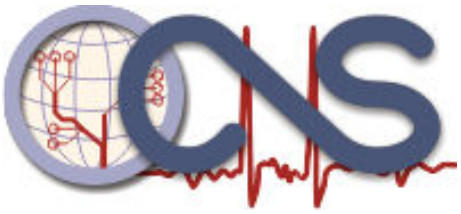

ALLEN INSTITUTE® – UNIVERSITY OF WASHINGTON® – SEATTLE, WA
CNS 2018

Contents

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



Overview

Organization for Computational Neurosciences (OCNS)

2018 Board of Directors

- **President: Astrid Prinz** (Emory University, Atlanta, USA).
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- **Local Org. Committee Rep. CNS 2018: Eric Shea-Brown** (University of Washington, Seattle, USA).
- **Local Org. Committee Rep. CNS 2019: Alex Roxin** (Centre de Recerca Matemàtica, Barcelona, Spain).
- **CNS Tutorials Organizer: Hermann Cuntz** (ESI and FIAS, Franfurt/Main, Germany).
- **CNS Workshop Organizer: Martin Zapotocky** (Czech Academy of Sciences, Prague, Czech Republic).
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- **CNS Registration Organizer: Leonid Rubchinsky** (Indiana University, Indianapolis, USA).
- **CNS Travel Awards: Taro Toyoizumi** (RIKEN Brain Science Institute, Saitama, Japan).
- **OCNS Member Approval: Maurice Chacron** (McGill University, Montreal, Canada).

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- **CNS Program Chair: Thomas Nowotny** (University of Sussex, UK).
- **CNS Publication Chair: Ingo Bojak** (University of Reading, UK).
- **Sacha van Albada** (Research Centre Jülich, Germany).
- **Maxim Bazhenov** (University of California San Diego, USA).
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- **Christoph Metzner** (University of Hertfordshire, UK).
- **Yaroslav Molkov** (Indiana University – Purdue University, Indianapolis, USA).
- **Tatyana Sharpee** (Salk Institute, San Diego, USA).
- **Tatjana Tchumatchenko** (Max Planck Institute for Brain Research, Frankfurt/Main, Germany).

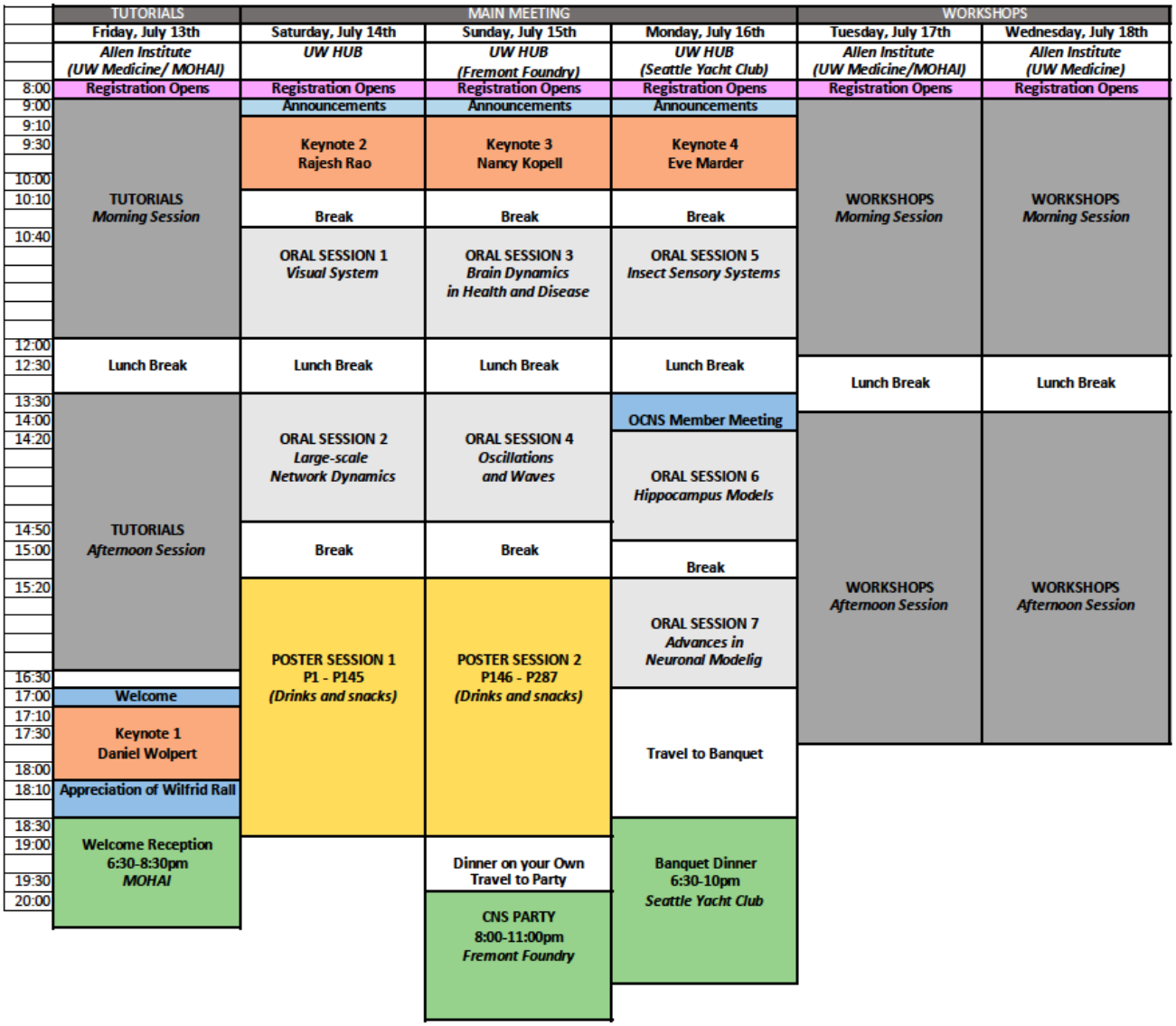
2018 Local Organizers

- **Christof Koch** (Allen Institute for Brain Science, Seattle, USA).
- **Adrienne Fairhall** (University of Washington, Seattle, USA).
- **Eric Shea-Brown** (University of Washington, Seattle, USA).

Fundraising

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

Timetable


TUTORIALS
MAIN MEETING
WORKSHOPS
Friday, July 13th
Allen Institute (UW Medicine/ MOHAI)
Saturday, July 14th
UW HUB
Sunday, July 15th
UW HUB (Fremont Foundry)
Monday, July 16th
UW HUB (Seattle Yacht Club)
Tuesday, July 17th
Allen Institute (UW Medicine/MOHAI)
Wednesday, July 18th
Allen Institute (UW Medicine)
8:00 Registration Opens
9:00 Announcements
9:10
9:30 Keynote 2 Rajesh Rao
10:00
10:10 Break
10:40 ORAL SESSION 1 Visual System
TUTORIALS Morning Session
12:00
12:30 Lunch Break
13:30
14:00 ORAL SESSION 2 Large-scale Network Dynamics
14:20
14:50 Break
15:00
15:20 POSTER SESSION 1 P1 - P145 (Drinks and snacks)
TUTORIALS Afternoon Session
16:30
17:00 Welcome
17:10
17:30 Keynote 1 Daniel Wolpert
18:00
18:10 Appreciation of Wilfrid Rall
18:30
19:00 Welcome Reception 6:30-8:30pm MOHAI
19:30
20:00
Registration Opens
Announcements
Keynote 3 Nancy Kopell
Break
ORAL SESSION 3 Brain Dynamics in Health and Disease
Lunch Break
ORAL SESSION 4 Oscillations and Waves
Break
POSTER SESSION 2 P146 - P287 (Drinks and snacks)
Dinner on your Own Travel to Party
CNS PARTY 8:00-11:00pm Fremont Foundry
Registration Opens
Announcements
Keynote 4 Eve Marder
Break
ORAL SESSION 5 Insect Sensory Systems
Lunch Break
OCNS Member Meeting
ORAL SESSION 6 Hippocampus Models
Break
ORAL SESSION 7 Advances in Neuronal Modelig
Travel to Banquet
Banquet Dinner 6:30-10pm Seattle Yacht Club
Registration Opens
WORKSHOPS Morning Session
Lunch Break
WORKSHOPS Afternoon Session
Registration Opens
WORKSHOPS Morning Session
Lunch Break
WORKSHOPS Afternoon Session

General Information

Meeting venues

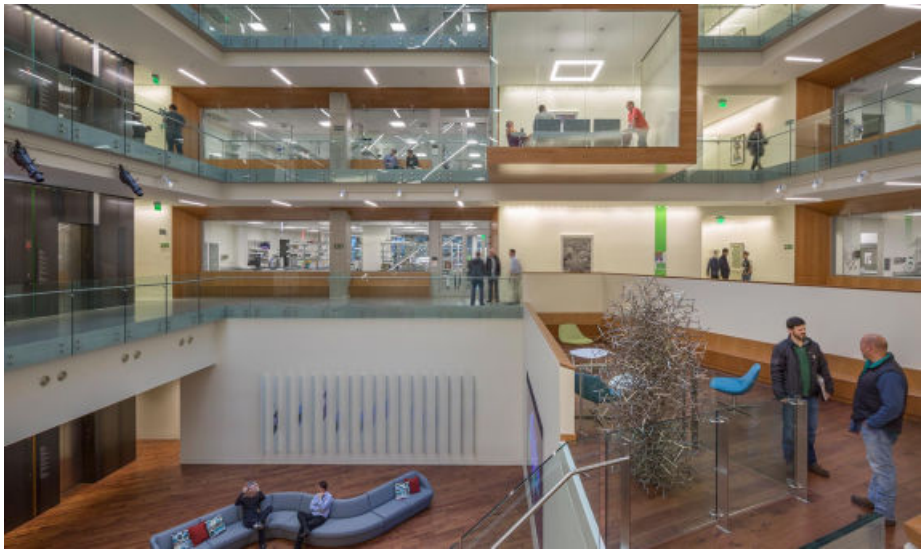
Allen Institute
615 Westlake Ave N, Seattle, WA, 98109

University of Washington Husky Union Building
4001 E Stevens Way NE, Seattle, WA 98195

The Allen Institute and University of Washington are thrilled to be hosting CNS 2018 in Seattle.
Founded in 2003 by Paul G. Allen, the Allen Institute has expanded from its initial pursuit of understanding the brain to encompass an investigation of the inner workings of cells and the funding of transformative scientific ideas around the world. The Allen Institute for Brain Science is a division of the Allen Institute and is dedicated to accelerating the understanding of how the human brain works in health and disease. Using a big science approach, the Allen Institute generates useful public resources used by researchers and organizations around the globe, drives technological and analytical advances, and discovers fundamental brain properties through integration of experiments, modeling and theory. The Allen Institute for Brain Science's data and tools are publicly available online at brain-map.org.

The University of Washington is a national leader in computational neuroscience, with award-winning research underway across the full spectrum of scales, mechanisms, and functions of the brain. Topics range from ion channel stochasticity in auditory processing to insect flight control to human/computer interfaces. Faculty members' interests span many areas of theory, computation and data analysis and interact extensively with colleagues in quantitative experimentation and imaging. The new UW Computational Neuroscience Center capitalizes on this strength, along with the UW Institute for Neuroengineering (UWIN) and the Center for Sensorimotor Neural Engineering (CSNE).

Tutorials and workshops locations:



Tutorials and workshops will be held at the Allen Institute, University of Washington Medicine - South Lake Union (UW Medicine SLU), and the Museum of History and Industry (MOHAI). Check in at the Allen Institute lobby before proceeding to all workshops and tutorials. Allow 10 minutes to walk from the Allen Institute to tutorial/workshop rooms in both UW Medicine SLU and MOHAI.

Ask at check in for the room number for each workshop and tutorial.

Please bring your conference badge to tutorials, workshops, and all other conference events.

Getting to the conference venues

From Sea-Tac Airport:

From the airport (SeaTac), the lowest cost and often fastest method of getting to Downtown or UW area is to take the light rail to downtown or to the UW station. Please check where your hotel is relative to the stations so you know your walk distance or if you need to call an Uber or Lyft. From the UW station you can walk a short distance (15-20 minutes) to the dorms or 25 minutes to the recommended hotels in the University District. The bus lines number 44 or 45 from the UW station will take you to the 45th and Roosevelt area of the University District, where the conference hotels are located.

The cost to ride light rail from the airport to either stations runs from $2.50 to $4 per person depending on how far you travel. A taxi or Uber from the airport can run from $55 to $75 per ride depending on traffic, and may take an hour during peak travel times.

Shuttle Express is a 24 hour service. Shuttle Express does pick ups and drop offs 24 hours a day. We highly suggest reservations from the airport; however, walk ups are taken. Rates are from $19.00 one way, per person.

TIP: There may be a wait, both with or without a reservation, at the airport as they wait to ensure they have enough people on board to justify making the trip to Seattle. Sometimes this can take a while, however, sometimes there is little to no wait depending on the time of day.

You may either book online www.shuttleexpress.com with Shuttle Express or call them at 425-981-7000.

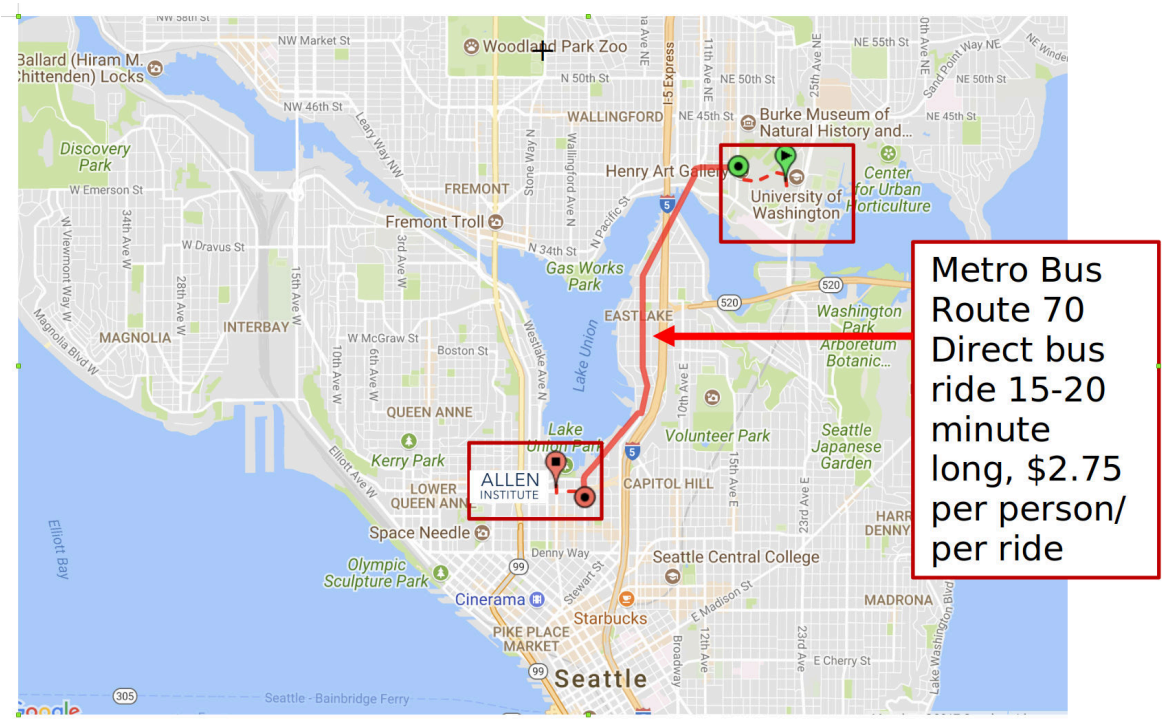
Between the University of Washington and the Allen Institute:

There is a direct bus line that runs between University of Washington and Allen Institute (metro bus # 70). The bus ride takes about 15-20 minutes and costs $2.75 a ride. You must have exact change; no change is made on the bus. You may also buy a transit card (Orca Card) at any light rail station vending machine. It can be loaded with cash or a credit card. You can plan your bus trip here https://tripplanner.kingcounty.gov/hiwire with departure and arrival times.

Parking is very limited at both the Allen Institute and University of Washington, and it is recommended to not drive if possible. For parking destinations near the Allen Institute, see:
https://seattle.bestparking.com/neighborhoods/south-lake-union-parking.
The best parking garage at the University of Washington is the Padelford garage, located off Pend Orielle Rd. Pay station parking is located on the lower levels.

The tutorials and workshops will be held at the Allen Institute, UW Medicine South Lake Union, and MOHAI. After checking in at the Allen Institute, you will be directed two blocks to UW Medicine South Lake Union or one block to MOHAI.

Seattle also offers two bike share companies with bikes located in various parts of the city, it is usually very easy to find an available bike. You can find the bike share information below: download their applications to create an account used to pay for bike time and find bikes.
Lime bikes https://www.limebike.com/
Ofo bikes http://ofo.com/



Welcome, first keynote, and reception:

The Welcome remarks and first keynote will be held on Friday July 13 at 5pm at the Allen Institute auditorium. **Please plan to arrive early if you wish to ensure a seat in the auditorium.** Overflow seating in rooms with a live video stream of the presentation will be located in other rooms in the Allen Institute.

The welcome reception will follow the keynote and will take place at MOHAI, located just one block from the Allen Institute. Attendees who do not attend the keynote may check in at MOHAI. Attendees who attended tutorials and/or the keynote should be sure to bring their conference badges.

Information for poster presentations:

The poster presentations will be held in the HUB Ballroom, located one floor above the main meeting room. Poster boards will be numbered. Pins will be provided.

Poster sessions will be held on July 14 and 15 at 3:20-7pm. Presenters are expected to be at the session until at least 6pm. The hall will be available starting at 1pm on both days for presenters to set up posters.

Posters should be removed promptly at the end of the poster session on both days. Presenters who leave before 7pm should take their posters with them at that time.

Please leave pins on poster boards at the end of the session.

Posters that are not removed by the end of the day of the session will be discarded. The organizers are not responsible for loss or damage to posters not removed by their owners.

Registration and locations

On the days of the main meeting, registration will be held in the University of Washington Husky Union Building (HUB) at the Lyceum, the primary meeting room.

On the days of the tutorials and workshops, registration will be held in the lobby of the Allen Institute, including for tutorials and workshops being held in neighboring buildings.

For those not attending the tutorials or opening keynote, registration at the Welcome Reception will also be available.

Registration hours:

Friday July 13, at the Allen Institute: 8 am to 5 pm
Friday July 13, at the Welcome Reception at MOHAI: 6 pm to 8 pm
Saturday July 14, at the UW HUB Lyceum: 8 am to 4 pm
Sunday July 15, at the UW HUB Lyceum: 8 am to 4 pm
Monday July 16, at the UW HUB Lyceum: 8 am to 4 pm
Tuesday July 17, at the Allen Institute: 8 am to 4 pm
Wednesday July 18, at the Allen Institute: 8 am to 4 pm

Please bring your conference badge to all conference events, including offsite social events.

Local information

Good to Know
Travel tips for Seattle are available at https://www.visitseattle.org/.

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

Insurance
The organizers do not accept responsibility for individual medical, travel or personal insurance. All participants are advised to take out their own personal insurance before traveling to Seattle.

Currency & Banking
Exchange of foreign currency is available at airports and at most hotels and banks throughout the city. International credit cards are accepted for payments in hotels, restaurants and shops. An increasing number of locations, especially small restaurants and food carts, are cashless.

Electricity
The US uses a 120 volt 60 Hz system. Travelers from outside of North America will likely require socket and/or voltage converters.

Shopping
Most stores in Seattle are open from 8am to 8pm. Some stores may open later on Sundays. A large shopping center called University Village with a grocery store, drugstore, and many other shops and restaurants is located approximately 0.5mi east of the main meeting location on the UW campus.

Time Zone
Seattle is on Pacific Daylight Time in July (GMT-7). Seattle is the northernmost city of over 1 million people in the United States, so days are long in summer. During the meeting, sunrise will be around 5:30am and sunset will be around 9pm.

Tipping
Gratuities are usually not automatically included in the bill in most bars and restaurants, but especially for groups larger than 6, an automatic gratuity may be applied. Standard tip is 18-20%.

Get around by public transportation
Seattle has an extensive bus network and a light rail that travels directly from the airport to downtown and the University of Washington. Bus fares can be paid in cash with exact change ($2.75, no matter what bus route or distance traveled) or with an Orca Card transit pass, which can be bought and loaded with fares at any light rail station. You can plan your bus trip here https://tripplanner.kingcounty.gov/hiwire with departure and arrival times. You can transfer between bus routes without paying a second fare within 2 hours of boarding the first bus.

By bike
Seattle also offers two bike share companies with bikes located in various parts of the city, it is usually very easy to find an available bike. You can find the bike share information below: download their applications to create an account used to pay for bike time and find bikes.
Lime bikes https://www.limebike.com/
Ofo bikes http://ofo.com/

By car
Parking is limited at both the Allen Institute and the University of Washington. For the Allen Institute and surrounding activities, see https://seattle.bestparking.com/neighborhoods/south-lake-union-parking for parking locations. At the University of Washington, proceed to the Padelford parking garage, accessed from Pend Orielle Rd, and use the pay stations on the lower levels as directed.

On foot
Many fun activities, interesting sights, and local restaurants are within walking distance of all conference venues. Recommended restaurants for each primary location are listed below.
The University District hotels are within walking distance of the main meeting location at the Husky Union Building. Ask at the hotel desk for a campus and neighborhood map.

Weather
July is the warmest month in Seattle. The average high is around 75F/24C and low around 55F/12C. Rain is relatively rare in July, but be prepared for surprise storms rolling in from Puget Sound.

