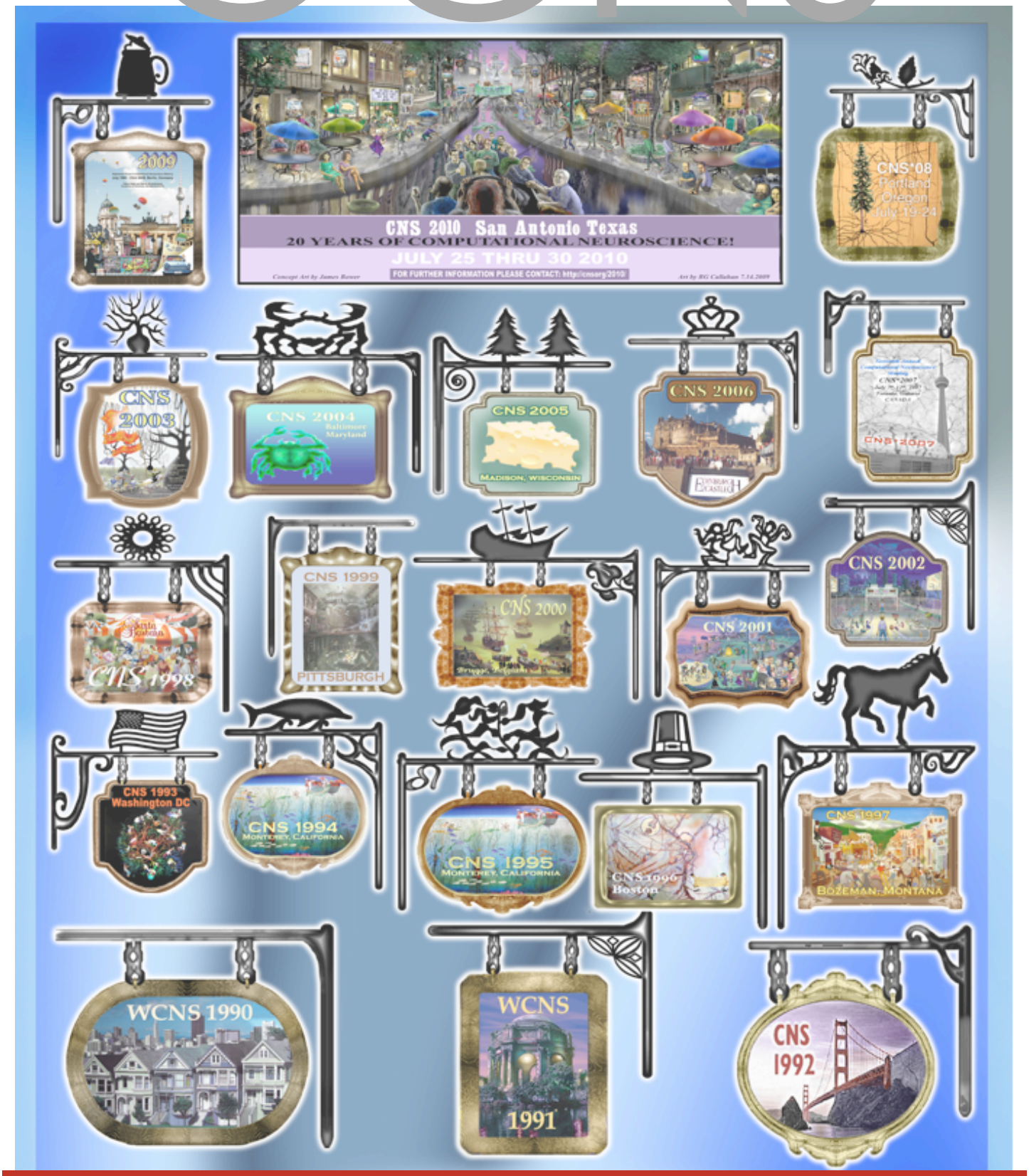
OCNS 2010

PROGRAM 2010



Sheraton Gunter Hotel, San Antonio TX
July 24-30, 2010

OCNS*2010 Local Organizers

Charles Wilson (U Texas San Antonio, USA)
James Bower (U Texas Health Sciences Ctr San Antonio, USA)
Todd Troyer (U Texas San Antonio, USA)

Cover art by Bonnie Callahan

OVERVIEW

PROGRAM

ABSTRACTS

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CNS 2010 is dedicated to the memory of Dr. Phillip Ulinski.

Mary and Phil Ulinski at CNS 2002 in Chicago, for which they served as the local organizers

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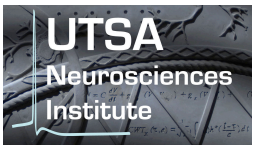


San Antonio Life Sciences Institute (UTSA/UTHSCSA SALSI)

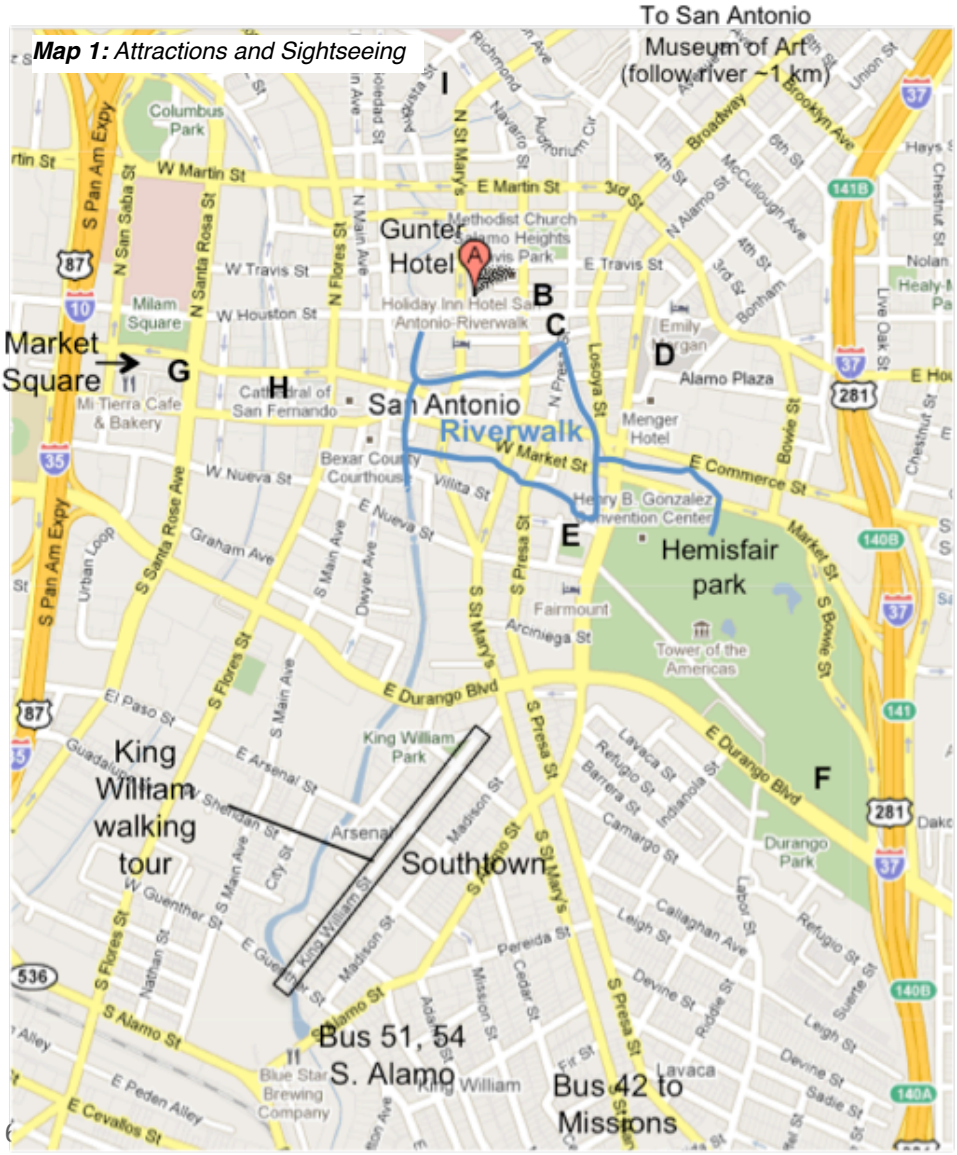
SAN ANTONIO INFORMATION

Mark Twain called San Antonio one of four 'unique' cities the U.S. Sometimes called the northernmost city in Mexico, it also has been influenced by Anglo settlers from the old South, by late 19th century German immigrants, by it's long standing status as a military outpost, as by its recent explosive growth. Finally, it is the most popular tourist destination in the State of Texas. You can find more extensive local information on the web: www.cbi.utsa.edu/cns. The main tourist information center is right across from the Alamo.

Top three things to see in San Antonio: The Alamo, the Riverwalk, and the San Antonio Missions.

Getting around: Downtown is small and walkable. You can also get around using the local trolley buses ($1.10 per ride - $. 15 transfer or $4 day pass from the tourist info center). The missions, museums, and several other attractions can also be reached by VIA city buses that pass through downtown (same fare as the trolleys - they're really one system). San Antonio is not a dense city, so many lines don't run frequently and stop early (around 10 p.m.). Check a schedule (www.viainfo.com).

Places to see downtown (letters refer to map, below): (**D**) the Alamo, (**H**) the Spanish Governor's palace (105 Plaza De Armas), Hemisfair park, (**E**) La Villita, King William district (get walking tour online).



Map 1: *Attractions and Sightseeing*

Museums (downtown):

(G) Museo Alameda, Tu-Sun, 12-6 p, $4/$2 students, Free on Tues; 101 S Santa Rosa – Latino Arts, affiliate of the Smithsonian.

(F) Institute of Texan Cultures, M-S 9a-5p, Su 12-5p, $8/$6 students; Far end of Hemisfair park.

(B) San Antonio Children's Museum, M-F 9a-5p, Sa 9a-6p, Su 12-5p, $7; 305 E Houston.

(C) Buckhorn Saloon and Texas Rangers Museum (banquet/party).

Museums (other):

- McNay Art Museum, Su 12-5p, Tu,W,F 10a-4p, Th 10a-9p, Sa 10a-5p; $8/$5 students, 6000 N New Braunfels – Modern Art. VIA bus 8.
- Witte science museum, M-S 10a-5p (Tu til 8p), Su 12-5p, $8; 3801 Broadway, VIA bus 9,10 or 14.
- San Antonio Museum of Art, Tu 10a-9p, W-Sa 10a-5p, Su 12-6p, $8/$5 students, Free Tu 4-9p; 200 W Jones Av. (North of downtown on the river.)
- Southwest School of Art and Craft, M-S 10a-5p, Su 11a-4p, Free; 300 Augusta, N. along the River.

Outdoor movie: Thursday July 29, Hemisfair park: Star Trek (2009)

Music and entertainment (see local free weekly paper for listings - (**www.sacurrent.com**).

Other things to do around downtown:

Take a walk north or south along the river. The riverwalk 'loop' can feel like an amusement park, but further away the river makes for a great evening walk. At dusk people gather to watch the Mexican free-tailed bats emerge from the IH-35 overpass at Camden street (there is a tour starting at 8 p.m. on July 27, starting at the turning basin near Pearl Brewery).

- **Hemisfair park** including the **Tower of the Americas** (which has a nice view but there's not a whole lot to see). Check out the fun 4D movie guide to Texas at the bottom.

‣**Instituto Cultural de Mexico,** Tu-F 10a-5p, Sa-Su 11a-5p, Free; Hemisfair park – often has interesting art exhibits.
‣**Ghost hunts of San Antonio tours**, 9 pm. Alamo plaza across from the (haunted) Menger Hotel. Even if you aren't into the whole ghost thing it's a decent walking tour of downtown.
‣**VFW post 76** (10th street and avenue B). An active veterans of foreign wars post in an old Victorian building. Like most San Antonio cultural institutions, it has a bar.

Dining, Drinking, and Dancing: There are A LOT of places to eat, drink and entertain yourself downtown, many of them along the Riverwalk. Here are just a few places that some of the locals say they like (letters refer to map location). The local web pages have links and some more info.

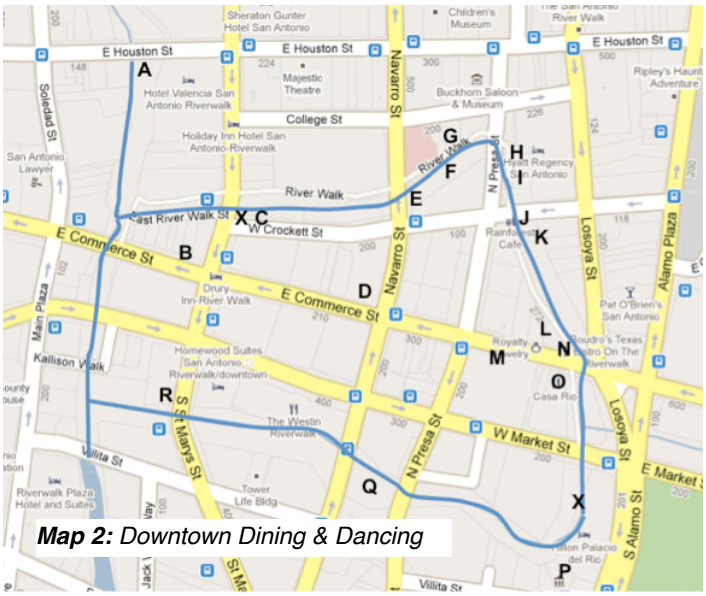
Food: *(see Map 2)*
Higher end: **N**. Boudros, **R**. Biga, **P**. Fig Tree, **F**. Paesano's

Mid Range: **A**. Acenar (TexMex), **K**. Zuni Grill (TexMex), **C**. Delores Del Rio (Italian)

Informal: **O**. Casa Rio (TexMex), **L**. Rio Rio(TexMex), **F**. County Line (BBQ), **M**. Schilo's (Deli/German), **J**. Rainforest Café (Mixed), **B**. Jerry's Chicago Style Hot Dogs, Mi Tierra (TexMex – located near market square, open 24 hrs), **D**. Justin's Ice Cream

Bars and Pubs: F. Howl at the Moon, **H**. Mad Dog's, **Q**. Durty Nellys, **E**. Dick's Last Resort, **G**. Swig (Martinis), **I**. The Landing (Jazz/swing)

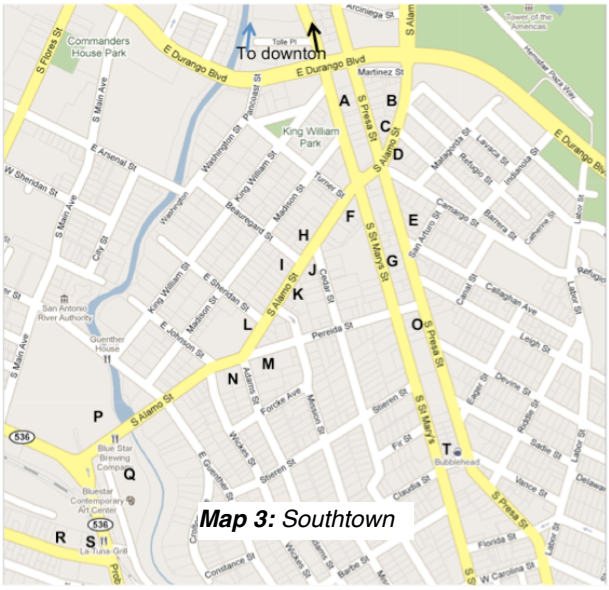
Dance Clubs: G. Polly Esther's, **D**. Tabu Lounge, Bonham Exchange, 411 Bonham St, behind the Alamo (GBLT); **X**. Loading for Riverboat Tours.



Map 2: *Downtown Dining & Dancing*

Southtown:

Just south of Downtown is a residential area known as Southtown. In the 70s and 80s this area was cultivated as an arts district and today it has a mix of galleries, restaurants, bars and music and arts venues. Roughly 1 mi (1.6 km) from downtown, it can be reached by a nice walk south along the river (or Blue Line Trolley or busses 51 and 54 along S. Alamo). The far anchor is the Blue Star arts complex, a converted warehouse that houses a brew pub, a martini bar, theater space, a bike shop, a coffee shop, and live/work spaces for artists. All letters beliow reference *Map 3*.



Map 3: *Southtown*

Food: K. Oloroso ($$), **C**. Azuca (Latin, $ $), **D**. La Frite (Belgian, $$), **L**. Liberty Bar ($), **S**. La Tuna Grill, **H**. Tito's (TexMex), **F**. Rosario's (TexMex), **A**. El Mirador (Family TexMex), **G**. Cascabel (Mexican), **O**. Frank's Hog stand (Burgers/Shakes – motorcycle theme), **E**. Mr. Tim's (southern, cheap!), **H**. Friendly spot ('Mexican tapas'), **P**. Guenter House (breakfast and lunch in a restored 1860 house).

Tea: I. Mad Hatter's (tea, sandwiches), **T**. Bubblehead Tea (& smoothies)

Bars, Pubs and Ice Houses: S. La Tuna (outdoor ice house), **Q**. Blue Star Brewing Co., **Q**. Joe's (martini bar), **R**. The Pedicab, **J**. B&D Ice, **N**. Casbeer's, **M**. Beethoven's Mannerchor (German singing society – they have a bar), **H**. Friendly Spot (outdoor bar, playground for kids), **C**. Bar America (cheap long necks/pool), **B**. Acapulco (ex drive in, vintage car crowd).

Dancing: C. Azuca (Latin/salsa), **R**. Pedicab

Other Useful Info:

‣**Taxis:** AAA Taxi (210-599-9999); San Antonio Taxis (210-444-2222); National Cab (210-434-4444); Yellow cab (210-222-2222);

‣**Markets/Deli:** Riverwalk Market and Deli, 126 E Houston; Delivery Market, 310 East Houston. (There really isn't a cheap market downtown.)

‣**Pharmacy/Drug store**: Walgreens, 300 E Houston; CVS, 211 Losoya.

‣**Other shopping:** River Center Mall, 849 E Commerce, around the corner from the Alamo.

‣**Travel gifts:** La Villita (group of shops in old part of town on S. Alamo across from Hemisfair park). Market Square, west between Houston and Commerce. For less touristy Mexican goods, take the bus to Fiesta on Main, 2025 N. Main.

