified conductances can then be perturbed in the living system either via dynamic clamp or pharmacological manipulation to validate the modeling results.

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O17 Parallel coding of first and second order stimulus attributes

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Natural stimuli often have time varying first (i.e. mean) and second order (i.e. variance) attributes that each carry critical information for perception and can vary independently over orders of magnitude. We recorded the responses of midbrain electrosensory neurons in the weakly electric fish Apteronotus leptorhynchus to stimuli with first and second order attributes that varied independently in time. We found two distinct groups of midbrain neurons: the first group responded to both first and second order attributes while the other responded selectively to second order attributes. Using computational analyses, we show how inputs from a heterogeneous population of ON- and OFF-type afferent neurons are combined in order to give rise to response selectivity to second order stimulus attributes in midbrain neurons. Our study thus uncovers, for the first time, generic and widely applicable mechanisms by which selectivity to second order stimulus attributes emerges in the brain.

O18 Short term synaptic depression with stochastic vesicle dynamics imposes a high-pass filter on presynaptic information

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The filtering properties of synapses are modulated by a form of short term depression arising from the depletion of neurotransmitter vesicles. The uptake and release of these vesicles is stochastic in nature, but a widely used model of synaptic depression does not take this stochasticity into account. While this model of synaptic depression accurately captures the trial-averaged synaptic response to a presynaptic spike train [1], it fails to capture variability introduced by stochastic vesicle dynamics [2]. Our goal is to understand the impact of stochastic vesicle dynamics on filtering and information transfer in depressing synapses.

We derive compact, closed-form expressions for the synaptic filter induced by short term synaptic depression when stochastic vesicle dynamics are taken into account and when they are not. We find that stochasticity in vesicle uptake and release fundamentally alters the way in which a synapse filters presynaptic information. Predictably, the variability introduced by this stochasticity reduces the rate at

which information is transmitted through a synapse. Additionally, this variability introduces frequency-dependence to the transfer of information through a synapse: a model that ignores synaptic variability transmits slowly varying signals with the same fidelity as faster varying signals [3, 4], but a model that takes this variability into account transmits faster varying signals with higher fidelity than slower signals (Figure 1). Differences between the models persist even when the presynaptic cell makes many contacts onto the postsynaptic cell. We extend our analysis to the population level and conclude that a slowly varying signal must be encoded by a large presynaptic population if it is to be reliably transmitted through depressing synapses, but faster varying signals can be reliably encoded by smaller populations. Our results provide useful analytical tools for understanding the filtering properties of depressing synapses and have important consequences for neural coding in the presence of short term synaptic depression.

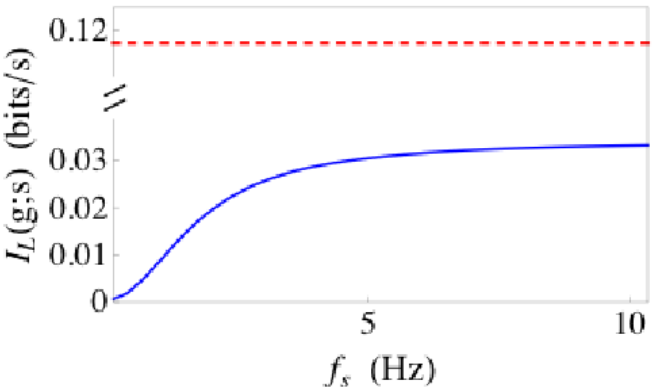

Figure 1: The linear information rate, $I_L(g;s)$, which represents the information per unit time available to an optimal linear decoder that estimates a rate-coded presynaptic signal, $s(t)$, by observing a postsynaptic conductance, $g(t)$. The linear information rate is plotted as a function of the peak frequency, f_s, of the signal. When stochastic vesicle dynamics are ignored (dashed red line), $I_L(g;s)$ is independent of f_s [3,4]. When stochastic vesicle dynamics are accounted for (solid blue line), information transfer is reduced and high-frequency signals are transferred more reliably than low-frequency signals.

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O19 Drug-dominated dopamine circuits spiral addicts down to a cognitive/behavioral conflict: A neurocomputational theory

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Long-term addicts find themselves powerless to resist drugs, despite knowing that drug-taking may be a harmful course of action, and an explicit motivation to quit. In controlled experiments, human addicts show a self-described mistake characterized by an inconsistency between drug-seeking response and their reported subjective value. We provide a unified computational theory for this inconsistency by showing how addictive drugs gradually produce a motivational bias toward drug-seeking at low-level habitual

decision processes, despite the low abstract cognitive values. This pathology emerges within the hierarchical reinforcement learning (HRL) framework when chronic drug-exposure pharmacologically hijacks the dopaminergic spirals that cascade reinforcement signal down the ventro-dorsal cortico-striatal hierarchy.

$$\delta_t^n = [r_t + V^{n+1}(s_{t+1}) - Q^n(s_t, a_t)] + D$$

$$Q^n(s_t, a_t) \leftarrow Q^n(s_t, a_t) + \alpha.\delta_t^n$$

Here, r_t is the rewarding value of the outcome, be it natural rewards or addictive drugs. These equations show that in order to compute the prediction error signal for updating the value (Q) of state-action pairs at the n-th level of decision hierarchy, the value of the temporally-advanced state (s_{t+1}) comes from one higher level of abstraction ($n+1$). This captures the role of dopamine-dependent serial connectivity linking the ventral to the dorsal striatum (known as dopamine spirals), which is suggested to integrate information across the segregated cortico-basal ganglia loops, thereby allowing more abstract levels to tune the reinforcement signal used at more detailed levels [1]. The pharmacological effect of addictive drugs on increasing the extracellular concentration of dopamine within the striatum is incorporated into this model by adding a positive term D to the prediction error signal. Simulation results (Figure 1) show that drug-induced dopamine-release puts a bias on the transfer of reinforcement signal from one level of abstraction to the next. The accumulation of these biases along the rostro-caudal axis progressively induces a significant discrepancy in the value of drug-seeking behaviors at the top and bottom extremes of the hierarchy, thereby, an inconsistency between cognitive plans and motor-level habits.

Beside this central phenomenon, our model also accounts for several behavioral and neurobiological aspects of addiction, such as the gradual insensitivity of drug-seeking to drug-associated punishments (compulsivity), the delayed development of cue-elicited dopamine efflux in addicts' dorsal striatum, and the occurrence of blocking effect for drug rewards. It also suggests key testable predictions and beyond that, sets the stage for a view of addiction as a pathology of hierarchical decision making processes.

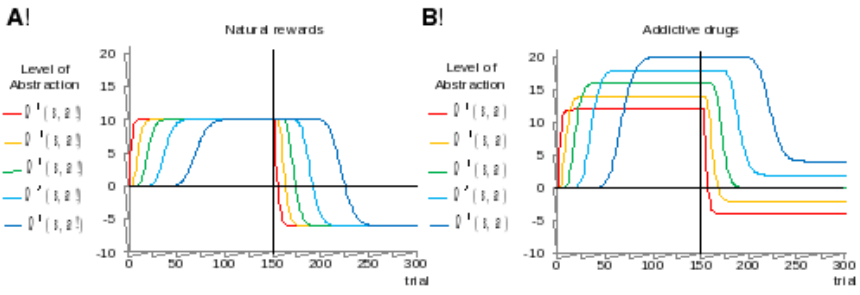

Figure 1. In the first 150 trials, the value of seeking natural rewards at all levels converge to r=10 (a). For the case of drug, however, the direct pharmacological effect of drug (D=2) results in the value of drug seekimg to become much higher than at the detailed levels, than at the abstract levels (b). If both of these actions be followed by punishment of magnitude -16 (the last 150 trials), then whereas cognitive loops assign a negative value to drug-seeking choice, motor-level loops find drug-seeking desirable (assign a positive value to it).

References

1. Haruno M, and Kawato M (2006). Heterarchical reinforcement-learning model for integration of multiple cortico-striatal loops: fMRI examination in stimulus-action-reward association learning. Neural Networks, 19: 1242-1254.