Free Wifi
Wifi is provided at the meeting venues. The University of Washington main campus (HUB) and UW Medicine South Lake Union will have the same wifi login information. The Allen Institute has its own separate wifi. See the registration desk for each venue for the login information.

Car Services
Taxis are available, but they can be quite expensive and congestion between the University District hotels and the Allen Institute is severe. Traveling by car during rush hour is not recommended.

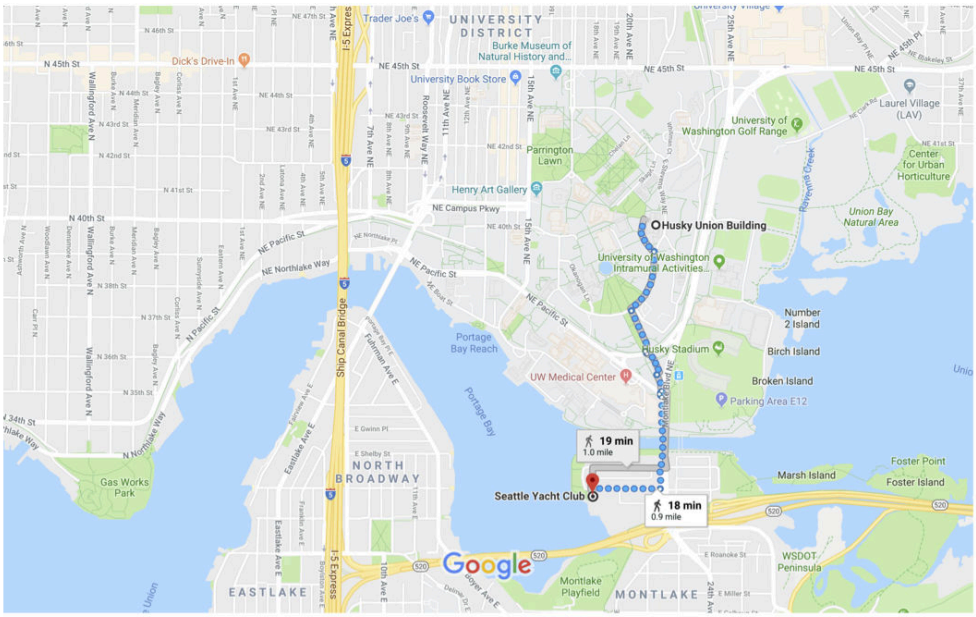
Uber and Lyft are available throughout the city. Traveling by public transportation or on foot in the University District, especially around University Avenue between 43rd and 50th, is not recommended after dark.

Gala Dinner

Date: Monday, July 16, 2018
Time: 6:30pm PM
Venue: Seattle Yacht Club, 1807 E Hamlin St, Seattle, WA 98112 https://www.seattleyachtclub.org/
Recommended dress code: Casual
How to get there: The best way to get to the Seattle Yacht Club from the main meeting venue at the University of Washington Husky Union Building is on foot or by bike. The distance is 1 mile and is entirely downhill or flat. For those who choose to bike, bike shares are generally abundant on campus. Conference staff will lead walking groups from the main meeting location at the HUB to the Yacht Club, departing from the registration desk between 5:30 and 6:00 pm.

The bike and pedestrian route travels through part of the University of Washington campus, down the Rainier Vista quad with panorama views of the Cascade Mountains and especially Mount Rainier, across the historic Montlake Bridge, and into the Montlake neighborhood.

There are no public transit routes that travel directly between those locations. Montlake Avenue and the Montlake Bridge are generally extremely congested at that time of day, so car share services will be time consuming.

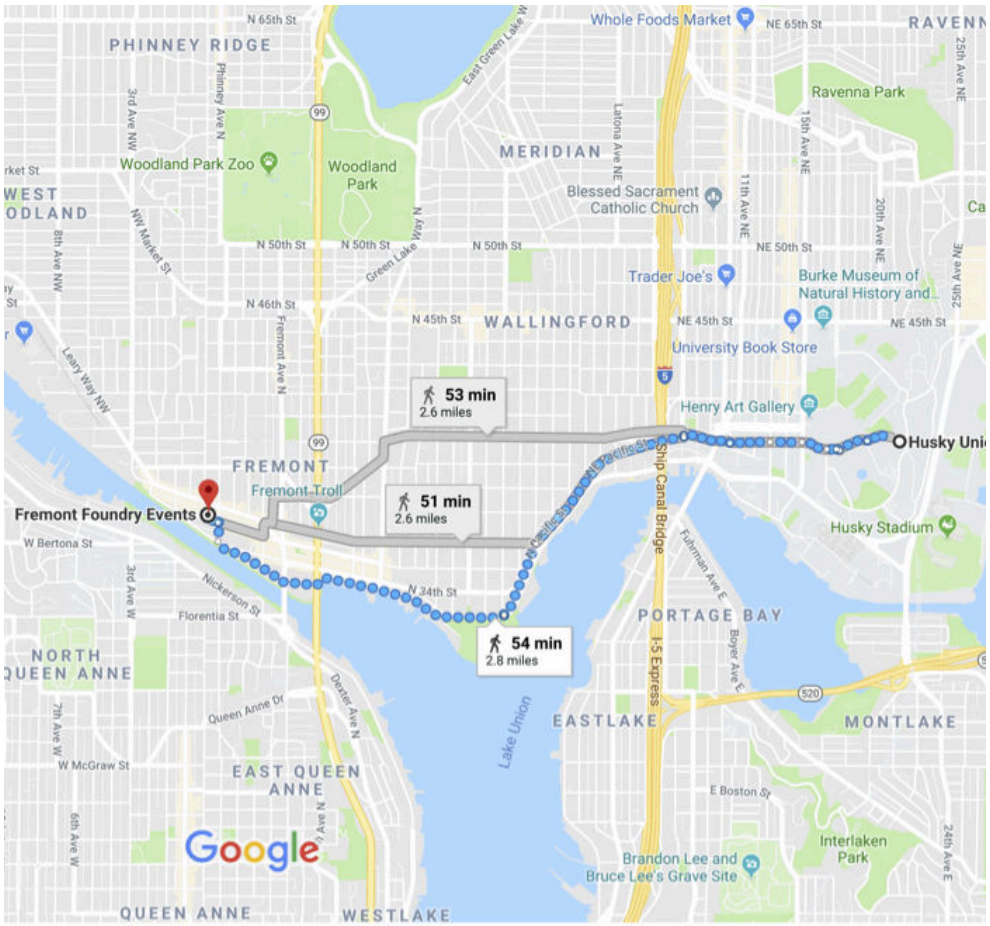

UNIVERSITY DISTRICT
Husky Union Building
Seattle Yacht Club
19 min
1.0 mile
18 min
0.9 mile
NORTH BROADWAY
EASTLAKE
MONTLAKE
Google
Map data ©2018 Google
1000 ft

CNS Party

Date: Sunday, July 15, 2018
Time: 8:00 PM
Venue: Fremont Foundry, 154 N 35th St, Seattle, WA 98103
Recommended dress code: Casual

The CNS Party will be held at the Fremont Foundry. Originally an artists' metal-working foundry, it was converted to an event space for parties, weddings, and other events.

Fremont is about 3 miles from the University of Washington. It can be reached from the University of Washington Husky Union Building and from the University District hotel area via the #31 or #32 bus lines (approximately 30 minutes travel), by carshare services (approximately 15 minutes travel), or by bikeshare services (20 minutes travel, all flat or downhill) via a protected mixed-use bike pedestrian trail. Pedestrians can also use the mixed-use trail. Plan to eat dinner before the party, as only light refreshments and drinks will be provided. Recommended restaurants for dinner near the party are listed below.


PHINNEY RIDGE
N 65th St
Whole Foods Market
NE 65th St
RAVENN
Ravenna Park
Woodland Park Zoo
Woodland Park
MERIDIAN
Blessed Sacrament Catholic Church
WEST ODLAND
N 50th St
NE 50th St
Trader Joe's
Burke Museum of Natural History and...
N 46th St
N 45th St
WALLINGFORD
NE 45th St
University Book Store
53 min
2.6 miles
Henry Art Gallery
Husky Uni
FREMONT
Fremont Troll
51 min
2.6 miles
Fremont Foundry Events
Husky Stadium
W Bertona St
N 34th St
Florentia St
PORTAGE BAY
54 min
2.8 miles
NORTH QUEEN ANNE
Queen Anne Dr
EASTLAKE
MONTLAKE
W McGraw St
EAST QUEEN ANNE
E Boston St
Google
Brandon Lee and Bruce Lee's Grave Site
Interlaken Park
QUEEN ANNE
WESTLAKE
Map data ©2018 Google
2000 ft

Restaurants

Reservations are recommended for groups larger than 6 at most restaurants in Seattle. Most restaurants in Seattle have vegetarian options.

Restaurants near the University of Washington in the University Village shopping area:

Ba Bar
Vietnamese
$$
http://babarseattle.com/university-village/

Elemental pizza
Wood-fired pizza
$$
https://elementalpizza.com/

Evergreens
Salads
$
http://evergreens.com/

Rachel's Ginger Beer with Ma'ono Fried Chicken
Ginger beer and cocktails, fried chicken
$
https://rachelsgingerbeer.com/pages/university-village

Eureka
Burgers and pub food, extensive tap list
$$
http://eurekarestaurantgroup.com/eat/

Molly Moon's
Ice cream
$
http://www.mollymoon.com/

For a full list of University Village restaurants, see https://uvillage.com/directory/

Selected restaurants near the University of Washington in the the University District hotel area:

U:Don
Udon and tempura bar
$
https://freshudon.com/

Chili's
South Indian
$ lunch, $$ dinner
http://chilissouthindianrestaurant.com/

Big Time Brewery
Pub food, beer brewed on site

$$
http://bigtimebrewery.com/

Agua Verde
Homestyle Mexican, view of lake
$ lunch, $$ dinner
http://aguaverde.com/cafe/

Cafe Allegro
Coffee
$
https://seattleallegro.com/

Cafe Solstice
Coffee, sandwiches, beer (evening)
$
https://www.cafesolsticeseattle.com/u-district/

For more recommendations, see http://www.cnsorg.org/cns-2018-local-info and scroll down to the map.

Selected restaurants near the Allen Institute, UW Medicine South Lake Union, and MOHAI:

Uptown Espresso
Coffee
$

100 Pound Clam
Seafood
$$
http://www.100poundclam.com/

El Chupacabra
Mexican, view of lake
$ to $$
http://www.elchupacabraseattle.com/menu/food/

Ballard Pizza Co
Pizza
$$
http://www.ballardpizzacompany.com/

Portage Bay Cafe
Brunch
$$
https://www.portagebaycafe.com/

Many food trucks are located around South Lake Union. Head south (away from the lake) on any road and you will probably find one!

Note that the Allen Institute Cafe is accessible to employees only.

For more recommendations, see http://www.cnsorg.org/cns-2018-local-info and scroll down to the map.

Selected restaurants near the Fremont Foundry:

Agrodolce
Southern Italian
$$

http://www.mariahinesrestaurants.com/restaurants/agrodolce/?activeTab=0

The Red Door
Gastropub
$$
http://reddoorseattle.com/

Rock Creek Seafood and Spirits
Seafood, modern NW
$$$
http://rockcreekseattle.com/

Dumpling Tzar
Dumplings
$
http://dumplingtzar.com/

Cafe Turko
Turkish
$$
http://cafe-turko.com/

Manolin
Seafood
$$ http://www.manolinseattle.com/menu-2/

Sights

The CNS locations circle Lake Union, located in the center of Seattle. Getting around the conference provides an opportunity to travel the city and see some sights!

The top tourist destination in Seattle is Pike Place Market, which is home to many small restaurants, food stands, shops, artisans, and the famous fish-tossing seafood market. Weekday mornings are likely to be the least crowded.

Other popular destinations include the Space Needle, located in Seattle Center, Kerry Park (the "Frasier" view), boating on Lake Union, and central neighborhoods including Capitol Hill, Fremont, Wallingford, Ballard, the University District, and more.

The University District farmer's market is about half a mile from campus and is held from 9-1 on Saturday mornings. It is the largest farmer's market in the state and has an abundance of locally grown produce, small artisan food companies, and ready-to-eat lunch bites.

See more recommendations for touring and sights:
General top destinations: https://www.visitseattle.org/ or https://www.buzzfeed.com/ariannaodell
Near the University of Washington: https://www.visitseattle.org/neighborhoods/university-district/
Near the Allen Institute: https://www.google.com/search?q=south+lake+union&ie=utf-8&oe=utf-8&client=firefox-b-1-ab
Nearest the Fremont Foundry: https://fremont.com/
Near the Fremont Foundry: https://www.fodors.com/world/north-america/usa/washington/seattle/neighborhoods/ballard

Program

Tutorials

T1 **Allen Institute Brain Observatory and Brain Modeling Toolkit tutorial**
Allen Institute Training Room, Friday July 13, 09:00 - 16:30

Yazan Billeh, Allen Institute, USA
Sergey Gratiy, Allen Institute, USA
Saskia E. J. de Vries, Allen Institute, USA

T2 **Multiscale modeling from molecular level to large network level**
Allen Institute Auditorium, Friday July 13, 09:00 - 16:30

Salvador Dura-Bernal, SUNY Downstate, USA
Robert McDougal, Yale University, USA
William Lytton, SUNY Downstate, USA

T3 **Simulation of large-scale neural networks**
UW Medicine SLU Brotman Auditorium, Friday July 13, 09:00 - 16:30

Sacha J. van Albada, Julich Research Centre and JARA, Germany
Philipp Weidel, Julich Research Centre and JARA, Germany

T4 **Neuroinformatics resources for computational modelers**
Allen Institute 288/289, Friday July 13, 09:00 - 12:00

Padraig Gleeson, University College London, UK

T5 **Modeling and analysis of extracellular potentials**
Allen Institute 286/287, Friday July 13, 09:00 - 12:00

Gaute Einevoll, Norwegian University of Life Sciences & University of Oslo, Norway
Espen Hagen, Dept. of Physics, University of Oslo, Norway

T6 **Single cell RNA-seq analysis for transcriptomic type characterization**
Allen Institute 286/287, Friday July 13, 13:30 - 16:30

Zizhen Yao, Allen Institute, USA
Lucas Graybuck, Allen Institute, USA

Room assignments are subject to change. Please check with registration in the Allen Institute lobby for the final room assignments for tutorials.

Main Meeting

Friday July 13

8:00 – 17:00		**Registration (Allen Institute)**
9:00 – 16:30		**Tutorials (Allen Institute & nearby UW Medicine at South Lake Union)**
17:00 – 17:10		**Welcome and Announcements (Allen Institute)**
17.10 – 18:10	K1	**Keynote 1:** ***Probabilistic models of sensorimotor control and decision making*** Daniel Wolpert
18:10 – 18:30		**Appreciation of Wilfrid Rall**
18:30 – 20:30		**Welcome Reception/Registration (nearby Museum of History and Industry (MOHAI))**

Saturday July 14

8:00 – 9:00		**Registration (University of Washington, Husky Union Building Lyceum, First Floor)**
9:00 – 9:10		**Announcements**
9:10 – 10:10	K2	**Keynote 2:** ***The Bayesian brain: from predictive coding to decision making*** Rajesh Rao
10:10 – 10:40		**Break**
		Oral Session I: Visual System
10:40 – 11:20	F1	**Featured Oral 1:** ***Predictive computations in the primary visual cortex*** Jan Homann*, Michael Berry, Sue-Ann Koay, Alistair M. Glidden, and David W. Tank
11:20 – 11:40	O1	***Generative model of visual cortex with short- and long-range recurrent interactions*** Federica Cappareli*, Klaus Pawelzik, David Rotermund, and Udo Ernst
11:40 – 12:00	O2	***Info in a bottleneck: exploring the compression of visual information in the retina*** Gabrielle Gutierrez*, Eric Shea-Brown, and Fred Rieke
12:00 – 13:30		**Lunch Break**

Oral Session II: Large-scale Network Dynamics

13:30 – 13:50 O3 ***Structural and dynamical properties of local cortical networks result from robust associative learning***
Danke Zhang, Chi Zhang, and Armen Stepanyants*

13:50 – 14:10 O4 ***Reduced models of an attractor neural network's response to conflicting external inputs***
Kathryn Hedrick*

14:10 – 14:30 O5 ***Topologies of repetitive functional network motifs vary dynamically with age in the developing human brain: Evidence from very high-dimensional invasive brain signals***
Caterina Stamoulis*, Phillip Pearl

14:30 – 14:50 O6 ***Revealing principles of cortical computation using the Allen Brain Observatory: A large, standardized calcium imaging dataset from the mouse visual cortex***
Michael A. Buice, Saskia E. J. de Vries*, Gabriel Ocker, Michael Oliver, Peter Ledochowitsch, Daniel Millman, Eric Shea-Brown, Christof Koch, Jianghong Shi, and R Clay Reid

14:50 – 15:20 **Break**

15:20 – 19:00 **Poster Session 1 (Posters 1 - 145) (University of Washington, Husky Union Building North Ballroom, Second Floor (Drinks and Snacks Provided))**

Sunday July 15

8:00 – 9:00 **Registration (University of Washington, Husky Union Building Lyceum, First Floor)**

9:00 – 9:10 **Announcements**

9:10 – 10:10 K3 **Keynote 3:**
Coordination, modulation and functional implications of brain rhythms
Nancy Kopell

10:10 – 10:40 **Break**

Oral Session III: Brain Dynamics in Health and Disease

10:40 – 11:20 F2 **Featured Oral 2:**
Response to deep brain stimulation in essential tremor: predictions beyond noisy data with a Wilson-Cowan model
Benoit Duchet*, Gihan Weerasinghe, Christian Bick, Hayriye Cagnan, and Rafal Bogacz

11:20 – 11:40 O7 ***Characterization of the brain's dynamical repertoire in the psychedelic state***
Louis-David Lord*, Paul Expert, Robin Carhart-Harris, Morten Kringelbach, and Joana Cabral

11:40 – 12:00 O8 ***Understanding the bispectrum as a measure of cross-frequency coupling***
Christopher Kovach*

12:00 – 13:30 **Lunch Break**

Oral Session IV: Oscillations and Waves

13:30 – 13:50 O9 ***Spinal interneurons and locomotor speed and gait control in quadrupeds***
Ilya Rybak*, Simon Danner, and Natalia Shevtsova

13:50 – 14:10 O10 ***A simplified model of network bursts in the pre-Botzinger complex***
Yury Sokolov*, Jonathan Rubin

14:10 – 14:30 O11 ***Traveling waves in single cortical regions: mechanisms and emerging computational principles***
Lyle Muller*, Terrence Sejnowski

14:30 – 14:50 O12 ***Excitable dynamics of NREM sleep: a unifying model for neocortex and hippocampus***
Daniel Levenstein*, György Buzáki, and John Rinzel

14:50 – 15:20 **Break**

15:20 – 19:00 **Poster Session II (Posters 146 - 287) (University of Washington, Husky Union Building North Ballroom, Second Floor (Drinks and Snacks Provided))**

19:00 – 20:00 **Break (Time Allocated for Dinner and Travel to Party)**

20:00 – 23:00 **CNS Party (Fremont Foundry, 154 North 35th Streeet, Seattle)**

Monday July 16

8:00 – 9:00 **Registration (University of Washington, Husky Union Building Lyceum, First Floor)**

9:00 – 9:10 **Announcements**

9:10 – 10:10 K4 **Keynote 4:**
Differential resilience to perturbation of circuits with similar performance
Eve Marder

10:10 – 10:40 **Break**

Oral Session V: Insect Sensory Systems

10:40 – 11:20 F3 **Featured Oral 3:**
A molecular odorant transduction model and combinatorial encoding in the Drosophila Antennae
Aurel A. Lazar, Chung-Heng Yeh*

11:20 – 11:40 O13 ***Biological mechanisms for learning: A computational model of olfactory learning in the Manduca sexta moth***
Charles Delahunt*, Jeffrey Riffell, and J. Nathan Kutz

11:40 – 12:00 O14 ***Modeling of TRP channel mediated noxious cold sensation in Drosophila sensory neurons***
Natalia Maksymchuk*, Atit Patel, Nathaniel Himmel, Daniel Cox, and Gennady Cymbalyuk

12:00 – 13:30 **Lunch Break**

13:30 – 14:20 **OCNS Member Meeting (University of Washington, Husky Union Building Lyceum, First Floor)**

Oral Session VI: Hippocampus Models

14:20 – 14:40 O15 ***A geometric attractor mechanism for the self-organization of entorhinal grid modules***
Louis Kang*, Vijay Balasubramanian

14:40 – 15:00 O16 ***Simulating in vivo context-dependent recruitment of CA1 hippocampal interneuron specific 3 (IS3) interneurons***
Alexandre Guet-McCreight*, Frances Skinner

15:00 – 15:20 **Break**

Oral Session VII: Advances in Neuronal Modeling

15:20 – 15:40 O17 ***Quantitative simplification of detailed microcircuit demonstrates the limitations to common point-neuron assumptions***
Christian A Rössert, Giuseppe Chindemi, Andrew Davison, Dimitri Rodarie, Nicolas Perez Nieves, Christian Pozzorini, Csaba Eroe, James King, Taylor Newton, Max Nolte, Srikanth Ramaswamy, Michael Reimann, Willem Wybo, Marc-Oliver Gewaltig, Wulfram Gerstner, Henry Markram, Idan Segev, and Eilif Muller*

15:40 – 16:00 O18 ***A novel synaptic plasticity rule for detailed model neurons with realistic dendrites***
Christian Ebner, Claudia Clopath, Peter Jedlicka*, and Hermann Cuntz