‣**Bike rental:** Charles A. James, 329 North Main, 210-226-7812 $30/day; Blue star bike rentals, 1414 South Alamo, 210-858-0331, $25 for 24 hours $15 for 6 hours; Alamo bike rentals, 1016 N. Flores, 210-226-BIKE, $28/day – further away but they have a shuttle.

‣**Riverboat tours:** 35-40 minutes, $8.25. Tickets available on-line. Seating is first come/first served. Boarding side closest to hotel: Aztec theater river level near Commerce. River Taxi: $5 one-way, $10 all day.

‣**Regional and family attractions (see local info pages for links):** Six Flags Fiesta Texas, Sea World, Zoo, Botanical Gardens, Rodeo, Natural Bridge Caverns, Natural Bridge Wildlife Ranch, River tubing, Water parks, Texas Hill Country, Austin, Gulf Coast and Corpus Cristi. (Plenty of car rentals downtown).

MEETING OVERVIEW

SATURDAY JULY 24, 2010

09:00 - 16:30 Tutorials (3rd Floor Meeting Rooms)
13:00 - 23:00 Registration (Mezzanine)
17:00 - 19:30 Opening reception (Crystal Ballroom)

SUNDAY JULY 25, 2010

08:30 Registration (Mezzanine)
09:00 Welcome: Erik De Schutter (OCNS President) Charles Wilson, Jim Bower and Todd Troyer (Local Organizers)
09:20 **Keynote**: Miguel Nicolelis
10:20 Break
10:40 **Oral Session I**: O-01 to O-04
12:00 Lunch Break
13:30 **Featured Oral**: F-01
14:10 Break
14:30 **Oral Session II**: O-05 to O-07
15:30 Break
15:50 **Oral Session III**: O-08 to O-11
17:20-19:30 Dinner Break
19:30-22:30 **Poster Session I**, P1-P64 (Bluebonnet/Magnolia)

MONDAY JULY 26, 2010

08:30 Registration (Crystal Ballroom Mezzanine)
08:45 Meeting Announcements
09:00 **Invited Lecture**: Vivian Mushahwar
10:00 Break
10:20 **Symposium I AM**: M. Hasselmo, J. Miller
12:00 Lunch Break
13:10 **Symposium I PM**: C. Linster, S. Crook
14:50 Break
15:20 **Symposium I PM**: M. A. Blackwell, U. Bhalla
17:00-19:30 Dinner Break
19:30-22:30 **Poster Session II**, P65-P129 (Bluebonnet/Magnolia)

TUESDAY JULY 27, 2010

08:30 Registration (Crystal Ballroom Mezzanine)
08:45 Meeting Announcements
09:00 **Invited Lecture**: Jonathan Wolpaw
10:00 Break
10:20 **Symposium II AM**: J. Rinzel, R. Calabrese
12:00 Lunch Break
13:10 **Symposium II PM**: A. Destexhe, B. Olshausen
14:50 Break
15:20 **Symposium II PM**: R. Montague, Discussion
17:00-19:30 Dinner Break
19:30-22:30 **Poster Session III,** P130-P194 (Bluebonnet and Magnolia)

WEDNESDAY July 28, 2010

08:30 Registration (Crystal Ballroom Mezzanine)
08:45 Meeting Announcements
09:00 **Featured Oral**: F-02
09:40 Break
10:00 **Oral Session IV**: O-12 to O-14
11:00 Business Meeting
11:40 General Business Meeting
12:00 Lunch Break
13:10 **Featured Oral**: F-03
13:50 Break
14:10 **Oral Session V**: O-15 to O-17
15:10 Break
15:30 **Oral Session VI**: O-17 to O-21
18:00 - 22:00 Banquet, Buckhorn Saloon & Texas Rangers Museum.

THURSDAY/FRIDAY July 29/30 2010

9:00-5:00 Workshops (2nd &3rd Floor Meeting Rooms)

MEETING AT-A-GLANCE

	TUTORIALS	MEETING				WORKSHOPS	
	Sat 7/24	Sun 7/25	Mon 7/26	Tues 7/27	Wed 7/28	Thurs 7/29	Fri 7/30
08:00		REGISTRATION (08:00-17:00)					
08:10							
08:20							
08:30							
08:40							
08:50		MEETING ANNOUNCEMENTS					
09:00	TUTORIALS AM	WELCOME	INVITED LECTURE L-2 V. Mushahwar	INVITED LECTURE L-3 J. Wolpaw	FEATURED ORAL F-02	WORKSHOPS AM	WORKSHOPS AM
09:10							
09:20		KEYNOTE L-1 M. Nicolelis					
09:30							
09:40					BREAK		
09:50							
10:00			BREAK	BREAK	ORAL O-12		
10:10							
10:20		BREAK	SYMPOSIUM S-01 M. Hasselmo	SYMPOSIUM S-07 J. Rinzel	ORAL O-13	BREAK	BREAK
10:30							
10:40		ORAL O-01			ORAL O-14	WORKSHOPS AM	WORKSHOPS AM
10:50							
11:00		ORAL O-02			BUSINESS MEETING		
11:10			SYMPOSIUM S-02 J. Miller	SYMPOSIUM S-08 R. Calabrese			
11:20		ORAL O-03					
11:30							
11:40		ORAL O-04					
11:50							
12:00	LUNCH BREAK	LUNCH BREAK	LUNCH BREAK	LUNCH BREAK	LUNCH BREAK	LUNCH BREAK	LUNCH BREAK
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13:20			SYMPOSIUM S-03 C. Linster	SYMPOSIUM S-09 A. Destexhe	FEATURED ORAL F-03		
13:30	TUTORIALS PM	FEATURED ORAL F-01				WORKSHOPS PM	WORKSHOPS PM
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13:50					BREAK		
14:00							
14:10		BREAK	SYMPOSIUM S-04 S. Crook	SYMPOSIUM S-10 B. Olshausen	ORAL O-15		
14:20							
14:30		ORAL O-05			ORAL O-16		
14:40							
14:50		ORAL O-06	BREAK	BREAK	ORAL O-17		
15:00						BREAK	BREAK
15:10		ORAL O-07			BREAK		
15:20						WORKSHOPS PM	WORKSHOPS PM
15:30		BREAK	SYMPOSIUM S-05 A. Blackwell	SYMPOSIUM S-11 R. Montague	ORAL O-18		
15:40							
15:50		ORAL O-08			ORAL O-19		
16:00							
16:10		ORAL O-09			ORAL O-20		
16:20							
16:30	BREAK	ORAL O-10	SYMPOSIUM S-06 U. Bhalla	PANEL DISCUSSION	ORAL O-21	DINNER BREAK	
16:40							
16:50		ORAL O-11					
17:00	RECEPTION				BREAK		
17:10							
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18:00		DINNER BREAK	DINNER BREAK	DINNER BREAK	BANQUET		
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18:30						MENTORING WORKSHOP	
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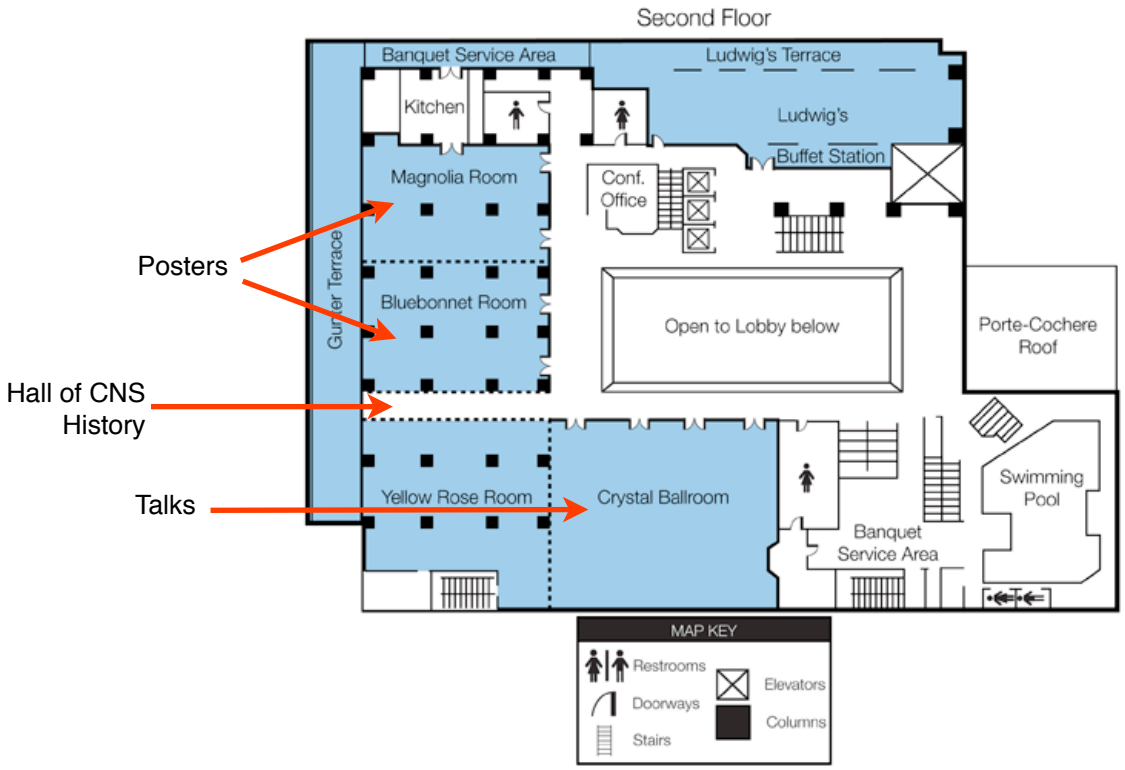
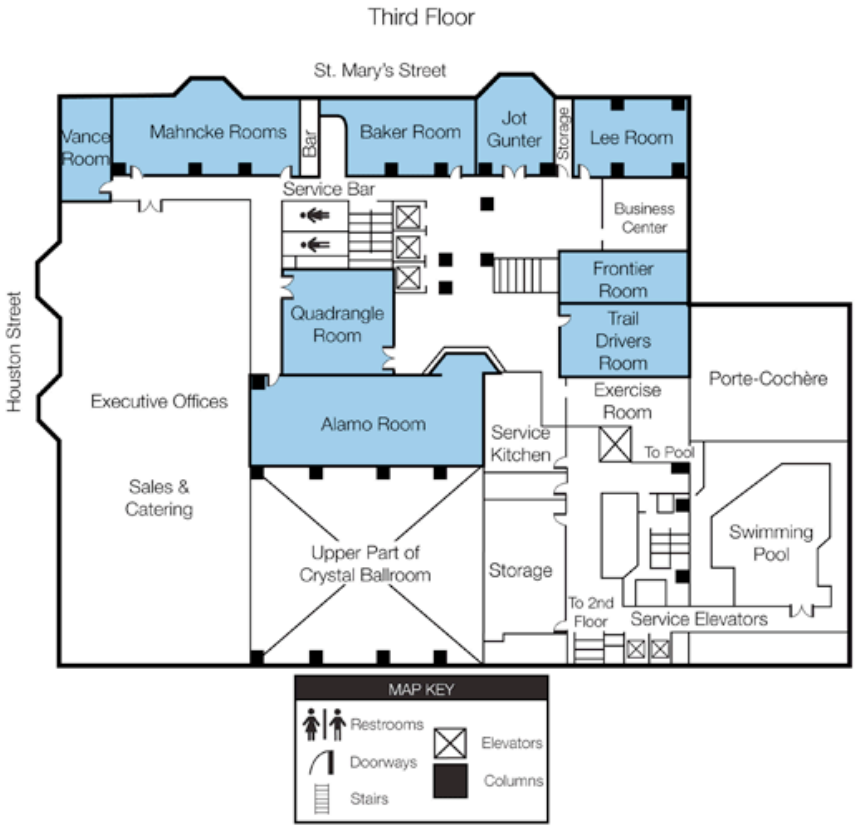
MEETING FLOOR PLAN

MAIN MEETING



TUTORIALS/ WORKSHOPS



TUTORIALS *Saturday, July 24*

AM Tutorials

Neural Control Engineering -
The Emerging Intersection of Control Theory and Neuroscience (6 hours, Ludwig's Room) Session I
Steven Schiff (Penn State Univ, USA)

Abstract: With the advent of model based ensemble techniques to track and control nonlinear systems in real time, the intersection between formal control theory and computational neuroscience is emerging as a powerful new area for exploration. This tutorial will explore how common models from computational neuroscience can be placed within a control theoretic framework, using a variety of cellular and network modeling frameworks. The route to real time feedback control systems will be explained with algorithm and code examples. A detailed discussion of formalizing model inadequacy will be covered. Applications to rhythmic hippocampal oscillations, seizures, Parkinson's disease, and cortical wave formation will be discussed (abstract truncated).

Neural Signal Processing Algorithms for Neural Spike Trains (3 hours, Alamo Room)
Emery N. Brown (MIT, USA)

One of the principal ways through which neurons represent and transmit information is in their spiking activity. Methods to analyze neural spike trains therefore play an important role in helping to understand function in the brain and central nervous system. In this tutorial we will review methods for single and multiple neural spike train data analysis. Lecture 1 will review of the theory of point processes and the use of the generalized linear model to relate spiking activity from single neurons to implicit and explicit stimuli. Lecture 2 will review likelihood methods for simultaneous analysis of multiple single neurons. Lecture 3 will present methods for dynamic analyses of neural spike trains including point process adaptive filters and neural spike train decoding algorithms. All methods will be illustrated using actual experimental examples.

Network Models of Short-term Memory, Persistent Neural activity, and Neural Integration
(3 hours,Yellow Rose Room).
Mark Goldman (University of California Davis, USA)

Neural activity that persists following the offset of a stimulus has been identified as a neural correlate of memory in a wide variety of systems. This tutorial will provide a mathematical foundation for building models of the neural activity observed in memory-storing networks. Current challenges to the field will be addressed and discussed. Topics to be covered include:
Linear network theory: positive and negative feedback; eigenvector and eigenvalue characterization of memory states; feedforward memory networks and failures of eigenvector analysis Robustness: How can networks built of neurons and synapses with brief decay time constants give rise to networks that can maintain memories for tens of seconds? What "tricks" might biology play that neural network modelers have failed to capture? Nonlinear networks: How can we construct memory networks with nonlinearities that cause linear systems techniques to break down?

Brute Force Exploration of High-Dimensional Neuronal Parameter Spaces (3 hours, Mahncke Room).
Astrid Prinz (Atlanta, USA)

The electrical activity generated by neurons and neuronal networks depends on cellular and synaptic parameters in a complex, often non-intuitive manner. This tutorial will cover a computational method that examines this activity-to-parameter relationship by systematically exploring the high-dimensional parameter spaces of neuron and network models. We will discuss advantages and disadvantages of parameter space exploration as compared to other methods of neuron and network analysis, technical issues regarding the implementation and execution of computational brute-force parameter exploration, available software tools and model databases generated with the method, and analysis and visualization techniques related to parameter space exploration. The tutorial will include hands-on exploration and visualization of an example neuronal parameter space by participants.

TUTORIALS *Saturday, July 24*

PM Tutorials

Neural Control Engineering - The Emerging Intersection of Control Theory and Neuroscience (6 hours, Ludwig's Room) Session II
Steven Schiff (Penn State Univ, USA)

Multi-Scale Modeling with MOOSE (3 hours, Mahncke Room).
Upinder S. Bhalla and Aditya Gilra (National Center for Biological Sciences, India)

The Multi-scale Object Oriented Simulation Environment (MOOSE) is an open-source simulator for computational neuroscience and systems biology written in C++. It promotes simulator inter-operability and model sharing by supporting standards like SBML and NeuroML. It has a PyQt GUI with 3D OpenGL visualization, and can run in parallel mode on a multi-core machine / cluster. We will guide the attendees through MOOSE installation on their linux / Windows® / Mac® notebooks and demonstrate some simple models distributed with MOOSE.

We will use Python to import a biochemical model of a secondary messenger pathway specified in SBML and a biophysical neuronal model specified in NeuroML to form a composite model in MOOSE. The secondary messenger pathway modifies the synaptic efficacies in the neuronal model while the activity of the latter modulates the former. The attendees will learn how to set up, tweak and simulate such multi-scale models in MOOSE.

Dynamical Systems Approaches to Understanding Neural Models (3 hours, Alamo Room).
Bard Ermentrout (Pittsburgh, USA.)

I will use an open source software package, XPPAUT, to show how dynamical systems methods can be used to investigate a number of problems in computational neuroscience. These range from simulations of single channels, through neurons, and networks. I will briefly touch on a number of numerical and mathematical methods that can be used to understand synchrony, spatial patterns, and the role of noise.

Introduction to Computational Motor Control (3 hours, Baker Room).
Reza Shadmehr (Johns Hopkins, USA),

This lecture introduces the problem of motor control from a computational perspective. The act of making a movement involves solving four kinds of problems:

1) We need to learn the costs that are associated with our actions as well as the rewards that we may experience upon completion of that action.
2) We need to learn how our motor commands produce changes in state of our body and our environment.
3) Given the cost structure of the task and the expected outcome of motor commands, we need to find those motor commands that minimize the costs and maximize the rewards.
4) Finally, as we execute the motor commands, we need to integrate our predictions about sensory outcomes with the actual feedback from our sensors to update our belief about our state.

In this framework, the function of basal ganglia appears related to learning costs and rewards associated with our sensory states. The function of the cerebellum appears to be related to predicting sensory outcome of motor commands and correcting motor commands through internal feedback. Together, reward driven optimal feedback control theory appears as a consistent framework to explain a number of disorders in human motor control.