O20 The Open Source Brain Initiative: enabling collaborative modelling in computational neuroscience

Padraig Gleeson[1⋆], Eugenio Piasini[1], Sharon Crook[2], Robert Cannon[3], Volker Steuber[4], Dieter Jaeger[5], Sergio Solinas[6], Egidio d'Angelo[6], and Angus Silver[1]

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[2] *School of Mathematical and Statistical Sciences, School of Life Sciences, Arizona State University, Arizona, USA*
[3] *Textensor Limited, Edinburgh, UK*
[4] *School of Computer Science, Science and Technology Research Institute, University of Hertfordshire, UK*
[5] *Department of Biology, Emory University, Atlanta, USA*
[6] *Department of Neuroscience, University of Pavia, Pavia, Italy*

While an increasing number of biophysically detailed neuronal models (featuring (semi-) realistic morphologies and voltage and ligand gated conductances) are being shared across the community through resources like ModelDB, these usually only represent a snapshot of the model at the time of publication, in a format specific to the original simulator used. Models are constantly evolving however, to take account of new experimental findings and to address new research questions, both by the original modellers, and by other researchers who help provide quality control/debugging of original scripts and convert the model (components) for use in other simulators. This crucial part of the model life cycle is not well addressed with currently available infrastructure.

The Open Source Brain (OSB) repository is being developed to provide a central location for researchers to collaboratively develop models which can be run across multiple simulators and can interact with the range of other applications in the NeuroML "ecosystem". NeuroML [1] is a simulator independent language for expressing detailed single cell and network models, which is supported by an increasing number of applications for generating, visualising, simulating and analysing such models as well as by databases providing the base components (e.g. reconstructed morphologies, ion channels) for use in the models (http://www.neuroml.org/tool_support). The OSB repository differs from existing model databases which have traditionally concentrated on frozen, published models. The cell, ion channel, synapse and network models in this repository develop over time to ensure they reflect best practices in neurophysiological modelling and allow continuously improving, bug-free simulations. Multiple views of the model elements are available to encourage feedback from modellers, theoreticians and experimentalists. Links can be made to previous versions of the models in ModelDB, and deep links will be used to ensure cross referencing to other neuroinformatics resources such as NeuroMorpho and NeuroLex.

The system is based around a Mercurial version control repository with models organised into projects illustrating a number of neurophysiologically relevant aspects of the cell and network behaviour. The history is recorded of all changes to each project by contributors who can be distributed worldwide. There is close integration with the application neuroConstruct [2], allowing the models to be examined with a 3D graphical user interface, and scripts automatically generated for use on a number of widely used neuronal

simulators. A number of models are already available in the repository, including cell and network models from the cerebellum, detailed cortical and hippocampal pyramidal cell models and a 3D version of a single column thalamocortical network model [3]. While most of the models available are conversions of existing published models, some have been developed during original research projects using the tools and formats discussed here [4]. The repository is currently in alpha stage of development and is being tested with a small number of labs. The resource can be accessed at http://opensourcebrain.org:8080. This work has been primarily funded by the Wellcome Trust (086699/095667).

References

1. Gleeson P, Crook S, Cannon RC, Hines ML, Billings GO, Farinella M, Morse TM, Davison AP, Ray S, Bhalla US, Barnes SR, Dimitrova YD, Silver RA (2010). NeuroML: A language for describing data driven models of neurons and networks with a high degree of biological detail. PLoS Comp. Biol., 6(6):e1000, 815.
2. Gleeson P, Steuber V, and Silver RA (2007). neuroConstruct: a tool for modeling networks of neurons in 3D space. Neuron, 54(2):219-235.
3. Traub RD, Contreras D, Cunningham MO, Murray H, LeBeau FE, Roopun A, Bibbig A, Wilent WB, Higley MJ, and Whittington MA (2005). Single column thalamocortical network model exhibiting gamma oscillations, sleep spindles, and epileptogenic bursts. Journal of Neurophysiology, 93(4):2194–232.
4. Vervaeke K, Lorincz A, Gleeson P, Farinella M, Nusser Z, Silver A (2010). Rapid desynchronization of an electrically coupled interneuron network with sparse excitatory synaptic input. Neuron, 67(3):435–51.

M1 NSF: Theory and Applications

Ken Whang*

Ken Whang, Program Director, Division of Information and Intelligent Systems, National Science Foundation

Place: Evans, Basement AB
Day: Mon, 12:30:13:30

This session will be part tutorial, but mostly informal discussion,aimed at answering your questions about NSF and giving you a better model of how NSF works.

Ken will be pleased to take questions in advance about any aspects of NSF or your experiences with NSF: anonymous questions may be posted here: http://www.surveymonkey.com/s/Z6BLC8G.

Workshops

W1 Behavior Informatics: data bases, data mining and experiments in virtual worlds

Bullock, Room 209 W A+B, Day(s): Thu, 9:00:18:00

Ansgar Koene, RIKEN BSI, Tokyo, Japan

The goal of this workshop is to explore the desirability and possible implementation of a Behaviour-Informatics platform for the accumulation and sharing of behavioural data, and related analysis tools. Various Neuroinformatics platforms have in recent years been established to facilitate data sharing and integration for digital atlases of brain structure and anatomy, for fMRI and electrophysiology data, for modeling of spiking neural networks and many more. These Neuroinformatics efforts promise to provide a more coherent picture of the complete brain architecture. Similar efforts in behavioural studies would facilitate a more complete understanding of the relation between behavioural traits at the micro and macro levels and their dependence on environmental conditions. In addition to the pooling and standardization of data from behavioural experiments, another pillar of behaviour informatics could be a concerted effort to use virtual environments, like massively multi player games, to gather information on human behaviour in complex dynamic (social) environments with relatively mininmal effort.
If computational NeuroEthology aims to study "the interaction between environment, body and nervous system that results in behavior" then clearly an informatics platform for sharing of behavioural data is just as important as platforms for anatomical and functional neural data.

Introduction: Ansgar Koene
Brief pre-view of the topics that will be discussed by the speaker indicating how each talk connects to the bigger picture of Behaviour-Informatics.

Neuro-Informatics: Pontus Holm (INCF or member of Neuroinformatics Japan Node at RIKEN)
Overview of what has been achieved in neuro-informatics so far, what it promises to achieve for neuro-science and how it is practically implemented.

Behavioural data mining: MIT Human Dynamics Lab (Prof. A. Pentland or one of his postdocs)
Examples of the use of behavioural data bases and data mining techniques for understanding human behaviour.

Real data from Virtual Worlds: W. Bainbridge (NSF Division of Information and Intelligent Systems or a coauthor of the NSF NetLab Workshop Report or Dmitri Williams of USC Annenberg)
Virtual Worlds as tool for studying interaction between humans.

Data mining from online games: M. Szell or S. Thurner Quantification of human group behavior in a massively multiplayer online game

Agent based modeling: Rosaria Conte (or one of her post-docs at CNR Laboratory for Agent- Based Social Simulation, Italy) Behaviour databases as basis for agent based modeling to link neuroethological

understanding of behavior at the individual level to the ramification of this behaviour when it occurs in groups of individuals.

Discussion: Ansgar Koene (moderator), all speakers & audience
Draft outline of a Behaviour Informatics platform

Speakers:
- Ansgar Koene (RIKEN BSI)
- Pontus Holm (INCF Program Officer; alternate speaker: Prof. Shiro Usui, Neuroinformatics Japan Node at RIKEN BSI or one of his post-docs)
- Prof. A. Pentland (MIT Human Dynamics Lab or one of his post-docs)
- W. Bainbridge (NSF Division of Information and Intelligent Systems; alternate speaker: Dmitri Williams, USC Annenberg)
- M. Szell or S. Thurner (Complex Systems Research Group, Medical University of Vienna)
- Rosaria Conte (or one of her post-docs at CNR Laboratory for Agent-Based Social Simulation, Italy)

W2 Computational Neuroethological Approaches to Problems in Social Neuroscience

Bullock, Room 210 E, Day(s): Wed, 9:00:18:00

Robert Liu, Emory University, Atlanta, GA, USA
Elizabeth Buffalo, Emory University, Atlanta, GA, USA

Workshop Sponsors: The **Center for Translational Social Neuroscience**, The **Computational & Life Sciences** and The **Department of Biology** at **Emory University**.