16:00 – 16:20 O19 ***Assisted construction of hybrid circuits: making easy the implementation and automation of interactions between living and model neurons***
Manuel Reyes-Sanchez, Irene Elices Ocon*, Rodrigo Amaducci, Francisco B Rodriguez, and Pablo Varona

16:20 – 16:40 O20 ***Deciphering the evolutionary route to the first neurons***
Oltman de Wiljes*, Ronald van Elburg, and Fred Keijzer

16:40 – 17:00 O21 ***Community models as the ultimate objective (and success) of computational neuroscience: exempli gratia: The cerebellar Purkinje cell***
James Bower*

17:00 – 18:30 **Break (Time Allocated for Travel to Banquet)**

18:30 – 21:00 **CNS Banquet (Seattle Yacht Club, 1807 E Hamlin St., Seattle)**

Tuesday July 17 and Wednesday July 18

Workshops (Allen Institute & nearby MOHAI, UW Medicine at South Lake Union)

9:00 – 12:30	**Workshop Morning Session**
12:30 – 14:00	**Break for Lunch**
14:00 – 18:00	**Workshop Afternoon Session**

Workshops

W1 **Methods of Information Theory in Computational Neuroscience**
Allen Institute Auditorium, Tue July 17 and Wed July 18, 9:00 to 18:00

Joseph T. Lizier, University of Sydney
Viola Priesemann, Max Planck Institute for Dynamics and Self-organisation
Justin Dauwels, Nanyang Technological University
Taro Toyoizumi, RIKEN Brain Science Institute
Alexander G Dimitrov, Washington State University
Lubomir Kostal, Czech Academy of Sciences
Michael Wibral, Goethe University, Frankfurt

W2 **Neuronal morphology and structure**
Allen Institute 286/287, Tue July 17, 9:00 to 18:00

Alexander Bird, Ernst Strüngmann Institute and FIAS, Frankfurt
André Castro, Ernst Strüngmann Institute and FIAS, Frankfurt
Hermann Cuntz, Ernst Strüngmann Institute and FIAS, Frankfurt

W3 **Bridging Spatial and Temporal Scales in Brain Connectomics**
MOHAI - Microsoft Lakefront Pavilion, Tue July 17, 9:00 to 18:00

Katharina Glomb, Lausanne University Hospital
Joana Cabral, Oxford University

W4 **Models for Perceiving and Learning Time Intervals and Rhythms**
Allen Institute Training Room, Tue July 17, 9:00 to 18:00

Áine Byrne, New York University
John Rinzel, New York University
Amitabha Bose, New Jersey Institute of Technology

W5 **Developing, Standardising, and Sharing Large Scale Network Simulations**
Allen Institute 288/289, Tue July 17, 9:00 to 12:30

Padraig Gleeson, University College London

W6 **Neuroscience Gateway and Large Scale Neural Systems Simulations and Tools**
Allen Institute 288/289, Tue July 17, 14:00 to 18:00

Amit Majumdar, University of California San Diego
Subhashini Sivagnanam, University of California San Diego
Ted Carnevale, Yale University

W7 **Dynamics of Rhythm Generation**
UW Medicine SLU Brotman Auditorium, Tue July 17, 9:00 to 18:00

Gennady Cymbalyuk, Georgia State University

W8 **Insights Gained by Detailed Dendritic Modeling**
Allen Institute 540 Lab, Wed July 18, 9:00 to 18:00

Dieter Jaeger, Emory University
Volker Steuber, University of Hertfordshire

W9 **Integrative Theories of Cortical Function**
Allen Institute Training Room, Wed July 18, 9:00 to 18:00

Hamish Meffin, The University of Melbourne
Stefan Mihalas, Allen Institute for Brain Science
Anthony Burkitt, The University of Melbourne

W10 **How Does Learning Reshape the Dimensionality of Collective Network Activity?**
UW Medicine SLU Brotman Auditorium, Wed July 18, 9:00 to 18:00

Rainer Engelken, Columbia University
Guillaume Lajoie, Université de Montréal
Merav Stern, University of Washington

W11 **Towards New Models for Cognitive Flexibility**
Allen Institute 288/289, Wed July 18, 9:00 to 18:00

Rajeev Rikhye, Massachusetts Institute of Technology

Room assignments are subject to change. Please visit registration at the Allen Institute for final room assignments.

Abstracts

Tutorials

T1 **Allen Institute Brain Observatory and Brain Modeling Toolkit tutorial**

Allen Institute Training Room, Friday July 13, 09:00 - 16:30

Yazan Billeh, Allen Institute, USA
Sergey Gratiy, Allen Institute, USA
Saskia E. J. de Vries, Allen Institute, USA

The first part of the tutorial will introduce the Allen Brain Observatory, an open dataset of neural activity recorded in the visual cortex of the awake mouse. Collected using a standardized 2-photon calcium imaging pipeline, this dataset contains recordings in response to a standard set of visual stimuli from 40,000 neurons in 200 experiments, spanning 6 cortical areas, 3 cortical layers, and 6 excitatory Cre-defined cell populations. This tutorial will introduce the scientific context for this pipelined dataset, and demonstrate how to download and access this data using the Allen Software Development Kits (Allen SDK). Working in a Python environment, participants will be led through example analyses of both single cell and population level sensory coding.

The second part of the tutorial will introduce the Brain Modeling ToolKit (BMTK). BMTK is a Python-based software package for building and simulating models of neuronal circuits. It supports simulations at four levels of resolution (biophysically detailed, point-neuron, population statistics, and machine intelligence) by providing wrappers to tools such as NEURON, NEST, diPDE, and TensorFlow. This tutorial will give an overview of BMTK and work through two examples to demonstrate how to build and run networks at different levels of granularity.

This tutorial requires a basic level of Python proficiency and using Python scientific packages such as numpy and pandas.

References

[1] Brain Observatory: observatory.brain-map.org/visualcoding

[2] BMTK: alleninstitute.github.io/bmtk/

T2 **Multiscale modeling from molecular level to large network level**

Allen Institute Auditorium, Friday July 13, 09:00 - 16:30

Salvador Dura-Bernal, SUNY Downstate, USA
Robert McDougal, Yale University, USA
William Lytton, SUNY Downstate, USA

Understanding brain function requires characterizing the interactions occurring across many temporal and spatial scales. Mechanistic multiscale modeling aims to organize and explore these interactions to determine how dynamics at one scale alter or are associated with dynamics at other scales. In this way, multiscale models provide insights into how changes at molecular and cellular levels, caused by development, learning, brain disease, drugs, or other factors, affect the dynamics of local networks and of brain areas. Large neuroscience data-gathering projects throughout the world (e.g. US BRAIN, EU HBP, Allen Institute) are making use of these tools – including the NEURON multiscale simulator – to better understand the vast amounts of information being gathered using many different techniques at different scales [1, 2].

This tutorial will present recent multiscale modeling tool development in the NEURON simulator [3], with an emphasis on reaction diffusion intracellular and extracellular modeling (chemophysiology complementing electrophysiology) and simulation of large biophysically detailed networks. The morning session will introduce 1) the basics of single cell modeling using the NEURON simulator and 2) NEURON's Reaction-Diffusion (RxD) module [4, 5]. RxD provides specification and simulation for molecular scale dynamics (genomics, proteomics, signaling cascades and reaction dynamics) coupled with the electrophysiological dynamics of the cell membrane. The afternoon session will introduce 1) basic network modeling in NEURON [6, 7], and 2) NetPyNE, a high-level Python interface (programmatic and GUI-based) to NEURON that facilitates the development, parallel simulation, and analysis of biological neuronal networks [8, 9, 10]. To finish, we will show an example of combining both tools to explore the effects of molecular-level dynamics in a large network.

References

[1] Markram H et al. (2015) Reconstruction and simulation of neocortical microcircuitry. Cell 163:456-492

[2] Hawrylycz M, Anastassiou C, Arkhipov A, Berg J, Buice M, Cain N, Gouwens NW, Gratiy S, et al. (2016) Inferring cortical function in the mouse visual system through large-scale systems neuroscience. PNAS, 113(27):7337-7344

[3] NEURON: https://neuron.yale.edu/

[4] McDougal R, Hines M, Lytton W (2013) Reaction-diffusion in the NEURON simulator. Front. Neuroinform. 7:28

[5] RxD: https://neuron.yale.edu/neuron/static/docs/rxd/index.html

[6] Migliore M, Cannia C, Lytton WW, Markram H and Hines ML (2006) Parallel network simulations with NEURON. Journal of Computational Neuroscience 21:119-129

[7] Lytton WW, Seidenstein AH, Dura-Bernal S, McDougal RA, SchÃ¼rmann F, Hines ML (2016) Simulation neurotechnologies for advancing brain research: parallelizing large networks in NEURON. Neural Comput. 28:2063-2090

[8] NetPyNE: www.netpyne.org

[9] NetPyNE-UI: https://github.com/MetaCell/NetPyNE-UI

[10] Dura-Bernal S, Neymotin SA, Suter BA, Shepherd G, Lytton WW (2018) Long-range inputs and H-current regulate different modes of operation in a multiscale model of mouse M1 microcircuits. bioRxiv 201707

T3 Simulation of large-scale neural networks

UW Medicine SLU Brotman Auditorium, Friday July 13, 09:00 - 16:30

Sacha J. van Albada, Jülich Research Centre and JARA, Germany
Philipp Weidel, Jülich Research Centre and JARA, Germany

This tutorial starts with an introduction to large-scale neuronal networks, giving examples of existing models and identifying some challenges these networks pose for modeling and simulation. This is followed by an introduction to the NEural Simulation Tool (NEST [1]), shedding light on its design principles, which address challenges for large-scale simulations. An overview of the features of NEST is provided, also touching upon advanced properties of neuronal networks like gap-junctions [2]. To familiarize participants with the basic usage of NEST, some simple networks are programmed in hands-on exercises. Next, the tutorial explains how NEST enables parallel simulations via both distributed and threaded computations. Threaded simulations are demonstrated on a cortical microcircuit model [3]. Finally, the tutorial provides an introduction to the NEST Modeling Language (NESTML [4]). In this final hands-on part of the tutorial, the participants learn how to create neuron models in NEST using NESTML.

The tutorial does not assume any prior knowledge of NEST. However, it is recommended that participants install NEST on their laptops beforehand [5]. Furthermore, it is recommended to have VirtualBox installed and to have at least 4 GB of free disk space available.

References

[1] Kunkel S, Morrison A, Weidel P, Eppler JM, Sinha A, Schenck W, Plesser HE (2017). NEST 2.12.0. Zenodo. http://doi.org/10.5281/zenodo.259534

[2] Hahne J, Helias M, Kunkel S, Igarashi J, Bolten M, Frommer A and Diesmann M (2015) A unified framework for spiking and gap-junction interactions in distributed neuronal network simulations Front. Neuroinform. 9:22

[3] Potjans TC, Diesmann M (2014) The cell-type specific cortical microcircuit: relating structure and activity in a full-scale spiking network model. Cereb. Cortex. 24(3):785-806

[4] Plotnikov D, Rumpe B, Blundell I, Ippen T, Eppler JM and Morrison A (2016) NESTML: a modeling language for spiking neurons. arXiv:1606.02882

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

T4 Neuroinformatics resources for computational modelers

Allen Institute 288/289, Friday July 13, 09:00 - 12:00

Padraig Gleeson, University College London, UK

Neuroinformatics resources are becoming an essential part of computational investigations in neuroscience. A movement towards making data and software freely available to the community means that more and more experimental datasets, general purpose analysis tools and infrastructure for computational modelling and simulation are available for computational neuroscientists to help build, constrain and validate their models.

This tutorial will give an overview of the range of neuroinformatics resources currently available to the community. The first half will give a brief introduction to a number of these under the headings; Experimental datasets; Structured data from literature; Analysis tools; Simulation environments; Model sharing; Computing infrastructure; Open source initiatives. The second half of the tutorial will involve hands on exercises where multiple resource will be accessed, data transformed and analysed and new models executed. Note that this tutorial will focus on neuroinformatics resources for cell and network modelling, and not cover the wide range of neuroimaging or genetics databases.

References

[1] Open source at: https://github.com/NeuralEnsemble/NeuroinformaticsTutorial

T5 Modeling and analysis of extracellular potentials

Allen Institute 286/287, Friday July 13, 09:00 - 12:00

Gaute Einevoll, Norwegian University of Life Sciences & University of Oslo, Norway
Espen Hagen, Dept. of Physics, University of Oslo, Norway

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

In the tutorial we will go through - the biophysics of extracellular recordings in the brain, - a scheme for biophysically detailed modeling of extracellular potentials and the application to modeling single spikes [5-7], MUAs [8] and LFPs, both from single neurons [9] and populations of neurons [8, 10-12], - LFPy (LFPy.github.io) [13], a versatile tool based on Python and the NEURON simulation environment [14] (www.neuron.yale.edu) for calculation of extracellular potentials around neurons and networks of neurons, as well as corresponding electroencephalography (EEG) and magnetoencephalography (MEG) signals.

References

[1] KH Pettersen et al., Extracellular spikes and CSD in Handbook of Neural Activity Measurement, Cambridge (2012)

[2] G Buzsaki et al., Nat Rev Neurosci 13:407 (2012)

[3] GT Einevoll et al., Nat Rev Neurosci 14:770 (2013)

[4] GT Einevoll et al., Curr Op Neurobiol 22:11 (2012)

[5] G Holt, C Koch, J Comp Neurosci 6:169 (1999)

[6] J Gold et al., J Neurophysiol 95:3113 (2006)

[7] KH Pettersen and GT Einevoll, Biophys J 94:784 (2008)

[8] KH Pettersen et al., J Comp Neurosci 24:291 (2008)

[9] H Lindén et al., J Comp Neurosci 29: 423 (2010)

[10] H Lindén et al., Neuron 72:859 (2011)

[11] S Leski et al., PLoS Comp Biol 9:e1003137 (2013)

[12] E Hagen et al., Cereb Cortex 26:4461 (2016)

[13] H Lindén et al., Front Neuroinf 7:41 (2014)

[14] ML Hines et al., Front Neuroinf 3:1 (2009)

T6 Single cell RNA-seq analysis for transcriptomic type characterization

Allen Institute 286/287, Friday July 13, 13:30 - 16:30

Zizhen Yao, Allen Institute, USA

Lucas Graybuck, Allen Institute, USA

The functional interplay of neural cell types gives rise to the complex, emergent function of neural tissues. To fully understand the biology of the brain, we need to be able to distinguish and describe these cell types, and identify markers that can be used to selectively label cell types for further study [1]. One scalable and comprehensive method for identifying cell types in the brain is single cell RNA-sequencing. High-quality and large scale scRNA-seq datasets provide data about the expression of thousands of genes from thousands of individual cells. With this starting point, we can perform clustering analyses to identify the cell types of mouse and human brains.

In the first half of this tutorial, we will first give an introduction of single cell RNA-seq technology, with an overview of multiple single cell studies in CNS, and commonly used computational tools. Then, we will focus on the recent comprehensive survey of mouse cortical cell types conducted by the Allen Institute for Brain Science, and give a summary of what we have learned about cell types in this study. In the second half of the tutorial, we will introduce the single cell analysis tools we have developed at the Allen Institute for Brain Science. To enable users to apply our analysis methods to their own datasets, we have developed the scrattch suite for R, which includes scrattch.iterclust (iterative clustering methods), scrattch.vis (data visualization methods), and scrattch.io (file and format handling). In the tutorial, we will demonstrate how these packages can be used to cluster scRNA-seq data generated for 1,679 cells from Tasic, et al. 2016. Nat. Neurosci [2].

References

[1] Poulin JF, Tasic B, Hjerling-Leffler J, Trimarchi JM, Awatramani R. Disentangling neural cell diversity using single-cell transcriptomics. Nat Neurosci. 2016;19(9):1131-1141

[2] Tasic B, Venon M, et al. Adult Mouse Cortical Cell Taxonomy by Single Cell Transcriptomics. Nat Neurosci. 2016; 19(2): 335-346

[3] Tasic B, Yao Z, et al. Shared and distinct transcriptomic cell types across neocortical areas. bioRxiv 229542; doi: https://doi.org/10.1101/229542

[4] Macosko EZ, Basu A, Satija R, et al. Highly parallel genome-wide expression profiling of individual cells using nanoliter droplets. Cell. 2015;161(5):1202-1214

Invited Presentations

Daniel Wolpert *FMedSci FRS,*
Mortimer B. Zuckerman Mind Brain Behavior Institute,
Columbia University,
New York, NY, USA

K1 – Probabilistic models of sensorimotor control and decision making

The effortless ease with which humans move our arms, our eyes, even our lips when we speak masks the true complexity of the control processes involved. This is evident when we try to build machines to perform human control tasks. I will review our work on how humans learn to make skilled movements covering probabilistic models of learning, including Bayesian and structural learning as well as the role of context in activating motor memories. I will also review our work showing the intimate interactions between decision making and sensorimotor control processes. This includes the bidirectional flow of information between elements of decision formations such as accumulated evidence and motor processes such as reflex gains. Taken together these studies show that probabilistic models play a fundamental role in human sensorimotor control.

Rajesh Rao *Hwang Endowed Professor of Computer Science & Engineering and EE*
University of Washington,
Seattle, WA, USA

K2 – The Bayesian brain: from predictive coding to decision making

How can the structure of brain circuits inform large-scale theories of brain function? We explore this question in the context of Bayesian models of perception and action, which prescribe optimal ways of combining sensory information with prior knowledge and rewards to enact behaviors. I will briefly review two Bayesian models, deep predictive coding and partially observable Markov decision processes (POMDPs), and illustrate how circuit structure can provide important clues to systems-level computation.

Nancy Kopell *Professor, Mathematics & Statistics,*
Director, Cognitive Rhythms Collaborative,
Co-Director, CompNet,
Boston University,
Boston, MA, USA

K3 – Coordination, modulation and functional implications of brain rhythms

The neuroscience community is just beginning to understand how brain rhythms take part in cognition and how flexible are the kinds of computations that can be made with rhythms. In this talk, I will discuss some case studies demonstrating this enormous flexibility and important functional implications. Each of the case studies is about some form of coordination. Examples include the interaction of multiple intrinsic time scales in a cortical rhythm in response to a periodic input; the ability of a slow rhythm in the striatum to modulate two other rhythms in different phases of its period; and the ability of a parietal rhythm to guide the formation, manipulation and termination of a kind of working memory.

Eve Marder *Professor of Biology,*
Member, US National Academy of Sciences,
Volen National Center for Complex Systems,
Brandeis University,
Waltham, MA, USA

K4 – Differential resilience to perturbation of circuits with similar performance

Experimental work on the crustacean stomatogastric ganglion (STG) has revealed a 2-6 fold variability in many of the parameters that are important for circuit dynamics. At the same time, a large body of theoretical work shows that similar network performance can arise from diverse underlying parameter sets. Together, these lines of evidence suggest that each individual animal, at any moment in its life-time, has found a different solution to producing “good enough” motor patterns for healthy performance in the world. This poses the question of the extent to which animals with different sets of underlying circuit parameters can respond reliably and robustly to environmental perturbations and neuromodulation. Consequently, we study the effects of temperature, pH, high K+, and neuromodulation on the pyloric rhythm of crabs. While all animals respond remarkably well to large environmental perturbations, extreme perturbations that produce system “crashes” reveal the underlying parameter differences in the population. Moreover, models of homeostatic regulation of intrinsic excitability give insight into the kinds of mechanisms that could give rise to the highly variable solutions to stable circuit performance.

Contributed Talks

F1 Predictive computations in the primary visual cortex

Jan Homann*, Michael Berry, Sue-Ann Koay, Alistair M. Glidden, and David W. Tank

Princeton University, Department of Neuroscience, Princeton, NJ, United States

Predictions about the future are important for an animal in order to interact with its environment. Therefore, predictive computation might be a core operation carried out by neocortical microcircuits. We explored whether the primary visual cortex can perform such computations by presenting repeated temporal sequences of static images with occasional unpredictable disruptions. Simultaneous recordings of 150-250 neurons were performed using two-photon Ca++ imaging of layer 2/3 neurons labeled with GCaMP6f in awake mice, who were head-fixed but free to run on a styrofoam ball. In our visual stimuli, each spatial frame consisted of either an oriented grating or a random superposition of Gabor filters.

We found that most of the neurons ($\sim$98%) showed a strong reduction in activity over a few repeats of the temporal sequence. When we presented a frame that violated the temporal sequence, these neurons responded transiently. In contrast, a small fraction ($\sim$2%) had activity that ramped up over several repeats, before reaching a steady, sequence-modulated response. This partitioning of the neural population into transient and sustained responses was observed for all temporal sequences tested. At the same time, the identity of which neurons were transient versus sustained depended on the temporal sequence.

These features – adaptation to a repeated temporal sequence and a transient response to a sequence violation – are hallmarks of predictive coding. After a few repeats, the temporal sequence becomes predictable and can be efficiently represented by a small subset of the neural population. The unpredictable frame then elicits an error signal because it encodes a potentially important novelty. In order to explore whether neural novelty signals could be useful to the animal, we performed behavioral experiments with matched visual stimuli that demonstrated that mice could easily learn to lick in response to a violation of an ongoing temporal sequence.