MAIN MEETING *Sun July 25 - Wed July 28*

SUNDAY JULY 25, 2010

09:00 **Welcome & Announcements**

KEYNOTE

09:20 Frontiers in Computational Neuroscience Lecture:

L-1 COMPUTING WITH NEURAL ENSEMBLES. *Miguel A. L. Nicolelis (Duke University)*

10:20 Break

ORAL SESSION I

10:40 **O-01 Firing pattern regulation in hypothalamic vasopressin neurons: roles of synaptic inputs and retrograde signaling.** Alexander O. Komendantov, Ion R. Popescu, Jeffrey G. Tasker

11:00 **O-02 Computational model explaining two types of bursting found in inspiratory pacemakers.** Natalia Toporikova, Robert Butera

11:20 **O-03 Coregulation of ionic currents maintaining the duty cycle of bursting.** William Barnett, Martin Anquez, Gennady Cymbalyuk

11:40 **O-04 Neuronal bursting: interactions of the persistent sodium and CAN currents.** Justin R. Dunmyre, Jonathan E. Rubin

12:00 -13:30 Break for Lunch

FEATURED ORAL I

13:30 **F-1 High storage capacity of synfire chains in large-scale cortical networks of conductance-based spiking neurons.** Chris Trengove, Cees van Leeuwen, Markus Diesmann

14:10 Break

ORAL SESSIONS II & III

14:30 **O-05 Spike Threshold Dynamics Shape the Response of Subthalamic Neurons to Cortical Input.** Michael A Farries, Hitoshi Kita, Charles J Wilson

14:50 **O-06 Fine temporal structure of beta-band synchronization in Parkinson's disease: experiments, models and mechanisms** Leonid L Rubchinsky, Choongseok Park, Robert M Worth

15:10 **O-07 A computational approach to understanding the longitudinal changes in cortical activity associated with intensive meditation training.** Manish Saggar, Stephen R. Aichele, Tonya. L. Jacobs, Anthony P. Zanesco, David A. Bridwell, Katherine A. Maclean, Brandon G. King, Baljinder K. Sahdra, Erika L. Rosenberg, Phillip R. Shaver, Emilio Ferrer, B. Alan Wallace, George R. Mangun, Clifford D. Saron, Risto Miikkulainen

15:30 Break

15:50 **O-08 A computational model of associative learning and chemotaxis in the nematode worm C. elegans.** Peter A. Appleby and Netta Cohen

16:10 **O-09 Tracking Neuronal Dynamics During Seizures.** Ghanim Ullah, Steven J Schiff

16:30 **O-10 Dynamical effects of antiepileptic drugs on neurons affect network synchronizability.** Theoden Netoff, Bryce Beverlin II

16:50 **O-11 Sparse coding models demonstrate some non-classical receptive field effects.** Mengchen Zhu, Christopher J Rozell

19:30-22:30 **POSTER SESSION I**, P1-P64 (Bluebonnet/Magnolia)

MAIN MEETING *Sun July 25 - Wed July 28*

MONDAY JULY 26, 2010

INVITED LECTURE

09:00 **L-2 SPINAL CORD-MACHINE-INTERFACE FOR RESTORING STANDING AND WALKING** Vivian Mushahwar (U of Alberta, Edmonton)

SYMPOSIUM I: AM Session

10:20 **S-01 20 years of oscillations and memory: The long and winding road linking cellular mechanisms to behavior.** Michael E. Hasselmo (Boston University)

11:10 **S-02 Analysis of invertebrate nervous systems as models for understanding complex function.** John Miller (Montana State University)

12:00 -13:10 Break for Lunch

SYMPOSIUM I: PM Session

13:10 **S-03 The olfactory system, still computing, but how??** Christiane Linster (Cornell University)

14:00 **S-04 Learning from the past: Approaches for Reproducibility in Computational Neuroscience.** Sharon Crook (Arizona State University)

14:50 Break

15:20 **S-05 Calcium: the Answer to Life, the Universe, and Everything.** Avrama Blackwell (George Mason University)

16:10 **S-06 Still looking for the memories: molecules and synaptic plasticity.** Upinder Bhalla (NCBS- Bangalore India)

19:30-22:30 **POSTER SESSION II**, P65-129 (Bluebonnet/Magnolia)

TUESDAY JULY 27, 2010

INVITED LECTURE

09:00 **L-2 Brain-Computer interfaces come of age: Traditional assumptions meet emerging realities.** Jonathan R. Wolpaw, **(**Wadsworth Center, Albany NY)

SYMPOSIUM II: AM Session

10:20 **S-07 Modeling neuronal dynamics - our trajectory.** John Rinzel (NYU)

11:10 **S-08 The more we look, the more biological variation we see: How has and should this influence modeling of small networks?** Ron Calabrese (Emory University)

12:00 -13:10 Break for Lunch

SYMPOSIUM II: PM Session

13:10 **S-09 The Nervous System, still noisy after all these years?** Alain Destexhe (CNRS - France)

14:00 **S-10 Learning about vision: questions we've answered, questions we haven't answered, and questions we haven't yet asked.** Bruno Olshausen (University of California Berkeley)

14:50 Break

15:20 **S-11 Reinforcement learning models then and now: From single cells to modern neuroimaging.** Reed Montague (Baylor College of Medicine)

16:10 PANEL DISCUSSION, All speakers.

19:30-22:30 **POSTER SESSION III**, P130-P194 (Bluebonnet/Magnolia)

MAIN MEETING *Sun July 25 - Wed July 28*

WEDNESDAY JULY 28, 2010

AM Session

FEATURED ORAL

09:00 **F-2 A game of pool, a game of tag: AMPA trafficking in the post-synaptic density.** Fidel Santamaria, George J. Augustine, Sridhar Raghavachari

09:40 Break

ORAL SESSION IV

10:00 **O-12 Phase response analysis during *in vivo*-like high conductance states; Dendritic SK determines the mean & variance of responses to dendritic excitation.** Nathan W. Schultheiss, Jeremy R. Edgerton, & Dieter Jaeger

10:20 **O-13 Role of active dendritic conductances in subthreshold input integration.** Michiel Remme, John Rinzel

10:40 **O-14 Analysis of the mechanisms underlying windup using a detailed biophysical model of WDR neurons.** Paulo_Aguiar, Mafalda Sousa, Deolinda Lima

11:00 Business Meeting (Crystal Ballroom)

12:00 -13:10 Break for Lunch

PM Session

FEATURED ORAL

13:10 **F-3 Coding signal strength by correlated activity in bursting neurons.** Oscar Ávila Åkerberg, Maurice J Chacron

13:50 Break

ORAL SESSIONS V & VI

14:10 **O-15 Selecting appropriate surrogate methods for spike correlation analysis.** Sonja Grün, Christian Borgelt, George Gerstein, Sebastien Louis, Markus Diesmann

14:30 **O-16 Feedback Control of the Spatiotemporal Firing Pattern of a Basal Ganglia Microcircuit Model.** Jianbo Liu, Karim G. Oweiss, Hassan K. Khalil

14:50 **O-17 Stimulus-dependent suppression of intrinsic variability in recurrent neural networks.** Kanaka Rajan, Laurence F Abbott, Haim Sompolinsky

15:10 Break

15:30 **O-18 Causal networks in the rat barrel cortex provide a signature of stimulus encoding.** Seif Eldawlatly and Karim Oweiss

15:50 **O-19 Sparse codes of harmonic sound and their interaction explain harmony-related response of auditory cortex.** Hiroki Terashima, Haruo Hosoya

16:10 **O-20 How Good is Grid Coding versus Place Coding for Navigation Using Noisy, Spiking Neurons?** Alexander Mathis, Martin Stemmler, Andreas Herz

16:30 **O-21 Sparse coding models demonstrate some non-classical receptive field effects.** Mengchen Zhu, Christopher J Rozell

THURSDAY WORKSHOPS (9am - 4:30pm)

*This is a partial list of Workshops that have been scheduled for OCNS*2010. Comprehensive information on each of the Workshops listed below may be found at http://www.cnsorg.org/2010/workshops.shtml. Room assignments and information on ad hoc additions to the schedule will be available at the registration desk.*

Methods of Information Theory in Computational Neuroscience (Day 1 of 2, All Day)

Organizers: Todd P. Coleman, Aurel Lazar and Simon R. Schulz

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.

Advancing Brain-Computer Interface (All Day)

Organizer: DGirijesh Prasad (University of Ulster, N. Ireland, UK) & Günter Edlinger (CEO g.tec, Austria)

A brain-Computer interface (BCI), also known as brain-machine interface (BMI), utilizes neurophysiological correlates of voluntary cognitive tasks to facilitate direct communication between human brain and computing devices without the involvement of neuro-muscular pathways. This emerging research area has the potential to contribute significantly to enhancing the accessibility of ICT systems for the elderly and disabled people. It is, in general, progressing in two main areas: BCI for communication for improving independence & quality of life of severely disabled people such as sufferers of motor neurone disease (MND) and spinal chord injury, and BCI for rehabilitation purposes, e.g. motor restoration in paralysis due to stroke. Current BCI systems however, lack sufficient robustness and the performance variability among users is quite high. One of the critical limitations is because of the non-stationary characteristics of the brain's neurophysiological responses, which makes it very hard to extract timeinvariant stable features unique to voluntary cognitive tasks. Under these inherent limitations, devising realworld BCI applications for constant use is a real challenge. The workshop aims to discuss recent developments in robust BCI design and practical real-world applications made possible through advances in one or more BCI design phases: paradigm design, invasive and non-invasive brain signal selection and acquisition, signal pre-processing, feature selection and extraction, feature classification, and application interface design.

Computational models for movement control and adaptation during BMI operation (All Day)

Organizer: Miriam Zacksenhouse (Technion)

The development of Brain-Machine Interfaces (BMIs) has been motivated by insights about neural encoding in the motor cortex, and in particular the evidence that cortical motor neurons encode the direction of movement. However, it is evident that cortical motor neurons encode multiple signals and that encoding may change with task. Of particular interest are the changes that may occur following changes in the motor environment, and, specifically, when switching to operate a BMI. Computation motor control models may facilitate the investigation of these changes and their potential interpretation and decoding. This workshop is targeted at bringing together researchers investigating neural encoding and decoding in the motor cortex, and in particular those working on BMIs, with researchers developing computational motor control models, to further explore neural changes during BMI operation and their potential interpretation within the context of computational motor control. It is expected that such a workshop would both motivate further development of computational motor control models and facilitate the development of BMIs.

WORKSHOPS *Thur July 29 - Fri July 30*

THURSDAY WORKSHOPS (cont)

Reaction-diffusion modeling for neurobiology

Organizers: Avrama Blackwell (George Mason U) and Bill Lytton (SUNY Downstate)

New methods developed for Computational Systems Biology (CSB) are being increasingly used in Computational Neuroscience to study the interaction of currents and voltage with e.g. second messenger cascades; transcriptional regulation and protein interactions. This workshop on reaction-diffusion (RD) modeling will attempt to address three somewhat divergent goals: 1. provide an introduction to research questions being addressed using reaction-diffusion modeling; 2. provide an introduction to modeling tools for interfacing RD with traditional computational neuroscience methods; 3. introduce some people from CSB to our community and vice versa in order to encourage cross-fertilization and new collaborations.

Modeling the dynamics and function of cerebellar neurons and circuits

Organizers: Dieter Jaeger (Emory) & Volker Steuber (Univ. of Hertfordshire)

The quasi-crystalline structure of the cerebellar cortex has inspired many functional theories and modeling studies over the past 50 years. This workshop is aimed at presenting and discussing the current state of the field of cerebellar modeling. We will describe models at different levels of abstraction from the subcellular to the network level. We will encourage debate on how well these models reflect the biological cerebellum at the present time and what future work is needed to generate accurate models of cerebellar function.

RTXI Test Drive a RealTime Linux Based Dynamic Clamp System on Your Laptop (Half Day)

Organizers: John White (University of Utah), David Christini (Cornell), & Robert Butera (Georgia Tech)

RTXI is an open-source, real-time Linux-based dynamic clamp system (http://www.rtxi.org). While realtime Linux systems have a reputation of being difficult to install and use, we believe RTXI overcomes such issues. In this workshop, an introductory talk will describe the system, and two applications talks will demonstrate its utility. Next, workshop participants will receive a bootable USB stick (containing a live Ubuntu-based RTXI system) with which to test drive RTXI on their laptops.

Using models to collaborate, communicate, and publish: An introduction to GENESIS 3.0 and the future of computational neurobiology

Organizers: Hugo Cornelis, Allan D. Coop, Mando Rodriguez, David Beeman, & James M. Bower.

The GENESIS Development Team announces a one day workshop to introduce the new GENESIS 3.0 (G-3) simulator. The G-3 simulator is a modular reimplementation of the GEneral NEural SImulation System software platform and provides both substantial functional enhancement over earlier versions of GENESIS and a fundamentally new “modern” architecture for modeling software. Implemented software components include realistic modeling solvers, a storage system for models, a flexible run-time scheduler, modules to implement various experimental designs, and a GUI interface for users that supports model-based communication including publication (abstract truncated).

Mechanisms, interactions and functions of neural oscillations

Organizers: Vassilis Cutsuridis and Nathan Schultheiss (Boston U)

Brain oscillations have been associated with diverse cognitive processes including perceptual binding, attention, and memory. Oscillations appear in the brain in various frequency bands, which may occur simultaneously or interact with one another. They contribute to coordination of activity between local and distant neuronal populations during both normal brain functioning and in disease states. Oscillations in the beta and gamma range establish synchronization with great precision in local cortical networks, whereas lower frequencies preferentially establish synchronization over longer distances. The goal of the workshop is to provide a resume of the state-of-the-art in computational and mathematical investigations of the mechanisms, interactions and functions of neural oscillations in both normal and diseased brain.

WORKSHOPS *Thur July 29 - Fri July 30*

Postdoc Career Strategy Workshop (Thursday Evening, Crystal Ballroom)

Organizers: Nathan Schultheiss (Boston U)

The computational neuroscience (CNS) community is both international and interdisciplinary, and there are many possible roads to success in this field. However, the challenges faced by current or soon-to-be postdocs in CNS are also diverse, and excellent mentorship from primary investigators is an invaluable resource for the development of future leaders in research. This workshop is intended to provide postdocs 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 short talks formatted as testimonials and will target issues faced in academia by junior faculty having recently transitioned from postdoc status, researchers working outside of their home countries, and/or researchers working in departments other than their primary field of training. Additional talks will be given by more senior researchers who are often responsible for steering departmental hiring and review board policies, decisions, and indeed the science itself. Postdocs and students are encouraged to ask questions of the speakers and participate in discussion of career strategies with a panel of distinguished researchers.

FRIDAY WORKSHOPS (All Full Day)

Methods of Information Theory in Computational Neuroscience (Day 2 of 2)

Organizers: Todd P. Coleman, Aurel Lazar and Simon R. Schulz

Neurodesign: Using computational modeling for the design of neurotechnology

Organizers: Douglas Weber (U Pittsburgh) & Ranu Jung (Arizona State U)

High-throughput 3D microscopy and high-performance computing for multi-scale modeling and simulation of large-scale neuronal circuits

Organizers: Yoonsuck Choe & Louise C. Abbott, (Texas A&M U)

Rapid advances in high-throughput, high-volume 3D microscopy technology is enabling the acquisition of neuronal-level data at the scale of whole small animal organs such as the mouse brain. Techniques that allow 3D molecular imaging and ultra high-resolution electron microscopy imaging provide a complementary perspective, where detailed local circuit function can be investigated. These microscopy technologies, together with high-performance computing power becoming available are enabling a data-driven, multi-scale modeling and simulation of large-scale neuronal circuits (such as the complete connectome of the mouse). This workshop will give a timely update on this burgeoning field and provide a forum for intensive discussion to shape the immediate and future direction of data-driven computational modeling and simulation of the brain.

Phase Locking in the Presence of Biological Noise

Organizers: Carmen Canavier (LSU) and Leonid Rubchinsky (IUPUI)

Two or more oscillators can temporally coordinate their activity via common inputs or coupling. If these oscillators are biological neurons or modules comprised of multiple biological neurons, the presence of noise is inevitable. In the presence of noise, it is not obvious whether oscillators have a consistent phasic relationship, or phase locking. At a minimum, circular statistics are required to determine whether there is a preferred phase difference. Phase slips or other phase dynamics may episodically or persistently interrupt periods of temporal coordination, thus the existence of a phase locking in the presence of noise is not trivial to ascertain as it is in its absence. Ghosts of attractors that are nearby in parameter space may also influence the dynamics. We will discuss approaches to detection of phase locking in noisy experimental data, and the implications of such a locking for information processing under normal and abnormal conditions in the nervous system.

WORKSHOPS *Thur July 29 - Fri July 30*

Multistability in Neurodynamics

Organizer: Gennady Cymbalyuk

This workshop is focused on the co-existence of regimes of activity of neurons. Such multistability enhances potential flexibility to the nervous system and has many implications for motor control, dynamical memory, information processing, and decision making. The goal of this workshop is to identify the scenarios leading to multistability in the neuronal dynamics and discuss its potential roles in the operation of the central nervous system under normal and pathological conditions. It is intensively studied on different levels. On the cellular level, multistability is co-existence of basic regimes like bursting, spiking, sub-threshold oscillations and silence. On the network level, examples of multistability include co-existence of different synchronization modes, “on” and “off” states, polyrhythmic bursting patterns, and co-existence of pathological and functional regimes.

Further Down The River - What Will the Next 20 Years of Computational Neuroscience Bring

Navigator: James M. Bower (University of Texas Health Science Center San Antonio)

As the CNS meeting starts its 20th year, where are we headed? Will progress flow slowly and evenly, with an occasional run through the rapids? Will the temperature be hot, but the river cool? Will we all be headed in the same direction, or will some fight the current and try to head upstream? And how many in the general population will watch and wonder at what we are doing? During this workshop the answers will be yes, yes, yes, and probably not, and perhaps 30,000. Meet in the lobby of the Gunther Hotel at 8:30 - and bring your bathing suit, sunscreen and water proof ID and all your ideas on what the future holds, further down the river.

Methods in Neuroinformatics

Organizers: Malin Sandström (INCF Secretariat), Raphael Ritz (INCF Secretariat), Jeff Grethe (UCSD)

Neuroinformatics is the research area that intersects neuroscience, informatics and modeling. As such, it offers solutions to and perspectives on many problems in computational neuroscience. The goal of this workshop is to bring together researchers in neuroinformatics to present and discuss work on current neuroinformatics initiatives that deal with specific issues relevant to computational neuroscience, among them model description.

Frontiers in Computational Neuroscience Lecture:

L-1 COMPUTING WITH NEURAL ENSEMBLES

Miguel A. L. Nicolelis, MD, PhD
Anne W. Deane Professor of Neuroscience
Departments of Neurobiology, Biomedical Engineering and Psychological and Brain Sciences
Co-Director, Duke Center for Neuroengineering

I review a series of recent experiments demonstrating the possibility of using real-time computational models to investigate how ensembles of neurons encode motor information. These experiments have revealed that brain-machine interfaces can be used not only to study fundamental aspects of neural ensemble physiology, but they can also serve as an experimental paradigm aimed at testing the design of modern neuroprosthetic devices. I also describe evidence indicating that continuous operation of a closed-loop brain machine interface, which utilizes a robotic arm as its main actuator, can induce significant changes in the physiological properties of neurons located in multiple motor and sensory cortical areas. This raises the hypothesis of whether the properties of a robot arm, or any other tool, can be assimilated by neuronal representations as if they were simple extensions of the subject's own body.