EMORY UNIVERSITY
DEPARTMENT OF BIOLOGY

EMORY UNIVERSITY | Center for Translational Social Neuroscience

The burgeoning field of Social Neuroscience investigates the neurobiological mechanisms underlying the interactions that occur between individuals. Interest in this area has grown as social deficits have become recognized as a key component of several mental health disorders. A cornerstone of this field is the idea

that understanding the neural activity, circuits and neurochemicals involved in processing social information and social rewards requires using stimuli and contexts that are more ethologically relevant than those traditionally applied in laboratory studies. However, natural stimuli and contexts are complex, so that designing controlled experiments and interpreting the data can be difficult. This is where computational neuroethology can make tangible contributions. This workshop highlights examples across species where using computational and/or quantitative methods in addressing problems in social neuroscience has helped advance our understanding of the neurobiological and evolutionary mechanisms underlying social interactions.

Confirmed speakers include:
Bruce Carlson (Washington University)
Eric Fortune (Johns Hopkins)
Asif Ghazanfar (Princeton)
Hans Hofmann (University of Texas at Austin)
Warren Jones (Marcus Autism Center)
Michael Platt (Duke)
Larry Young (Emory).

W3 Examining the dynamic nature of neural representations with the olfactory system

Bullock, Room 210 E, Day(s): Thu, 9:00:18:00

Christopher Buckley, RIKEN BSI, Tokyo, Japan
Taro Toyizumi, RIKEN BSI, Tokyo, Japan
Thomas Nowotny, University of Sussex, Sussex, UK

In the last few years rapid progress has been made in our understanding of olfactory information processing. The theoretical aspects of these advances not only have the potential to elucidate the mechanisms of olfaction but also to shed some light on neural information processing more broadly. Consequently, it seems timely to organise a focussed workshop to discuss these ideas. In particular, we will examine the dynamic phenomena at the core of olfactory information processing. For example, recent work (e.g., [1,2,3,4]) has begun to shed light on the nature of population rate codes. We will discuss the controversy over whether odours are represented as discrete dynamical states [1] or as smooth, but nonlinear, superposition of the responses to individual chemical components [2]. Other work has begun to elucidate the roles and mechanisms of stimulus evoked oscillations (e.g., [5,6]). In particular, we will discuss the possibility that stimulus specific transient synchrony between inhibitory neurons is important for coding odour identity and/or intensity. Lastly, work in Drosophila has demonstrated that inter-ŋ-glomerular interactions serve to normalise the responses of component specific glomeruli in a divisive manner [7]. We will discuss the prospects of using these insights from the olfactory system to examine the dynamic action of divisive normalisation in the central nervous system in general.

The workshop is aimed at the many researchers working on the olfactory system but also is likely to attract a wider audience of theoreticians interested in core computational neuroscience topics such as

stimulus evoked oscillations and non-ŋ-linear rate dynamics. The workshop should also be of particular interest to all who are working with nonlinear dynamical systems theory.

Tentative list of speakers: M. Bazhenov, T. A. Cleland, R. Galan, D. Martinez, T Nowotny, M. Stopfer, U. Bhalla, G. Galizia, A. Khan, C. Linster, J. Niessing, A. Khan, G. Turner

References:

1. Niessing et. al., Olfactory pattern classification by discrete neuronal network states. Nature, 2010.

2. Khan et. al., Odor Representations in the Rat Olfactory Bulb Change Smoothly with Morphing Stimuli. Neuron, 2011

3. Roberto F. Galán et. al., Sensory memory for odors is encoded in spontaneous correlated activity between olfactory glomeruli. Neural Computation, 2006.

4. C. L. Buckley and T. Nowotny. Multi-ŋ-scale Model of an Inhibitory Network Shows Optimal Properties near Bifurcation, Physical Review Letters, 2011.

5. Ito, I, et. al., Frequency transitions in odor-ŋ-evoked neural oscillations. Neuron, 2009.

6. Assisi C et al., Using the structure of inhibitory networks to unravel mechanisms of spatiotemporal patterning. Neuron, 2011.

7. Olsen et. al., Divisive Normalization in Olfactory Population Codes, Neuron, 2010

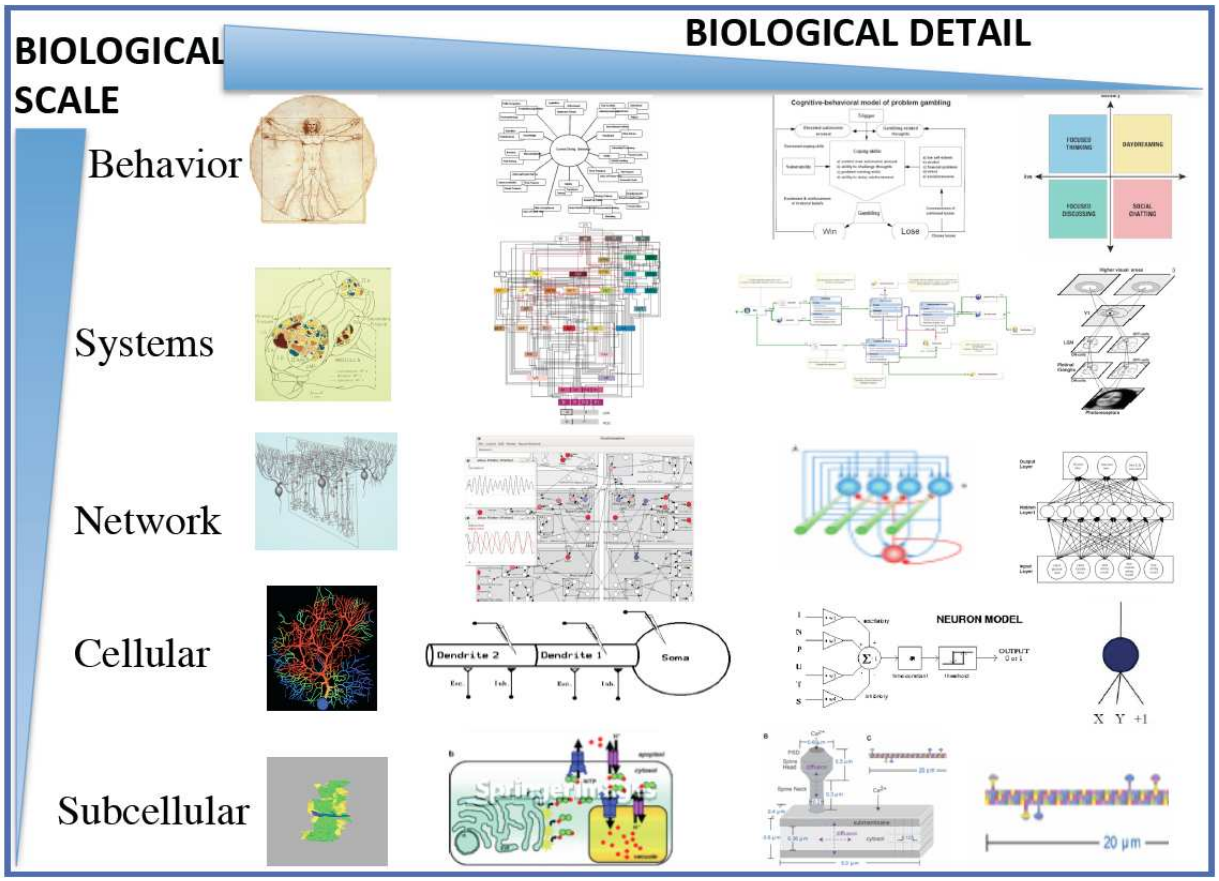
W4 Multi-Scale Modeling in Computational Neuroscience II: Challenges and Opportunities

Evans, Room Basement AB, Day(s): Wed, 9:00:18:00

James Bower, UTSA, San Antonio, Texas, USA
Ilya Rybak, Drexel University, Philadelphia, PA, USA

Following last year's highly successful CNS 2011 workshop, we will once again consider and discuss challenges and issues in multi-scale modeling as they apply to understanding nervous systems. Specifically, last year's workshop produced an outline for a taxonomy for multi-scale modeling (see figure), which was proposed as a mechanism for better understanding the structure of multi-scale modeling efforts. This coming workshop will focus on the further exploration of this taxonomy, with several short initial presentations on its development and applications over the last year. The workshop is being organized by the co-chairs of the Computational Neuroscience Working Group of IMAG, a multi-federal agency consortium based at the National Institutes of Health, tasked with exploring and developing multi-scale modeling in biology. including the U.S. National Institutes of Health, the U.S. National Science Foundation. ().