F2 Response to deep brain stimulation in essential tremor: predictions beyond noisy data with a Wilson-Cowan model

Benoit Duchet[1]*, Gihan Weerasinghe[1], Christian Bick[2], Hayriye Cagnan[1], and Rafal Bogacz[1]

[1]*University of Oxford, Nuffield Department of Clinical Neurosciences, Oxford, United Kingdom*
[2]*University of Oxford, Mathematical Institute, Oxford, United Kingdom*

Thalamic deep brain stimulation (DBS) is a therapy option for Essential tremor (ET), the most common movement disorder. Clinically available DBS delivers constant, high frequency electrical stimulation and could be improved in terms of efficacy, reduction of side effects, and decrease in power usage.

Given phased locked stimulation data, we propose a method to study the effects of stimulation along both the tremor oscillation phase axis and the tremor oscillation amplitude axis, with the goal of better informing stimulation strategies. Because of noise in tremor recordings and experimental limitations, the amplitude axis is especially difficult to access by direct data analysis in the phasic paradigm. We show that a Wilson-Cowan model can be fitted to data, and thanks to isochronal and isostable coordinates, we obtain response curves and surfaces for the noiseless model. The noiseless 2D phase response curves and amplitude response curves show good agreement with the response curves obtained directly from experimental data. The 3D response surfaces give us the ability to make predictions beyond what the noise level of the data can let us see. In that sense, our method can be seen as a way of de-noising the experimental response to stimulation. Although mathematically inspired by a canonical neuroscience model, our model includes the various neural populations thought to be involved in the generation of ET, and allows for the stimulation of the most common target for ET DBS, the ventral intermediate nucleus of the thalamus.

Our model predicts that only certain phases are conducive to amplitude reduction through stimulation, the best of which being the phase that brings the system closer to the fixed point, where there are no pathological oscillations. This particular phase is amplitude dependent, but in general the optimal stimulation phase occurs during the descending part of the oscillations, slightly before the trough. Moreover, the response to stimulation is linearly dependent on stimulation magnitude. We also find that the best phase to stimulate corresponds to the maximum positive slope of the PRC. Finally, we report that the effects of stimulation are reduced as the amplitude of the oscillations increases, and therefore predict that phasic stimulation will be less effective when delivered at higher oscillation amplitudes.

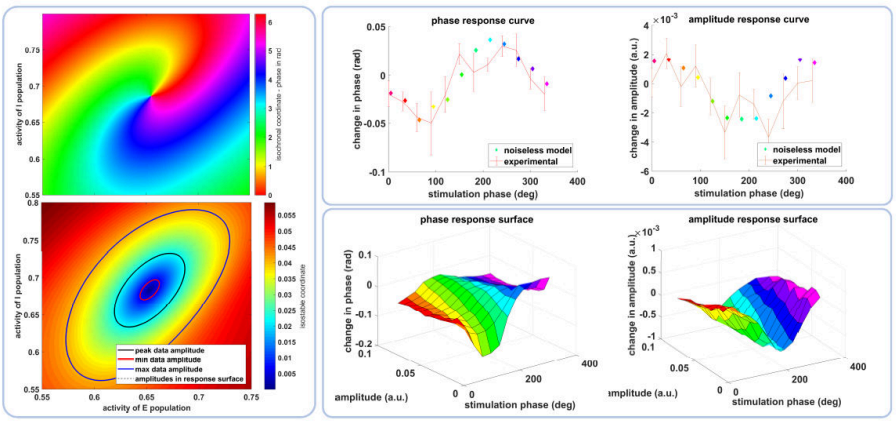


Figure 1: Response curves and surfaces from isochronal and isostable coordinates for patient 1. The model response curves agree with experimental data.

F3 A molecular odorant transduction model and combinatorial encoding in the Drosophila Antennae

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A key functionality of olfactory sensory neurons (OSNs) in the *Drosophila* antennae is to jointly encode both odorant identity and odorant concentration. The identity of an odorant is combinatorially encoded by the set of responding OSN groups expressing the same receptor type, and the size of OSN set varies as the concentration changes. The temporal response of an OSN simultaneously represents the information of odorant concentration and concentration gradient. These two aspects of olfactory coding, *identity* and *concentration*, originate in the odorant transduction process. However, detailed molecular models of the odorant transduction process are scarce for fruit flies.

To address these challenges we advance a comprehensive model of fruit fly OSNs as a cascade consisting of an odorant transduction process (OTP) and a biophysical spike generator (BSG). We model identity and concentration in OTP by an odorant-receptor binding rate tensor modulated by the odorant concentration profile and an odorant-receptor dissociation rate tensor, and quantitatively describe the ligand binding/dissociation process.

To biologically validate our modeling approach, we first propose an algorithm for estimating the affinity and the dissociation rate of an odorant-receptor pair. We then apply the algorithm to electrophysiology recordings and estimate the affinity and dissociation rate for three odorant-receptor pairs, (*acetone*, *Or59b*), (*methyl butyrate*, *Or59b*), and (*butyraldehyde*, *Or7a*). Second, we evaluate the temporal response of the OSN model with a multitude of stimuli, including step, ramp and parabolic odorant waveforms for all three odorant-receptor pairs. The output of the model closely reproduces the temporal responses of OSNs obtained from *in vivo* electrophysiology recordings for all three odorant-receptor pairs across all three types of stimuli. Lastly, we evaluate the model at the OSN antennae population level. We first empirically estimate the odorant-receptor affinity using the spike count records in the DoOR database for 24 receptor types in response to 110 odorants. With estimated affinity values, we simulate the temporal response of the OSN population to staircase odorant waveforms. The output of simulated OSN population demonstrates that the odorant identity is encoded in the set of odorant-activated OSN groups expressing the same receptor type, and, more importantly, the size of the set expands or reduces as the odorant concentration increases or decreases.

The fruit fly OSN model presented here provides a theoretical foundation for understanding the neural code of both odorant identity and odorant concentration. It advances the state-of-the-art in a number of ways. First, it models on the molecular level the combinatorial complexity of the transformation taking place in *Drosophila* antennae OSNs. The resulting *concentration-dependent combinatorial code* determines the complexity of the input space driving olfactory processing in the downstream neuropils, such as odorant recognition and olfactory associative learning. Second, the model is biologically validated using multiple electrophysiology recordings. Third, the OSN model demonstrates that the currently available data for odorant-receptor responses only enables the estimation of the affinity of the odorant-receptor pair. Our model calls for new experiments for massively identifying the odorant-receptor dissociation rates of relevance to flies.

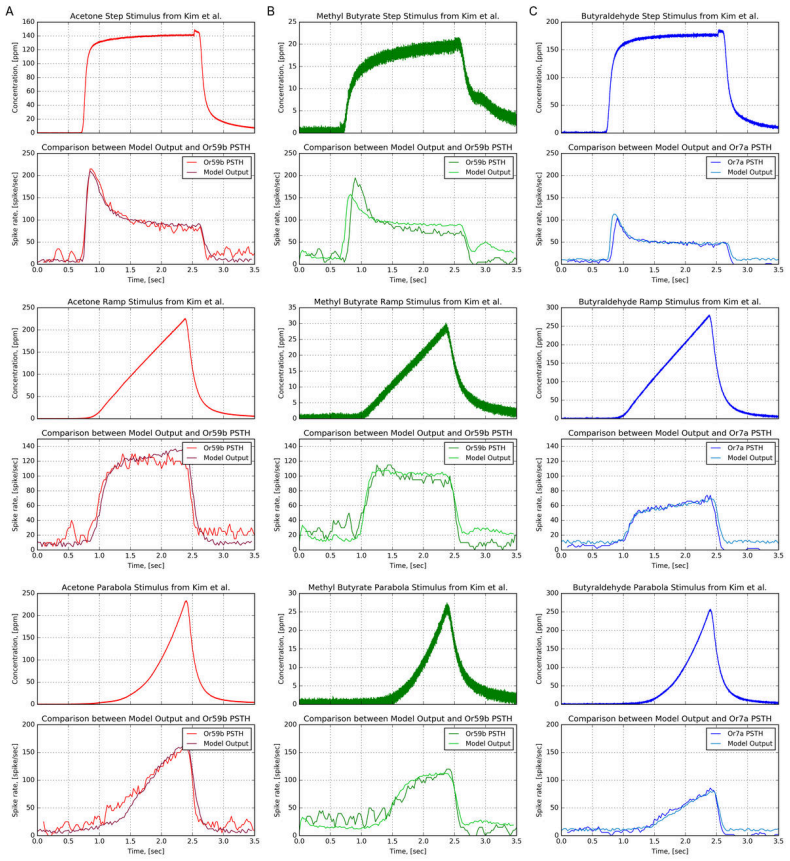

Figure 1: Characterization of the fruit fly OSN model with multiple odorants and receptor types. Three odorant-receptor pairs are tested. (A) (Or59b, acetone) (B) (Or59b, methyl butyrate). (C) (Or7a, butyraldehyde). (Odd rows) Stimuli. (Even rows) PSTH from the model output and experimental recordings.

O1 Generative model of visual cortex with short- and long-range recurrent interactions

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In V1, neuronal responses are sensitive to context: responses to stimuli presented within the classical receptive field are modulated by stimuli in the surround. Recently, sparse coding models [1] have been successful in explaining part of these modulatory effects [2]: Their dynamics implements an inference process to seek an optimal (w.r.t. accuracy and sparseness) representation of a visual input in terms of fundamental features. This is achieved through a competition between similarly tuned neurons with overlapping input fields, which also mediates contextual modulation.

However, this connection scheme implies that neurons with non-overlapping input fields do not interact. Therefore, the proposed mechanism does not provide a satisfactory explanation of the mechanisms behind these phenomena, since contextual effects are usually caused by surround stimuli positioned far from the cRF (e.g. Mizobe et al 2001 report collinear modulation for distance center-surround up to 12 deg). To overcome this limitation, we propose an extension of the classical framework [2] by defining a new generative model for visual scenes that includes dependencies among different features in spatially well-separated locations. To perform inference in this model, we also derive a dynamical system that can be mapped to a neural circuit and a lateral connection scheme for optimally processing local and contextual information.

The result can be interpreted as a neural network where units are linked by short range horizontal connections within the same hypercolumn and by long range connections between different hypercolumns (Fig. 1b). Each hypercolumn contains units that receive input from a localized region of the visual field and builds a sparse representation of its input as if it was presented in isolation. In parallel, these local representations are combined by providing contextual information to each other. In our simulations connections are learned from natural images. Long-range connections reflect the co-occurrence of features in different visual field locations: this predicts a connectivity structure linking neurons with similar orientation and spatial frequency preferences, which is similar to the typical patterns found for long-ranging (3-4mm) horizontal axons in visual cortex [3]. Subjected to contextual stimuli typically used in empirical studies, our model replicates several hallmark effects of contextual processing. Hereby local and long-range interactions act hand-in-hand, for example in realizing two different origins of near and far surround suppression, respectively [4]. In summary, our model provides a novel framework for contextual processing in the visual system proposing a well-defined functional role for horizontal axons.

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Acknowledgements

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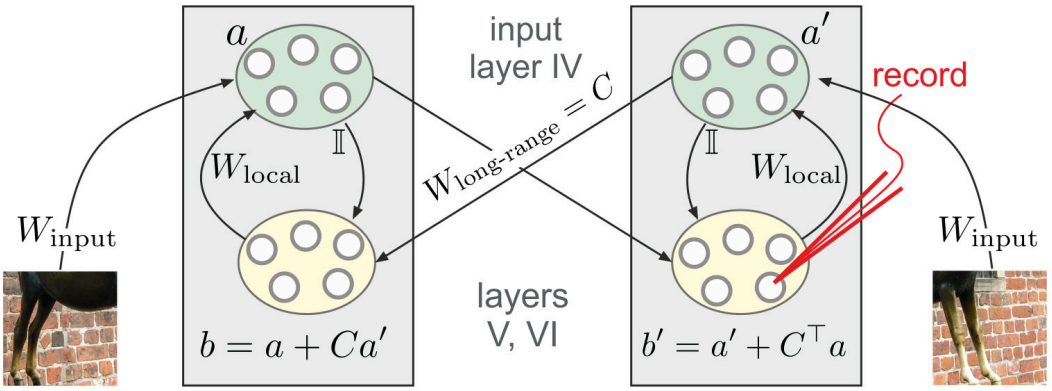

Figure 1: (A) Example of stimuli from a natural scene (top) and dictionary of fundamental features (bottom) (B) Scheme of the generative model (C) Network architecture to perform inference in the generative model

O2 Info in a bottleneck: exploring the compression of visual information in the retina

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The retina is organized in convergent and divergent layers that compress and expand signals before passing visual information along to the brain. Receptive fields anatomically correspond to the collection of inputs that converge upon a single retinal output cell. This subunit circuit structure produces an information bottleneck because information is compressed along the pathway to an output neuron. We wondered whether the structure of the retina combined with its adaptation properties serve to preserve information given this bottleneck.

A remarkable property of the retina is its ability to adapt its processing to environmental conditions. Adaptation to background luminance shifts the nonlinear response filters of the subunits over a timescale of about a minute. This has the effect of adjusting the linearity of responses in a manner that is dependent on the luminance environment. Another feature of the retina is the diversity of cell types present at the output layer. Within types, there are ON and OFF versions of cell types which have sensitivities that are complementary but not symmetrical. Having complementary cell types combined with adaptation mechanisms may allow the retina to leverage these redundancies under certain conditions while having the flexibility to adapt to an efficient or predictive code in other conditions. We want to know whether the retina adapts its processing to maximize visual information transmission by adjusting the subunit response functions in the circuit.

To quantify the amount of information that is preserved in the signals exiting the retina under this kind of set up, we estimate the mutual information between a naturalistic stimulus set and the output from our model retina circuit. We use a binless estimator to account for the fact that the input signals and the outputs are continuous. Consistent with past studies, our preliminary results indicate that the optimal thresholds for the nonlinear subunits depend on the amount of input noise given a naturalistic distribution of stimulus contrasts. Our work builds on past studies by incorporating the known subunit structure into the circuit which produces information compression. Under circumstances where subunits receive independent inputs, rather than correlated inputs, the circuit is optimal when ON and OFF subunits redundantly encode the most prevalent stimuli for a broad range of subunit noise levels. Our preliminary results suggest novel ways in which adaptation mechanisms, along with the particular bottleneck structure of the retina, enable the retina to adapt the computations it produces in different contexts.

O3 Structural and dynamical properties of local cortical networks result from robust associative learning

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Many ubiquitous features characterize the structure and dynamics of local cortical networks. At the level of pairwise connectivity, it is known that the probabilities of excitatory connections are generally lower than those for inhibitory, and the majority of reported probabilities lies in the $0.10 - 0.19$ range if the presynaptic cell is excitatory and $0.25 - 0.56$ range if it is inhibitory. It is also known that the distributions of connection weights have stereotypic shapes with the majority of measured coefficients of variation (CV) of unitary postsynaptic potentials in the $0.85 - 1.1$ range for excitatory connections and slightly lower values for inhibitory, $0.78 - 0.96$. At the level of connectivity within 3-neuron clusters, several overrepresented connectivity motifs have been discovered. Information becomes scarce as one considers larger clusters of neurons, but even here deviations from random connectivity have been reported for clusters of 3-8 neurons. Similarly, many universal features characterize activity of neurons in local cortical networks. For example, individual neurons exhibit highly irregular spiking activity, resembling Poisson processes with close to one CV in inter-spike-intervals. Spike trains of nearby neurons are only marginally correlated, $0.04 - 0.15$, and, at the network level, spiking activity can be described as sustained, irregular, and asynchronous.

In this study, we pursue a hypothesis that associative learning alone is sufficient to explain these network features. To test this hypothesis, we trained recurrent networks of excitatory and inhibitory McCulloch and Pitts neurons [1,2] on memory sequences of varying lengths and compared network properties to those observed experimentally. Learning in the network is mediated by changing connection weights in the presence of biologically inspired constraints. (1) Input connection weights of each neuron are sign-constrained to be non-negative if the presynaptic neuron is excitatory and non-positive if it is inhibitory. (2) Input weights of each neuron are homeostatically constrained to have a predefined *l1-norm*. (3) Each neuron must attempt to learn its associations robustly, so that they can be recalled correctly in the presence of a given level of postsynaptic noise. We explore structural and dynamical properties of associative networks in the space of these constraints, and show that there is a unique region of parameters that is consistent with all of the above-described experimental observations. In this region, local cortical circuits are loaded with associative memories close to their capacity and memories can be successfully retrieved even in the presence of noise comparable to the baseline variations in the postsynaptic potential, which provides an independent validation of the theory in terms of the hypothesized network function. Confluence of these results suggests that many structural and dynamical properties of local cortical networks are simply a byproduct of associative learning.

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O4 Reduced models of an attractor neural network's response to conflicting external inputs

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The theory of attractor neural networks has been influential in our understanding of the neural processes underlying spatial, declarative, and episodic memory. Many theoretical studies focus on the inherent properties of an attractor, such as its structure and capacity. Relatively little is known about how an attractor neural network responds to external inputs, which often carry conflicting information about a stimulus. In this talk I will present analytical results concerning the behavior of an attractor neural network's response to conflicting external inputs. My focus is on analyzing the emergent properties of the megamap model, a quasi-continuous attractor network in which place cells are flexibly recombined to represent a large spatial environment (Hedrick and Zhang, 2016). In this model, the system shows a sharp transition from the winner-take-all mode, which is characteristic of standard continuous attractor neural networks, to a combinatorial mode in which the equilibrium activity pattern combines embedded attractor states in response to conflicting external inputs. I derive a numerical test for determining the operational mode of the system a priori. I then derive a linear transformation from the full model to a reduced 2-unit model that has similar qualitative behavior. The analysis of the reduced model and explicit expressions relating the parameters of the reduced model to the megamap elucidate the conditions under which the combinatorial mode emerges and the dynamics in each mode given the relative strength of the attractor network and the relative strength of the two conflicting inputs. Although my focus on a particular attractor network model, I describe a set of conditions under which the reduced model can be applied to more general attractor neural networks.

The reduced 2-unit model captures the amplitude of each activity bump but not its radius. I extend this reduced model to examine the spatial effects on the system's behavior by approximating the activity bump and recurrent connections using two-dimensional Gaussian tuning curves. Analysis of this reduced model reveals that these spatial effects underlie the nonlinearities observed in the full megamap model but not in the reduced 2-unit model. I compare these results to numerical simulations and electrophysiological data from an experiment in which hippocampal place cells resolve conflicting external inputs from the medial entorhinal cortex (MEC) and lateral entorhinal cortex (LEC) when local and global cues are rotated in opposite directions (Knierim and Neunuebel, 2016). In this experiment, place cells in the CA3 (which are believed to form attractor neural networks) coherently follow the noisy inputs from the LEC rather than the much stronger spatial inputs from the MEC. The reduced model predicts that this surprising response is due to three factors: (1) CA3 place cells are initially driven by the LEC input only, (2) the attractor network acts in the WTA mode, and (3) connections from MEC to CA3 are governed by fast Hebbian synaptic plasticity. To bridge the gap between the idealistic theory and the noisy electrophysiological data, I run numerical simulations using the conductance-based integrate and fire model and unsupervised Hebbian plasticity. The noise in the model leads to the partial remapping observed experimentally.

O5 Topologies of repetitive functional network motifs vary dynamically with age in the developing human brain: Evidence from very high-dimensional invasive brain signals

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Throughout the course of the day, or even an hour, functional brain networks are continuously recruited to process thousands of inputs from the outside world and respond to the demands of countless behaviors and cognitive processes. Across scales of organization, these networks' small-world and scale-free topologies facilitate optimally efficient neural information processing. However, the building blocks of these networks (modules or motifs), their emergence, re-organization during development and time-dependent stereotypy remain poorly understood. Unrelated theoretical work has shown that specific network patterns emerge as a result of a dynamic system's propensity towards a stable configuration. There is also growing evidence from both animal and human studies that a relatively small number of such modules are combined (in potentially infinite ways) to give rise to the observed functional network topologies. In this study, we investigated the organization, size and stereotypy of functional network motifs in the developing human brain, using very high-dimensional invasive human electrophysiological signals, collected continuously over long periods of time (typically several days) from a relatively large number of children and young adults (n = 39, age $<$1 to 23 years) with intracerebral electrode grids covering different parts of the brain. All patients had recordings from a relatively large number ($>$70) of electrodes. Information theoretic and contraction theoretic measures were used to estimate functional connectivity, identify sub-network patterns (motifs) that occurred repetitively over time and independently of the area of the brain being spatially sampled, and characterize their stability (using an eigenvalue analysis).

A relatively small number of functionally active nodes were estimated, which formed stable patterns that occurred repetitively across temporal scales and brain regions. The size of these patterns (number of activated nodes) changed with age, with progressively smaller sub-graphs (3-4 nodes) emerging as a function of neural maturation. Across ages, identified motifs were consistently correlated with network stability. These results indicate the although stable functional network motifs may be in place early in life to process multi-modal sensory information, re-organization of the brainâ€™s neural circuitry as a function of neural maturation may lead to increasingly parsimonious modules to facilitate increasingly efficient neural information processing. These modules may also constitute a network-level biomarker of neural maturation at the macroscale sampled by invasive human recordings.

O6 Revealing principles of cortical computation using the Allen Brain Observatory: A large, standardized calcium imaging dataset from the mouse visual cortex

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A prominent question of sensory processing is how information is represented and transformed by the neural circuit through multiple layers and across multiple areas in order to create perceptions and ultimately guide behavior. In order to facilitate uncovering these principles, we have created the Allen Brain Observatory. This is a public dataset of neural responses collected from visual areas of awake mouse cortex using 2-photon calcium imaging. We systematically recorded responses from over 50,000 neurons in over 500 experiments, using a high-throughput imaging pipeline. Data were collected from 6 cortical areas and 4 cortical layers. GCaMP6f was transgenically expressed, driven by 13 different Cre lines which limit expression to specific subsets of excitatory (10 Cre lines) or inhibitory cells (3 Cre lines). Visual responses were imaged in response to an array of both artificial and natural stimuli, including drifting gratings, static gratings, locally sparse noise, natural scenes and natural movies while the mouse was awake and free to run on a running disc. Several metrics were computed to describe the visual responses of the neurons, including orientation and direction selectivity, image selectivity, lifetime sparseness, and receptive field areas.