INVITED LECTURES

L-2 SPINAL CORD-MACHINE-INTERFACE FOR RESTORING STANDING AND WALKING

Vivian K. Mushahwar PhD
Department of Cell Biology and Centre for Neuroscience
University of Alberta
Edmonton, Alberta, Canada

Loss of standing and walking is a devastating side effect of neural injuries including spinal cord injury, head trauma, and stroke. In this talk, I will summarize our lab's efforts at restoring functional standing and walking capacity through the use of a miniature intraspinal neuroprosthetic interface. I will describe pertinent anatomical features of the spinal cord (in comparison to cortical regions of the brain), long-term performance of microwires interfacing with spinal cord tissue, models of locomotor function, and software and hardware implementations of models of locomotion. I will particularly focus on important features of the neuronal locomotor circuitry and show results from in vivo experimentation demonstrating the effectiveness of the intraspinal neuroprosthetic interface in producing robust over-ground walking.

This work is supported by the National Institute of Health, the Canadian Institutes of Health Research, the International Spinal Research Fund and the Alberta Heritage Foundation for Medical Research.

L-3 BRAIN-COMPUTER INTERFACES COME OF AGE: *TRADITIONAL ASSUMPTIONS MEET EMERGING REALITIES*

Jonathan R. Wolpaw, M.D.
Wadsworth Center
New York State Department of Health
Albany, NY

Brain-computer interfaces (BCIs) could provide important new communication and control options for people with severe motor disabilities. Much BCI research has been based on four assumptions: (1) that intended actions are fully represented in the cerebral cortex; (2) that neuronal action potentials can provide the best picture of an intended action; (3) that, therefore, the best BCI is one that records action potentials and decodes them; and (4) that ongoing mutual adaptation by the BCI user and the BCI system is not very important. It is increasingly clear that none of these assumptions is defensible. Intended actions are the products of many areas, from the cortex to the spinal cord, and the contributions of each area change continually as the CNS adapts to optimize performance. BCIs must guide and track these adaptations if they are to achieve and maintain good performance. Furthermore, it is not yet clear which categories of brain signals will prove most effective for which BCI applications. In human studies to date, low-resolution EEG-based BCIs and high-resolution cortical neuron-based BCIs perform similarly. In sum, BCIs allow their users to develop new skills in which the users achieve their goals through brain signals rather than muscles. Thus, the central task of BCI research is to determine which brain signals users can best control, to maximize that control, and to translate it accurately and reliably into actions that accomplish the users' intentions. The most difficult aspect of this task is probably not the realization of many degrees of freedom, but rather the achievement of highly reliable performance. Much better reliability is essential if BCIs are to advance from laboratory demonstrations to systems of significant practical value in daily life.

SPECIAL SYMPOSIUM

Computational Neuroscience: What have we learned in 20 years and what do we still need to know?

It has been 20 years since plans were made to organize the worlds first open international meeting in computational neuroscience. Quoting from the introduction to the proceedings of the first meeting in 1990, "Sensing that recent leaps in computational power and knowledge of the nervous system may have set the state for a revolution in theoretical neurobiology, our motive was to organize a conference focused on emerging modeling tools and emerging neurobiological concepts". As part of CNS*2010, we have invited key participants in the last 20 years of CNS meetings to present their views on where computational neuroscience has been and where it is going. This special symposium will allow for both reflection and predictions on the future of our computational approach to understanding how brains work.

S-01 20 years of oscillations and memory: The long and winding road linking cellular mechanisms to behavior

Michael E. Hasselmo (Boston University)

The techniques of computational neuroscience are particularly relevant to understanding the mechanism and functional role of oscillations in neural structures. Researchers from the CNS meeting have played a central role in our understanding of neural oscillations over the past twenty years. The mechanisms of neural oscillations have been explained using mathematical analysis and detailed biophysical simulations in GENESIS and NEURON. This work has demonstrated the role of circuit dynamics, synaptic time constants and intrinsic properties in contributing to network oscillations. Specifically in the field of memory research, experimental data indicates an important role of hippocampal theta rhythm oscillations for learning and memory. The phenomenon of theta phase precession in hippocampal place cells, first reported by O'Keefe and Recce in 1993, provides a particularly exciting link between cellular mechanisms and memory function. Their first paper on theta phase precession proposed that it arose from oscillatory interference, but this model suffered from the problem that oscillatory interference would produce multiple firing fields. This problem became an experimentally verified prediction when grid cells with a repeating array of firing fields were described in the entorhinal cortex by the Moser laboratory. O'Keefe and Burgess then predicted that the difference in spacing between firing fields of entorhinal grid cells along the dorsal to ventral axis of entorhinal cortex would scale with a difference in the intrinsic frequency of entorhinal neurons. Supporting this prediction, whole cell patch data subsequently showed that the frequency of cellular resonance and membrane potential oscillations differed along the dorsal to ventral axis of entorhinal cortex (Giocomo et al., 2007; Giocomo and Hasselmo, 2008). This and other experimentally verified predictions support the model of grid cell firing in which input from speed-modulated head direction cells regulates interference between oscillations to perform functional path integration and generate realistic grid cell spiking activity in a virtual rat. A related model generates grid cell firing (Hasselmo, 2008a) using velocity modulation of neurons showing persistent spiking in the entorhinal cortex (Yoshida et al., 2008) and in the postsubiculum (Yoshida and Hasselmo, 2009). Grid cell models have been used in network models (Hasselmo, 2008b) that can replicate data on firing of hippocampal neurons in spatial tasks (Lee et al., 2006) and can encode and retrieve spatiotemporal trajectories as a potential mechanism for episodic memory. However, a number of important experimental questions remain concerning the link of these cellular mechanisms to behavior.

Giocomo, L.M., Hasselmo, M.E. (2008) Time constant of I(h) differs along dorsal to ventral axis of medial entorhinal cortex. J. Neurosci., 28:9414-25

Giocomo LM, Zilli EA, Fransen E, Hasselmo ME. (2007) Temporal frequency of subthreshold oscillations scales with entorhinal grid cell field spacing. Science, 315:1719-22.

Hasselmo M.E. (2008a) Grid cell mechanisms and function: Contributions of entorhinal persistent spiking and phase resetting. Hippocampus. 2008;18(12):1213-29.

Hasselmo, M.E. (2008b) Temporally structured replay of neural activity in a model of entorhinal cortex, hippocampus and postsubiculum. Eur. J. Neurosci. 28:1301-1315

Lee I, Griffin AL, Zilli EA, Eichenbaum H, Hasselmo ME (2006) Gradual translocation of spatial correlates of neuronal firing in the hippocampus toward prospective reward locations. Neuron, 51: 639-50.

Yoshida, M., Fransen, E., Hasselmo, M.E. (2008) mGluR-dependent persistent firing in entorhinal cortex layer III neurons. Eur. J. Neurosci. 28(6):1116-26.
Yoshida M., Hasselmo M.E. (2009) Persistent firing supported by an intrinsic cellular mechanism in a component of the head direction system. J Neurosci. 29(15):4945-52.

S-02 Analysis of invertebrate nervous systems as models for understanding complex function

John Miller (Montana State University)

Analyses of invertebrate nervous systems have illuminated many fundamental principles in neuroscience, starting as far back as the late 1800s with Ramon y Cajal's anatomical studies and continuing to the present. A significant fraction of the theoretical and modeling studies at CNS meetings (as well as many other meetings!) over the past 20 years have been based on invertebrate nerve cell- and system- level research. However, it seems to be a prevalent (though not universal!) attitude among neuroscientists that the generalizability and relevance of invertebrate research stops short of large-scale cortical function in vertebrates; *i.e.*, that our own brains work in a way that is fundamentally different than the way that the brains of even the most complex invertebrates work. What is some of the evidence on both sides of this argument? To put it from a different perspective, just how far does the multi-dimensional "invertebrate-system-level-research space" extend into and overlap with the "how-does-the-mammalian-brain-work space"?

S-03 The olfactory system, still computing, but how??

Christiane Linster (Cornell University)

Computational olfaction originally focused on the role of dynamics, attractors and synchronization properties in the olfactory bulb and cortex. These approaches were followed by a large body of work focusing more on activity patterns and their dependence on behavioral states, followed by a more recent revival of the importance of dynamics. I will review the history of thinking in computational olfaction and conclude with a general model of olfactory function that incorporates much of what has been proposed during the last 25 years.

S-04 Learning from the past: Approaches for Reproducibility in Computational Neuroscience

Sharon Crook (Arizona State University)

The independent verification of results is a critical step in the scientific process, and it would seem that achieving reproducibility should be much easier for computational scientists than for experimentalists. However, problems with reproducibility have received wide attention in recent years in many fields of computational inquiry, and some refer to the inability to routinely achieve reproducibility as a true crisis in computational science. Reproducibility in computational neuroscience requires descriptions of complex models that are precise and unambiguous, and some recent approaches aimed toward achieving reproducibility include model sharing databases, suggestions for publication standards, software for tracking computational "experiments", and simulator independent model description languages. In this talk, I will discuss the past, present and future of the independent verification of computational results in our field. Looking to the past, I will make the case that reproducibility is not only good for science but also good for scientists. In addition, I will provide an overview of the current status of efforts in the community and their trajectories. Finally, as more computational scientists focus on complex multiscale and collaborative approaches, systemic and automated avenues for reproducibility are becoming increasingly important, and I will challenge the CNS community to address this potential crisis.

SYMPOSIUM *Mon-Tues July 26-27*

S-05 Calcium: the Answer to Life, the Universe, and Everything

Avrama Blackwell (George Mason University)

Calcium is a crucially important molecule for diverse functions such as synaptic plasticity and neurotransmitter release in neurons, and for excitation-contraction coupling in other excitable cells. In the field of plasticity, an elevation in intracellular calcium concentration is critical for induction of both LTP and LTD. Several experiments have suggested that the amplitude of the calcium elevation predicts whether an induction paradigm will produce potentiation or depression: a large calcium elevation produces potentiation, whereas a small calcium elevation produces depression. Other experiments suggest that calcium concentration by itself is not always sufficient to predict the direction of plasticity.

Calcium imaging has revealed much about the spatial and temporal regulation of intracellular calcium, which is controlled by multiple mechanisms. Sources of calcium include influx through voltage dependent channels, synaptically activated channels, as well as calcium release channels of the endoplasmic reticulum. Sinks of calcium include membrane pumps and intracellular buffers. Diffusion interacts with these mechanisms to influence the size of spatial microdomains. Calcium dynamics in neurons are complicated both because of diffusion within the complex morphology and because of multiple feedback loops between membrane potential and calcium concentration. On a fast time scale, calcium influx through voltage dependent channels activates calcium dependent potassium channels, which in turn modifies membrane potential. On a slower time scale, calcium leads to activation of kinases and phosphotases that modify properties of synaptic channels and intrinsic channels, which in turn modifies the rate of calcium influx. The non-linear interactions between different sources of calcium and its multiple target molecules impede predicting the consequences of neural activity without using quantitative dynamical models.

Both the importance of calcium and quantity of calcium imaging data makes it an attractive target for modeling, especially since the fluorescent calcium indicators are fast calcium buffers, and thus prevent the plasticity changes of interest. Nonetheless, the complexity of processes controlling calcium makes it extremely difficult to model. This talk discusses the history of modeling calcium dynamics, emphasizing the discoveries made with the models, and the evolution in size and complexity of the models.

S-06 Still looking for the memories: molecules and synaptic plasticity.

Upinder Bhalla (NCBS- Bangalore India)

Computational neuroscientists have been playing around with plastic synapses for several decades. Interestingly, mechanistically detailed models of synaptic plasticity started around the same time as the CNS meetings. This was when the associative properties of the NMDA receptor were demonstrated, first setting out the molecular and mechanistic underpinnings of synaptic plasticity. Some 20 years ago there was little reason to to expect that the underlying biology would turn out to be as outrageously complicated as we now find it. Associativity seemed to be established by the NMDA receptor especially through the work of Collingridge, and there were already a couple of candidate mechanisms for how to maintain synaptic weights: the CaMKII autocatalytic process found by several people and first modeled by Lisman, and the PKA story from Kandel.

These leads led into a maze. Even ten years ago, there were 200 known molecules implicated in synaptic plasticity. My own first models of plasticity had some dozen signaling pathways - a far cry from what was known. The field as a whole is still playing catch-up. Nevertheless, most of the key properties of plasticity have had a good share of models, at various levels of detail. Associativity and long-term sustenance of plasticity were among the first to be looked at. Several groups have now looked at pattern selectivity in triggering plasticity. The question of biochemical noise at the synapse, and how it affects sustained retention of information, has been addressed in increasing detail since the early CaMKII models of Lisman, and has been extended to many other signaling pathways. Most of the key plasticity findings - stages of plasticity, metaplasticity, STDP, synaptic tagging, activity-dependent dendritic protein synthesis - have now been modeled at biochemical levels of detail.

Granted that there are lots more molecules lurking out there, surely we have enough of the key ones to account for many features of plasticity? Only if we believe the results of all the models in isolation. I would hazard to say that the

field as a whole is still trying to make sense of the parts list. We now know enough to realize that these phenomena have to be put together in the context of each other and of the cell. Not only do the molecules have to talk to each other, but they must also diffuse, talk to the electrical events, cause structural changes, and interact with protein synthesis and transport processes. I think that these interfaces between scales and cellular processes are where the most interesting questions await us. As many of us felt, 20 years ago, we're again at a fascinating time where the experiments, the databases, and the computational tools are just coming together to address these questions.

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S-07 Modeling neuronal dynamics - our trajectory

John Rinzel (NYU)

Dynamics dynamics dynamics... timing timing timing... noise noise noise. The temporal aspects of neuronal activity are being increasingly appreciated. Some awakenings for the roles of dynamics will be reviewed in this talk: dendritic currents and signaling, STDP - synaptic plasticity with precise timing, the nature of spike threshold, the resurrection of Hodgkin's classification (1948), coincidence detection mechanisms, the color and correlation of noisy inputs, and, where might our trajectory carry us?

S-08 The more we look, the more biological variation we see: How has and should this influence modeling of small networks?

Ron Calabrese (Emory University)

Variability in intrinsic membrane and synaptic parameters and their implication for network activity has received considerable theoretical attention (Prinz et al., 2004; Prinz, 2007). It is now increasingly clear that many biological neuronal networks, and CPGs in particular, can display a 2-5 fold range of intrinsic membrane currents and synaptic strengths while still producing stereotypical output (Bucher et al., 2005; Marder and Goaillard, 2006; Marder et al., 2007; Goaillard et al., 2009). Moreover, there are indications that average values for such strength parameters may be misleading in that models constructed from average values may not produce stereotypical output.

One of the long-range aims of my lab has been a complete model of how a CPG controls intersegmental motor outflow. In our system, the leech heartbeat CPG, output from premotor interneurons of the well-characterized CPG onto motor neurons is all inhibitory. We began by choosing an exemplar recording of spiking activity in all the intersegmental premotor interneurons as the temporal input for our model of motor neuron coordination into a fictive motor pattern (Norris et al., 2006). We then focused on a quantitative assessment of inhibitory synaptic strength and dynamics and associated conduction delays of premotor interneurons to be used as parameters in the model (Norris et al., 2007a, b). Using values averaged across animals for these synaptic strengths allowed us to construct a model that captured the gross intersegmental coordination but fell well short of quantitative verisimilitude with phase data for the fictive pattern averaged across animals (Garcia et al., 2008). This led us to reassess not only animal-to-animal variability in the pattern of synaptic strengths but also in the temporal pattern of interneuronal input and of motor neuron output. We found that all of these patterns, while on average true to the general conception of the fictive pattern, showed uncorrelated variability.

Given that we did not even consider variability in motor neuron intrinsic properties then it becomes clear that we are faced with nearly impossible task in arriving at a quantitatively accurate model of even the simplest neuronal networks: parameter and characteristic measurement must all be made in the same animal. Perhaps then there is merit in using average data for models and sacrificing quantitative accuracy for mechanistic understanding. On the other hand it may be desirable to tune a model based on average data to a desired output using automated search algorithms (with as many parameters set free as computational feasible) and then determine whether the parameters found fall within the biological range.

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S-09 The Nervous System, still noisy after all these years?

Alain Destexhe (CNRS - France)

The talk will overview the notion of "noise" in the nervous system and it will emphasize the role played by theoretical and computational neuroscience. There are multiple sources of noise in neurons, and it was classically considered as detrimental to information processing. However, the last decades have seen a tremendous paradigm shift, where the "noise" is often considered as beneficial, and even integral part of neural coding. The talk will review the early attempts to investigate the sources of noise, and how strongly researchers had to fight to convince their colleagues that the noise (and the high conductances) seen in vivo is not due to bad recording conditions, but is present at all levels of integration. Today, many concepts about the role of noise have been integrated by the theoretical/computational neuroscience community, in part because of its close relationship with communities like complex systems or the mathematics of stochastic systems. These concepts also led to new methods to analyze experimental data, and conceive new experimental paradigms where stochastic aspects are explicitly taken into account. These models and experiments have revealed, for example, that the average level of input is sometimes irrelevant, but it is the variations, the fluctuations and the "noise" that are the most pertinent variables. Thus, models are expected to become more and more "noisy" in the future.

S-10 Learning about vision: questions we've answered, questions we haven't answered, and questions we haven't yet asked.

Bruno Olshausen (University of California Berkeley)

The past twenty years have seen steady progress in the study of vision: Advances in adaptive optics have enabled us to resolve and stimulate individual cones within the living human eye; functional magnetic resonance imaging has revealed a detailed map of visual areas in the human brain, some with a striking degree of functional specificity; psychophysical studies have revealed the existence of intermediate-level representations of 3D surfaces and occlusion; and computational studies have shown how aspects of neural coding and behavior are related to natural image statistics through principles of efficient coding and Bayesian inference. In parallel with these developments, the field of computer vision has also undergone important advances - in particular, the discovery of stable and unique keypoint detectors and invariant feature descriptors has enabled dramatic improvements in object recognition and the recovery of 3D scene structure from multiple views.