The results of this discussion will be added to the IMAG wiki and will be presented to the Multi-scale Modeling Consortium at NIH. This workshop therefore represents an opportunity for the CNS community to influence the direction of future funding for modeling in general and multi-scale modeling efforts in particular.



W5 Methods of Systems Identification for Studying Information Processing in Sensory Systems

Bullock, Room 209 W A+B, Day(s): Wed, 9:00:18:00

Aurel Lazar, Columbia University, New York, NY, USA
Mikko Juusola, University of Sheffield, Sheffield, UK

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

W6 Methods of Information Theory in Computational Neuroscience

Presser, Room Gaines Auditorium, Day(s): Wed & Thu, 9:00:18:00

Todd Coleman, UCSD, San Diego, CA, USA
Michael Gastpar, EPFL, Lausane, Switzerland
Conor Houghton, Trinity College, Dublin, Ireland
Aurel Lazar, Columbia University, New York, NY, USA
Simon Schultz, ICL, London, UK
Tatyana Sharpee, Salk Institute, San Diego, USA

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

W7 Modern evolutionary algorithms in computational neuroscience : tools to parameterize, explore model properties & design model structures

Evans, Room Basement C, Day(s): Wed, 9:00:18:00

Benoît Girard, UPMC/CNRS, Paris, France
Denis Sheynikhovich, UPMC/CNRS, Paris, France
Jean-Baptiste Mouret, UPMC/CNRS, Paris, France
Stephane Doncieux, UPMC/CNRS, Paris, France

In the last 10 years, evolutionary algorithms (EA) have been occasionally used as tools to help parameterizing computational models of the brain. As models grow more and more in complexity, manually adjusting parameters become unreasonable, while automatic approaches, like EA, can provide acceptable solutions. Modern EA can also help computational neuroscience beyond optimization: the use of multiple-ŋ-objectives EA (MOEA) allows to find multiple trade-ŋ-off solutions to the studied problem, revealing intrinsic properties of the problem itself, and thus actively participating in the investigation process. Modern generative EA can also be used to fully generate the structure of brain networks models based on known anatomical and electrophysiological data and thus directly participate in the model design.

The goal of this workshop is to gather neuroscientists around the current use of EA in computational neuroscience, to advertise the possibilities of this approach, as well as to discuss the emerging and future applications of EA in our field.

Confirmed speakers:
A. Korngreen (Bar-Ilan University, Israel) **Optimizing ion channel models using a parallel genetic algorithm on graphical processors**.
D. Jaeger (Emory University, USA) **Using particle swarm evolutionary algorithm to improve ion channel kinetics**.
J.-C. Quinton (Pascal Institute / Polytech Clermont-Ferrand, France) **Tuning and learning with evolutionary methods: anticipatory representations and dynamic neural fields**.
A. J. Nevado-Holgado (University of Oxford, UK). **Multiobjective evolutionary algorithms to fit realistic computational models of brain networks to extensive experimental data**.
J.-B. Mouret (Université Pierre & Marie Curie / CNRS, France) **Recent advances in evolutionary algorithms with examples from neuro-evolution**.
J. Liénard (Université Pierre & Marie Curie / CNRS, France) **Integration of detailed primate anatomical and electrophysiological data in a model of the basal ganglia using multi-objective evolutionary algorithms**.

W8 Principles of motor pattern generation: experiments and modeling

Evans, Room Basement C, Day(s): Thu, 9:00:18:00

Carmen Canavier, LSU HSC, New Orleans, LA, USA
Andrey Shilnikov, GSU, Atlanta, USA

A central pattern generator (CPG) is a neural microcircuit of cells that are capable of producing rhythmic patterns that underlie motor behaviors such as heartbeat, respiration, and locomotion in animals and humans. These patterns do not require sensory input or external patterned inputs, but rather are an emergent property of the network. Burst firing activity is usually associated with CPG activity. In some cases intrinsically or conditionally bursting pacemaker neurons are thought to be responsible for the rhythm, whereas in other cases the basic pattern generating unit is though to be a half center oscillator in which synaptic escape or release of each oscillator in turn drives the network rhythm. Here we focus both on the mechanisms for robustness in dedicated CPGs and for flexibility in multifunctional CPGs that can produce distinct rhythms, such as swimming versus crawling, and alternation of blood circulation patterns in leeches. Robustness to heterogeneity and noise of individual modes allows for phase constancy under a wide variety of conditions. Flexibility may be attributed to switching between rhythms by input-dependent switching between attractors of the CPG. We will focus on experimental approaches, often using the dynamic clamp, combined with various mathematical and computational techniques for understanding the underlying principles sub-serving pattern generation.

Speakers: Allen Selverston (UCSD)
Akira Sokurai (GSU) "A natural example of different circuit architectures for analogous behaviors in different species"
Thomas Nowotny (Sussex, UK) "Central patterns generation with heteroclinic orbits"
Maxim Bazhenov (UCR)
Erik Shwerwood (Utah)
Brian Mulloney (UC Davis)
Carmen Canavier (LSU)
Andrey Shilnikov (GSU) "Bifurcation of polythythmic patterns in 3-cell bursting motifs"

W9 Dynamics of rhythm generation

Bullock, Room G9, Day(s): Wed & Thu, 9:00:18:00

Roman Borisyuk, University of Plymouth, Plymouth, UK
Ronald Calabrese, Emory University, Atlanta, GA
Alan Roberts, University of Bristol, Bristol, UK
Gennady Cymbalyuk, Georgia State University, Atlanta, GA

The ability of distinct anatomical circuits to generate patterns of rhythmic activity is widespread among vertebrate and invertebrate species. These patterns correspond to different functions like motor behaviors and odor recognition and pathological events like seizure episodes. The dynamics of the circuits producing such patterns are based on the basic principles conserved across phyla. For example, the swimming behavior of the tadpole is sporadic and transient, lasting for a few tens of periods. In contrast the heart-beating of the leech must sustain through its life and the pattern of the activity stays constant even in the variable environment.

We would like to bring together experts applying experimental approaches and the methods developed in the neuroscience, neurophysics, neuro-informatics, neuroethology, and the bifurcation theory to determine the basic principles of the transient and steady dynamics of rhythm generation from different phyla.