Surveying these metrics across areas, layers and Cre-defined cell populations, several patterns emerge. Layer 4 exhibited clear differences across areas and cell populations, but these differences were reduced in the other layers. This pattern is consistent with layer 4 predominately carrying feedforward thalamocortical input, while layers 2/3, 5 and 6 represent higher order responses.

One of the most striking results in this dataset is the small numbers of responsive cells and the remarkable variability of the responses of these cells. Only 57% of cells in the Brain Observatory dataset respond to any of the visual stimuli presented. Further, even responsive cells show large trial-to-trial variability. We fit these neurons to a simple wavelet pyramid model with simple (linear-nonlinear) and complex components (the “energy” model). Roughly 15% of neurons in the dataset show significantly predictable responses to visual stimuli via this model, with relatively low explainable variance. All cells also show some degree of “complex” behavior, ie. there are no purely “simple” cells according to this model. We compare the representations in each layer and area to responses generated by standard Convolutional Neural Networks, a model derived from the canonical understanding of the cat visual system. We find that the mouse cortex are most similar to early middle areas of ConvNets, rather than the initial Gabor-like layer thought to describe responses in V1 of cats.

Finally, we examine the correlation structure of population activity, showing that correlations in neural responses have an impact on information transmission in an area and layer dependent fashion. Furthermore, we show that the “noise” and “signal” correlations are positively correlated throughout the mouse visual system, providing strong evidence against certain types of theories that exhibit “explaining away”, ie. theories in which neurons with similar mean tuning properties will functionally inhibit one another, such as the sparse coding model of Olshausen and Field and some probabilistic coding models. This dataset provides a testbed for theories of cortical computations and will be a valuable resource for the community.

O7 Characterization of the brain's dynamical repertoire in the psychedelic state

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Brain activity can be understood as the exploration of a dynamical landscape of activity configurations over both space and time. This dynamical landscape may be defined in terms of spontaneous transitions within a repertoire of discrete metastable states of functional connectivity (FC), or "FC states", which underlie different mental processes. It however remains unclear how the brain's dynamical landscape might be disrupted in altered states of consciousness, such as the psychedelic state. The present study investigates changes in the brain's dynamical repertoire in a rare fMRI dataset consisting of healthy participants intravenously injected with the psychedelic compound psilocybin; the active compound in magic mushrooms. We employed a data-driven approach to study brain dynamics in the psychedelic state, which focuses on the dominant FC pattern captured by the leading eigenvector of dynamic FC matrices, and enables the identification of recurrent FC patterns ("FC-state"), and their transition profiles over time. We found that a FC state closely corresponding to the fronto-parietal control system was strongly destabilized by the drug, while transitions toward a globally synchronized FC state were enhanced. These differences between brain state trajectories in normal waking consciousness and the psychedelic state suggest that psilocybin induces an alternative type of unconstrained functional integration at the expense of locally segregated activity specific networks supporting executive function. These results provide a mechanistic perspective on the acute psychological effects of psychedelics, and further raise the possibility that mapping the brain's dynamical landscape may help guide pharmacological interventions in neuropsychiatric disorders.

O8 Understanding the bispectrum as a measure of cross-frequency coupling

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Interest in the origin and significance of cross-frequency coupling in electrophysiological signals has grown rapidly over the last several years, with particular emphasis on phase-amplitude coupling (PAC). Much of this recent attention has focused on measures of PAC obtained from filtered analytic signals through the comparison of phase and analytic envelope. As use of these measures has increased, so has an appreciation of their ambiguities, attested by an expanding cautionary literature on the topic.

Meanwhile, “classical” statistically motivated measures of cross-frequency coupling derived from spectral representations of higher moments have remained at the periphery of the latest surge of attention, due in large part to a common perception that such measures are comparatively difficult to interpret and that they relate to a form of cross-frequency coupling distinct from PAC. Recently, we have shown that common PAC measures are, in fact, fundamentally normalized bispectral estimators which yield smoothed estimates of the true signal bispectrum [1]. Differences between the measures relate to properties of the respective smoothing kernels. In light of this observation, classical bispectral estimators can claim a number of advantages over recently introduced PAC measures, including more favorable bias properties and freedom from the constraints on range and resolution that are inherent in PAC measures.

Interpretation of the bispectrum is commonly explained in terms of “quadratic” phase coupling between spectrally narrow signal components; in demonstrating the relationship to PAC measures, we develop an alternative approach to interpretion through a decomposition of the signal into spectrally broad transient components. The relationship between PAC measures and the bispectrum can be understood by considering the case of a low-frequency transient, corresponding to the “slow” oscillation (SO), accompanied by a transiently windowed high-frequency “fast” oscillation (FO). As detailed in Figures 1 and 2, windowing of the FO at the scale of the SO implies that the the bispectrum contains a straightforward representation of the spectrum of the SO and the power spectrum of the FO, from which both might be directly recovered to good approximation. Moreover, within the range of the FO, the phase bispectrum encodes the relative delay between the SO and the FO modulating window. With these insights we develop guidelines for the evaluation of PAC from bispectral statistics. This framework addresses a number of the recently identified limitations and ambiguities of PAC measures.

Finally, some extensions of this framework towards the blind recovery of recurring transient signal features are briefly considered. The feasibility of this application is demonstrated through the identification of auditory evoked responses in human intracranial recordings from both controlled stimuli (click trains) and uncontrolled ecologically meaningful stimuli (a video soundtrack) with no foreknowledge of the stimulus.

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O9 Spinal interneurons and locomotor speed and gait control in quadrupeds

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To effectively move in a complex and dynamic environment, limbed animals should vary locomotor speed and adapt gaits to the desired speed and the environment. With increasing locomotor speed, quadrupedal animals, including mice, switch locomotor gait from walk to trot and then to gallop and bound. Centrally, the locomotor gaits are controlled by interactions between four central pattern generators (CPGs) located on the left and right sides of the lumbar and cervical enlargements of the cord, and each producing rhythmic activity controlling one limb. The activity of these CPGs are coordinated by commissural interneurons (CINs), projecting across the midline to the contralateral side of the cord, and by long propriospinal neurons (LPNs) that connect the cervical and lumbar CPG circuits in both directions.

We use computational modeling to investigate how the CIN and LPN connections between the cervical and lumbar, left and right CPGs can be organized and what roles different CIN and LPN pathways play in the control and speed-dependent expression of different gaits. Our model contains four rhythm generators (RGs) with left-right cervical and lumbar CIN interactions and homolateral and diagonal ascending and descending LPN interactions. These interactions are organized via several interneuronal pathways mediated by genetically identified neuron types and are based on their suggested functions and connectivity. Supraspinal (brainstem) drives excite all RGs, thereby controlling oscillation frequency, and inhibit some CINs and LPNs, which allows the model to reproduce the speed-dependent gait transitions observed in the intact mice [1].

The model reproduces the experimentally observed loss of particular gaits after selective removal of genetically identified neurons (V2a, V0V, or all V0) and the speed-dependent disruption of hind limb coordination after deletion of ascending (cervical-to- lumbar) LPNs [2]. The model suggests that (1) V0D and V0V CINs together secure left-right alternation, whereas V3 CINs promote left-right synchronization, and that (2) V0D LPNs support diagonal alternation, whereas V0V LPNs promote diagonal synchronization. Thus, V0D CINs and LPNs together stabilize walk and V0V CINs and LPNs stabilize trot. The transition from trot to gallop and bound occurs when the activity of V3 CINs overcomes the activity of (brainstem-drive inhibited) V0V CINs and diagonal LPNs.

Our simulations have also shown that external inputs to CINs and LPNs, other than supraspinal drives controlling locomotor frequency, can induce gait changes independent of speed. These inputs may represent activities of sensory afferents, which is consistent with multiple experimental data showing that CINs and LPNs receive direct and indirect inputs from sensory afferents. Based on the results of these simulations we suggest that CINs and LPNs represent the main neural targets for different local/intraspinal, supraspinal, and sensory inputs to control interlimb coordination and adjust locomotor gait to various internal and external conditions.

The model proposes a series of testable predictions, including the anticipated effects of the deletion of particular identified types of CINs and LPNs, and can be used as a test bed for simulating various spinal cord perturbations and injuries.

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O10 A simplified model of network bursts in the pre-Botzinger complex

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Network (population) bursts are a signature neuronal activity in a critical brainstem region for respiratory rhythm generation, the pre-Botzinger complex (pre-BotC). During the initiation of a network burst, the pre-BotC shows a consistent pattern of dynamic transitions. Starting with mostly silent neurons, the pre-BotC transitions to an intermediate state with a positive fraction of firing neurons that may include tonically spiking and bursting neurons. When a sufficient number of neurons becomes engaged in firing, the pre-BotC network finally undergoes a transition to a population burst, characterized by a high fraction of simultaneously bursting neurons.

Over the last few decades several models of population bursts in the pre-BotC have been proposed, including conductance-based models featuring various ionic currents, such as INaP and ICAN. While the main objective of these models was to identify the bio-physical driving sources underlying network burst initiation, the role of the synaptic connection patterns in shaping neuronal activity has been relatively overlooked. The main reason for this omission is that the models are too complicated for a full analytical treatment and, due to computational limitations, it is difficult to gain full insight into the influence of connectivity.

To overcome these obstacles, we propose a simplified model, which is based on a bootstrap percolation process, and is defined as follows. For a given graph, every node has three possible states: inactive, weakly- active, and fully-active, which correspond to silence, tonic spiking and bursting, respectively. We initialize all nodes to the weakly-active state with probability p1 and to the fully-active state with probability p2, independently of other nodes. As the process evolves, an inactive node will transit to the weakly-active state if the amount of activity among its neighbors exceeds a threshold k1, and if the amount is greater than k2, it will transit to the fully-active state. Similarly, a weakly-active node becomes fully-active if the amount of activity among its neighbors exceeds k2. Nodes cannot reduce their activity levels, and those nodes that are fully-active will not change their states until the end of a trial.

We analyze this process analytically and computationally on various random graph models and address three questions. First, we determine values p1 and p2 as functions of k1 and k2 for which the network reaches a population burst at the end of a trial. Our findings suggest possible reasons why the network may fail to generate a population burst after the deletion of a fixed fraction of arbitrary nodes in the network, which is consistent with laser ablation of rhythmogenic pre-BotC (Dbx1) neurons in experiments. Second, we investigate how structural features of different graph models affect the duration of the process. Lastly, we describe how using nodal measures we may identify nodes that, when activated initially, are particularly well suited to ignite a population burst. This result shows that local properties of graphs are good descriptors of the spread of bursting activity and also addresses the extent to which successive population bursts may feature similar or different initiation mechanisms.

O11 Traveling waves in single cortical regions: mechanisms and emerging computational principles

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With new multichannel recording technologies, neuroscientists can now record from single cortical regions with high spatial and temporal resolution. Early recordings during anesthesia found spontaneous and stimulus-evoked waves traveling across single cortical regions. For a long time, however, these waves were thought to disappear in awake animals and during high-input regimes. By introducing new signal processing methods for moment-by-moment detection and characterization of spatiotemporal patterns under noise, our recent work has found that small visual stimuli evoke waves traveling out from the point of thalamocortical input to primary visual cortex in the awake monkey [1]. Further, using a measure of directed information transfer across recording sites in V1 of anesthetized monkey, another group has found that traveling waves can influence intracortical dynamics during viewing of natural stimuli [2]. These results indicate that traveling waves can play a role in organizing neural activity during natural sensory processing. Their overall computational role in sensory cortex, however, remains poorly understood.

Here, we introduce a spiking model that captures a general network-level mechanism for traveling waves in cortex. We study networks in the self-sustained activity regime [3], where conductance-based networks of neurons can create an internally generated noise [4] consistent with the irregular-asynchronous (IA) background activity state in cortex [5]. We find that a microscopic property – the axonal conduction velocity – profoundly controls the spatiotemporal structure of the spontaneous background state. While previous work has generally considered the time delays from intraregional recurrent fibers to be negligible, these can range up to tens of milliseconds over a few millimeters of the cortical surface, and their inclusion shapes self-sustained activity patterns into spontaneous traveling waves matching those observed in recordings from cortex. By studying networks from 104 to 106 neurons through a range of connectivity regimes, from very sparse (100 synapses/cell) to that found in cortex (10,000 synapses/cell, [6]), we identify spatiotemporal patterns ranging from *dense waves*, where the fraction of individual neurons participating in a passing wave is nearly unity, to *sparse waves*, where this fraction becomes very low. The sparse wave regime offers a unique operating mode, where many waves can coexist while weakly interacting during their propagation across the network. Finally, in collaboration with the laboratory of John Reynolds (Salk Institute), we show how spontaneous, sparse traveling waves can affect visual processing in the awake marmoset, leading to dynamic shifts in perceptual thresholds.

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O12 Excitable dynamics of NREM sleep: a unifying model for neocortex and hippocampus

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During non-rapid eye movement (NREM) sleep, the neocortex continuously alternates between states of neuronal spiking (UP states) and inactivity (DOWN states). Similarly, the hippocampus also shows continuous alternations between brief periods of neuronal activity (SPW-Rs) and relative inactivity. While the durations of active/inactive states are dramatically different in the two regions, the hippocampus and neocortex are both cortical tissue and are under similar neuromodulatory influence during NREM. Thus, it prompts one to wonder whether the neocortical UP/DOWN states and hippocampal SPW-Rs might be explained by similar mechanisms. Furthermore, the mechanisms by which alternation dynamics in the two regions interact to support NREM function are unclear. To address these questions, we used an idealized firing rate model of UP/DOWN alternations with four distinct dynamical regimes, which are distinguished by the stability or transience of UP/DOWN states and encompass those seen in previous studies. By directly matching model dynamics with experimental observations in naturally-sleeping rats, we found that the alternation dynamics observed in neocortex and hippocampus during NREM reflect two distinct regimes of excitable activity that show characteristically asymmetric durations of UP/DOWN states. Specifically, we find that the neocortical dynamics reflect a stable UP state interrupted by transient DOWN states (slow waves), while the hippocampal dynamics reflect a stable DOWN state with transient UP states (sharp waves). We further considered the effects of including an inhibitory population in the model. We find that under conditions of balanced excitation and inhibition, neocortical UP->DOWN transitions can be evoked by excitatory input and are followed by a high frequency oscillation at the DOWN->UP transition, as is observed in vivo. We propose that during NREM sleep, hippocampal and neocortical populations are in excitable states, from which small fluctuations can evoke the transient events that support NREM function. The excitable dynamics we describe suggest a mechanism by which the two structures could show a form of communication through “stochastic synchronization" of spontaneous population events during NREM sleep.

O13 Biological mechanisms for learning: A computational model of olfactory learning in the Manduca sexta moth

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The moth olfactory network, which includes the antennal lobe (AL), mushroom body (MB), and ancillary structures, is a relatively simple biological neural system that is capable of learning. Its structural features include motifs that are widespread in biological neural systems, such as a cascade of networks, large dimension shifts from stage to stage, sparsity, noise, and randomness. Learning is enabled by a neuromodulatory reward mechanism of octopamine stimulation of the AL, whose increased activity induces rewiring of the MB through Hebbian plasticity.

The goal of this work is to analyze how these various components interact to enable learning. To this end, we build a computational model of the moth olfactory network, including the dynamics of octopamine stimulation, which is closely aligned with the known biophysics of the AL-MB and with in vivo AL firing rate data of moths during learning. To our knowledge this is the first full, end-to-end neural network model that demonstrates learning behavior while also closely matching the structure and behavior of a particular biological system. The model is able to robustly learn new odors, and provides a valuable tool for examining the role of octopamine in learning. This octopamine mechanism during learning is of particular interest, since how it promotes the construction of new codes in the MB is not understood.

Specifically, our experiments elucidate key biological mechanisms for fast learning from noisy data that rely on an interaction between cascaded networks, sparsity, Hebbian plasticity, and neuromodulatory stimulation by octopamine.

O14 Modeling of TRP channel mediated noxious cold sensation in Drosophila sensory neurons

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Intracellular Ca2+ concentration usually correlates with the neuronal pattern and behavioral response. However, noxious cold sensation in Drosophila presents a paradox with these associations. Pkd2 and Trpm channels are required to trigger nociceptive full body contraction (CT) under acute cold [1]. *Trpm* mutants exhibit an increase in [Ca2+]i levels above control and display reduction of CT behavior, whereas *Pkd2* mutants showed reductions in [Ca2+]i level and inhibition of behavior [1].

We developed a Hodgkin-Huxley-type model of the cold sensitive CIII neurons to investigate interaction of Pkd2, Trpm and SK currents and to explain the experimental paradox. Our main mechanism assumes that the mutation of *Trpm* is homeostatically accompanied by a compensatory increase of the total Pkd2 current conductance, which leads to an amplified rise of [Ca2+]i under noxious cold temperatures. This higher [Ca2+]i activates stronger SK current which hyperpolarizes the membrane potential and suppresses spiking. This leads to inhibition of the stereotyped CT behavior under noxious cold stimuli. This model prediction is supported by the experiments, which showed 2-fold increase of *Pkd2* mRNA levels in Trpm mutants relative to control, while no change in *Trpm* mRNA levels was observed in *Pkd2* mutants.

Basic models of the CIII neuron describing responses of Control, *Trpm* and *Pkd2* mutants show transitions from silence at room temperature to spiking activity below 18 degrees Celsius, but have distinct features. Models of Control and *Trpm* mutants reach a maximum spike frequency near 14.5 degrees Celsius, while *Pkd2* mutants exhibited a maximum frequency at 6 degrees Celsius and had a smaller frequency compared to Control and *Trpm* mutants. The decrease of maximum frequency in *Pkd2* mutants as well as absence of spiking activity for most of the temperature range in *Trpm* mutants may explain the inhibition of CT behavior under noxious cold.

The [Ca2+] responses of the three models describing control, *Trpm* and *Pkd2* mutants are in agreement with the corresponding experimental data [1]. [Ca2+]i signal of CIII neurons under noxious cold is the strongest in *Trpm* mutants and the weakest in *Pkd2* mutants. Thus, the model and experimental results suggest that cold-evoked CT behavior is tuned to an optimal Ca2+ level which does not always functionally represent level of neuronal excitation.

Also, the basic model currently exhibits a wide spectrum of qualitatively different activity regimes. Depending on the parameter set, the model could show different regimes which are associated with different levels of [Ca2+]i and could be arranged into an alternative scheme of the temperature coding following the sequence of transitions between regimes: small amplitude spiking, period doubling cascade, bursting, large amplitude spiking, and rest state along with the temperature going down. These two coding schemes provide robust and generic mechanisms of coding modality-specific activity patterns by coordinated modality-specific activation of two TRP currents.

Acknowledgements

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O15 A geometric attractor mechanism for the self-organization of entorhinal grid modules

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The grid system of the mammalian medial entorhinal cortex (mEC) exhibits striking modularity. Rat grid cell recordings reveal that spatial grid scales cluster around discrete values separated by constant ratios reported in the range 1.3-1.8. Although this modular organization has been shown to be a robust and efficient encoding of spatial location, its origin is unknown. We present the first proposed mechanism through which geometric sequences of grid scales arise naturally. A series of continuous attractor networks along the longitudinal mEC axis that would otherwise generate a smooth distribution of grid scales forms modules separated by discrete jumps in scale when excitatory connections are introduced. Moreover, constant scale ratios between successive modules arise through robust geometric relationships between commensurate triangular grids, whose lattice constants are separated by [sqrt(1.7)] or other ratios, or between grids containing local lattice modulations called discommensurations. These relationships persist in single neuron spatial rate maps due to faithful path integration and are unaffected by perturbations to model parameters. We speculate on how excitatory connections between attractor networks can be realized by the known architecture of the mEC and suggest analyses and experiments that test our model.

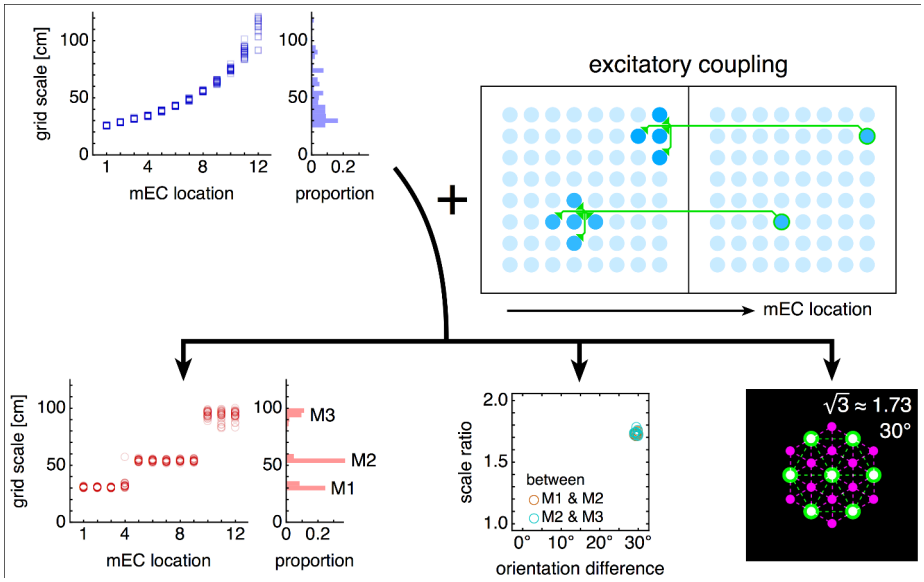

Figure 1: Grid cells with smoothly distributed scales self-organize into discrete modules when excitatory connections along the medial entorhinal cortex (mEC) are added. Adjacent modules have fixed scale ratios and orientation differences due to robust geometric relationships between commensurate triangular lattices.

