
CNS 2013
July 13-18, Paris, France
T&M BML

Contents

Overview

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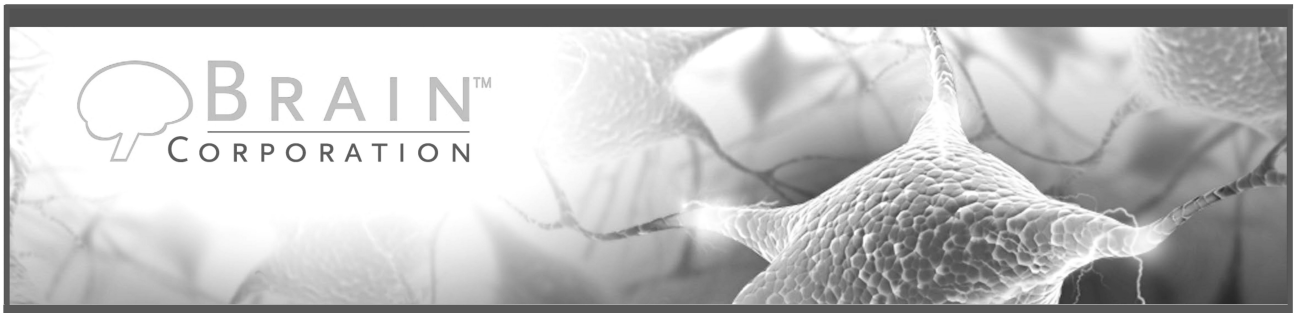

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Experience with neural networks and machine learning.
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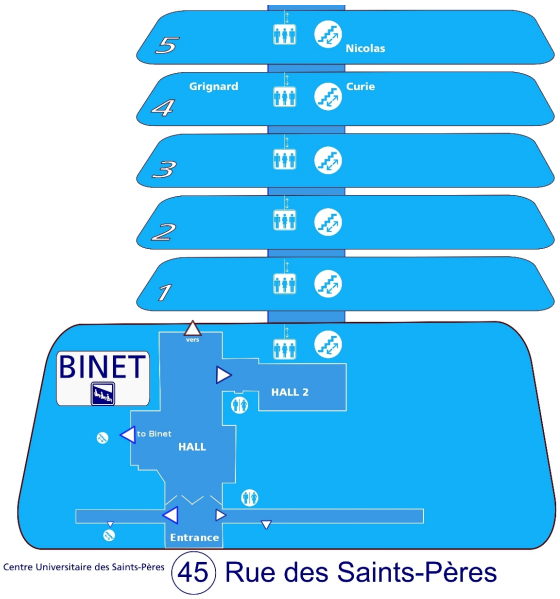
Timetable