Speakers:

Barreto, Ernest (George Mason University, School of Physics, Astronomy) - Ion concentration dynamics as a unifying mechanism for bursting of many different morphologies
Borisyuk, Roman (University of Plymouth, School of Computing, Communication, and Electronics) -
Calabrese, Ronald (Emory University, Biology Department) - Harnessing experimentally observed individual variation in neuronal network models.
Cohen, Netta (University of Leeds, School of Computing) -
Cymbalyuk, Gennady (Georgia State University, Neuroscience Institute) -
Dzakpasu, Rhonda (Georgetown University, Dept. of Physics) - The effects of synaptic potentiation modulation on in vitro neuronal network dynamics
Edwads, Donald (Georgia State University, Neuroscience Institute) - A neuromechanical model of locomotion in crayfish
Nadim, Farzan (New Jersey Institute of Technology, Dept. of Mathematical Sciences) -
O'Donovan, Michael (NIH/NINDS, Developmental Neurobiology Section) -
Prinz, Astrid (Emory University, Biology Department) -
Prilutsky, Boris (GaTech, School of Applied Physiology) -
Roberts, Alan (University of Bristol, School of Biological Sciences) - Starting, running and stopping the tadpole swimming network
Rybak, Ilya (Drexel University, Dept. of Neurobiology and Anatomy) -
Sakurai, Akira (Georgia State University, Neuroscience Institute) - Potential mechanisms underlying termination of a transient rhythmic motor behavior
Spardy, Lucy (University of Pittsburgh, Dept. of Mathematics) -
Wiggins, Tim (University of Minnesota, Dept. of Neuroscience, Masino's Lab) -
Zochowski, Michal (Univeristy of Michigan, Dept. of Physics) -

W10 Disease dynamics: Computational modeling of neurological diseases

Evans, Room Basement AB, Day(s): Thu, 9:00:18:00

Sharmila Venugopal, UCLA, Los Angeles, CA, USA
Ranu Jung, FIU, Miami, FL, USA

The goal of this workshop is to bring together a diverse group of neuroscientists to discuss and present recent advances in computational modeling of dynamical disease phenomena. Computational modeling holds great promise in improving our understanding of disease dynamics and in designing pharmacological, surgical and neuroprosthetic interventional strategies for neurological disorders. In this workshop, we will focus on how computational models can enhance our understanding of disease dynamics and predict emergence of pathological excitability states. Examples of pathological excitability states include hyper-excitability of sensorimotor reflex circuits leading to spasticity, hyper-synchrony of cortical networks leading to epileptiform activity and Parkinsonian rhythms. The models discussed will represent cellular, network and behavioral disease constructs encompassing multiple levels of nervous system organization. The workshop will promote cross-talk on pathogenic mechanisms in multiple neurological conditions while providing opportunities for an open dialog amongst experimental, computational and clinical neuroscientists to further stimulate the utilization of computational models in predicting disease dynamics.

Speakers:
Ranu Jung, Ph.D. (Florida International U) - Introduction to the Workshop
Steven J. Schiff, M.D, Ph.D. (Penn. State U)
Ivan Soltesz Lab (U of California Irvine)
Sharmila Venugopal, Ph.D. (U of California Los Angeles) - Modeling multiple channelopathies in Amyotrophic Lateral Sclerosis
Sheriff M Elbasiouny, Ph.D. (Northwestern University)
Jonathan Rubin, Ph.D. (U of Pittsburg)
Alla Borisyuk, Ph.D. (U of Utah)
Michael Hasselmo Lab (Boston U)

W11 Neuromechanical modeling of posture and locomotion

Bullock, Room 102 W, Day(s): Wed, 9:00:18:00

Boris I. Prilutsky, Georgia Institute of Technology, Atlanta, GA, USA
Alexander N. Klishko, Georgia Institute of Technology, Atlanta,GA, USA

Neuromechanics is a new, quickly growing field of neuroscience research that merges neurophysiology, biomechanics and motor control and aims at understanding living systems and their elements through interactions between their neural and mechanical dynamic properties. Although Neuromechanics is not limited by computational approaches, neuromechanical modeling is a powerful tool that allows for integration of massive knowledge gained in the past several decades in organization of motion related brain and spinal cord activity, various body sensors and reflex pathways, muscle mechanical and physiological properties and detailed quantitative morphology of musculoskeletal systems. Recent work in neuromechanical modeling has demonstrated advantages of such an integrative approach and led to discoveries of new emergent properties of neuromechanical systems. The goal of this workshop is to bring together neuromechanics researchers, discuss new developments in the field of neuromechanical modeling and inform Computational Neuroscience community of this new and exciting area of research.

Speakers:
Shinya Aoi, PhD, Department of Aeronautics and Astronautics, Kyoto University, Japan
Thomas J. Burkholder, PhD and Nathan E. Bunderson, PhD. School of Applied Physiology, Georgia Tech, USA
Donald H. Edwards, Jr, PhD, Neuroscience Institute, Georgia State University, USA
Gennady Cymbalyuk, PhD, Neuroscience Institute, Georgia State University, USA
Sergey N. Markin, PhD, Department of Neurobiology and Anatomy, Drexel University College of Medicine, USA
Jonathan Rubin, PhD, Department of Mathematics, University of Pittsburgh, USA
Lena Ting, PhD, Department of Biomedical Engineering, Georgia Tech/Emory, USA
Sergiy Yakovenko, PhD, Center for Neuroscience, West Virginia University, USA

W12 Postdoc and Student Career Strategy Workshop

Bullock, Room G9, Day(s): Wed, 18:00:20:00

Nathan W. Schultheiss, Boston University, Boston, MA, USA

The computational neuroscience (CNS) community is both international and interdisciplinary, and there are many possible roads to success in the field. However, the challenges faced by current or soon-to-be postdocs are also diverse, and excellent mentorship from primary investigators is an invaluable resource for the development of future leaders in research or industry. This workshop is intended to provide postdocs and students in CNS an opportunity to hear about several very successful career paths and/or strategies from current leaders in the CNS community. The workshop will consist of testimonial insights from junior faculty having recently transitioned from postdoc status, researchers working outside of their home countries, researchers working in departments other than their primary field of training, and senior faculty who have witnessed and steered search committees, reviewing boards, and indeed the field of computational neuroscience itself through both 'fat' and 'lean' funding periods and through its exciting continued development. Postdocs and students are encouraged to ask questions of the speakers and participate in discussion of topics of universal interest or specific concerns. (Our own concerns are often more universal that we realize until we voice them!) Given the considerable participation in recent years, this year after a general discussion, we may have separate panels to specifically address student topics and postdoc topics.

Recent faculty mentors have included:

Jim Bower	Tim Lewis	Tobias Niemann	Dieter Jaeger
Rob Butera	Tay Netoff	Frances Skinner	John White
Ron Calabrese	Astrid Prinz	Tomasz Smolinski	

Our continued thanks to you!!

Posters

Link to poster abstracts on BMC Neuroscience web site:
http://www.biomedcentral.com/bmcneurosci/supplements/13/S1

Note: Asterisk* indicates contact author.

Sunday Posters
Posters P1 – P63

P1 Spike-Timing Dependent Plasticity Facilitates Excitatory/Inhibitory Dysbalances in Early Phases of Tinnitus Manifestation

Christoph Metzner[1,2⋆], Fabian Guth[1], Achim Schweikard[1], and Bartosz Zurowski[3]

[1]*Institute for Robotics and Cognitive Systems, University of Luebeck, 23538 Luebeck, Germany*
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[3]*Department of Psychiatry, University Clinics Schleswig-Holstein, 23538 Luebeck, Germany*

P2 Information theoretic and machine learning approaches to quantify non-linear visual feature interaction underlying visual object recognition

Alireza Alemi-Neissi[1⋆], Carlo Baldassi[2,3], Alfredo Braunstein[2,3], Andrea Pagnani[2], Riccardo Zecchina[2,3], and Davide Zoccolan[1]

[1]*Area di Neuroscienze, Scuola Internazionale Superiore di Studi Avanzati (SISSA), Trieste, Italy*
[2]*Dipartimento di Scienza Applicata e Tecnologia, Politecnico di Torino, Torino, Italy*
[3]*Human Genetic Foundation (HuGeF), Torino, Italy*

P3 Model of a Sparse Encoding Neuron

Praveen Yenduri[1⋆], Anna Gilbert[2], and Jun Zhang[3]

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[2]*Department of Mathematics, University of Michigan, Ann Arbor, MI 48109, USA*
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P4 Modeling Cuttlefish Behavioural Chromatophore Response

Zach Zboch[1], James Peterson[2⋆]

[1] *Department of Biological Sciences, Clemson University, Clemson, SC 29634, USA*
[2] *Department of Biological Sciences, Clemson University, Clemson, SC 29634, USA*

P5 Temperature-fastened Sodium inactivation accounts for energy efficient cortical action potentials in mammalian brains

Yuguo Yu[1,2⋆]

[1] *Center for Computational Systems Biology, Fudan University, Shanghai, 200433, China*
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P6 Properties of cortical axons for energy efficient cortical action potentials

Yuguo Yu[1,2⋆]