O16 Simulating in vivo context-dependent recruitment of CA1 hippocampal interneuron specific 3 (IS3) interneurons

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Obtaining recordings from individual cells during behaviour is technically challenging, especially for the diverse interneuron subtypes that tend to be smaller, less accessible, and less identifiable relative to excitatory cells. As such, it is difficult to determine inhibitory cell contributions but it is clear that consideration of interneuron subtypes is critical to understanding brain function and behavior (Kepecs & Fishell, 2014). To address this, we use computational approaches. We focus on the hippocampal CA1 interneuron specific 3 (IS3) cell, a cell type that has not yet been recorded from in vivo. Notably, though IS3 cells represent a small fraction of interneurons in CA1 hippocampus, they possess unique circuitry properties in that they only inhibit other inhibitory neurons, such as Oriens Lacunosum Moleculare (OLM) interneurons. In vitro, photo-activation of IS3 cells at theta frequencies has been shown to elicit theta-timed spiking in OLM cells (Tyan et al, 2014). To explore the potential contributions of IS3 cells during in vivo contexts, we use multi-compartment IS3 cell models to generate predictions of input populations that could either enhance or dampen IS3 cell activities during behavior.

We have developed data-driven multi-compartment models of IS3 cells with active dendritic properties (Guet-McCreight et al, 2016), determined realistic synaptic parameters along the dendritic morphology of the models (Guet-McCreight et al, 2017), and estimated numbers of active synapses and presynaptic spike rates to generate in vivo-like states for IS3 cell models. Here, we consider context-dependent recruitment of IS3 cells during simulated states of theta rhythms and sharp-wave associated ripples (SWRs). During these states, we use our models to predict the contributions of different presynaptic inhibitory and excitatory input populations.

Our results show that excitatory theta-timed inputs from CA3 and entorhinal cortex can modulate the timing of IS3 cell spiking during theta rhythms. Moreover, depending on their relative contributions, the timing of the IS3 cell model's spiking can occur anywhere between the rising phase and peak of the theta cycle. As well, we show that inhibitory inputs can dampen spike recruitment of IS3 cells regardless of phase, though less so for inhibitory inputs that are the most antiphase relative to excitatory inputs. For our simulated SWR context, we show that transiently bursting CA3 inputs alone are sufficient to recruit the IS3 cell model to spike. We also show that the presence of feedforward inhibition on the proximal dendrites of the model can sufficiently dampen IS3 cell spiking during a SWR context. In summary, we have simulated in vivo-like contexts where IS3 cell spike recruitment can be either enhanced or dampened. Our results highlight possible IS3 cell spiking scenarios and thus their potential contributions to brain function and behavior.

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O17 Quantitative simplification of detailed microcircuit demonstrates the limitations to common point-neuron assumptions

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A first-draft detailed simulation of a piece of the rat neocortex has recently been reported by an international collaboration [1]. This work integrated the current state of experimental knowledge on the detailed 3D anatomy and physiology of the various neuron types, and their synaptic properties and connectivity, and was shown to reproduce findings from a range of in vivo experiments reported in the literature without parameter tuning. On the other hand, for large-scale network simulations, point-neuron models are typically used for describing and analyzing network dynamics and functions. The properties and connectivity structure of point neuron models generally are not constrained by biological data and thus use ad hoc simplifying assumptions. This makes some of the mathematically tractable models somewhat disconnected from experimental neuroscience. To bridge the gap between these two extremes (the detailed and the oversimplified), we aimed to derive point-neuron network models from data-driven detailed network models in an automated, repeatable and quantitatively verifiable manner. The simplification occurs in a modular workflow, in an in vivo-like state. First, synapses are displaced from dendrites to the soma while correcting for dendritic filtering using low-pass filters for the synaptic current numerically calibrated for each dendritic compartment. Next, point-neuron models for each neuron in the microcircuit are fitted to their respective morphologically detailed counterparts. Here, generalized integrate-and-fire point neuron models are used, leveraging a recently published fitting toolbox [2]. The fits are constrained by currents and voltages computed in the morphologically detailed reference neurons with soma-displaced synapses, as described above. Benchmarking the simplified network model to the detailed microcircuit model for a range of simulated in vivo and in vitro protocols, we found good agreement for both quantitative and qualitative aspects. Our automated approach not only makes it possible to continuously update the simplified circuit as the detailed network integrates new data, but the modularity of the simplification process also makes it applicable to other point neuron and synapse models, network models, and simulators. In addition to providing an extensive assessment of validity for carefully reduced point neuron network models, our approach is fundamentally important and informative, in particular in cases when network functionalities are lost during the simplification pipeline. By taking the simplification further to evaluate common simplifying assumptions, we further illustrate the contributions of specific synaptic and cellular dynamics to the overall response of the detailed network, revealing limitations for several common approaches.

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O18 A novel synaptic plasticity rule for detailed model neurons with realistic dendrites

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Numerous experiments have been conducted in the past in order to monitor the complex interactions that drive activity-dependent long-term plasticity of synapses. Spike timing, firing rate and synaptic location have been found to be important factors that dynamically contribute to the outcomes of plasticity induction protocols. While several theoretical models that implement plasticity rules already exist, they have not yet been used in depth to study plasticity in neuron models with detailed morphology. Here, we extend previous phenomenological voltage-based plasticity rules by developing a new framework based on three signaling pathways. We apply it to a L5 pyramidal cell model with active dendritic properties and realistic propagation of voltage. We show that our novel rule not only reconciles outcomes of several experiments but also predicts spatiotemporal patterns of plasticity that are characteristic for individual stimulation protocols and their impact on local processes at the synapse, including protocols inducing local plasticity in tuft dendrites. Due to this focus on local voltage signals, our framework can explain synaptic plasticity in the absence of postsynaptic action potentials, as suggested in recent studies. We thereby link experimental results that would intuitively seem to require entirely different rules, showing that a unifying rule might explain the vast majority of experiments in cortical pyramidal cells if key biophysical pathways are taken into account. Ultimately, we can now study how the cell-type specific electrotonic properties can explain differences in emerging plasticity by incorporating our plasticity rule in a variety of existing detailed compartmental models such as models of hippocampal pyramidal or granule cells. To summarize, a simple plasticity rule that utilizes pre- and postsynaptic plasticity pathways can explain experimental results with a large variety of induction protocols when the plasticity rule is incorporated in the compartmentalized structure of a detailed dendritic model.

O19 Assisted construction of hybrid circuits: making easy the implementation and automation of interactions between living and model neurons

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Closed-loop interactions with the nervous system are a powerful approach to characterize neural dynamics and control network functions [1,2]. In particular, neuron models can interact with living neurons in hybrid circuits once proper adaptation is achieved in both directions [3,4]. Such adaptations are not easy to accomplish in a manual trial-and-error process, and are better determined with closed-loop protocols based on real-time event detection [5] and well-defined interaction goals and performance measurements. This work presents a set of algorithms for the assisted construction of hybrid circuits. These algorithms have been implemented in RTHybrid, an open-source cross-platform real-time model library [6].

Our real-time algorithms for assisted construction of hybrid circuits are based in a general closed-loop paradigm designed to be modular and effective. The algorithms perform as a function of their online measured input parameters the following tasks: (1) temporal and amplitude scaling, (2) drift compensation, (3) synaptic tuning/calibration, (4) model turning/calibration, (5) automatic activity control, (6) automatic mapping of the dynamics. The temporal and amplitude scales are evaluated and matched online to create compatible working regimes between the model and living neurons [4]. All protocols use three steps: event detection, activity and connection characterization and target performance evaluation. The events detected online include: spikes, bursts, hyperpolarization intervals, voltage ranges, temporal structures, phases, etc. The interaction characterization measures include event timings, instantaneous periods, synchronization levels, target phases, and working/dynamic range assessments. When the interaction goal is not fulfilled, the target evaluator algorithm changes in an informed and automatic manner the parameters of the hybrid circuit. Our algorithms have been validated in a hybrid circuit to study the presence of dynamical invariants in CPGs.

In conclusion, hybrid circuits require experiment-specific adaptations to work properly, and the parameters of the implementation must be evaluated dynamically on each preparation and even adapted during the same experiment. These algorithms can also be used to automatically map the parameter space to achieve a given goal, and in general to control/explore/unveil bifurcations and circuit dynamics.

Acknowledgements
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O20 Deciphering the evolutionary route to the first neurons

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We do not yet know how the very first nervous systems and their constituting neurons evolved within the animal kingdom. One important difficulty comes from the lack of examples of intermediate neuronal stages within currently existing animals. Such examples would bridge the gap between non-neuronal and neuronal configurations. However, on the one hand there are basic animals like sponges and placozoa who do not have neurons or a nervous system. On the other hand, even the most basic forms of animals with nervous systems, such as jellyfish (cnidarians) and comb-jellies (ctenophores) already exhibit a nervous system built from complete neurons. So far it is unknown how the three fundamental ingredients of modern neurons—electrical signaling, synapses, and neuronal elongations—came together in the first neurons and why this happened. Compared to modern animals, very little is known about the earliest possessors of nervous systems. Essentially modern nervous systems complete with eyes and a central nerve cord are known from the beginning of the Cambrian period, so the very origin of nervous systems must predate that period. However, Precambrian animal fossils are enigmatic and difficult to interpret, providing insufficient information about the behavioural and neuronal makeup of these organisms. Molecular phylogenetic studies do provide important clues concerning the cellular building blocks present to these animals but do not allow a clear view of the organization of the animals living in these times.

Computational neuroscience provides an important additional instrument to enhance our understanding of the neuronal and behavioural mechanisms that were potentially present in very early animals. Modelling very basic animal configurations, using primitive features such as cell-to-cell signalling that can be assumed to have been present at this stage, provides a way to assess the behavioural capacities of such configurations. Such modelling also allows a step by step investigation of potential evolutionary sequences of various proto-neuronal features and the behavioural effects they induce. All in all, these models provide rigorous thought experiments that enable a systematic investigation of various (proto-)neuronal features on coordination in a simple body.

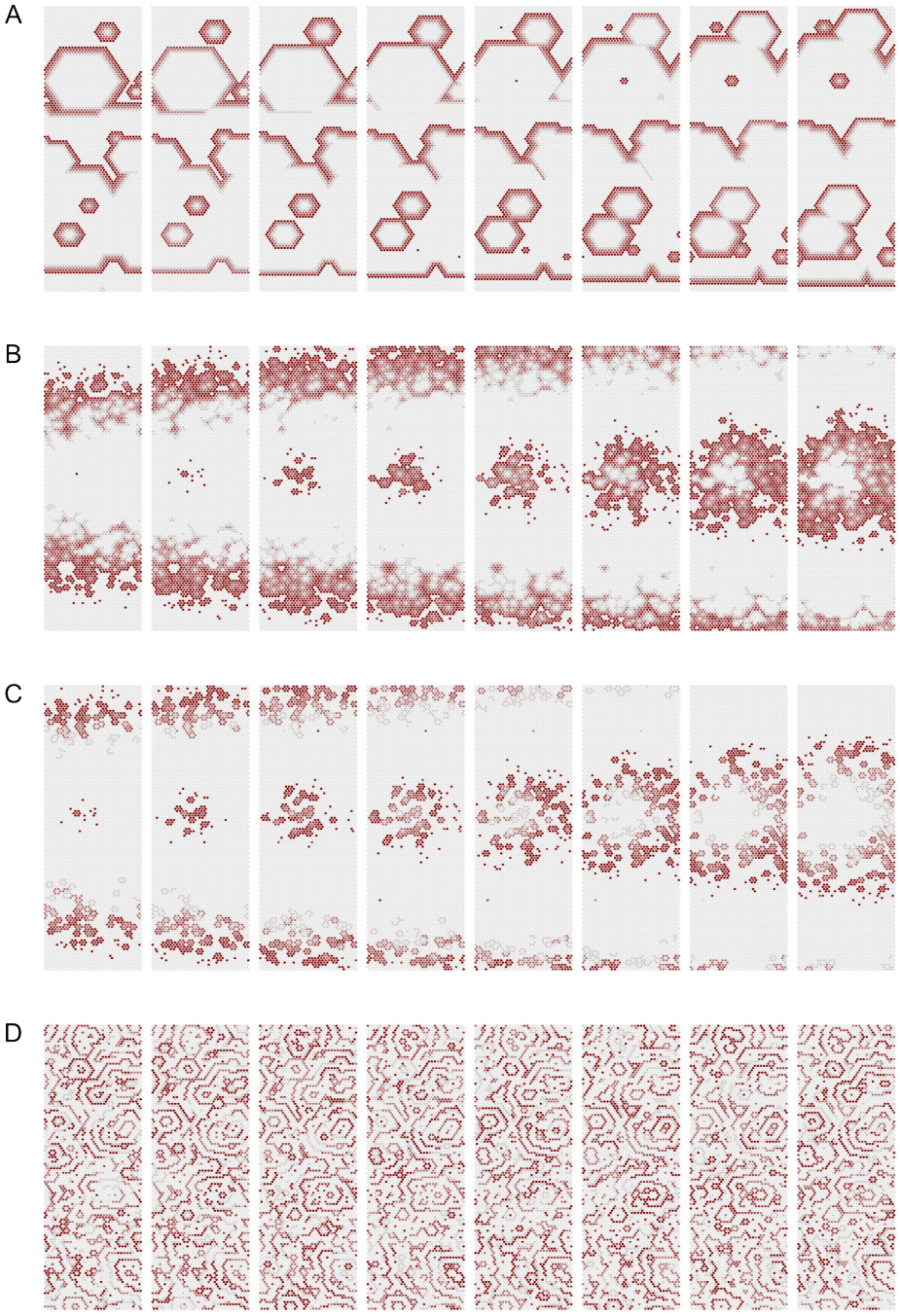

Figure 1: Various degrees of emergent coordination on a larger (32 cells in circumference, 128 in length) worm-shaped body. Four different experiments, showing 8 frames each: A, lacking elongations; B, 10% of cells exhibiting elongations; C, same as B, but no nearest-neighbour connections between cells lacking elongations; D, same as B, but with very low transmission speed.

O21 Community models as the ultimate objective (and success) of computational neuroscience: exempli gratia: The cerebellar Purkinje cell

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On its main web page, the Organization for Computational Neuroscience (OCNS) defines Computational Neuroscience as “the study of brain function in terms of the information processing properties of the structures that make up the (sic) nervous system”. As nervous systems ARE information processing structures, this definition begs the question how the field of Computational Neuroscience distinguishes itself from neuroscience as a whole? The definition of Computational Neuroscience provided by OCNS makes an effort to addresses this conundrum by further defining CNS as “an interdisciplinary science that links the diverse fields of neuroscience, cognitive science and psychology with electrical engineering, computer science, mathematics and physics.” In this presentation, I will propose that THE key concept underlying Computational Neuroscience is, in fact, the question of ’linkage’. More specifically, I will propose that ’linkage’ should not be an abstract ideal, but instead, specifically requires the development of computation tools and devices as well as an attitude towards science that supports the development of “community models” defined as actual mathematical models shared and developed collaboratively across the community of those interested in a particular neuronal feature or component. While one can argue that standards for academic advancement and the current publication process favor isolated models developed by individual research groups which therefore, continue to dominate computational neuroscience, I will suggest that only shared community models can truly support scientific communication, coordination and collaboration. Further, of necessity, to be effective I will assert that these community models must be ’realistic’, reflecting the actual physical and physiological structure of the components of the nervous systems being studied. Not only do community models of this type provide a basis for real collaboration, they also, in effect, represent the current state of our understanding of neuronal structure / function relationships mathematically. In this presentation, these assertions will be considered with respect to the development over the last 40 years of a model of the cerebellar Purkinje cell as one of the first computational models used across multiple laboratories as well as the historical context provided by the emergence of ’realistic’ community models in Physics in the 16th century. In a companion submission, I will consider, with several of my long-term colleagues, how the development of shared simulation platforms when combined with a new approach to scientific publication can drive the development and use of community models.

Figure 1: The first poster created for the CNS meeting, intended to represent the initial somewhat disorganized state of the field.

Workshops

W1 Methods of Information Theory in Computational Neuroscience

Allen Institute Auditorium, Tue July 17 and Wed July 18, 9:00 to 18:00

Joseph T. Lizier, University of Sydney
Viola Priesemann, Max Planck Institute for Dynamics and Self-organisation
Justin Dauwels, Nanyang Technological University
Taro Toyoizumi, RIKEN Brain Science Institute
Alexander G Dimitrov, Washington State University
Lubomir Kostal, Czech Academy of Sciences
Michael Wibral, Goethe University, Frankfurt

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.

Please see our website http://bit.ly/cns2018itw for full abstracts, schedule and additional contributed talks (to be announced).

Speakers:

- Braden Brinkman (Stony Brook University, New York, US) “Signal-to-noise ratio competes with neural bandwidth to shape efficient coding strategies”
- Mireille Conrad (University of Geneva, Geneva, Switzerland) “Mutual information vs. transfer entropy in spike-based neuroscience”
- Benjamin Cramer (University of Heidelberg, Heidelberg, Germany) “Information theory reveals a diverse range of states induced by spike timing based learning in neural networks”
- Alexander Dimitrov (Washington State University Vancouver, Vancouver, US) “Modeling of perceptual invariances in biological sensory processing”
- Eva Dyer (Georgia Tech, Atlanta, US) “Finding low-dimensional structure in large-scale neural recordings”
- Justin Gardner (Stanford University, Stanford, US) “Optimality and heuristics for human perceptual inference”
- Jim Kay (University of Glasgow, Glasgow, UK) “Partial Information Decompositions based on Dependency Constraints”
- Joseph T. Lizier (The University of Sydney, Sydney, Australia) “Pointwise Partial Information Decomposition Using the Specificity and Ambiguity Lattices”
- Leonardo Novelli (The University of Sydney, Sydney, Australia) “Validation and performance of effective network inference using multivariate transfer entropy with IDTxl”
- Tatyana Sharpee (Salk Institute for Biological Studies, La Jolla, US) “Information-theoretic constraints on cortical evolution”
- Nicholas M. Timme (Indiana University, Bloomington, and Purdue University Indianapolis, US) “From neural cultures to rodent models of disease: examples of information theory analyses of effective connectivity, computation, and encoding”
- Taro Toyoizumi (RIKEN Brain Science Institute, Tokyo, Japan) “Emergence of Levy Walks from Second-Order Stochastic Optimization”

- Siwei Wang (Hebrew University of Jerusalem, Jerusalem, Isreal) “Closing the gap from structure to function with information theoretic design principles”
- Plus additional contributed talks ...

W2 Neuronal morphology and structure

Allen Institute 286/287, Tue July 17, 9:00 to 18:00

Alexander Bird, Ernst Strüngmann Institute and FIAS, Frankfurt
André Castro, Ernst Strüngmann Institute and FIAS, Frankfurt
Hermann Cuntz, Ernst Strüngmann Institute and FIAS, Frankfurt

Neurons are complex structures and their morphologies display both great diversity and the potential for remarkable specificity in function and connectivity. Theoretical neuroscience has always had a key role to play in analysing neuronal structure, starting with Cajal's insight that neurons must balance the material and functional costs associated with their dendritic trees. Recent advances in experimental techniques allow us to study dendrites from new perspectives, but have also created new challenges in reconstruction, quantification, and comparison. For example, large scale studies of connectivity have reinforced the importance of single cell morphology within microcircuits, whilst an ever-expanding library of genetic and physical manipulations shed new insights into the processes leading to the development of these morphologies. The goal of the workshop is to provide a resume of the state-of-the-art in experimental, computational and mathematical investigations into the morphology of neurons in a variety of systems.

Speakers:

- Uygar Sümbül (Allen Institute, Seattle, USA) "Quantifying neuroanatomy"
- Ruth Benavides-Piccione (Instituto Cajal, Madrid, Spain) "The microanatomy of pyramidal cells"
- Erik De Schutter (OIST, Okinawa, Japan) "The Purkinje cell dendrite causes its unique firing rate-dependent phase response curve"
- Lida Kanari (EPFL, Lausanne, Switzerland) "Randomness and structure in artificially generated neuronal networks"
- Kurt Haas (University of British Columbia, Vancouver, Canada) "Dynamic morphometrics: Rapid time-lapse imaging and quantification of experience-driven dendrite growth"
- Hollis Cline (Scripps Institute, San Diego, USA) "In vivo time-lapse imaging analysis of neuronal structure and functional plasticity"
- Casey Schneider-Mizell (Allen Institute, Seattle, USA) "The neuroanatomy of connectivity in the Drosophila larva"
- Staci Sorensen (Allen Institute, Seattle, USA) "Morphological, electrophysiological and transcriptional descriptions of cortical cell types"
- Sophie Laturnus (Universität Tübingen, Tübingen, Germany) "A systematic comparison of neuronal morphology representations for cell type discrimination"
- Hongkui Zeng (Allen Institute, Seattle, USA) "Morphology as a key feature for neuronal cell type classification"

W3 **Bridging Spatial and Temporal Scales in Brain Connectomics**

MOHAI Microsoft Lakefront Pavilion, Tue July 17, 9:00 to 18:00

Katharina Glomb, Lausanne University Hospital
Joana Cabral, Oxford University

In this workshop we will explore Dynamic Functional Connectivity on different temporal and spatial scales. We aim to review recent results and put them in perspective to understand common points and discrepancies across different neuroimaging communities. In particular, we will target the difficulties faced by methodological approaches when bridging scales due to the differences in how neural dynamics are described.