In light of these great strides, one may be tempted to think we are getting close to understanding how visual systems work. However, there are still many basic questions that remain open: How do the tiny nervous systems of bees and jumping spiders enable their sophisticated abilities in navigation and object recognition? How do the

different lamina within mammalian visual cortex interact and contribute to visual processing? What is the role of the massive feedback pathways between cortical areas and between cortex and thalamus? To these and many other questions we come up empty handed. And despite the advances in computer vision, we have yet to see any artificial vision system approach the level of robustness, speed, and low power consumption, let alone intelligence, found in biological vision systems. One gets the sense we are missing something that is quite fundamental.

In order to move forward, we must first acknowledge the magnitude of the problem before us. Scientists by their nature are eager to test hypotheses, or to "tell a story" about how a given set of facts or findings fit together and explain perceptual phenomena. But nervous systems present us with stunning complexity. Most of the hypotheses and stories we tell are far too simple minded and ultimately turn out to be wrong.[1] Worse yet, they can be misleading and stifling because they encourage one to look at the data through a narrow lens. I shall argue here that, given the state of our knowledge in neuroscience, we are better served by taking an exploratory approach that casts a wide net and seeks to reveal interesting phenomena, rather than carefully designing experiments to test a specific set of hypotheses. In particular, what is needed is a better understanding of *neural dynamics* and the unique computational properties exhibited by neural systems. We also need new insight into the perception-action cycle and the principles governing sensorimotor loops. These areas remain vastly under investigated. Indeed, some of the most important questions have yet to be asked.

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S-11 Reinforcement learning models then and now: From single cells to modern neuroimaging

Reed Montague (Baylor College of Medicine)

Reinforcement learning models represent a large and productive genre of computational approaches to synaptic plasticity, cell spiking, and they even reach all the way up to behavioral control and neuroimaging experiments in humans. These models are the result of a fusion of ideas that emerged from the area of optimal control in the late 50's and the area of trial and error learning familiar to psychology over the last 30 years. Reinforcement learning approaches initially found their biggest neurobiological 'hit' in their application to single cell recordings from midbrain dopamine neurons during reward learning tasks, but they have matured dramatically in recent years and are being used to capture sequential choice and real-world decision-making in models that can connect sensibly to underlying neural substrates. This talk will look back over the last 20 years of development of this approach to motivated learning, show how the computational models made a real difference in the interpretation of the neurobiological data, and look to new directions and applications for the future.

FEATURED ORALS

F-1

High storage capacity of synfire chains in large-scale cortical networks of conductance-based spiking neurons

Chris_Trengove[1], Cees van Leeuwen[2], Markus Diesmann[13]

[1] Brain and Neural Systems Team, RIKEN, Wako City, Saitama, 351-0198, Japan
[2] Perceptual Dynamics Laboratory, RIKEN, Wako City, Saitama, 351-0198, Japan
[3] Diesmann Research Unit, Brain Sciences Institute, RIKEN, Wako City, Saitama, 351-0198, Japan
E-mail: ctrengove@brain.riken.jp

We demonstrate stable dynamics of synchronous pulse packet propagation and global asynchronous irregular (AI) activity in a sparse network of 10^5 neurons in which excitatory connectivity is derived from a random superposition of synfire chains having a total length of up to 80,000 pools. This is the largest amount of synfire chains to have been stably embedded in a network model of spiking neurons: about two orders of magnitude great than achieved previously [1-3]. Key features of the model which allow this high storage capacity are: (1) conductance-based rather than current-based synapses; and (2) inhibitory neurons in the synfire chain pools. As noted recently [3] the use of conductance over current based results in a narrower membrane potential distribution with a mean closer to threshold, which is more favourable for pulse-packet propagation at realistic levels of AI activity and background input.

Recurrent dynamics of the AI state is driven by the propagating pulse packets which deliver random excitatory and inhibitory 'background input' to the rest of the network. The equilibrium dynamical behaviour of the network depends on a critical level of excitatory and inhibitory background input above which synfire wave propagation ceases to be viable. In our simulation protocol synfire waves are initiated by ongoing, intermittent delivery of external input pulses. In the stable regime, an upper limit in the number of simultaneously propagating pulse packets is reached, typically ~ 5-30 waves, above which further waves are only successfully initiated at the expense of existing waves.

Using a semi-analytical framework, storage capacity, and the number of pulse packets that can propagate simultaneously, is calculated. Mean field analysis is used to estimate the stability and spiking rate of the AI state, combined with a numerical determination of the stability of wave propagation. Results agree qualitatively with simulations and correctly predict the order of magnitude of storage capacity. Departures from mean field theory are observed, most notably the greater fluctuations in the AI state particularly as the upper limit of storage is approached, which impacts upon the stability of pulse packet propagation and reduces the maximum number of simultaneously propagating waves.

We also demonstrate in a modified version of this architecture that includes branching chains how synfire waves can be generated and maintained endogenously, using the same mechanism which stabilises the number of synfire waves at upper limit determined by the critical level of background input.

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F-2

A game of pool, a game of tag: AMPA trafficking in the post-synaptic density

Fidel Santamaria[1], George J. Augustine[2], Sridhar Raghavachari[2]

[1] Biology Department/ Neurosciences Institute, University of Texas at San Antonio, TX 78249 USA
[2] Department of Neurobiology, Duke University Medical Center, P.O. Box 3209, Durham, NC, 27710
E-mail: fidel.santamaria@utsa.edu

One mechanism of information storage in neurons is the plasticity in the strength of synaptic contacts. The strength of an excitatory synapse is partially due to the concentration of a particular type of ionotropic glutamate receptor (AMPAR) in the post-synaptic density (PSD). AMPAR concentration in the PSD has to be plastic, to allow the storage of new memories; but it also has to be stable to preserve important information. Although much is known about the molecular identity of synapses, the biophysical mechanisms by which AMPAR can enter, leave and remain in the synapse are unclear.

We implemented Monte Carlo simulations to determine the influence of PSD structure and activity in maintaining homeostatic concentrations of AMPARs in the synapse. The model consisted of tracking the movement of individual AMPAR in and around a PSD. The membrane was simulated as a square mesh in which each position could be present a diffusing AMPAR, and anchored PSD molecule, or be empty. PSD molecules could be active, in which case, they could bind with a certain binding energy to AMPAR. If PSD molecules were inactive they acted as obstacles [1, 2].

We found that, the high concentration and excluded volume caused by PSD molecules result in molecular crowding. Diffusion of AMPAR in the PSD under such conditions is anomalous. Anomalous diffusion due to molecular crowding is a physical process fundamentally different from the steady-state excluded volume analyses such as tortuosity [3]. Anomalous diffusion of AMPAR results in retention of these receptors inside the PSD for periods ranging from seconds to several hours in the absence of any binding of receptors to PSD molecules. Our simulations were capable of reproducing several recent experimental results [4, 5]. Trapping of receptors in the PSD by crowding effects was very sensitive to the concentration of PSD molecules, showing a switch-like behavior for retention of receptors. Non-covalent binding of AMPAR to anchored PSD molecules allowed the synapse to become well-mixed, resulting in normal diffusion of AMPAR. Binding also allowed the exchange of receptors in and out of the PSD.

We propose that molecular crowding is an important mechanism to maintain homeostatic synaptic concentrations of AMPARs in the PSD without the need of energetically expensive biochemical reactions. Binding of AMPAR with PSD molecules could collaborate with crowding to maintain synaptic homeostasis but could also allows synaptic plasticity by increasing the exchange of these receptors with the surrounding extra-synaptic membrane.

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F-3

Coding signal strength by correlated activity in bursting neurons

Oscar Ávila Åkerberg[1], Maurice J Chacron[1,2]

[1] Department of Physics, McGill University, Montreal, Quebec, Canada, H3A2T8
[2] Department of Physiology, McGill University, Montreal, Quebec, Canada, H3G1Y6
E-mail: oscar@cnd.mcgill.ca

Understanding how populations of neurons encode sensory information is of critical importance [1]. Correlations between the activities of neurons are ubiquitous in the central nervous system and, although their implications for encoding and decoding of sensory information has been the subject of arduous debates, there is a general consensus that their effects can be significant [2]. As such, there is great interest in understanding how correlated activity can be regulated. Recent experimental evidence has shown that correlated activity amongst pyramidal cells within the electrosensory lateral line lobe (ELL) of weakly electric fish can be regulated based on the behavioral context: these cells modulate their correlated activity depending on whether the fish is performing electrolocation or communication tasks without changing the mean firing rate of their response [3]. Moreover, it was shown in the same study that the changes in correlated activity were correlated with changes in bursting dynamics.

In this work we explore the role of intrinsic bursting dynamics on the correlated activity of ELL pyramidal

neurons. We use a combination of mathematical modeling as well as *in vivo* and *in vitro* electrophysiology to show that bursting dynamics can significantly alter the ability of neuronal populations to be correlated by common input. In particular, our model predicts that the ratio of output to input correlations (i.e. the correlation susceptibility [4]) is largely independent of stimulus amplitude when neurons are in the tonic firing model. In contrast, we find that the correlation susceptibility increases with stimulus amplitude when the neurons are in the bursting mode (Fig. 1). We then performed *in vivo* and *in vitro* experiments to verify this prediction. Our results show that intrinsic dynamics have important consequences on correlated activity and have further revealed a potential coding mechanism for stimulus amplitude through correlated activity.

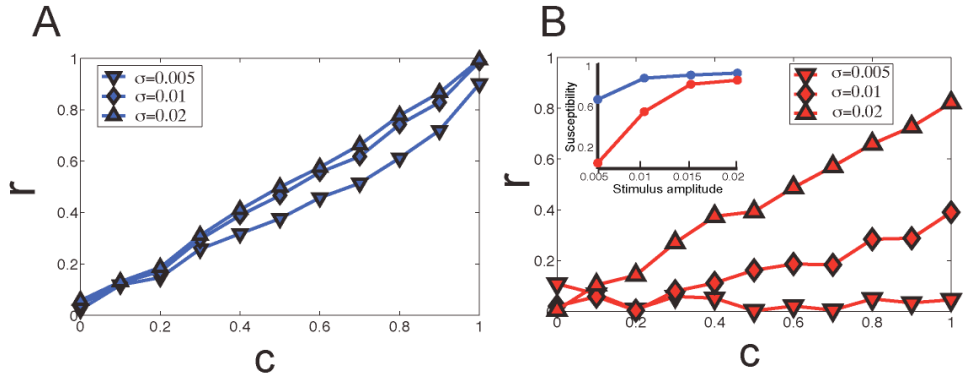

Figure 1. Modeling results: Output correlation coefficient, r, as a function of input correlation, c, for three different stimulus amplitudes in tonic firing neurons A and bursting neurons B. As shown in the inset the correlation susceptibility (i.e. the ratio of output correlation r to input correlation c) is roughly independent of stimulus amplitude σ when the neurons are in the tonic firing mode but increases with stimulus amplitude when the neurons are in the bursting firing mode.

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CONTRIBUTED ORALS

O-01

Firing pattern regulation in hypothalamic vasopressin neurons: roles of synaptic inputs and retrograde signaling

Alexander O. Komendantov[1], Ion R. Popescu[2], Jeffrey G. Tasker[2,3]

[1] Krasnow Institute for Advanced Study, George Mason University, Fairfax, VA, 22030, USA
[2] Department of Cell and Molecular Biology, Tulane University, New Orleans, LA 70118, USA
[3] Neuroscience Program, Tulane University, New Orleans, LA 70118, USA
E-mail: akomenda@gmu.edu

Magnocellular neurosecretory cells (MNCs) of the hypothalamus release the hormones oxytocin (OT) and vasopressin (VP) into the blood. These cells demonstrate enhancement of hormone release with bursting patterns of electrical activity. OT neurons fire synchronized bursts at long intervals during parturition and milk ejection; VP neurons generate an asynchronous phasic bursting in response to osmotic and cardiovascular stimuli. The mechanisms of bursting activity in VP are not known completely and are believed to be different *in vitro* and *in vivo*. Whereas *in vitro,* phasic bursting in VP neurons appears to be governed by intrinsic deterministic mechanisms, *in vivo* burst generation and termination significantly depends on synaptic activity. Mounting evidences suggest that retrograde signaling *via* endocannabinoids (eCBs) plays a prominent role in modulating MNC synaptic activity [1]. Our recent experiments suggest that bursts of action potentials are capable of suppressing glutamatergic input in VP neurons. We also found that blocking eCB receptors increased burst duration and intra-burst action potential frequency, consistent with a potential role in burst termination.

To investigate theoretically the role of synaptic inputs in the phasic bursting activity in VP neurons, we used an updated multicompartmental model of the MNC [2]. The model takes into account MNC morphology and electrotonic properties and includes a set of realistic voltage-gated and Ca^{2+}-activated ion currents, compartmental Ca^{2+} dynamics and reproduces several of the hallmark characteristics of MNC electrophysiological properties. Phasic bursting in the model is controlled by both intrinsic and synaptic mechanisms: bursts of action potentials arise from the summation of slow depolarizing afterpotentials superimposed on a tonic background activation of glutamatergic synaptic inputs; activity-dependent release of a retrograde messenger (eCB) from the dendrites of VP neurons attenuates tonic glutamate release and leads to burst termination. Background synaptic activity was simulated as independent excitatory and inhibitory inputs mediated by AMPA and $GABA_A$ conductances. Our computational studies also suggest that $GABA_A$ receptor activation promotes burst firing patterns, and stochastic synaptic inputs play an important role in the modulation of phasic activity in VP neurons.

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O-02

Computational model explaining two types of bursting found in inspiratory pacemakers.

Natalia Toporikova[1,2], Robert Butera[1,2]

[1] Laboratory for Neuroengineering, Georgia Institute of Technology, Atlanta, GA 30332, USA
[2] School of Electrical and Computer Engineering, Georgia Institute of Technology, Atlanta, GA 30332, USA
E-mail: ntoporikova3@mail.gatech.edu

The respiratory rhythm is generated within the network of inspiratory neurons in the pre-Bötzinger complex (pBC) and persists under highly variable neuronal input. The mechanism of pBC rhythm generation at the level of the network is an active area of debate and inquiry. A fraction of these inspiratory pBC neurons generate a stable bursting rhythm even when pharmacologically isolated from the network and likely contribute to the rhythm. Experiments indicate that the intrinsic bursting mechanism of these pacemaker neurons depends on either persistent sodium current or changes in intracellular Ca^{2+}. Motivated

by experimental evidence obtained from these subpopulations of bursting neurons, we developed a two-compartment mathematical model of a pBC pacemaker neuron with two independent bursting mechanisms. The model explains a number of contradictory experimental results and is able to generate a robust bursting rhythm over a large range of parameters, with a frequency adjusted by neuromodulators.

For the somatic compartment of our model, we used a previously developed model of pBC pacemaker neurons [1]. In this model, action potentials are generated by a fast sodium current (I_{Na}) and a delayed rectifier potassium current (I_K), and the burst is terminated by slow inactivation of a persistent sodium current (I_{NaP}). The bursting in the dendritic compartment of our model follows the Ca^{2+} oscillations arising from periodic Ca^{2+} release from intracellular stores. Briefly, the activation of a G_q-protein cascade leads to an increase in the concentration of IP_3, which binds to its receptor on the surface of the endoplasmic reticulum and initiates Ca^{2+} influx into the cytosol. A calcium-activated nonspecific cation current (I_{CaN}) then depolarizes the cell membrane in response to the increase in intracellular Ca^{2+} concentration. This depolarizing potential spreads to the soma and activates action potential-generating currents (I_{Na} and I_K), thus initiating the burst. Finally, the action potential propagates to the dendrite, producing a dendritic burst of smaller amplitude.

The model predicts that in synaptically isolated cells, the bursting mechanism depends on neuromodulators, endogenously released within the pBC. The neuromodulatory tone can bias the neuron to a somatic (I_{NaP}) or dendritic (Ca^{2+} and I_{CaN}) mode of bursting, or a hybrid of the two. In the dendritic mode, the period of bursting is largely modulated by the IP_3 concentration, whereas in the somatic mode the burst duration is modulated by the persistent sodium current. This model displays changes in burst duration and period that are consistent with experimentally published pharmacological manipulations, such as the application of ion channel blockers (FFA and Riluzole) as well as neuromodulatory manipulations.

O-03
Coregulation of ionic currents maintaining the duty cycle of bursting

William Barnett[1], Martin Anquez[2], Gennady Cymbalyuk[2,3]
[1] Department of Biology, Georgia State University, Atlanta, Georgia 30303, USA
[2] Department of Physics and Astronomy, Georgia State University, Atlanta, Georgia 30303, USA
[3] Neuroscience Institute, Georgia State University, Atlanta, Georgia 30303, USA
E-mail: gcymbalyuk@gsu.edu

Central pattern generators (CPGs) are the oscillatory neuronal networks which control rhythmic movements of animals. Some CPGs keep the phase relationships between the neurons' oscillatory activities over a wide range of the cycle periods. Maintenance of the duty cycle of the bursting activity could be a key feature for a variety of dynamical mechanisms supporting phase constancy in oscillatory neuronal networks. It is a form of cellular homeostasis of neuronal activity. Among other currents, hyperpolarization-activated currents and potassium currents have been shown to be a ubiquitous target for modulation and homeostasis [1,2].

Here we present a novel mechanism of coregulation of currents which preserves duty cycle of bursting activity over a range of cycle periods. We develop a generic low-dimensional Hodgkin-Huxley type model stemming from a model of the leech heart interneuron under certain pharmacological conditions [3]. Application of Co^{2+} and 4-AP blocks Ca^{2+} currents, the synaptic currents and most of the K^+ currents. The model contains the slow potassium current (I_{K2}), the fast sodium current (I_{Na}). Our new model also includes the hyperpolarization activated current (I_h). Bifurcation theory allows us to make predictions concerning the temporal characteristics of the dynamics of bursting nearby the critical transitions between activities.