[1] *Center for Computational Systems Biology*
[2] *School of Life Sciences, Fudan University, Shanghai, 200433, China*

P7 Statistics of natural scene structures and scene categorization

Xin Chen[1], Weibing Wan[1], and Zhiyong Yang[1,2,3⋆]

[1] *Brain and Behavior Discovery Institute,*
[2] *Department of Ophthalmology, and*
[3] *Vision Discovery Institute, Georgia Health Sciences University, Augusta, Georgia, 30912, USA*

P8 Statistics of eye movements in scene categorization and scene memorization

Xin Chen[1⋆], Weibing Wan[1], and Zhiyong Yang[1,2,3]

[1] *Brain & Behavior Discovery Institute,*
[2] *Department of Ophthalmology, and*
[3] *Vision Discovery Institute, Georgia Health Sciences University, Augusta, GA, 30912, USA*

P9 A visual code book–structured probability distributions in natural scenes

Weibing Wan[1], Zhiyong Yang[1,2,3⋆]

[1] *Brain and Behavior Discovery Institute*
[2] *Department of Ophthalmology*
[3] *Vision Discovery Institute, Georgia Health Sciences University, Augusta, Georgia, 30912, USA*

P10 Features of chaotic activity in a balanced network of Type II neuronal oscillators

Maximilian Puelma Touzel[1,2,3⋆], Michael Monteforte[1,2,3], and Fred Wolf[1,2,3]

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[2] *Faculty of Physics, Georg-August-University, Goettingen, Germany*
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P11 Synaptic activations of neuronal populations in the thalamocortical loop from LFP using kCSD and ICA

Szymon Leski⋆, Helena Glabska, Jan Potworowski, and Daniel K Wojcik

Department of Neurophysiology, Nencki Institute of Experimental Biology, Warsaw, 02-093, Poland

P12 Spike threshold dynamics reshape the phase response curve and increase the degree of synchronization among neurons coupled by excitatory synapses

Michael Farries⋆, Charles J. Wilson

Department of Biology, University of Texas San Antonio, San Antonio, TX 78240, USA

P13 A high-level, simulator independent, Python library for simulating small networks of multicompartmental neurons

Michael Hull⋆, David Willshaw

Institute for Adaptive and Neural Computation, University of Edinburgh, EH8 9AB, UK

P14 Signal analysis of whole-body shortening behavior in Hirudo verbana

Benjamin Migliori[1⋆], Chris Palmer[2], and William Kristan[2]

[1] *Department of Physics, University of California San Diego, La Jolla, CA 92092, USA*
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P15 Haloperidol effects on striatal dopamine and DOPAC levels and subcellular distribution

Lane Wallace⋆

Division of Pharmacology, College of Pharmacy, Ohio State University, Columbus, OH 43210, USA

P16 General anaesthetics induce tonic inhibition and modulate the gain of neural populations : a modeling study

Axel Hutt⋆, Thomas Voegtlin

INRIA CR Nancy - Grand Est, CS20101, 54603 Villers-ls-Nancy Cedex, France

P17 Optimal Information Encoding for Multiple, Simultaneously Presented Stimuli

Jan Pieczkowski[1,2⋆], Lawrence York[2], Jeanette Hellgren Kotaleski[1,3], and Mark van Rossum[2]

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P18 Action Recognition Using Natural Action Structures

Xiaoyuan Zhu[1,2⋆], Zhiyong Yang[1,3], and Joe Tsien[1,2]

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P19 Variability of inter-syllable gaps challenges the branched-chain model of sequence production in Bengalese finches.

Kristofer Bouchard[1,3], Anand Kulkarni[2⋆], Michael Brainard[1], and Todd Troyer[2]

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P20 Tracking sub-syllabic features in zebra finch song during development

Meagan Woodford⋆, Matthew Benavides, and Todd Troyer

Biology Department, Neurosciences Institute, UTSA, San Antonio, Texas, 78249

P21 A talkative Potts attractor neural network welcomes BLISS words

Sahar Pirmoradian⋆, Alessandro Treves

Cognitive Neuroscience Sector, SISSA, Trieste, 34136, Italy

P22 Brain ventricle volume correlates with effortful control in healthy young males

Rongxiang Tang[1,2], Yi-Yuan Tang[2,3⋆]

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P23 – Withdrawn –

P24 Dynamic Bayesian network modeling for intervention mechanism

Yan Sun[1,2], Yi-Yuan Tang[3,4⋆]

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P25 Coherent spontaneous resting EEG of frontal regions in human brain

Shaowei Xue[1], Yu-Qin Deng[1], and Yi-Yuan Tang[1,2⋆]

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P26 – Withdrawn –

P27 Optical imaging of motor cortical activation using functional near-infrared spectroscopy

Nicoladie D Tam[1⋆]

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

P28 Derivation of the evolution of empathic other-regarding social emotions as compared to non-social self-regarding emotions

Nicoladie D Tam⋆

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

P29 Computational Modeling of Light Activated Ion Channels

Roxana Stefanescu[1⋆], Shivakeshavan Ratnadurai[2], Paul R Carney[1,2], Pramod Khargonekar[3], and Sachin S Talathi[1,2]

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[2] *Department of Biomedical Engineering, and*
[3] *Department of Electrical Engineering, University of Florida, Gainesville, FL 32610*

P30 Genesis of interictal spikes in the CA1: A computational investigation

Shivakeshavan Ratnadurai[1⋆], Roxana Stefanescu[2], Pramod Khargonekar[3], Paul R Carney[1,2], and Sachin S Talathi[1,2]

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P31 Computational Modeling of Light Activated Ion Channels

Roxana Stefanescu[1⋆], Shivakeshavan Ratnadurai[2], Paul Carney[1,2], Pramod Khargonekar[3], and Sachin S Talathi[1,2]

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P32 STDP induced synchrony in inhibitory neural networks: Theory and Experiments

Zack B Kagan[1], Charles Frazier[2,5], and Sachin S Talathi[3,4,5⋆]

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P33 Charge balanced control of seizure like activity in a two dimensional cortical model

Prashanth Selvaraj[1⋆], Andrew Szeri[1,2]

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P34 Critical slowing in a Hodgkin-Huxley neuron near spiking threshold

Alex Bukoski[1⋆], D Alistair Steyn-Ross[2], and Moira L. Steyn-Ross[2]

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P35 Fractal-based linear model of resting state hemodynamic response in fMRI

Wonsang You[1⋆], Sophie Achard[2], and Jörg Stadler[1]

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P36 NeRvolver: A computational intelligence-based system for automated construction, tuning, and analysis of neuronal models

Emlyne Forren, Myles J Johnson-Gray, Parth Patel, and Tomasz G Smolinski*

Department of Computer and Information Sciences, Delaware State University, Dover, DE 19901, USA

P37 Analyzing conductance correlations involved in recovery of bursting after neuromodulator deprivation in lobster stomatogastric neuron models

Kenneth Shim[1], Astrid Prinz[2], and Tomasz G Smolinski[1*]

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P38 Modeling Na+- and Ca2+-dependent mechanisms of rhythmic bursting in excitatory neural networks

Ilya Rybak[1*], Patrick Jasinski[1], Yaroslav Molkov[1,2], Natalia Shevtsova[1], and Jeffrey Smith[3]

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[3]*Cellular and Systems Neurobiology Section, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD 20892, USA*

P39 Analysis of excitatory and inhibitory interactions at high temporal resolution in core circuits of the respiratory CPG

Yaroslav Molkov[1*], Anke Borgmann[2,3], Ruli Zhang[3], Ilya Rybak[4], and Jeffrey Smith[3]

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P40 A model for simulating Local Field Potential in the thalamus of Essential Tremor patient during deep brain stimulation.