As an example, similar results about the sources that contribute to dynamic connectivity patterns have been reported on different scales. On the one hand, there are changes in global coherence, sometimes described as standing or traveling waves. On the other hand, there are causal interactions between brain regions/neuronal populations which can be extracted by considering time delays. Ideally, the workshop will help to identify opportunities that have thus far remained unexplored.

Speakers:

- Amrit Kashyap (Georgia Tech, Atlanta, USA) “Brain dynamics viewed through BOLD, electrophysiology and computational modeling”
- Joana Cabral (University of Oxford, UK) “Mechanistic network models of MEG and fMRI functional connectivity”
- Louis-David Lord (University of Oxford, UK) “Characterization of the brain’s dynamical repertoire in the psychedelic state”
- Sebastien Naze (IBM, Thomas J. Watson Research Center, Yorktown Heights, USA) “Sensitivity analysis of the connectome harmonics and implications in neurodegenerative diseases”
- Jeremie Lefebvre (Krembil Research Institute, Toronto, Canada) “State-Dependent Entrainment of Cortical Oscillations with Periodic Stimulation”
- Gijs Plomp (University of Fribourg, Switzerland) “Fast directed interactions between brain areas and cortical layers”
- Petra Ritter (Charite Berlin, Germany), TBA
- Katharina Glomb (CHUV, Lausanne, Switzerland) “Graph signal processing for anatomically constrained source-reconstructed EEG data”

W4 Models for Perceiving and Learning Time Intervals and Rhythms

Allen Institute Training Room, Tue July 17, 9:00 to 18:00

Áine Byrne, New York University
John Rinzel, New York University
Amitabha Bose, New Jersey Institute of Technology

Accurate time estimation is essential for survival, yet the neural bases remain elusive. Time processing has been widely studied in the context of decision making, language, memory and perception. Research on interval-timing, for sub to suprasecond scales, ranges from psychophysical experiments and imaging studies to theoretical models. Beat perception in music is particularly compelling, fast perception and learning of repetitive time intervals from 100 to 2000 ms. The abilities to recognize and predict rhythms appear inherent to humans. Hypotheses of neural mechanism involve sensory and motor area interaction (eg, listening and finger-tapping). We will bring together researchers that are developing models of timing and of prediction with frameworks that include drift-diffusion, neural resonance, coincidence detection and adapting neuronal oscillator circuits. We seek to promote discussion and linkage between the timing and prediction fields, both important for understanding beat perception.

Speakers:

- Jessica Grahn (Western University, Canada) “The role of beat perception in auditory sequence processing”
- Sorinel Oprisan (College of Charleston, USA) “Models of interval timing”
- John Iversen (University of California San Diego, USA) “Audiomotor interactions in beat perception”
- Edward Large (University of Connecticut, USA) “How you got your groove: Modeling rhythm learning, perceptual narrowing, and enculturation”
- Sundeep Teki (University of Oxford, UK) “Contextual representation of time intervals in rhythmic sound sequences”
- Áine Byrne (New York University, USA) “A neuro-mechanistic model for beat generation”
- Hugo Merchant (National Autonomous University of Mexico, Mexico), “Neural population dynamics in the primate supplementary motor area during rhythmic tapping”
- Patrick Simen (Oberlin College, USA) “A drift-diffusion model of complex motor timing without a reset problem”

W5 Developing, Standardising and Sharing Large Scale Network Simulations

Allen Institute 288/289, Tue July 17, 9:00 to 12:30

Padraig Gleeson, University College London

A number of groups around the world are developing complex, experimentally constrained models of cortical function. Creating the software infrastructure to develop, simulate and share these types of models takes a significant amount of time for any of the groups involved and there can be a lot of overlap, duplication in work and repeated effort.

This workshop aims to highlight some of the initiatives currently underway to build detailed cortical models as well as those projects building the infrastructure to make it easier to develop, disseminate and compare the models. Attendees of this workshop will come away with a better idea of the state of the art in large scale cortical model development and the efforts underway to make these more accessible and reusable for other researchers.

The most recent program for the workshop can be found here:
http://www.opensourcebrain.org/docs/Help/Meetings#CNS_2018

Speakers:

- Anton Arkhipov (Allen Institute, Seattle, USA) and Eilif Muller (Blue Brain Project, Switzerland) “Data-Driven Modeling of Brain Circuits and the SONATA Data Format’"
- Markus Diesmann and Sacha van Albada (Jülich Research Centre, Germany) “Large scale model development from the NEST perspective”
- Salvador Dura-Bernal (SUNY Downstate Medical Center, Brooklyn, NY, USA) “Development of large scale data-driven network models in NetPyNE, a high-level interface to NEURON”
- Padraig Gleeson (University College London, UK) “Large scale cortical models in NeuroML format on Open Source Brain”
- Open Discussion: “How best to move forward and what needs of the community are not being met?”

W6 Neuroscience Gateway and Large Scale Neural Systems Simulations and Tools

Allen Institute 288/289, Tue July 17, 14:00 to 18:00

Amit Majumdar, University of California San Diego
Subhashini Sivagnanam, University of California San Diego
Ted Carnevale, Yale University

Large scale modeling and simulations, using supercomputing resources, are important components of computational neuroscience. Computational neuroscientists in the US, from the EU Human Brain Project and those involved with the recently (December, 2017) signed International Brain Initiative depend on High Performance Computing for research. The US NSF and NIH funded Neuroscience Gateway (NSG) project provides neuronal tools, pipelines, and libraries optimally implemented on HPC resources for the neuroscience community. NSG tools and libraries include NEURON, CARLSim, PGENESIS, NEST, Brian, PyNN, MOOSE, BluePyOpt, The Virtual Brain Pipeline, Matlab, EEGLAB, Freesurfer, Human Neocortical Neurosolver etc.; NSG provides tens of millions of supercomputing hours freely for computational neuroscientists, has over 600 users, and is a platform for dissemination of computational neuroscience tools. This workshop will bring together some of the developers of neuronal tools/libraries/pipelines available on NSG and neuroscience users that are using NSG for computational neuroscience research to discuss both tool development and research results enabled by NSG.

Speakers:

- Subhashini Sivagnanam, Kenneth Yoshimoto, Amit Majumdar (UCSD, La Jolla, CA, USA), Ted Carlevale (Yale U., New Haven, CT, USA) “Neuroscience Gateway - Enabling Large Scale Simulations and Data Processing in Neuroscience”
- Robert McDougal (Yale U., New Haven, CT, USA) “Strategies for Parallel NEURON Simulations”
- Ting-Shuo Chou, Hirak J. Kashyap, Jinwei Xing, Stanislav Listopad, Emily L Rounds, Michael Beyeler, Nikil Dutt, Jeffrey L Krichmar (UCI, Irvine, CA, USA) “CARLsim 4: An Open Source Library for Large Scale, Biologically Detailed Spiking Neural Network Simulation using Heterogeneous Clusters”
- Alexandre Guet-McCreight, Frances Skinner (Krembil Research Institute, University of Health Network and University of Toronto, Toronto, ON, Canada) “Using NSG to perform millions of simulations in order to characterize in vivo-like states for interneurons of the hippocampus”
- Richard C. Gerkin, Russell J Jarvis, Sharon M. Crook (Arizona State University, Tempe, AZ, USA) “NeuronUnit: Tools for data-driven validation of neuron and neural circuit models”
- Vijay Iyer (MathWorks Inc., Boston, MA, USA) “Neuroscience Modeling and Data Processing with Community-authored MATLAB-based Tools”

W7 Dynamics of Rhythm Generation

UW Medicine SLU Brotman Auditorium, Tue July 17, 9:00 to 18:00

Gennady Cymbalyuk, Georgia State University

The ability of distinct circuits to generate patterns of rhythmic activity is widespread among vertebrate and invertebrate species. These patterns correspond to different functions like control of different rhythmic movements and pathological events like seizure episodes. The dynamics of the circuits producing such patterns are based on the basic principles conserved across phyla. This workshop will investigate roles of interactions of processes on different time and space scales in attaining the robustness and flexibility, characteristic for living circuits. For example, we will discuss the roles played by Na+/K+ pump and ion exchangers in generation of functional and dysfunctional rhythms. We would like to bring together experts applying experimental approaches and the methods developed in the neuroscience, neurophysics, neuro-informatics, neuroethology, and the bifurcation theory to determine the basic principles of the transient, intermittent, and steady dynamics of rhythm generation from different phyla.

Speakers:

- Anatoly Buchin (Allen Institute for Brain Science, Seattle, USA) “Epileptic seizures as pathological oscillations in neural network and neural mass models”
- Gennady Cymbalyuk (GSU, Atlanta, USA) “Roles of the Na/K pump current in generation of bursting patterns”
- Irene Elices (Universidad Autónoma de Madrid, Madrid, Spain) “Dynamical invariants: cycle-by-cycle rhythm negotiation”
- Yaroslav Molkov (GSU, Atlanta, USA) “TRP channels and intracellular calcium dynamics in the pre-B́otzinger complex”
- Astrid Prinz (Emory University, Atlanta, USA) “Mechanisms for stabilizing rhythm generation”
- Nino Ramirez (Seattle Children’s Hospital, Seattle, USA) “Dynamic mechanisms underlying respiratory rhythm generation”
- Ilya Rybak (Drexel University, Philadelphia, USA) “Respiratory CPG: Insights from optogenetic and modeling studies”
- Yina Wei (Allen Institute for Brain Science, Seattle, USA) “Differentials roles of sleep spindles and sleep slow oscillations in memory consolidation”

W8 **Insights Gained by Detailed Dendritic Modeling**

Allen Institute 540 Lab, Wed July 18, 9:00 to 18:00

Dieter Jaeger, Emory University

Volker Steuber, University of Hertfordshire

Most abstract neural network models operate with single compartment neurons, i.e. without dendrites. In contrast, just about all mammalian neurons receive a majority of their synaptic inputs on dendrites. It is becoming increasingly clear that this is not just to provide more surface area and sample inputs in specific spatial configurations, but that dendrites supply neurons with important non-linear functions. This workshop will highlight modelling studies that explore the properties of dendritic computations through compartmental modelling. The distinct dendritic computational properties of different cell types will be highlighted.

Call for Contributed Talks: Open call for contributed short talks to our dendrite workshop - interested potential attendees please e-mail the organisers with a title and short abstract.

Speakers:

- Dieter Jaeger (Emory University, Atlanta, USA) “Introduction and Globus Pallidus neuron modelling”
- Volker Steuber (University of Hertfordshire, Hatfield, Hertfordshire, UK) “Dendritic morphology and information processing in cerebellar neurons”
- Carmen Canavier(LSU Health Sciences Center, New Orleans, LA, USA) “Intrinsic mechanisms of frequency selectivity in proximal dendrites of CA1 Pyramidal neuron”
- Arnd Roth(University College London, UK) “Active dendrites enable strong but sparse inputs to determine orientation selectivity”
- Alexandra Tran-Van-Minh (Francis Crick Institute, London, UK) “Dendritic properties of cerebellar stellate cells: information processing with sublinear dendrites”
- Monika Jadi (Yale University, New Haven, USA) “Inhibitory control of non-linear dendritic computations”
- Christof Koch (Allen Institute, WA, USA), “The astonishing diversity of mouse and human cortical dendrites”
- Bill Lytton (SUNY Dowstate Medical Center, NY, USA) “Dendritic plateaus could underlie hierarchical embedded ensembles”
- Avrama Blackwell (George Mason University, VA, USA) “Inhibition enhances spine-specific Calcium encoding of synaptic input patterns”
- Frances Skinner (UHN and Univ. of Toronto, ON, Canada) “How the specifics of dendritic ion channels in inhibitory cells of the hippocampus could contribute to function”
- Subutai Ahmed (Numenta, Inc., Ca, USA) “The predictive neuron: how active dendrites enable spatiotemporal computation in neocortex”

W9 **Integrative Theories of Cortical Function**

Allen Institute Training Room, Wed July 18, 9:00 to 18:00

Hamish Meffin, The University of Melbourne
Stefan Mihalas, Allen Institute for Brain Science
Anthony Burkitt, The University of Melbourne

The cerebral cortex is a brain region remarkable in similarity of structure between different mammalian species and between different areas in a species. This has led to developments of theories that parts of the cortex perform a similar set of operations, a dictionary of canonical cortical computations. In recent years, several theories for what these operations are have been developed. In concert with the theories multiple models have been developed implementing these proposed computations. This workshop aims to look at what progress has been made in understanding these local computations, how the global cortex functions arise from them, what experimental evidence can be used to differentiate between model, and what are the general integrative principles. We plan to foster a dialogue between theoreticians, experimentalists and modelers.

For an up to date list of talks and schedule please see http://www.nvri.org.au/events.php/40/cns2018-workshop-integrative-theories-of-cortical-function

Speakers:

- Tania Pasternak (U Rochester, USA) “Defining a role for prefrontal cortex in memory-guided sensory comparisons”
- Subutai Ahmad (VP Research Numenta, USA) “Locations in the neocortex: A Theory of sensorimotor prediction using cortical grid cells”
- Anitha Pasupathy (U Washington, USA) “Encoding things and stuff: multiplexed form and texture signals in primate V4"
- Markus Diesmann (Research Centre Jülich , Germany) “Reusable publication of a cortical multi-area model at cellular resolution”
- Hamish Meffin (U Melbourne, Australia) “The structure of non-linear receptive fields in cat primary visual cortex”
- Chang Sun Kim (Chonnam National University, Korea) “Computational implementation of the free energy principle in the brain”
- Stefan Mihalas (Allen Institute for Brain Science, USA) “Cortical visual systems perform deep integration of context”
- Christof Koch (Allen Institute for Brain Science, USA) “Cortex as the Physical Substrate of Consciousness”

W10 How Does Learning Reshape the Dimensionality of Collective Network Activity?

UW Medicine SLU Brotman Auditorium, Wed July 18, 9:00 to 18:00

Rainer Engelken, Columbia University
Guillaume Lajoie, Université de Montréal
Merav Stern, University of Washington

Large neural networks, biological or artificial, can learn complex input-output relations. During learning the network dynamics are often constrained to a low-dimensional manifold despite available high-dimensional space. The mechanism behind this space dimensionality confinement is yet unclear.

Current technological advances in chronic population recordings and optogenetics provide the tools to measure and manipulate the reorganization of this state-space structure in neural circuits in awake, behaving animals during learning.

We will bring together theoreticians and experimentalists to address a most fundamental question in neuroscience, that is, how learning reshapes collective network activity.

More specifically, we would like to explore:

How does the neural dimensionality of a learned task relate to the task complexity?

Which mathematical tools are suitable to identify low-dimensional neural manifolds and track their emergence during learning?

How does the dimensionality constrain the learning capabilities?

Speakers:

- SueYeon Chung (Harvard University) “Classification and geometry of neural manifolds, and the application to deep networks”
- Rainer Engelken (Columbia University) “Dimensionality and entropy rate of spontaneous and evoked neural rate dynamics”
- Kameron Decker Harris (University of Washington) “Connections between dimensionality and network sparsity”
- Zack Kilpatrick (University of Colorado Boulder) “Learning continuous attractors in recurrent neural networks”
- Guillaume Lajoie (Université de Montréal) “External perturbations modulate coding manifolds and dimensionality of motor cortex activity”
- Luca Mazzucato (Columbia University, University of Oregon) “Changes in effective network coupling mediate learning in a trace fear conditioning task”
- Stefano Recanatesi (University of Washington) “Explaining the dimensionality of the activity in RNNs through connectivity motifs”
- Merav Stern (University of Washington) “Increased correlations and decreased activity dimensions during task performance”
- Evelyn Tang (University of Pennsylvania) “Effective learning is accompanied by high dimensional and efficient representations of neural activity”
- Alex Williams (Stanford University) “Dimensionality reduction with single trial resolution”

W11 Towards New Models for Cognitive Flexibility

Allen Institute 288/289, Wed July 18, 9:00 to 18:00

Rajeev Rikhye, Massachusetts Institute of Technology

Cognitive flexibility is defined as the ability to make different inferences from the same stimulus depending on behavioral demands. This essential computation allows us to act intelligently in our dynamically changing environments. The prefrontal cortex (PFC) has traditionally been the focus of many computational theories of cognitive flexibility. However, several recent have identified many subcortical areas, such as the mediodorsal thalamus, as key players in controlling how the cortex flexibly switches between task sets. These new results suggest that the computations responsible for cognitive flexibility are more distributed and dynamic than previously thought.

In this workshop, we bring together theorists and researchers interested in flexibility at several levels. Our goal is to develop a unified view of the fundamental neural motifs – both cortical and subcortical – that underlie cognitive flexibility. We anticipate that this workshop will be of interest to anyone interested in cognitive flexibility and neural computation.

Speakers:

- Michele Basso (UCLA, US) “The role of the Basal Ganglia and Superior Colliculus in Decision Making”
- Timothy Hanks (UC Davis, US) “Flexibility of timescales of evidence weighting for decisions and confidence”
- Athena Akrami (Princeton University, US) “Role of posterior parietal cortex in mixing past with present information”
- Camilo Libedinsky (NUS, Singapore) “Heterogeneity in the prevalence of mixed-selectivity among different sub-regions of the lateral prefrontal cortex”
- Seth Egger (MIT, US) “Internal Models of sensorimotor integration regulate cortical dynamics”
- Nicolas Masse (University of Chicago, US) “TBA"
- Rajeev Rikhye (MIT, US), “Fronto-thalamic substrates of cognitive flexbility”

Posters

Poster Listing

Saturday Posters
Posters P1 – P145

P1 **MRI2MRI: A fully convolutional deep artificial network algorithm that accurately transforms between brain MRI contrasts**

Ariel Rokem[1⋆], Sa Xiao[2], Yue Wu[2], and Aaron Lee[2]

[1]University of Washington, eScience Institute, Seattle, WA, United States
[2]University of Washington, Department of Ophthalmology, Seattle, WA, United States

P2 **Closing the loop between neural network simulators and the OpenAI Gym**

Philipp Weidel[1⋆], Jakob Jordan[2], and Abigail Morrison[1]

[1]Juelich Research Centre, Institute for Advanced Simulation (IAS-6), Juelich, Germany
[2]University of Bern, Department of Physiology, Bern, Switzerland

P3 **Reproducing polychronization: a guide to maximizing the reproducibility of spiking network models**

Robin Pauli[1], Philipp Weidel[1⋆], Susanne Kunkel[2], and Abigail Morrison[1]

[1]Jülich Research Centre, Institute for Advanced Simulation (IAS-6), Juelich, Germany
[2]Norwegian University of Life Sciences, Faculty of Science and Technology, Ås, Norway

P4 **Localization of coherent activity based on multi-electrode local field potentials**

Robin Pauli⋆, Tom Tetzlaff, and Abigail Morrison

Jülich Research Centre, Institute for Advanced Simulation (IAS-6), Juelich, Germany

P5 **Exploring the role of striatal D1-MSNs and D2-MSNs in action selection using a robotic framework**

Jyotika Bahuguna⋆, Philipp Weidel, and Abigail Morrison

Jülich Research Centre, Institute for Advanced Simulation (IAS-6), Juelich, Germany

P6 **Calcium imaging spike deconvolution with minimal parameter tuning and limiting assumptions**

Nathan Lee[1⋆], Kameron Decker Harris[2], and Aleksandr Aravkin[1]

[1]University of Washington, Department of Applied Mathematics, Seattle, WA, United States
[2]University of Washington, Department of Computer Science, Seattle, WA, United States

P7 **Applying exact robust PCA to analyze mouse brain activity data**

Roman Levin⋆, Merav Stern, Eric Shea-Brown, and Aleksandr Aravkin

University of Washington, Department of Applied Mathematics, Seattle, WA, United States

P8 A theory of dendritic buckets

Hermann Cuntz[1⋆], Alexander Bird[2]

[1]*Frankfurt Institute for Advanced Studies (FIAS) & Ernst Strüngmann Institute (ESI), Computational Neuroanatomy, Frankfurt am Main, Germany*
[2]*Frankfurt Institute for Advanced Studies (FIAS), Computational Neuroanatomy, Frankfurt am Main, Germany*

P9 Predictive information as an organization principle for both sensory and cortical circuitry

Siwei Wang[1⋆], Idan Segev[1], Stephanie Palmer[2], Oren Amsalem[1], and Alexander Borst[3]

[1]*Hebrew University of Jerusalem, Department of Neurobiology, Jerusalem, Israel*
[2]*University of Chicago, Department of Organismal Biology and Anatomy & Department of Physics, Chicago, IL, United States*
[3]*Max Plack Institute, Department of Neurobiology, Munich, Germany*

P10 Distinct roles of anterior cingulate cortex and basolateral amygdala in reinforcement learning under perceptual uncertainty.