Shilnikov & Cymblayuk showed that the transition from bursting into tonic spiking (blue sky catastrophe) determines the dependence of the burst duration on the voltage of half-activation of I_{K2} (θ_{K2}) as one over square root of the parameter value[4]. Here we show that the half-activation potential of I_h (θ_h) controls the interburst interval as one over square root of the parameter value. We investigate the activity of the model to identify mechanisms of coregulation of I_{K2} and I_h maintaining the duty cycle. Bifurcation analysis of the model was performed using θ_{K2} and θ_h, as controlling parameters. We investigated the temporal characteristics of bursting activity. We identified a saddle node bifurcation for periodic orbits determining the blue sky catastrophe [4] and a saddle node bifurcation for stationary states (SNIC) [5]. We showed the temporal characteristics of bursting depend on the location of the bifurcation curves. By coordinating the two parameters, we were able to increase the period such that the burst duration and interburst interval

maintained constant proportion. The coregulation consists of a negative correlation of θ_{K2} and θ_h, which is steeper for the higher duty cycles.
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O-04

Neuronal bursting: interactions of the persistent sodium and CAN currents

Justin R. Dunmyre[1], Jonathan E. Rubin[2]

[1] Mathematics, University of Pittsburgh, Pittsburgh, PA, 15260, USA
[2] Mathematics and Center for the Neural Basis of Cognition, University of Pittsburgh, PA, 15260, USA
E-mail: jrd34@pitt.edu

The pre-Botzinger complex (pBC) is a heterogeneous neuronal network within the mammalian brainstem and has been experimentally found to generate robust, synchronous bursts [1]. Significant modeling research has been conducted on characterizing the dynamics of individual neurons within the pBC.[2,3] It is well known that the persistent sodium current (I_{NaP}) contributes to square-wave bursting seen in the pBC[4]. Recent experimental work within the pBC identified a signaling cascade that starts with presynaptic glutamate and ends with the release of intracellular calcium that activates a nonspecific cationic current (I_{CAN}) [5]. A subsequent model demonstrated that I_{CAN} may contribute to bursts within the pBC that exhibit depolarization block[6]. With these two mechanisms for generating bursts present within the pBC, an open question is how do they combine to generate the robust bursts seen in the network? The present work seeks to analyze the result of including both I_{NaP} and I_{CAN} within the same model. We consider the effects of heterogeneity in the conductance g_{NaP} of I_{NaP} and the conductance g_{CAN} of I_{CAN}; with this heterogeneity in mind, the model cell may be quiescent, tonically active, have only square-wave bursts, have only depolarization-block exhibiting bursts, or may show both types of bursting. Using the mathematical tools of bifurcation analysis and slow-fast decomposition, we illuminate the mechanisms underlying the transitions of a model cell between the types of dynamics listed above. Our results show that, in cases where g_{CAN} is relatively high, increasing g_{NaP} increases the range of g_{CAN} where the resultant cell has depolarization-block exhibiting bursts. On the other hand, when g_{CAN} is relatively low, increasing g_{NaP} may cause the cell to transition from quiescence, to square wave bursting, to tonic activity, to square wave bursts with high duty cycles, and finally further increase of g_{NaP} causes the cell to again be tonically active. The latter two transitions do not occur if I_{CAN} is absent. The interactions of I_{CAN} and I_{NaP} are relevant to many systems beyond the pBC. Individually, I_{CAN} and I_{NaP} have been focused on as important to rhythmic burst generation in other systems such as the entorhinal cortex [7]; however, it is likely that both currents are present in these systems. Thus, a detailed account for the interaction of I_{CAN} and I_{NaP} may help explain the rhythm generation encountered in other systems beyond the pBC.
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O-05

Spike Threshold Dynamics Shape the Response of Subthalamic Neurons to Cortical Input

Michael A Farries[1], Hitoshi Kita[2], Charles J Wilson[1]

[1] Department of Biology, University of Texas San Antonio, San Antonio, TX 78240, USA
[2] Department of Anatomy and Neurobiology, University of Tennessee Health Science Center, Memphis, TN 38163, USA
E-mail: michael.farries@utsa.edu

The subthalamic nucleus (STN) is a population of autonomously active glutamatergic neurons within the basal ganglia (BG) that innervates BG output nuclei and is reciprocally connected with the globus pallidus (GP). The STN receives cortical input and so forms a direct bridge to BG output nuclei that bypasses the striatum. The STN's response to cortical stimulation in vivo begins at very short latency (2-5 ms) and consists of two excitatory peaks divided by a brief period of inhibition. The brief inhibition is generally ascribed to disynaptic inhibition from the GP, but signs of cortically-evoked inhibition persist in some STN recordings made in rats with GP lesions. We investigated the contribution of intrinsic properties to the STN's response to cortical excitation by studying their response to cortical fiber stimulation in brain slices in the presence of GABAergic antagonists. Responses to relatively strong stimulation often exhibited two distinct excitatory peaks in the PSTH separated by a gap that resembles inhibition. The distribution of latencies to the first poststimulus spike could also exhibit this gap, so this effect cannot be attributed to the AHP of the first spike. We found that spikes fired shortly after the onset of large EPSPs were triggered at a substantially lowered threshold (2-7 mV). The threshold dropped rapidly (within 1-2 ms of EPSP onset) and rose quickly back to the baseline level (or to a slightly elevated threshold). This drop in threshold can explain the two peaks seen in PSTHs and latency distributions: the cell fires immediately when above the lowered threshold but must otherwise wait until reaching the higher baseline threshold if it misses the narrow low-threshold window. Thus, EPSP-evoked changes in spike threshold can both facilitate a rapid, short-latency response in the STN to strong cortical input and change the firing pattern evoked by that input. Smaller EPSPs advance the time of the next spike but evoke smaller changes in spike threshold that do not produce the appearance of an excitation-inhibition sequence. The change in firing pattern associated with large EPSPs could allow targets of STN projections to distinguish activity driven by sharp increases in cortical drive from autonomous or tonically-driven activity. A model of the STN's response to cortical input suggests that two modes of operation--a coincidence-detecting short latency response to sharp increases in excitation and a more subtle response to smaller fluctuations in synaptic drive--can coexist and operate in parallel.

CONTRIBUTED ORALS

O-06

Fine temporal structure of beta-band synchronization in Parkinson's disease: experiments, models and mechanisms

Leonid L Rubchinsky[1,2], Choongseok Park[1], Robert M Worth[3]

[1] Department of Mathematical Sciences and Center for Mathematical Biosciences
Indiana University Purdue University Indianapolis, Indianapolis, IN 46202, USA
[2] Stark Neurosciences Research Institute, Indiana University School of Medicine, Indianapolis, IN 46202, USA
[3] Department of Neurological Surgery, Indiana University School of Medicine, Indianapolis, IN 46202, USA
E-mail: leo@math.iupui.edu

Synchronous oscillatory activity in the beta frequency band is a characteristic feature of neuronal dynamics of basal ganglia in PD and is hypothesized to be related to disease's hypokinetic symptoms [1]. However the character and nature of this activity is very much opened question. The dynamics of these oscillations and their mechanisms are the subjects of this study. We simultaneously record spikes and LFP from subthalamic nucleus (STN) of patients with PD and analyze the phase locking between these signals as it develops in time in order to explore the variability of beta-band oscillations in BG. We then match the experimental results with the models of BG networks. STN LFP has mostly synaptic origin and (given the absence of lateral connections within STN) is representative of input signals to STN. The fragile temporal structure of this synchrony necessitates the use of time-sensitive data analysis methods, so we followed our earlier developments [2,3] to detect oscillatory episodes with synchronous dynamics.

To recover the fine temporal structure of these episodes we constructed the first-return maps for the difference of the phases of oscillations and quantified the transition between different regions of the maps. Thus we were able to identify the succession of synchronized and desynchronized periods within oscillatory episodes with overall tendency for synchrony. The synchronized dynamics is interrupted by essentially nonsynchronized periods. These desynchronization events are distributed in a specific way – there is a predominance of short desynchronization events. The signals go out of phase for just one cycle of oscillations more often than for two or a larger number of cycles. An alternative scenario (desynchronization events are longer but less frequent) would produce the same degree of average synchrony. However our results show that this alternative is not realized in the parkinsonian BG.

To understand the mechanisms of the observed synchronous activity, we developed conductance-based models for subthalamo-pallidal circuits (based on [4]). The model retains substantial amount of biological realism (both, membrane properties and anatomical organization of the inhibitory-excitatory network) and exhibits synchronous oscillatory patterns. We studied the model dynamics under the variation of parameters, which are modulated by dopamine and subjected the model output to the same data-analysis techniques, as we used for experimental data. Thus we identified the parameter domain, where the model dynamics and experimentally observed dynamics are similar in how the synchronized patterns evolve in time. This area is on the boundary of fully synchronous regime and is also characterized by a proximity to a non-synchronized (and presumably healthy) dynamics.

The realistic patterns arise in the model network, when synaptic projections (normally suppressed by dopamine) become stronger. The intermittent synchrony in the parkinsonian state may be a result of a propensity of BG circuits to be engaged in the brief synchronized episodes of activity needed for movement control. The low-dopamine state with stronger coupling may result in a partial suppression of this very transient (and hard-to-detect) character of neuronal dynamics, favoring only short desynchronization events, which interrupt mostly synchronous episodes.

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O-07

A computational approach to understanding the longitudinal changes in cortical activity associated with intensive meditation training

Manish Saggar[1], Stephen R. Aichele[2,3], Tonya. L. Jacobs[3], Anthony P. Zanesco[3], David A. Bridwell[4,3], Katherine A. Maclean[6,3], Brandon G. King[3], Baljinder K. Sahdra[2,3], Erika L. Rosenberg[3], Phillip R. Shaver[2], Emilio Ferrer[2], B. Alan Wallace[5], George R. Mangun[2,3], Clifford D. Saron[3], Risto Miikkulainen[1]

[1] Department of Computer Science, University of Texas at Austin, Austin, TX 78712 USA
[2] Department of Psychology, [3]Center for Mind and Brain, University of California-Davis, Davis, CA 95618 USA
[4] Department of Cognitive Science, Univ. of California-Irvine, Irvine, CA 92697 USA
[5] Santa Barbara Institute for Consciousness Studies, Santa Barbara, CA 93130 USA
[6] Department of Psychiatry and Behavioral Sciences, JHU School of Medicine, Baltimore, MD 21224 USA
E-mail: mishu@cs.utexas.edu

Meditation involves focusing one's attention on an object or a phenomenon. Numerous studies have discovered beneficial effects of meditation. However, very few scientific studies have attempted to formally understand the underlying mechanisms. In the present study, two 3-month retreats were conducted. 88-channel EEG data was collected while participants rested with their eyes closed before and after engaging in focused attention meditation. Second-order blind source separation was used for artifact removal. Reference-free data was obtained by estimating current source density. Changes in spectral power were assessed using a nonparametric cluster-based approach. Within-session (pre-meditation rest vs. meditation) results, in the alpha band, suggest that after 3-months there is a generalization of overall cortical activity during rest, such that the "default" mode of resting came to resemble the meditation mode. To formally understand the underlying mechanisms responsible for such a cortical change during rest, a computational model was employed. Attention regulation during focused meditation is hypothesized to involve cortico-thalamo-cortical interactions. Thus, to model EEG data during rest, the presented model architecture includes cortico-thalamic and thalamo-cortical interactions (Figure-1a). This model was initially developed by [1] and is capable of modeling both spatial and temporal dynamics of cortical activity using a 2-D continuum approach with inputs from sub-cortical structures. Three computational modeling experiments were run to examine – (1) if the model can fit the 88-channel EEG data, (2) if the inverse computational modeling can provide insights about the patterns observed on the scalp, and (3) if the stability analysis of model equations can explain for the observed cortical activity changes on the scalp. As shown in Figure-1b, the model-estimated spectrum is in accordance with the experimental spectrum without crossing physiologically plausible limits for the parameter set. Further, inverse computational modeling of the estimated spectral data indicates that after three months of training intrathalamic gain was significantly reduced in the right-parietal location (Figure-1c). This reduction suggests that participants after training were more alert even during rest. This sustained alertness during rest may be reflected as a change in the "default" mode after three months of training. Lastly, the stability analysis of model equations showed that reduction in the intrathalamic gain parameter provided more overall stability to the system. Altogether, these computational experiments along with the spectral analysis provide a more insightful formal theory of what might be happening inside the brain than does the spectral analysis alone.

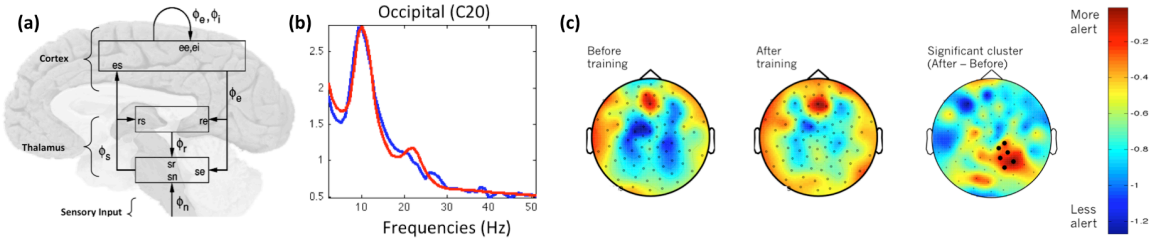

Figure 1(a) Model architecture for a single site (adapted from [1]). **(b)** Model estimated (in red) and experimental (in blue) spectra for a sample channel and subject. **(c)** Topographic map of the intrathalamic gain parameter before and after training. Reduction in intrathalamic gain has been linked to increase in alertness [2]. Thus, the model not only fits experimental data but also provides a concrete and testable hypothesis about the changes seen on the scalp.

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O-08

A computational model of associative learning and chemotaxis in the nematode worm *C. elegans*

Peter A. Appleby and Netta Cohen

School of Computing, University of Leeds, Leeds LS2 9JT, United Kingdom
E-mail: n.cohen@leeds.ac.uk

The nematode worm *C. elegans* is an exciting model system for experimentalists and modelers alike. It has a relatively small nervous system, made up of just 302 neurons in the adult hermaphrodite, that has been mapped in detail using serial section electron microscopy. Despite the simplicity of its nervous system *C. elegans* displays a range of interesting behaviors. This includes thermo- and chemotaxis and the modulation of locomotion strategies in response to the presence of food. In chemotaxis *C. elegans* will move up or down a chemical gradient dependent on whether the chemical acts as an attractant or repellent. It does this in two distinct ways, first by gradually steering left or right until the worm points up or down the gradient and second by modulating the probability of initiating a sharp series of turns (known as a piroeutte) along with the final orientation of the worm after the pirouette has finished. Experimental work has shown that the chemotaxis response is dynamic and that the degree of influence a particular chemical has on navigation can be changed, or even reversed depending on experience. Changes are reversible, specific to the chemical in question, and can be generated by classical conditioning experiments. All of these are hallmarks of associative learning, a sophisticated process that requires integration of multiple signals to produce a coordinated change in a behavioral response.

Despite the wealth of experimental data on learning in chemotaxis in *C. elegans*, comparatively little is known about how the known circuitry of *C. elegans* carries out the computations that underlie it. Even less is known about how that circuitry changes during associative learning. Here, we focus on the worm's chemotaxis and the ability of *C. elegans* to learn associations between salt (NaCl) concentrations and food. We draw upon existing experimental data from a variety of sources including electrophysiological and anatomical data to construct a simplified NaCl chemotaxis circuit in *C. elegans*. This circuit consists of the left and right ASE amphid sensory neurons, which comprise the dominant NaCl sensation pathway in *C. elegans*, eight pairs of interneurons, and ten head motor neurons. Where possible the properties of individual neurons are constrained by electrophysiological and calcium imaging data.

We next define a set of experimentally observed behaviors we wish to reproduce, including gentle turning, modulation of pirouette frequency, control of final orientation following a pirouette, and associative learning. In particular, we are interested in the alteration in behavioral response to NaCl that arises due to the pairing of high concentrations of NaCl with food or starvation. We use this to derive a family of model networks with specific synaptic polarities, time scales of neuronal responses, and intrinsic neuronal properties that have the capacity to generate the specified set of behaviors. We implement one of these models and record the behaviour of model worms that are placed in a variety of simulated environments. We observe qualitatively realistic chemotaxis behavior and adaptation and demonstrate that our model is robust and tolerant to noise.

Our proposed chemotaxis circuit leads to a number of distinct predictions that could be used to test the model experimentally. This includes postulating the computational role of each neuron in the network and the locus and nature of the plasticity underpinning the experimentally observed associative learning. Our model also suggests that this plasticity be expressed not by changes in synaptic strength but by changes in the sensory neurons themselves. Thus, contrary to the prevailing view in the *C. elegans* community, plasticity in our model of chemotaxis is expressed at a neuronal rather than synaptic level.

C. elegans offers a unique opportunity to push the boundaries of systems neuroscience. The ability to model a neuronal circuit in such detail is a remarkable opportunity to study associative learning in an animal displaying a sophisticated set of behaviors and nontrivial learning. A biologically grounded model of behavior and learning in *C. elegans* has great potential to offer detailed and integrated understanding of sensory processing, synaptic plasticity and associative learning. Lessons learned from such models can be applied

to other sensory and sensorimotor modalities in the worm with the eventual goal of producing an integrated model of the worm's sensorimotor system. We believe that theoretical insights gained from this endeavour will be invaluable in our study of larger, more complex nervous systems.

O-09

Tracking Neuronal Dynamics During Seizures

Ghanim Ullah[1], Steven J Schiff[1,2]

[1] Center for Neural Engineering, Department of Engineering Science and Mechanics, Pennsylvania State University, University Park, PA 16802, USA

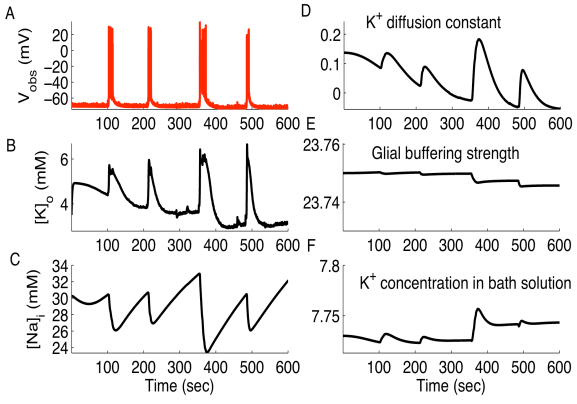
[2] Departments of Neurosurgery and Physics, Pennsylvania State University, University Park, PA 16802, USA

E-mail: ghanim@psu.edu

We demonstrate that a meaningful estimation of the neuronal network dynamics from single measured variable is now possible by incorporating biophysical neuronal models into a model-based control framework. Specifically, we assimilate noisy membrane potential (V) measurements from individual hippocampal neurons to reconstruct the dynamics of networks of these cells, their extracellular microenvironment, and the activities of different neuronal types during seizures.