Ishita Basu[1*], Daniela Tuninetti[1], Daniel Graupe[1,2], and Konstantin Slavin[3]

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P41 Using model databases to determine dendritic distributions of Ih channels in oriens-lacunosum/moleculare hippocampal interneurons

Vladislav Sekulic[1,2⋆], Josh Lawrence[3,4], and Frances Skinner[1,2,5]

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P42 A declarative model specification system allowing NeuroML to be extended with user-defined component types

Robert Cannon[1⋆], Padraig Gleeson[2], Sharon Crook[3], and Angus Silver[2]

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P43 Motif statistics and spike correlations in neuronal networks

Yu Hu[1⋆], James Trousdale[2], Kresimir Josic[2,3], and Eric Shea-Brown[1,4]

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P44 Speed and Accuracy in Decision Making: Input correlations and performance

Nicholas Cain⋆, Eric Shea-Brown

Applied Mathematics, University of Washington, Seattle, WA 98388, USA

P45 A simple mechanism for higher order correlations in integrate and fire neurons

David Leen[1⋆], Eric Shea-Brown[1,2]

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P46 Dendrites equip neurons with a range of resonant frequencies

Jonathan Laudanski[1], Benjamin Torben-Nielsen[2,3⋆], Idan Segev[3], and Shihab Shamma[4]

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P47 Modeling the effects of molecular crowding on cerebellar long term depression

Horace Troy Deans, Fidel Santamaria⋆

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P48 A neuromechanical computational model of spinal control of locomotion

Sergey Markin[1⋆], Alexander Klishko[2], Natalia Shevtsova[1], Michel Lemay[1], Boris Prilutsky[2], and Ilya Rybak[1]

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P49 Modeling [Ca2+]o- and [K+]o-dependent oscillations in spinal Hb9 interneurons

Natalia Shevtsova[1⋆], Sabrina Tazerart[2,3], Laurent Vinay[2], Frédéric Brocard[2], and Ilya Rybak[1]

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P50 Modeling Dose-dependent Temperature Responses to Methamphetamine

Yaroslav Molkov[1⋆], Dmitry Zaretsky[2], Maria Zaretskaia[2], and Dan Rusyniak[2]

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P51 A closed model for the respiratory system in mammals

Choongseok Park[1⋆], Yaroslav Molkov[2], Alona Ben-Tal[3], Natalia Shevtsova[4], Jeffrey Smith[5], Ilya Rybak[4], and Jonathan Rubin[1]

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P52 Can a central pattern generator produce multiple motor patterns? Modeling scratch rhythms in turtle

Abigail Snyder⋆, Jonathan Rubin

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P53 Synchronizing and desynchronizing effects of nonlinear delayed feedback deep brain stimulation in Parkinson's disease

Andrey Dovzhenok[1], Choongseok Park[1], Robert Worth[2], and Leonid Rubchinsky[1,3⋆]

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P54 Mechanisms of pathological synchrony in Parkinson's disease induced by changes in synaptic and cellular properties due to dopamine

Choongseok Park[1,2⋆], Leonid Rubchinsky[1,3]

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P55 Biologically realistic excitatory and inhibitory cell properties emerge from a sparse coding network

Mengchen Zhu[1⋆], Christopher Rozell[2]

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P56 Role of morphological changes in newly born granule cells of hippocampus after status epilepticus induced by pilocarpine in hyperexcitability

Julian Tejada[1,2], Norberto Garcia-Cairasco[2], and Antonio Roque[1⋆]

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P57 Automated model optimization to study spike shape modulation in Layer 2/3 cortical pyramidal neurons

Michael Vella⋆, Hugh P.c Robinson

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P58 The role of electrical coupling in the decision to initiate swimming in young frog tadpoles

Michael Hull[1⋆], David Willshaw[1], and Alan Roberts[2]

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P59 A model for dynamical switching in tristable perception for visual plaids

Gemma Huguet[1⋆], Jean Michel Hupé[2], and John Rinzel[1,3]

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P60 Divisive feedback can underlie phasic firing but is precise coincidence detection adequately robust?

Xiangying Meng[1,3], Gemma Huguet[2⋆], and John Rinzel[2,3]

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P61 Local field potentials in the auditory brain stem described by idealized biophysically-based models of the medial superior olive

Joshua Goldwyn[1,2⋆], John Rinzel[1,2]

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P62 Specifying production times in the ACT-R cognitive modeling system using evoked response potential latency

Daniel Cassenti*, Anthony Ries

Human Research and Engineering Directorate, U.S. Army Research Laboratory, APG, MD 21005, USA

P63 Sunk costs account for rats' decisions on an intertemporal foraging task

Andrew Wikenheiser[1*], A. David Redish[2]

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Monday Posters
Posters P64 – P126

P64 Phase-locking, quasiperiodicity and chaos in periodically driven noisy neuronal models: a spectral approach

Alla Borisyuk*, Firas Rassoul-Agha

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P65 Cellular and Nuclear Morphology...and Calcium Signaling: Revealing the Interplay between Structure and Function

Markus Breit[1], Peter Bengtson[2], Anna Hagenston[2], Hilmar Bading[2], and Gillian Queisser[1*]

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P66 A compartmental model of an identified Drosophila larval motoneuron for investigating functional effects of ion channel parameters

Cengiz Gunay[1*], Logesh Dharmar[1], Fred Sieling[1,2], Richard Baines[3], and Astrid A Prinz[1]

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P67 Reducing the maximal calcium conductance in models of the pyloric network after decentralization prevents recovery

Claire Tang[1⋆], Amber Hudson[2], and Astrid A Prinz[1]

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P68 Effect of intrinsic membrane conductances on Phase Resetting Curves in a conductance-based neuron model

Wafa Soofi[1⋆], Astrid A Prinz[2]

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P69 Investigating synaptic plasticity in the crab Cancer borealis pyloric circuit and in a computational pyloric model network database

Santiago Archila⋆, Astrid A Prinz

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P70 Paw-shake response and locomotion: Can one CPG generate two different rhythmic behaviors?

Alexander Klishko[1⋆], David Cofer[2], Gennady Cymbalyuk[2], Donald Edwards[2], and Boris Prilutsky[1]

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P71 Amplitude modulated photostimulation for probing neuronal network dynamics

Jonathan Newman[1⋆], Tatjana Tchumatchenko[2], Ming-Fai Fong[1,3], and Steve M. Potter[1]

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P72 Influence of inhibition on encoding vocalizations in the mouse auditory midbrain

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P73 – Withdrawn –

P74 Classification and visualization of neural patterns using subspace analysis statistical methods

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P75 Evaluation of target search efficiency for neurons during developmental growth

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P76 Effects of synaptic connectivity inhomogeneities for propagation of activity in neural tissue

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P77 Resistance between channels may lead to increased action potential efficiency

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P78 Bifurcation structure of phase locked modes in Type I excitable cells based on phase and spike time resetting curves

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P79 The influence of network structure on neuronal network dynamics

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P80 Ih modulates theta rhythm and synchrony in computer model of CA3

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P81 Modeling and prediction of conduction delay in an unmyelinated axon

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P82 Neural mechanism of binding amplitude information of echo sound with its frequency one in echolocating bat

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P83 Decoding in the dark: extracting information from spontaneous activity in primary visual cortex

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P84 Fano factor constancy and scale-invariant sampling in recurrent networks with probabilistic synapses

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P85 Temperature dependent transitions in excitability predicted by an electrodiffusion model of membrane potential

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P86 – Withdrawn –

P87 GPU Facilitated Unsupervised Visual Feature Acquisition

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P88 – Withdrawn –

P89 Classification of vocalizations by recordings from the auditory midbrain

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P90 Reinforcement learning of 2-joint virtual arm reaching in motor cortex simulation

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P91 CPP alters theta/gamma oscillations in rat hippocampus: simulation and experiment

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P92 Determining information flow through a network of simulated neurons

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P93 Modeling predicts that parameters shaping action potentials and synaptic responses differ in pyramidal neurons of the visual and prefrontal cortices

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P94 What can be learned from high-resolution sleep data using ECoG

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P95 Interpretation of seizure evolution pathways via a mean-field cortical model

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P96 Optimal Neural coding in networks of heterogeneous neurons

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P97 Spike Train Distance Metric Reveals Plasticity in Discrimination of Salient Calls by Putative Excitatory Cells of the Auditory Cortex

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P98 Heterogeneous short-term plasticity enables spectral separation of information in the neural spike train