Alexandra Stolyarova[1⋆], Megan Peters[2], Hakwan Lau[1], and Alicia Izquierdo[1]

[1]*University of California, Los Angeles, Department of Psychology, Los Angeles, CA, United States*
[2]*University of California, Riverside, Bioengineering, Riverside, CA, United States*

P11 Efficient search with Lévy flights emerges from stochastic optimization

Lukasz Kusmierz⋆, Taro Toyoizumi, and Alireza Gourdarzi

RIKEN Brain Science Institute, Neural Computation and Adaptation, Wako, Japan

P12 A multi-scale data-based network model of lateral inhibition in mouse olfactory bulb

Daniel Zavitz[1⋆], Isaac Youngstrom[2], Matt Wachowiak[2], and Alla Borisyuk[1]

[1]*University of Utah, Department of Mathematics, Salt Lake City, UT, United States*
[2]*University of Utah, Department of Neurobiology & Anatomy, Salt Lake City, UT, United States*

P13 Assessing phase-locking and entrainment in oscillatory networks using one-dimensional maps

Casey Diekman, Amitabha Bose⋆

New Jersey Institute of Technology, Department of Mathematical Sciences, Newark, NJ, United States

P14 Functional role of 5-HT1A receptors in serotonergic modulation of active exhalation

William Barnett[1⋆], Yaroslav Molkov[1], Lucas Koolen[2], Adrian Newman-Tancredi[3], Mark Varney[3], and Ana Abdala[2]

[1]*Georgia State University, Department of Mathematics & Statistics, Atlanta, GA, United States*
[2]*University of Bristol, School of Physiology, Pharmacology & Neuroscience, Biomedical Sciences Faculty, Bristol, United Kingdom*
[3]*Neurolixis Inc, Dana Point, CA, United States*

P15 Analyzing how Na+/K+ pump influences the robust bursting activity of half-center oscillator (HCO) models

Ronald Calabrese, Anca Doloc-Mihu⋆

Emory University, Department of Biology, Atlanta, GA, United States

P16 Experimental directory structure (Exdir): An alternative to HDF5 without introducing a new file format

Svenn-Arne Dragly[1], Milad Hobbi Mobarhan[2], Mikkel Lepperød[2], Simen Tennøe[3], Gaute Einevoll[4⋆], Marianne Fyhn[2], Torkel Hafting[5], and Anders Malthe-Sørensen

[1]University of Oslo, Department of Physics, Oslo, Norway
[2]University of Oslo, Department of Biosciences, Oslo, Norway
[3]University of Oslo, Department of Informatics, Oslo, Norway
[4]Norwegian University of Life Sciences, Faculty of Science and Technology, Aas, Norway
[5]University of Oslo, Institute of Basic Medical Sciences, Oslo, Norway

P17 A mathematical framework for modeling large scale extracellular electrodiffusion surrounding morphologically detailed neurons

Gaute Einevoll[1], Geir Halnes[1⋆], Andreas Solbrå[2], Aslak Wigdahl Bergersen[3], Jonas van den Brink[3], and Anders Malthe-Sørensen[2]

[1]Norwegian University of Life Sciences, Faculty of Science and Technology, Aas, Norway
[2]University of Oslo, Department of Physics, Oslo, Norway
[3]Simula Research Laboratory, Fornebu, Norway

P19 Modeling the perceived perils of sodium channel anticonvulsants in Dravet Syndrome

Andrew Knox⋆

University of Wisconsin, Department of Neurology, Madison, WI, United States

P20 Spatial modeling of AMPA receptor trafficking and sorting at the Endosome

Erik De Schutter⋆, Sarah Nagasawa, Iain Hepburn, and Andrew R. Gallimore

Okinawa Institute of Science and Technology, Computational Neuroscience Unit, Onna-Son, Japan

P21 Neural representation of perceptual texture dimensions in macaque area V4

Taekjun Kim⋆, Wyeth Bair, and Anitha Pasupathy

University of Washington, Department of Biological Structure, Seattle, WA, United States

P22 Object encoding in macaque inferior temporal cortex under partial occlusion

Tomoyuki Namima⋆, Anitha Pasupathy

University of Washington, Department of Biological Structure, Seattle, WA, United States

P23 The impact of propagation delay in a Linsker-type network

Catherine Davey⋆, David Grayden, and Anthony Burkitt

University of Melbourne, Department of Biomedical Engineering, Melbourne, Australia

P24 A biologically plausible neural model of visual pathways based on efficient coding

Yanbo Lian[1⋆], Hamish Meffin[2], David Grayden[1], Tatiana Kameneva[3], and Anthony Burkitt[1]

[1]University of Melbourne, Department of Biomedical Engineering, Melbourne, Australia
[2]National Vision Research Institute, Carlton, Australia
[3]University of Melbourne, Electrical and Electronic Engineering, Parkville, Vic, Australia

P25 Building and simulating a biophysically detailed network model of the mouse primary visual cortex

Yazan Billeh*, Sergey Gratiy, Kael Dai, Ramakrishnan Iyer, Nathan Gouwens, Stefan Mihalas, Christof Koch, and Anton Arkhipov

Allen Institute for Brain Science, Modelling, Analysis and Theory, Seattle, WA, United States

P26 Nonlinear dynamics tools unfold brain activity in optogenetic experiments

Jessica Helms[1], Xandre Clementsmith[1], Sorinel Oprisan[1]*, Tams Tompa[2], and Antonieta Lavin[3]

[1]*College of Charleston, Department of Physics and Astronomy, Charleston, SC, United States*
[2]*University of Miskolc, Miskolc, Hungary*
[3]*Medical University of South Carolina, Charleston, SC, United States*

P27 On the subthreshold resonance properties of neurons

Rodrigo F. O. Pena*, Vinícius Cordeiro, Cesar C. Ceballos, and Antônio C. Roque

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P28 Implementation of the Potjans-Diesmann cortical microcircuit model in NetPyNE/NEURON with rescaling option

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P29 Effects of spike frequency adaptation on dynamics of a multi-layered cortical network with heterogeneous neuron types

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P30 Information processing from external inputs to the entorhinal cortex grid cells

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P31 Understanding action potential evolution in axon due to focal geometric deformation using a hybrid 1D-3D model

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P32 A quantitative model for estimating the scale of photochemically induced ischemic stroke

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P33 Visualization of pre-motor and parietal network activity patterns during free behavior in rats

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P34 Mean field theory of large and sparse recurrent networks of spiking neurons including temporal correlations of spike-trains

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P35 Probabilistic analysis of high-dimensional stochastic firing rate models: Bridging neural network models and firing rate models

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P36 Input oscillations may stabilize working memory activity

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P37 Artificial evolution of networks of artificial adaptive exponential neurons for multiplicative operations

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P38 Artificial evolution of very small spiking neural network robust to noise and damage for recognizing temporal patterns

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P39 Population and single-neuron measures of multisensory integration

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P40 Optimizing deep convolutional network architectures to match visual cortex

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P41 SIMNETS: a novel mathematical framework to detect functional neuronal sub-ensembles

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P42 Finite size effect for spiking neural network with spatially dependent coupling

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P43 A space-time continuum in the hippocampus?

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P44 Bayesian filtering of uncertain sensory data in the brain: Hamilton's principle approach

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P45 Simultaneous recording of micro-electrocorticography and local field potentials for decoding rat forelimb movement

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P46 Predicting the effects of deep brain stimulation using a coupled oscillator model

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P47 Retinal motion-detection under noisy conditions

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P48 Using information theory and a Bayesian model to examine the factors that influence the decision to consume alcohol in a rodent model of alcoholism

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P49 Stochastic facilitation of encoding process of a dynamical pattern in mouse retina

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P50 Diverse dynamics in small recurrent networks: A case study of coupled recurrent and coupled inhibitory neurons

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P51 Morpho-electric properties and computational simulation of human dentate gyrus granule cells from the epileptogenic hippocampus

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P52 Development of realistic single-neuron models of mouse V1 capturing in vitro and in vivo properties

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P53 A multi-modal discovery platform toward studying mechanisms-of-action of electric brain stimulation

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P54 Kernel current source density revisited

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P55 Modelling the electrical impedance of neural tissue based on its cellular building blocks

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P56 Local and global neuronal network structure influence synchronous events

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P57 Interplay of synaptic noise and chaos determines limits of cortical reliability

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P58 Shedding light on the cellular origins of voltage-sensitive dye imaging: an in silico study

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P59 Reconstruction and simulation of a full-scale model of rat hippocampus CA1

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P60 The SONATA data format: A new file format for efficient description of large-scale neural network models

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P61 Stability of synaptic weights in a biophysical model of plasticity in the neocortical microcircuit without explicit homeostatic mechanisms

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P62 Biophysical modeling of synaptic plasticity in the somatosensory cortex

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P63 Dynamic Worm: Moving model of Caenorhabditis elegans worm controlled by the nervous system

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P64 Modeling network connectivity for dopamine-mediated olfactory learning in mosquitos.

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P65 Detection of spatio-temporal spike patterns in motor cortex during a reach-to-grasp task

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P66 Random contrastive Hebbian Learning as a biologically plausible learning scheme

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P67 Activity of neural circuit in V1 during locomotion demystified

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P68 Dimensionality of recurrent neural networks trained to classify spatially clustered inputs

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P69 How connectivity motifs shape the dimensionality of network response

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P70 Action potential propagation in axons: Effect on sodium conductance of collateral and sub-branch distance from soma

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P71 Action potential propagation in axons: How sodium conductance can estimate propagation as collateral and sub-branch length vary

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P72 Dentate gyrus network model

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P73 The ratio of specialist and generalist neurons in the feature extraction phase determines the odor processing capabilities of the locust olfactory system

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P74 Regulation of neural threshold in Kenyon cells through their sparse condition improves pattern recognition performance

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P76 Local excitatory/inhibitory imbalances shape global patterns of activity: A model for desynchronized activity under anesthesia in Alzheimer's disease

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P77 Neural automata

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P78 Predictable variability in sensory-evoked responses in the awake brain: optimal readouts and implications for behavior

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P79 Selectivity and sensitivity of cortical neurons to electric stimulation using ECoG electrode arrays

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P80 Patterns of gastrointestinal motility and the effects of temperature and menthol: A modelling approach

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P81 Mechanisms underlying locomotion and paw-shaking rhythms in cat multifunctional central pattern generator

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P82 The role of Na+/K+ pump in intrinsic intermittent bursting dynamics in model neuron of the Pre-Bötzinger Complex

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P83 Changes in relaxation time predict stimulus-induced reduction of variability at the single-cell level

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P84 Intracellular fluxes contributing to [Ca2+]i responses in rat magnocellular neurons

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P85 Spatiochromatic integration by double opponent neurons in macaque V1

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P86 A dynamic causal modeling of voltage sensitive dye imaging (VSDI-DCM) in the rodent hippocampus

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P87 Estimation of effective connectivity in the microcircuits of the mouse barrel cortex using dynamic causal modeling of calcium imaging

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P88 Predictions of neuronal connectivity from axonal and dendritic arbors

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P89 Optimal wiring imposes fixed cortical hypercolumn sizes

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P90 Dendritic branching statistics explained from minimal wiring constraints

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P91 Dissecting the structure and function relationship in Drosophila dendrite development with the help of computational modelling

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P92 Dimensionality reduction of brain signals of rats by Spectral Principal Component Analysis (SPCA)

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P94 Advancing computational studies of the nervous system: Publishing models not paper descriptions of models

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P95 Disentangling diverse patterns of synaptic efficacy in vivo and their causes

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P96 Optimizing stimulation protocols for prosthetic vision based on retinal anatomy

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P97 Effect of use dependent plasticity on information transfer at hippocampal synapses

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P98 A neural network model of complementary learning systems

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P99 Separation of hemodynamic signals from GCaMP fluorescence measured with widefield imaging

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P100 Brains on board: Neuromorphic control of flying robots

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P101 Gamma genesis and phase-amplitude coupling in a model of striatal fast-spiking interneurons

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P102 Brain activity in a spherical geometry via neural field theory

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P103 Model of plasticity in re-learning auditory and visual localization cues

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P104 Optimal readout of neural activity near criticality

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P105 Robust dendritic computations with sparse distributed representations

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P106 Sparse coding and dimensionality reduction in the brain

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P107 Network interactions can mask intrinsic dynamics in rhythmic circuits

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P108 Modeling predicts altered ion channel mechanisms and firing properties in striatal neurons of the Q175 mouse model of Huntington's disease

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P109 Influence of cortical network topology and delay structure on EEG rhythms in a whole-brain connectome-based thalamocortical neural mass model

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P110 Characterizing neural selectivity in multidimensional sensory feature space

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P111 Twin fingerprinting: Optimal mapping of heritable traits in the human connectome

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P112 Spike-timing-dependent plasticity effect on the patterns of neural synchrony

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P113 Modeling the variability of spontaneous astrocyte calcium activity and responses to repeated stimuli

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P114 From connectivity to activity: Community detection reveals multiple simultaneous dynamical regimes within networks

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P115 A generalized platform for modeling electric field effects on neuronal dynamics

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P116 Synchronization by uncorrelated noise: interacting rhythms in interconnected neuronal networks

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P117 Classification of morphological and electrophysiological types in mouse visual cortex

Nathan Gouwens*, Staci Sorensen, Jim Berg, Changkyu Lee, Tim Jarsky, Jonathan Ting, Michael Hawrylycz, Anton Arkhipov, Hongkui Zeng, Christof Koch, Susan Sunkin, David Feng, Colin Farrell, Hanchuan Peng, Ed Lein, Lydia Ng, Amy Bernard, and John Phillips

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P118 Soma-axon coupling configurations that enhance neuronal coincidence detection

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P119 Short-term plasticity of GABAergic synapses in the Substantia Nigra pars reticulata

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P120 Simulating pharmacological blockade of persistent sodium currents in respiratory circuits

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P121 Weak-noise-induced transitions with inhibition and modulation of neural oscillations

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P122 Randomness and structure in artificially generated neuronal networks

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P123 Moving towards the Single Cell Projectome: A multi-modal approach to assessing single-cell morphology and connectivity for classification of layer 2/3 neurons in mouse V1

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P124 Oscillatory and broadband contributions to directed functional connectivity in the human cortex

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P125 Facilitatory mechanisms during the encoding of frequency-modulated sweeps in the auditory pathway

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P126 A rigorous statistical test supports a new model of homeostatic plasticity

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P127 Novel approaches to optimize biophysically detailed computational models of single neurons

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P128 Construction of a biochemically detailed single-compartment model for post-synaptic long-term potentiation: application to cortical plasticity

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P129 What is the resistivity of the human brain? Insights from direct electrical stimulation, electrocorticographic recordings of the human cortex, and analytic models

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P130 Improvement of computational efficiency of a biochemical plasticity model

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P131 Modeling traveling wave dynamics in the visual cortex

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P132 Cusps enable line attractors and graded information channels in neural computation

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P133 Population vector decoding for optical imaging with fNIRS (functional near-infrared spectroscopy)

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P134 Firing-rate based network modeling of the dLGN circuit: Effects of cortical feedback on spatiotemporal response properties of relay cells

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P135 Computational modeling of neuron-astrocyte interactions: Evolution, reproducibility, comparability and future development of models

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P136 Data-driven study of synchronous population activity in generic spiking neuronal networks: How much do we capture using the minimal model for the considered phenomena?

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P137 Fast gabaergic neurotransmission inhibits diversely AMPA and NMDA receptor mediated network dynamics in cortical cultures: A model-driven experimental study

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P138 A neural mass model to predict electrical stimulation evoked responses in human brain

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P139 Missing data for an electrodiagnostic nerve test

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P140 Neural model of the multi-stable dynamics of the perception of body motion

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P141 Detecting and classifying neocortical population codes via deep artificial neural networks

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P142 Blind recovery of transient responses with higher-order spectra

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P143 Early spontaneous activity predicts structural changes in layout of orientation domains during early development

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P144 Multispike Tempotron performance under different task-related neural spiking statistics

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P145 Modeling mouse visual cortex

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P146 On the correspondence between receptive fields derived from spikes versus calcium

Peter Ledochowitsch[1]*, Nicholas Cain[1], Joshua Siegle[1], Xiaoxuan Jia[2], Michael Oliver[1], Ulf Knoblich[3], Lawrence Huang[3], Brian Hu[1], Gabriel Ocker[1], Daniel Millman[1], Séverine Durand[1], Ramakrishnan Iyer[1], Lu Li[3], Shawn Olsen[1], R Clay Reid[1], Hongkui Zeng[1], Stefan Mihalas[1], Saskia E. J. de Vries[1], and Michael A. Buice[1]

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P147 The structure of population activity and coding in mouse visual cortex.

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P148 Online biologically plausible decoding of clusters in retinal population activity

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P149 Towards a computational account of theta band (4-8 Hz) power modulation in the subthalamic nucleus during response conflict condition.

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P150 Mediodorsal thalamus permits cognitive flexibility by suppressing conflicting prefrontal representations

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P151 Long memory in dynamic recurrent networks

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P152 Stimulus-dependent tuning in cortical area MST of macaques

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P153 Validation and performance of effective network inference using multivariate transfer entropy with IDTxl

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P154 Generative models on accelerated neuromorphic hardware

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P155 Modeling rhythmic control of brain sequential dynamics

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P156 An excitation / inhibition ratio impacts on organization of neural connectivity and information transfer

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P157 Intrinsically bursting neurons enlarge timescales of fluctuations in firing rates

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P158 Acetylcholine modulation in a biophysical model of cortical neuron

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P159 Anesthesia modifies subthreshold critical slowing in a stochastic Hodgkin-Huxley neuron exposed to inhibitory synaptic noise

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P160 Identifying 'influential seizers' in a network model of focal epilepsy

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P161 Rich dynamical repertoire in the balanced state

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P163 Prefrontal oscillations bias pathways for thought and action

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P164 From single neurons to perception: Examining the basis for sensory deficits in autism

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P165 Cortical information integration with critical subnetworks: Large capacity, high accuracy, and rapid detection.

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P166 Neuroscience gateway: Enabling large scale simulations and data processing and dissemination of neuroscience tools/software

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P167 Computational model of the conditional probability of decision-making process as an optimization process

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P168 PyRates - A Python framework for rate-based neural simulations

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P169 A stochastic model of single serotonergic fibers

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P170 Neural model for the recognition of agency and social interaction from abstract stimuli

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P171 Learning oscillatory brain dynamics: van der Pol meets LSTM

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P172 A cross-platform real-time model library to build hybrid neural circuits

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P173 Unveiling and characterizing dynamical invariants in central pattern generators

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P174 Point process-based dynamic functional connectivity with source-reconstructed EEG data

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P175 Modeling the spatial inhomogeneous degradation of nitric oxide shows a key role of anatomically localized NO production

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P176 A Bayesian, biophysical framework for spike sorting

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P177 A detailed model of the hippocampal formation for the generation of Sharp-Wave Ripples and Theta-nested Gamma oscillations

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P178 A mechanistic model explains auditory evoked responses as a reflection of network properties of the entire auditory cortex

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P179 Noisy deep networks with short-term plasticity make similar errors as mice in a detection of change task

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P180 Statistical properties of strengths of structural and functional connectivity

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P181 Plasticity of information coding by cerebellar Purkinje cells during sensorimotor learning

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P182 A systematic comparison of neural morphology representations in the context of cell type discrimination

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P183 Online accurate spike sorting for hundreds of channels

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P184 Time step sensitivity in large scale compartmental models of the neocortex

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P185 Electrical coupling of perisomatic and distal apical regions of a layer 5 pyramidal neuron compartmental model

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P186 Impact of small world connectivity on a multi-region model of cerebral cortex

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P187 Transcranial direct current stimulation (tDCS) is impacted by neuronal morphology and spatial configuration

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P188 Simulating extracellular signatures of action potentials using single compartment neurons and geometrical filtering

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P189 Learning the payoffs and costs of actions

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P190 A network of intrinsic oscillators can drive forward locomotion in C. elegans

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P191 Computational validation of a closed loop neuromorphic controller for ventilatory control

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P192 Modeling the altered function of canonical feedback inhibitory circuits in chronic epilepsy

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P193 Investigating impact of synaptic inputs in seizure models

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P194 Fundamental neuromechanical components of robust forward locomotion in C. Elegans

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P195 A reservoir computing model of motor learning with parallel cortical and basal ganglia pathways

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P196 Dynamic features of neural responses to triplet-streaming simulated by integrate-and-fire networks of core auditory cortex

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P197 Reduction of conductance-based neuron models for neuromodulation studies

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P198 System identification of neuronal dynamics

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P199 Neuromorphic hyperpolarized bursting

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P200 Robust regulation of neuronal dynamics by the Na/K pump

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P201 Gender differences in intrinsic oscillations of the resting brain following brief mindfulness intervention

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P202 Tonic-to-bursting transitions in synchronous gap junction coupled neurons

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P204 In the footsteps of learning: Changes in network dynamics and dimensionality with task acquisition

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P209 A dynamic resource model for sequential working memory

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P210 Retinal development of cortical functional circuits

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P215 Laminar contributions to auditory feature processing

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P220 Functional connectivity in mouse visual cortex revealed by large scale recordings

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P228 The emergence of spatiotemporal spike patterns and feature binding relations within a spiking neural network model of the primate visual cortex: a cortical implementation of capsule networks.

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P230 Learning to be modular: Interplay between dynamics of synaptic strengths and neuronal activity in the brain results in its modular connection topology

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P242 Multiplexed coding using differentially synchronized spikes: Part 2, experiments.

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P244 Multiplexed coding using differentially synchronized spikes: Part 1, theory and simulations

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P254 Spatial patterns of synchrony from electrical synapses in the inferior olive

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P273 Axonal dynamics: Signal propagation and collision

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P275 Oscillations and chaos in adaptive neural networks

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P277 Balancing of orientation preference in primary visual cortex

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P280 Cortical dynamics on multiple time-scales drive growth of smooth maps to- gether with local heterogeneity

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P281 Large-scale cortical model based on structural connectivity on aging APOE-4 allele carriers

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P282 Approximate Bayesian inference for a neural mass model of anaesthesia

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P284 Effect of floating point precision on dynamics of membrane potential in neural simulation

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P285 Decomposing adaptable elements of optokinetic response into cerebellar and non-cerebellar contributions by modeling and cerebellectomy approach

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P286 Differential functions of calcium dynamics in synaptic plasticity

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