We used two-compartmental models for the pyramidal cells (PCs) and interneurons (INs): a cellular compartment and the surrounding extracellular microenvironment. The membrane potentials of both cells were modeled by Hodgkin-Huxley equations containing sodium, potassium, calcium-gated potassium, and leak currents. The current equations were augmented with dynamic variables representing the intra- and extracellular ion concentrations (K^+, Na^+, and Ca^{2+}). To estimate and track the dynamics of the neuronal networks, we applied a nonlinear ensemble version of the Kalman filter, the Unscented Kalman Filter (UKF) [1]. Details of the model and UKF implementation can be found in [2,3].

In Fig.1 we show an example where we use V measurements to estimate the rest of the dynamics of CA1 PC. Fig.1A shows an intracellular recording from a PC during seizures, and plot the estimated $[K]_o$ in Fig.1B. As is clear from the figure the $[K]_o$ oscillates as the cell goes into and out of seizures. The $[K]_o$ begins to rise as the cell enters seizures and peaks with the maximal firing frequency, followed by decreasing $[K]_o$ as the firing rate decreases and the seizure terminates. Higher $[K]_o$ makes the PC more excitable by raising the reversal potential for K^+ currents. The increased K^+ reversal potential causes the cell to burst fire spontaneously. Changes in the $[Na]_i$ are closely coupled with the changes of $[K]_o$ (Fig.1C). As shown in panels (1D-F) wereconstructed the parameters controlling the microenvironment of the cell. These parameters included the diffusion constant of K^+ in the extracellular space, K^+ buffering strength of glia, and K^+ concentration in the reservoir of the perfusing solution *in vitro* (or in the vasculature *in vivo*) during seizures.



In conclusion, we demonstrated that estimating the neuronal microenvironment and neuronal interactions can be performed by embedding our improving neuronal models within a model-based state estimation framework. This approach can provide a more complete understanding of otherwise incompletely observed neuronal dynamics during normal and pathological brain function.

Figure 1: Assimilating spontaneous seizure data by whole cell recording from CA1 hippocampal pyramidal neurons. (A) Measured V (red) from single PC during spontaneous seizures. Estimated (black) [K]o (B), [Na]i (C), K+ diffusion constant (D), glial buffering strength (E), and K+ concentration in bath solution (F).

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O-10

Dynamical effects of antiepileptic drugs on neurons affect network synchronizability

Theoden_Netoff[1], Bryce Beverlin II[2]

[1] Biomedical Engineering, University of Minnesota, Minneapolis, MN 55455, USA
[2] Physics, University of Minnesota, Minneapolis, MN 55455, USA
E-mail: tnetoff@umn.edu

Epilepsy is characterized by periods of excessive neuronal activity called seizures. While much is known about population behaviors of neurons during seizures, as measured by EEG electrodes, very little is known about the activity at the cellular level. The etiology of the disease can often be traced to specific mutations in particular ion channels [1]. These same ion channels are often the targets of antiepileptic drugs. Bridging the molecular scale causes and treatment of epilepsy to the network scale phenotype is a multi-scale problem that needs to be solved in order to develop more rational approaches to treating epilepsy.

Our research seeks to understand the basic mechanisms of epilepsy by understanding how network synchrony is affected by molecular level changes caused by epileptogenic mutations and antiepileptic drugs. Our approach is guided by experimental evidence, in a rat model of epilepsy, indicating that synchrony in the network changes over the different phases of the seizure [2]. Changes in synchrony may hold a key to understanding the causes and developing novel treatments for epilepsy. However, why synchrony changes during a seizure is still a mystery.

To better understand how neurons synchronize, we use pulse coupled oscillator theory [3]. The dynamics of the neuron are reduced to a simple input-output relationship, measuring how synaptic inputs applied at different phases of a periodically firing neuron advances or delays the spike, resulting in a Phase-Response Curve (PRC). From the measured PRC, it is possible to predict how a network of neurons will synchronize [4, 5]. We then measure how epileptogenic mutations and antiepileptic drugs affect the neuron's PRC to infer how it changes the synchronizability of the network. By measuring the effects of these changes at the molecular level we know causes epilepsy, we can bridge the effect to a population.

Computational simulations and *in vitro* experiments measuring PRCs from neurons will be presented. We find that epileptogenic mutations in voltage gated sodium channels and potassium channels affect the neurons' PRCs to increase network synchrony while antiepileptic drugs decrease synchrony. We hypothesize that while many antiepileptic drugs have very different mechanisms of action, their common feature may be that they decrease network synchrony.

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CONTRIBUTED ORALS

O-11

Decorrelation of low-frequency neural activity by inhibitory feedback

Tom_Tetzlaff[1], Moritz Helias[2], Gaute T. Einevoll[1], Markus Diesmann[2,3]

[1] Department of Mathematical Sciences and Technology, Norwegian University of Life Sciences, Ås, Norway
[2] RIKEN Brain Science Institute, Wako City, Japan
[3] Brain and Neural Systems Team, RIKEN Computational Science Research Program, Wako City, Japan
E-mail: tom.tetzlaff@umb.no

To correctly judge the functional role of cooperative neural activity it is essential to understand how neural correlations are determined by the structure and dynamics of neural networks. Shared presynaptic input is one of the major sources of correlated synaptic activity in such systems. In the asynchronous state of recurrent neural network models, however, spike correlations are considerably smaller than what one would expect based on the amount of shared presynaptic sources [1,2]. A similar lack of correlations in the spiking activity of neighbouring cortical neurons has been observed experimentally [3]. Recently, it has been pointed out that shared-input correlations can be actively suppressed by the dynamics of recurrent networks [4]. Here, we show that both in networks with purely inhibitory coupling (Fig.A) and in those with mixed excitatory-inhibitory coupling (Fig.B) this active decorrelation affects mainly the activity at low frequencies (<20 Hz). High-frequency activity, in contrast, is rather unaffected. Simulations rule out that this phenomenon is the result of refractoriness. By means of a simple linear population-rate model we demonstrate that the effect is essentially explained by inhibitory feedback.

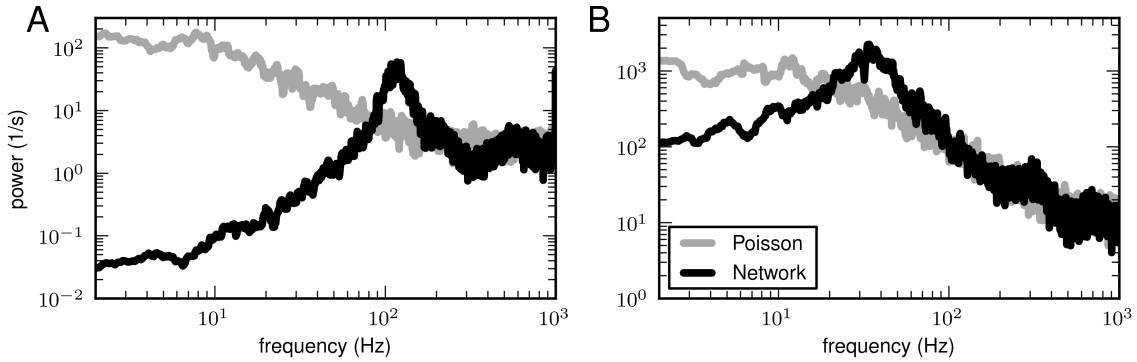

Figure 1: Population-rate power spectra for an inhibitory (**A**) and a balanced recurrent network (**B**) of leaky integrate-and-fire model neurons (black curves). Grey curves represent population-rate spectra of ensembles of unconnected neurons receiving stationary Poisson input spike trains with the same shared-input structure as in the respective recurrent cases (black curves).

Acknowledgements
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CONTRIBUTED ORALS

O-12

Phase response analysis during *in vivo*-like high conductance states; Dendritic SK determines the mean & variance of responses to dendritic excitation

Nathan W. Schultheiss[1], Jeremy R. Edgerton[2], & Dieter Jaeger[2]

[1] Department of Psychology, Boston University, Boston, MA 02215, USA
[2] Department of Biology, Emory University, Atlanta, GA 30322, USA
Email: nschult@bu.edu

A neuron's phase response curve (PRC) describes how synaptic inputs at different times during the spike cycle affect the timing of subsequent spikes, and PRC analysis is a powerful technique for predicting and interpreting the emergence of synchronous modes in synaptically coupled networks and neuronal populations receiving common input. However, neuronal PRCs are typically measured during intrinsic pacemaking which may not reflect neuronal excitability and dynamics during high conductance states generated by complex network activity *in vivo*. Using a full morphological model of a globus pallidus (GP) neuron we have recently demonstrated that during intrinsic pacemaking, somatic PRCs for GP neurons are type I, i.e. excitatory inputs at all phases of the spike cycle advance the spontaneous spiking rhythm[1]. We also demonstrated that synaptic *excitation* of the distal dendrite can paradoxically *delay* subsequent spiking when delivered at some phases of the spike cycle (yielding a type II PRC) as a consequence of dendritic activation of the small conductance calcium-activated potassium current, SK[1]. Since during high conductance states spike timing is determined by a balance between intrinsic mechanisms and synaptic input fluctuations, in this study we investigated how somatic and dendritic phase response properties of the GP model are affected by ongoing stochastic synaptic background activity.

We generated high conductance states in the model by applying synaptic backgrounds composed of randomly-timed excitatory and inhibitory synaptic inputs at 1022 GABA synapses and 100 AMPA synapses distributed throughout the dendrite. By varying the synaptic gain and input frequency parameters across the physiological range for inputs to GP, we achieved a diverse set of high conductance states characterized by sub-threshold voltage fluctuations, irregular spiking, and elevated membrane conductance (Figure 1A&B) and spanning the range of spike frequencies observed *in vivo* (1545 Hz). For each synaptic background parameter set, we generated 100 single-trial PRCs by delivering a single 2.5 nS AMPA-synaptic input to either the soma or distal dendrite at each of 72 time-points (in separate simulations) within the first spike cycle of each of 100 control spike trains (Figure 1C&D). We then averaged the single-trial PRCs for each synaptic background to evaluate the dependence of PRC shape on stimulus location, spike frequency, and dendritic SK conductance, in addition to the gain and input frequencies of synaptic backgrounds. Next, we analyzed on a trial-by-trial basis the interactions of PRC stimuli with transient fluctuations in the synaptic background leading to added or skipped spikes. We determined that dendritic SK underlies both the incidence of skipped spikes and the dependence of skipped spike events on stimulus phase. Interestingly, the input-phase dependence of skipped spike events mirrored the phase-dependent variance in the PRC itself (which we plotted as phase response-variance curves, PRVCs). This indicates that dendritic SK determines not only the mean response, but also the variance of responses, to excitatory dendritic inputs. Average somatic and dendritic PRCs were type I and type II, respectively, and are likely to represent the average behavior of populations of GP neurons in response to shared excitation. Furthermore, in-as-much as the GP model and simulated synaptic backgrounds are physiologically realistic, the variance in the model's responses across trials directly reflects a major source of spike time variance across populations of GP neurons *in vivo*.

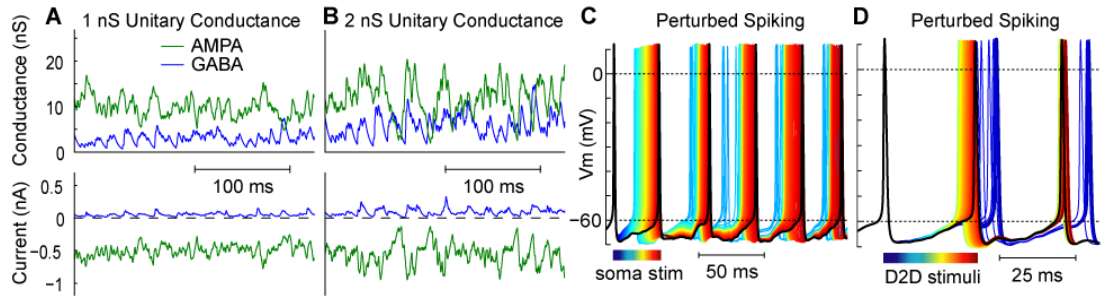

Figure 1. A&B. Total synaptic conductance (top) and current (bottom) for synaptic backgrounds composed of 1 nS (**A**) or 2 nS (**B**) unitary inputs. **C&D.** Perturbations of control spike trains (black) by somatic (**C**) or dendritic (D2D)(**D**) stimuli. (Trace color indicates input phase.)

CONTRIBUTED ORALS

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O-13

Role of active dendritic conductances in subthreshold input integration

Michiel Remme[1], John Rinzel[1,2]

[1] Center for Neural Science, New York University, New York, NY 10003, USA
[2] Courant Institute of Mathematical Sciences, New York University, New York, NY 10003, USA
E-mail: michiel.remme@nyu.edu

A large body of data has demonstrated the presence of voltage-dependent conductances in the dendrites of many types of neurons. A subset of these conductances are active at subthreshold membrane potentials and can greatly affect the integration of synaptic inputs. To understand the computations neurons perform it is key to understand the role of active dendrites in subthreshold input processing. Here, we examine how active dendritic conductances affect postsynaptic potentials that propagate along dendrites and the interaction between such signals. We focus in particular on coincidence detection, one of the most basic operations a neuron can perform, in which a neuron needs to detect the occurrence of two or more EPSPs in a short time interval (e.g. down to ~10 μs in auditory brainstem).

To systematically study the effects of active dendritic conductances on synaptic inputs, we make use of the so-called quasi-active description of dendritic cables [1], an extension of classical passive cable theory, which relies on linearizing the voltage-dependent conductances. Though the linearized description does not capture the full dynamics of the active currents, the results can serve as a solid reference for the effects of active dendritic currents on propagating EPSPs. This approach allows us to categorize active dendritic currents into two types: restorative currents (e.g. I_h), which function as a negative feedback and counteract changes of the membrane potential, and regenerative currents (e.g. I_{NaP}), which act as a positive feedback and amplify membrane potential changes.

The two types have qualitatively different effects on subthreshold EPSP propagation and interaction. Compared to a passive cable's response, the EPSP halfwidth is decreased by restorative currents and increased by regenerative currents. Moreover, these effects increase as the EPSP propagates along the active cable. Interestingly, restorative dendritic currents can maintain a constant EPSP halfwidth or even gradually narrow the EPSP as it moves along a dendrite. We find there is an optimal activation time constant of the active dendritic currents to exert their maximal effect on the EPSP halfwidth. Finally, since narrow EPSPs will only summate on short intervals, coincidence detection of dendritic inputs is enhanced by restorative currents: the coincidence window is narrower and is less dependent on the exact locations of the inputs along the dendrite. Conversely, coincidence detection is less precise when the dendrites have regenerative currents, which cause a wider coincidence window and stronger dependence of the window on input locations.

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O-14

Analysis of the mechanisms underlying windup using a detailed biophysical model of WDR neurons

Paulo_Aguiar[1,2], Mafalda Sousa[2], Deolinda Lima[2,3]

[1] CMUP – Centro de Matemática da Universidade do Porto, Porto, Portugal
[2] IBMC – Instituto de Biologia Molecular e Celular , Porto, Portugal
[3] Faculdade de Medicina da Universidade do Porto, Portugal
E-mail: pauloaguiar@fc.up.pt

Windup is characterized as a frequency-dependent increase in the number of evoked action potentials in dorsal horn neurons, in response to electrical stimulation of afferent C-fibers. This phenomenon was first described in the mid-sixties but the core mechanisms behind it still remain elusive. Several factors affecting its dynamics have been identified but the distinction between modulating mechanisms from generating mechanisms is not always clear. Several mechanisms contribute to the excitation of dorsal horn neurons exhibiting windup and one of our main aims was to help making this distinction. The approach presented here relies on mathematical and computational analysis to study the mechanism(s) underlying windup. To

our knowledge, this is the first biophysical model addressing the windup mechanism, and one of the few existing models for information processing in the spinal cord' dorsal horn. From experimentally obtained windup profiles, we extract the time-scale of the facilitation mechanisms which are capable of supporting the characteristics of windup. Guided by these values and using simulations of a biologically realistic compartmental model of a wide dynamic range (WDR) neuron, we are able to assess the contribution of each mechanism for the generation of action potentials windup. We show that the key mechanisms giving rise to windup is the temporal summation of AMPA and NMDA long-lasting post-synaptic responses taking place on top of a membrane potential cumulative depolarization. Calcium activated non-specific cationic currents driven by calcium influx from L-type calcium channels and synaptic currents support this cumulative depolarization and plateau formation in WDR neuron membrane potential. The effects of different non-homogeneous stimulation protocols are explored and their important role in clarifying many aspects of the windup generation is demonstrated. The models were constructed in the well established simulation environment NEURON and are used to produce several predictions which can be tested experimentally.
Acknowledgements: The authors would to thank Pascal Fossat and Frédéric Nagy for providing the nociceptive flexion reflex data used in the regression analysis. The first author was supported by the Centro de Matemática da Universidade do Porto, financed by FCT through the programmes POCTI and POSI, with Portuguese and European Community structural funds, and by a FCT Grant PTDC/SAU-NEU/68929/2006

O-15

Selecting appropriate surrogate methods for spike correlation analysis

Sonja Grün[1], Christian Borgelt[2], George Gerstein[3], Sebastien Louis[1], Markus Diesmann[1,4]

[1] RIKEN Brain Science Institute, Wako-Shi, Japan
[2] European Centre for Soft Computing, Mieres (Asturias), Spain
[3] Department of Neuroscience, University of Pennsylvania, USA
[4] RIKEN Computational Science Research Program, Wako-shi, Japan
E-mail: gruen@brain.riken.jp

In the correlation analysis of experimentally recorded parallel spike trains one has to thoroughly consider the statistical features of the data in order to prevent false positive results [1]. Typically, the complexity of the data prevents us from using analytical expressions for evaluating the significance of observed correlations. Similarly, parametric tests presuppose models that are typically simplifications of the real neuronal data and thus may ignore important features. An alternative to these approaches is to use surrogate data, i.e. modified versions of the original data, to assess the significance [2]. The goal of this study is to develop selection criteria for suitable surrogate types.

To study the applicability of surrogates we defined data sets exhibiting different statistical features found in typical experimental data (non-stationary firing rates, cross-trial non-stationary rates, deviation from Poisson) in combinations of increasing complexity. To demonstrate the impact of surrogate schemes on correlation analysis, we examine these with different surrogate generation methods commonly used in the literature [1]. Common to all these methods is that they in one way or the other destroy the precise temporal relation of the spiking activities between the neurons, by e.g. shuffling the trial ids (tr-shu) [3], randomizing the spike times (sp-rnd), randomly dithering the whole spike train against the other (tr-di) [4,5], dithering of individual spike times (sp-di) [6,7], dithering spike times under conservation of the joint-ISI distribution (jisi-di) [8], or by exchanging spikes across trials under local preservation of spike counts (sp-exg) [9,10].