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P99 An Open Architecture for the Massively Parallel Emulation of the Drosophila Brain on Multiple GPUs

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P100 Conductance interaction identification by means of Boltzmann distribution and mutual information analysis in conductance-based neuron models

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P101 A minimal model for a slow pacemaking neuron

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P102 Impulse dynamics of coupled synchronous neurons

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P103 Encoding of touch location and intensity by neurons of the medicinal leech Hirudo medicinalis

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P104 Automated quantification of optokinetic responses based on head-movement

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P105 Behaviour Informatics: data integration for greater understanding of human and animal behaviour

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P106 Detecting Vibration Source for the Orientation Behavior of Sand Scorpions

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P107 Low-frequency EEG correlates of fMRI in the resting state

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P108 Data driven analysis of low frequency spatio-temporal dynamics in resting state MRI (rsMRI) data

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P109 Testing entropy-based search strategies for a visual classification task

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P110 Lethality of Complex Neuronal Network in Caenorhabditis elegans Nervous System based on Cell Attacks

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P111 Individual Gain/Loss Attitude, Conditional Cooperation, and Random Shifting in a Public Goods Game

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P112 Background firing rate affects the signal transfer of behavior locked input patterns from Purkinje cells to the cerebellar nuclei

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P113 Modeling the Chemical Dynamics of Chloride Ion Indicators

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P114 Individual differences in temperaments traits and complex network properties of fMRI

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P115 Modeling frequency-dependent action potential failures in CA3 pyramidal cell axons

Ximing Li, Bill Holmes⋆

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P116 Improved conditions for the generation of beta oscillations in the subthalamic nucleus-globus pallidus network

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P117 Dynamics of large-scale neuronal networks of the human cortex functional connectivity

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P118 Adaptive Control of 2-wheeled balancing robot by two hemispheric cerebellar neuronal network model

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P119 Reaction-Diffusion Modeling in the NEURON Simulator

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P120 Same ion channel populations and different excitabilities: Beyond the conductance-based model

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P121 From spinal cord to hippocampus: links between bifurcation structure, ion channel expression, and firing patterns in a variety of neuron types

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P122 Neuronal avalanches and the cortico-striatal network

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P123 Gamma band LFP in mouse barrel cortex is coupled to respiratory rhythm

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P124 Bistable persistent spiking of layer II and layer V medial entorhinal cortical neurons during theta frequency oscillations in vitro

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P125 – Withdrawn –

P126 Cell Assembly Detection with Frequent Item Set Mining

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Tuesday Posters
Posters P127 – P189

P127 Comparing the spatio-temporal organization of joint spiking and local field potential oscillations in motor cortex

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P128 Turing instabilities in a mean field model of electrocortical activity

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P129 Convergence analysis of efficient online learning in Bayesian spiking neurons

Andre van Schaik[1⋆], Levin Kuhlmann[2], Michael Hauser-Raspe[1], Jonathan Manton[2], Jonathan Tapson[1], and David Grayden[2]

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P130 STDP encodes oscillation frequency in the connections of recurrent networks of spiking neurons

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P131 Eye contact, a fundamental building block of social behavior, engages single unit activity in the monkey amygdala

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P132 Variations in spike times

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P133 A biophysically realistic computer model of Alzheimer pathology to guide the development of symptomatic drugs

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P134 Structure of the Afferent Terminals in the Terminal Ganglion of a Cricket and Persistent Homology

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P135 An agent-based approach to multi-scale neuronal network simulations using modified McCulloch-Pitts neurons

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P136 What Does Weber's Law tell us about Spike Statistics?

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P137 Tracking a trajectory of a moving stimulus by spike timing dependent plasticity

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P138 Ionic mechanisms of action potential propagation velocity changes in peripheral C-fibers. Implications for pain

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P139 Pattern variability in a computational model of respiratory rhythm generation

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P140 – Withdrawn –

P141 Coding, stability, and non-spatial inputs in a modular grid-to-place cell model

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P142 Cav3-KCa3.1 complex enhances detection of facilitating parallel fiber inputs in cerebellar Purkinje cells

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P143 Modeling realistic extracellular spiking activity in populations of neurons for the purpose of evaluating automatic spike-sorting algorithms

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P144 Simplified model of the frequency dependence of the LFP's spatial reach

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P145 Axonal anisotropy and connectivity inhomogeneities in 2D networks

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P146 Adaptation shapes spike train correlations: theory and applications to tinnitus

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P147 Taming the model zoo: A unified view on correlations in recurrent networks

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P148 Inhibitory interneurons enable sparse code formation in a spiking circuit model of V1

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P149 Computational Neuroscience Ontology: a new tool to provide semantic meaning to your models

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P150 Exploring the functional implications of brain architecture and connectivity: a multi-simulator framework for biophysical neuronal models

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P151 Exploring the functional implications of brain architecture and connectvity: a declarative language framework

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P152 A computational study of stochastic mechanisms in dendritic calcium spike generation

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P153 A computational study on the spatial correlation of granule cell firing in the cerebellar cortex

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P154 Improving Data Caching of the STochastic Engine for Pathway Simulation (STEPS)

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P155 Benchmarking Drosophila receptor neurons for technical applications

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P156 Determinants of associative memory performance in spiking and non-spiking neural networks with different synaptic plasticity regimes

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P157 Bistability of seizure-like bursting and silence

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P158 Homeostatic regulation in a single neuron model from the Pre-Bötzinger Complex

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P159 A computational model of posterior parietal circuits during decision making and sequential planning

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P160 Dorsal parietal area 5 only encodes the immediate reach in sequential arm movement

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P161 Compromise revisited: inhibitory synapse and electrical coupling effects on bilateral phasing in the leech heartbeat system

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P162 Understanding plasticity of chemotaxis in C. elegans, a computational model of associative learning

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P163 Size matters: modeling the effects of body shape on locomotive behavior in the nematode C. elegans

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P164 A computational study of the influence of synaptic cooperativity on synaptic plasticity in a hippocampal CA1 pyramidal cell

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P165 Geometric analysis of soft thresholds in action potential initiation and the consequences for understanding phase response curves and model tuning

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P166 Spatial Processing in Binocular Rivalry

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P167 Synchronization of entorhinal cortex stellate cells

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P168 Dysfunction of cross-frequency phase-phase coupling in primary dysmenorrhea: a resting magnetoencephalographic study

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P169 Bifurcation structure of Adaptation versus Depolarization block

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P170 Theta Entrainment of Gamma Modules: Effects of Heterogeneity and Non-stationarity

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P171 Analysis of Input-Output Relationships of CPG Elements and Their Contributions to Rhythmic Output

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P172 Exploring the relationships between neuronal parameters and network activity

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P173 Methodology for extracting activity from functional calcium imaging data

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P174 Fixed point topology and robustness to perturbations between pairs of coupled neurons

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P175 Dynamics of neuromodulatory feedback determines frequency modulation in respiratory network

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P176 Multiscale modeling with GENESIS 3, using the G-shell and Python

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P177 Neuronal transmission of timing precision: dependence on intrinsic and synaptic properties

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P178 Combining Computational Neuroscience and Body Sensor Networks to Investigate Alzheimer's Disease

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P179 Synergetic role of inhibition and excitation in bursting synchronization

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P180 A temporal model of neural activity and VSD response in V1

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P181 Inverse stochastic resonance induced by ion channel noise

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P182 Cessation of seizure-like oscillations by periodic stimulation in a neuron model with dynamic ion concentrations

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P183 Video Compressive Sensing for Dynamic MRI

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P184 Group Sparse Coding with a Collection of Winner-Take-All Networks

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P185 On the trade-off between single-neuron complexity and network size with respect to spike-timed computations

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P186 Voltage oscillations and response dynamics in a model of sensory hair cells

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P187 Modeling study of a Central Pattern Generator in the Melibe seaslug

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P188 Phase-lag return mappings for a 3 cell multifunctional central pattern generator

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P189 Chaotic dynamics as a possible mechanism of rapid change of hippocampal local field activity between theta rhythm and large irregular activity

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