To quantify the applicability of the various surrogates for significance estimation of spike correlation we concentrate on spike coincidences (allowed temporal precision: +/-1ms) and use their empirical count *n*emp as a test statistic. The p-value of *n*emp is obtained by comparing it to the surrogates' coincidence count distributions. To evaluate the true performance of the surrogates we study the false positive (FP) and false negative (FN) rates for different configurations of parameters implemented in simulated data (rate modulation, regularity, non-stationarity across trials, co-variation of rates).

Based on the FN and FP performances, we find spike train dithering (tr-di) as the most robust detector of excess coincidences amongst the selected surrogates methods. Its detection accuracy is seemingly unaffected by the level of complexity of the data and its sensitivity remains at acceptable levels. Still, tr-di smooths the firing rate profile on the time scale of the dither width, and it is expected to produce false positives is the case of abrupt transients in firing rate. With the aim of dealing with this issue, further work is being done on the development of novel methods taking into account the observed firing rate profile. Doing

so enables an approximate mapping of non-stationary processes to stationary ones, through which more accurate surrogates can be generated.

This study illustrates the serious need to select appropriate surrogate methods when evaluating the significance of correlations observed in a given data set. Not doing so can lead to false conclusions and misinterpretation of the data. We therefore strongly recommend to test the chosen method on synthetic data which is as similar as possible to the experimental data at hand, but yet does not contain the feature being tested for, before proceeding with the analysis to control for false positive results [11].

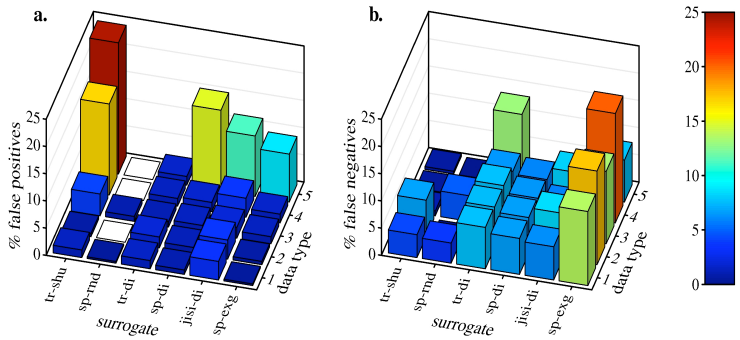

Figure 1: False positive (a.) and false negative (b.) percentages for all tested surrogate methods across five different data types. Colors code FP and FN percentages. White squares mark the position of bars of 100% FP.

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O-16

Feedback Control of the Spatiotemporal Firing Pattern of a Basal Ganglia Microcircuit Model

Jianbo Liu[1], Karim G. Oweiss[1,2], Hassan K. Khalil[1]

[1] Department of Electrical and Computer Engineering, Michigan State University, East Lansing, Michigan, U.S.A.
[2] Neuroscience Program, Michigan State University, East Lansing, Michigan, U.S.A.
E-mail: jliu@egr.msu.edu

One of the fundamental objectives in systems neuroscience is to precisely control the spatiotemporal firing patterns of cortical neurons to elicit a desired pattern of activity. In this work, we study the effects of intracortical micro-stimulation on the dynamics of a basal ganglia microcircuit model, and explore the feasibility of controlling the spatiotemporal firing patterns of the modeled population in the presence of unobserved inputs. Results from the simulation study suggest that properly designed Multiple-Input-Multiple-Output (MIMO) feedback control paradigm can force a subpopulation of observed output neurons to follow a prescribed spatiotemporal firing pattern despite the presence of unobserved inputs. The accuracy of the spike timing of the controlled neural firing with respect to the reference spike trains is in the order of tens of milliseconds.

CONTRIBUTED ORALS

O-17

Stimulus-dependent suppression of intrinsic variability in recurrent neural networks

Kanaka Rajan[1], Laurence F Abbott[2], Haim Sompolinsky[3]
[1] Lewis-Sigler Institute for Integrative Genomics, Princeton University, Princeton, NJ, USA
[2] Department of Neuroscience, Columbia University, New York, NY USA
[3] Racah Institute of Physics, Interdisciplinary Center for Neural Computation, Hebrew University, Israel
E-mail: krajan@princeton.edu

Trial-to-trial variability is an essential feature of neural responses and is likely to arise from a complex interaction between stimulus-evoked activity and ongoing spontaneous neural activity in the central nervous system. Response variability is often treated as random noise generated either by an external source like another brain area, or by stochastic processes within the circuit. A considerable amount of variability can also arise from the same circuitry and intrinsic network dynamics that generate responses to a stimulus. Indeed ongoing neural activity in the central nervous system is comparable in magnitude and complexity to activity evoked by sensory stimuli [1, 2].

How can we distinguish between external and internal sources of neuronal variability? We ask whether internal and external sources of variability depend on stimulus features in different ways, giving them distinct experimental signatures and functional interpretations. How are stimulus-evoked responses faithfully extracted from complex background activity to identify real features of the external world?

We use a neural network model that generates highly irregular and chaotic patterns of activity in the absence of stochastic input. On the basis of numerical simulations and mean-field calculations [1], we find a phase transition between two basic dynamic behaviors: a periodic state where the network is locked in phase and frequency to the external stimulus, and a chaotic state where neurons behave as noisy oscillators with only partial entrainment to the stimulus (Figure 1). We construct phase diagrams showing how these dynamics depend on the strength and frequency of the external input, the strength of the connectivity, and the residual imbalance between excitation and inhibition. We argue that sensory-evoked responses can actively suppress ongoing intrinsically generated fluctuations. This provides a theoretical basis and potential mechanism for the experimental observation that intrinsic neuronal variability is reduced by the presence of a stimulus [1-4].

We also show that the nonlinear interaction between the relatively slow intrinsic fluctuations and external stimulus results in a non-monotonic frequency dependence of this suppression. Consequently, measures of trial-to-trial variability of neural responses can be more sensitive to the amplitude and frequency of the stimulus, compared to the mean responses that are typically the focus of electrophysiological studies.

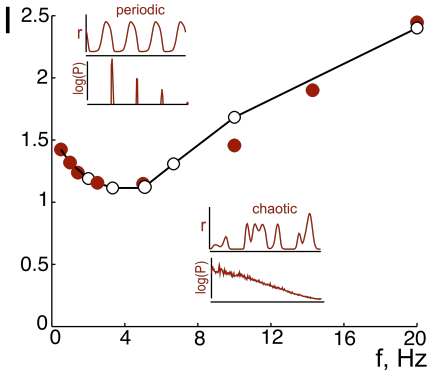

Figure 1. A phase transition curve showing critical input amplitudes that divide regions of periodic and chaotic activity as a function of input frequency, computed by mean-field theory (open circles) and by simulating a 10,000-neuron network (red circles). There is a resonant frequency at which it is possible for a periodic input to entrain the network by suppressing intrinsic chaos even though there are no resonant features apparent in the spontaneous activity. Inset traces show representative firing rates for the regions indicated along with the logarithm of the power spectrum of the activity across the network.

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CONTRIBUTED ORALS

O-18

Causal networks in the rat barrel cortex provide a signature of stimulus encoding

Seif Eldawlatly[1] and Karim Oweiss[1,2]

[1] Electrical and Computer Engineering Dept., Michigan State University, East Lansing, MI 48824, USA
[2] Neuroscience Program, Michigan State University, East Lansing, MI 48824, USA
E-mail: eldawlat@egr.msu.edu

Identifying stimulus-dependent cortical circuits is a fundamental goal in systems neuroscience. Graph-theoretic techniques provide an elegant tool to infer such circuitry. Recently, we have proposed to use Dynamic Bayesian Networks (DBN) as an efficient graphical model that explains the observed spike train data in local cortical circuits. A unique advantage of DBNs over other metrics of neuronal connectivity (such as crosscorrelograms) is its ability to explain the temporally precise activity of an observed neuron taking into account the activity of the entire observed population.

We recorded simultaneously the ensemble neural activity using 32-channel microelectrode arrays from Layer V of the primary somatosensory (barrel) cortex in 3 anesthetized rats. An average population size of 20±7 single units/rat was identified. Responses were recorded during stimulation of individual whiskers (3 whiskers/rat). Each whisker was horizontally deflected with a displacement of 80 μm for 100 ms at 1 Hz frequency. Spike trains within 100 ms of stimulus onset were analyzed using DBN. To rule out the existence of spurious connections inferred by co-modulation of firing rates, a jittered version of the spike trains was formed in which each spike was randomly jittered in a uniform interval of [-5,5] ms. Common connections inferred in the original and jittered data were eliminated.

Results demonstrate that stable stimulus-driven network states were obtained, with 22% of the inferred connections appearing in more than 35% of the trials. To examine the validity of these results, we computed the correlation between the response latency of each neuron and the number of inferred pre-synaptic/post-synaptic connections for each stimulated whisker. In a causal network context, we hypothesized that pre-synaptic neurons that fire before other post-synaptic neurons in the observed population should have shorter response latency reminiscent of a “signaling mechanism” to other neurons about stimulus presence. Therefore, these neurons were expected to have more post-synaptic connections than their large response latency counterparts. Correlation between the response latency and the number of post-synaptic (pre-synaptic) connections was -0.63 (+0.61) consistent with our hypothesis. Finally, we used 90% of the trials to obtain a template network model for each whisker stimulated. We then decoded the spike trains in the remaining 10% of the trials to determine the corresponding whisker identity. A decoding accuracy of 97% was obtained indicating that the obtained causal networks are stimulus-dependent in which the population acts cooperatively rather than independently to encode the underlying stimulus.

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CONTRIBUTED ORALS

O-19

Sparse codes of harmonic sound and their interaction explain harmony-related response of auditory cortex

Hiroki Terashima[1], Haruo Hosoya[2]

[1] Department of Complexity Science and Engineering, The University of Tokyo, Chiba 277-8561, Japan
[2] Department of Computer Science, The University of Tokyo, Tokyo 113-8654, Japan
E-mail: hterashima@mns.k.u-tokyo.ac.jp

Sparse coding and relevant theories have well explained the response properties of visual system [1] and early stages of auditory system, suggesting their adaptation to the statistics of natural stimuli. The present study continues this line of research in a higher layer of auditory system, specifically focusing on the harmonic structure in natural sounds. Harmony is often found in behaviourally important sounds like animal vocalization, and should be captured in higher stages of auditory system, since a peripheral auditory neuron typically responds to only one frequency component. Indeed, neurophysiological studies have revealed that monkey primary auditory cortex (A1) contains neurons with response properties related to harmony: some A1 neurons are activated or modulated by multiple frequencies, which are often harmonically related to each other or in ratio of simple integers [2].

We hypothesize that such harmonic relations emerge from sparse coding of harmonic natural sound. In order to test this, we first learn 'basis' spectra by applying the sparse coding model and algorithm in [1] to the frequency-domain representation of a sound source, which is an approximation of the output from peripheral auditory system. Next, we regard the obtained bases as model A1 neurons, and then investigate how in the model they respond to pure- and two-tone stimuli, following the experimental scheme used in [2]; the bases can be classified into two groups ('single-peaked' and 'multi-peaked') as in the experiment, and moreover, we find a similar harmonic tendency in the ratio distributions of their response and modulation peak frequencies. A comparison using three sound sources with different degrees of harmony shows that the level of the harmonic tendency correlates with the harmonic degree of the sound source used for learning, and it is notable that the level with a highly harmonic sound, namely piano performance, is quantitatively comparable to that of the A1 neurons. Considering together an absence of such relations with a non-harmonic sound source, we conclude that the harmonic relations emerge as a reflection of the harmonic structure in input sound.

This result suggests that the sparse coding still prevails in higher stages of sensory systems, supporting an idea of the adaptation to behaviourally important stimuli. Furthermore, the modulatory behaviours can be explained by divisive interaction between model neurons that partially share their harmonic structures. We propose physiological experiments to confirm such interaction and predict their results.

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CONTRIBUTED ORALS

O-20

How Good is Grid Coding versus Place Coding for Navigation Using Noisy, Spiking Neurons?

Alexander Mathis[1,2,3], Martin Stemmler[1,2], Andreas Herz[1,2]

[1] Division of Neurobiology, Ludwig-Maximilians-Universität München, 82152 Planegg-Martinsried, Germany
[2] Bernsteincenter for Computational Neuroscience Munich, University, 82152 Planegg-Martinsried, Germany
[3] Graduate School for Systemic Neuroscience, LMU Munich, 82152 Planegg-Martinsried, Germany
E-mail: herz@bio.lmu.de

Our understanding of how the brain encodes navigation through space underwent a revolution with the remarkable discovery of grid cells in the medial entorhinal cortex (MEC) of rodents [3]. A grid cell builds a hexagonal lattice representation of physical space, such that the cell fires whenever the rodent moves through a lattice point. In contrast, place cells in the hippocampus proper fire only at a single, specific location in space.

Different place cells encode different spatial locations, while different grid cells exhibit different lattice spacings, orientations, and phases. At the level of a single neuron, the multiple firing fields of a grid cell lead to an inherent ambiguity in the position estimate. Hence, for both codes precise information about position can only be gained from a population of grid and place cells respectively. We will present two different interpretations of the grid population code, one as an effective way of subdividing space with a high resolution and one based on modular arithmetic, similar to Fiete et.al. [2]. For a clarification of these concepts look at Fig 1 & 2 and in the appendix. We furthermore argue that the modular arithmetic interpretation is lacking robustness in both: capacity and resolution.

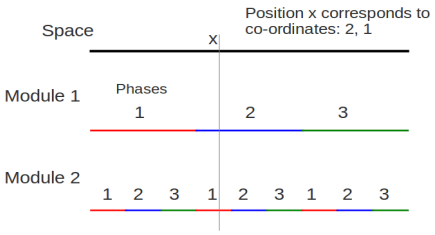

Figure 1: Interval nesting hypothesis

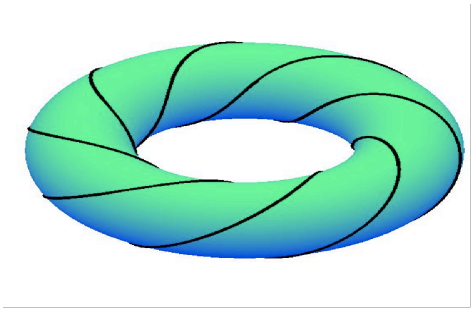

Figure 2: Modular arithmetic hypothesis

After these interpretations we set out to investigate the spatial resolution of the place code and the grid code on a limited, one-dimensional space with a finite amount of cells and families of tuning curves. Therefore, we built a stochastic population coding model as in Bethge [1]. The family of tuning curves convert the position into firing rates for statistically independent Poisson neurons. To compare the two coding schemes we calculate the maximum likelihood position estimate and compute the mean square error of the population code. We believe that the grid and place code should enable real-time readout of the rat's position *while* it is moving. For this reason, we have to consider short decoding times and hence, since Fisher information methods based on the Cramér Rao bound fail to estimate the mean error in such cases [1], we make use of Monte Carlo integration methods.

Under the condition of noisy, spiking neurons, we demonstrate that the grid code, if it is organized as in the interval nesting hypothesis outperforms the place code for any tuning width. On the other hand, if it is organized as in the modular arithmetic hypothesis, i.e. spatial periods that are far shorter than the length of space the grid code has a lower distortion than the best place codes.

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CONTRIBUTED ORALS

O-21

Sparse coding models demonstrate some non-classical receptive field effects

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

[1] Department of Biomedical Engineering, Georgia Institute of Technology, Atlanta, GA 30332, USA
[2] Department of Electrical and Computer Engineering, Georgia Institute of Technology, Atlanta, GA 30332, USA
E-mail: mczhu@gatech.edu

Non-classical receptive field (nCRF) effects include several response properties in V1 neurons not explained by a linear-nonlinear (LN) receptive field model, but instead requiring significant interactions between V1 neurons. Using a sparse coding model [1-2] and bar and grating stimuli, simulated physiology experiments were carried out that replicated several nCRF phenomena reported previously in neurophysiology experiments. These include: end-stopping [3] (Fig. 1), contrast invariance of orientation tuning [4] (Fig. 2), radius, orientation, and contrast tunings of surround suppression [5-6] (Fig. 3, 4, 5). The results suggest that a sparse coding model can explain many of the nonlinear effects in V1 cells, and is therefore a reasonable candidate for a functional model of striate cortex.

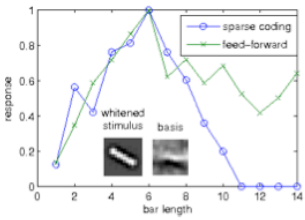

Figure 1. End-stopping. Comparison with a LN model.

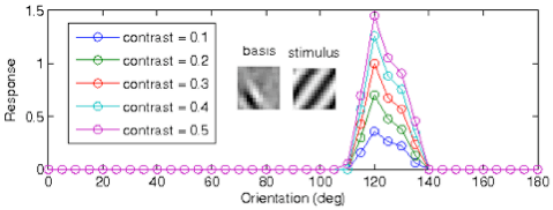

Figure 2. Contrast invariance of orientation tuning.

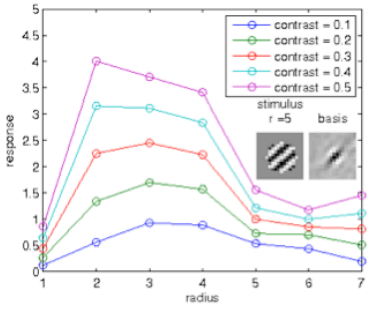

Figure 3. Surround suppression at different contrasts.

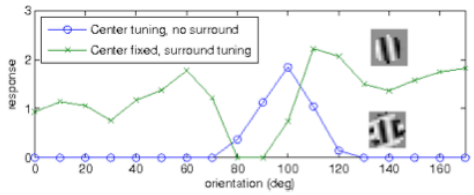

Figure 4. Orientation tuning of surround suppression.

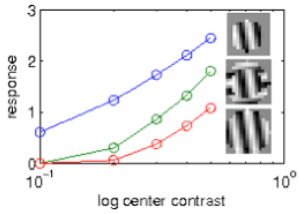

Figure 5. Surround orientation influences contrast tuning.

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