	TUTORIALS	MAIN MEETING			WORKSHOPS	
	Saturday, July 13	Sunday, July 14	Monday, July 15	Tuesday, July 16	Wednesday, July 17	Thursday, July 18
08:00	Registration	Registration	Registration	Registration	Registration	Registration
08:10						
08:20						
08:30						
08:40						
08:50						
09:00	**TUTORIALS*** *Morning session 1*	**Announcement**	**Announcement**	**Announcement**	**WORKSHOPS*** *Morning session 1*	**WORKSHOPS*** *Morning session 1*
09:10		**Keynote 2** *S. Laughlin*	**Keynote 3** *S. Denève*	**Oral session 6** *Motor Control* / **Featured Oral 3**		
09:20						
09:30						
09:40						
09:50				**Oral 16**		
10:00						
10:10	Break	Break	Break	Break	Break	Break
10:20						
10:30	**TUTORIALS** *Morning session 2*	**Oral session 1** *Synapses & Plasticty*: **Oral 1**, **Oral 2**, **Oral 3**, **Oral 4**	**Oral session 4** *Sensory Mechanisms*: **Oral 11**, **Oral 12**, **Featured Oral 2**	**Oral session 6** *Motor Control* **Oral 17**; **OCNS member meeting**	**WORKSHOPS** *Morning session 2*	**WORKSHOPS** *Morning session 2*
10:40						
10:50						
11:00						
11:10						
11:20						
11:30						
11:40						
11:50						
12:00		Lunch Break	Lunch Break *(Funding and other opportunities related to computational neuroscience – informal information session)*	Lunch Break		
12:10						
12:20	Lunch Break				Lunch Break	Lunch Break
12:30						
12:40						
12:50						
13:00						
13:10						
13:20						
13:30	**TUTORIALS** *Afternoon session 1*	**Oral session 2** *Visual System*: **Oral 5**, **Oral 6**, **Oral 7**, **Oral 8**	**Oral session 5** *Hippocampus*: **Oral 13**, **Oral 14**, **Oral 15**	**Oral session 7** *Network Structure & Dynamics*: Oral 18, Oral 19, Oral 20, Oral 21	**WORKSHOPS** *Afternoon session 1*	**WORKSHOPS** *Afternoon session 1*
13:40						
13:50						
14:00						
14:10						
14:20						
14:30						
14:40						
14:50						
15:00			**Poster session 2** **P146-P290**			
15:10						
15:20	Break	Break		Break	Break	Break
15:30						
15:40						
15:50	**TUTORIALS** *Afternoon session 2*	**Oral session 3** *Correlations*: **Oral 9**, **Featured Oral 1**, **Oral 10**		**Oral session 7** *Network…* Oral 22; Brain Corp. Prize Announcement; **Keynote 4** *R. Yuste*	**WORKSHOPS** *Afternoon session 2*	**WORKSHOPS** *Afternoon session 2*
16:00						
16:10						
16:15						
16:20						
16:30						
16:40						
16:50						
17:00	Welcome & Announcements	**Poster session 1** **P1-P145**	Travel to Gala Dinner *(Subway recommended. We are also providing buses on first come first serve basis)*	**Poster session 3** **P291-P435**		
17:10						
17:15						
17:20	**Keynote 1** *NK Logothetis*					
17:30						
17:40						
17:50						
18:00						**Postdoc and student career strategy workshop**
18:10						
18:15						
18:20	Welcome reception		**Gala Dinner!**			
18:30						
18:40						
18:50						
19:00						
19:10						
19:20						
19:30						
19:40						
19:50						
20:00						
20:10	*Enjoy your evening!*	*French National Day!* *Try to enjoy the view of the Eiffel Tower fireworks!*		*Enjoy your evening!*		
20:15						

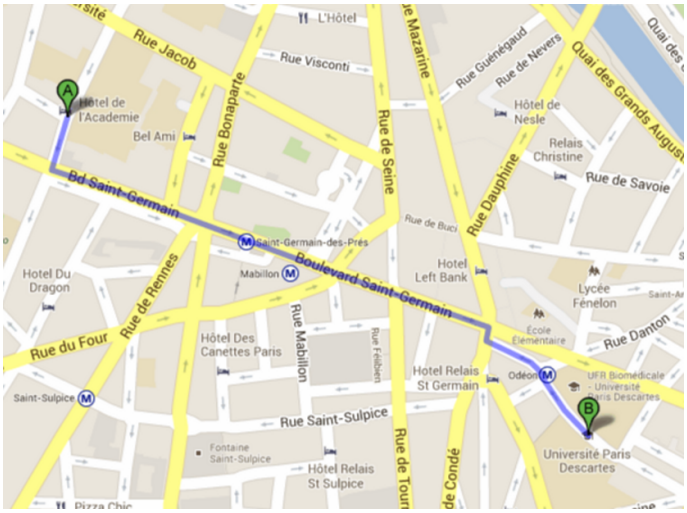
General Info

At the Meeting Venue

The main meeting, as well as all the tutorials and the workshops (apart from W2), will be held at the Paris Descartes University - Centre Universitaire des Saints-Pères, 45 rue des Saints-Pères (Map 1). The workshop W2 will be held at the University Headquartes (12 rue de l'École de Medécine) which are located at 15 minutes walking distance from the Centre Universitaire des Saints-Pères. Directions are given in Map 2.



Map 1



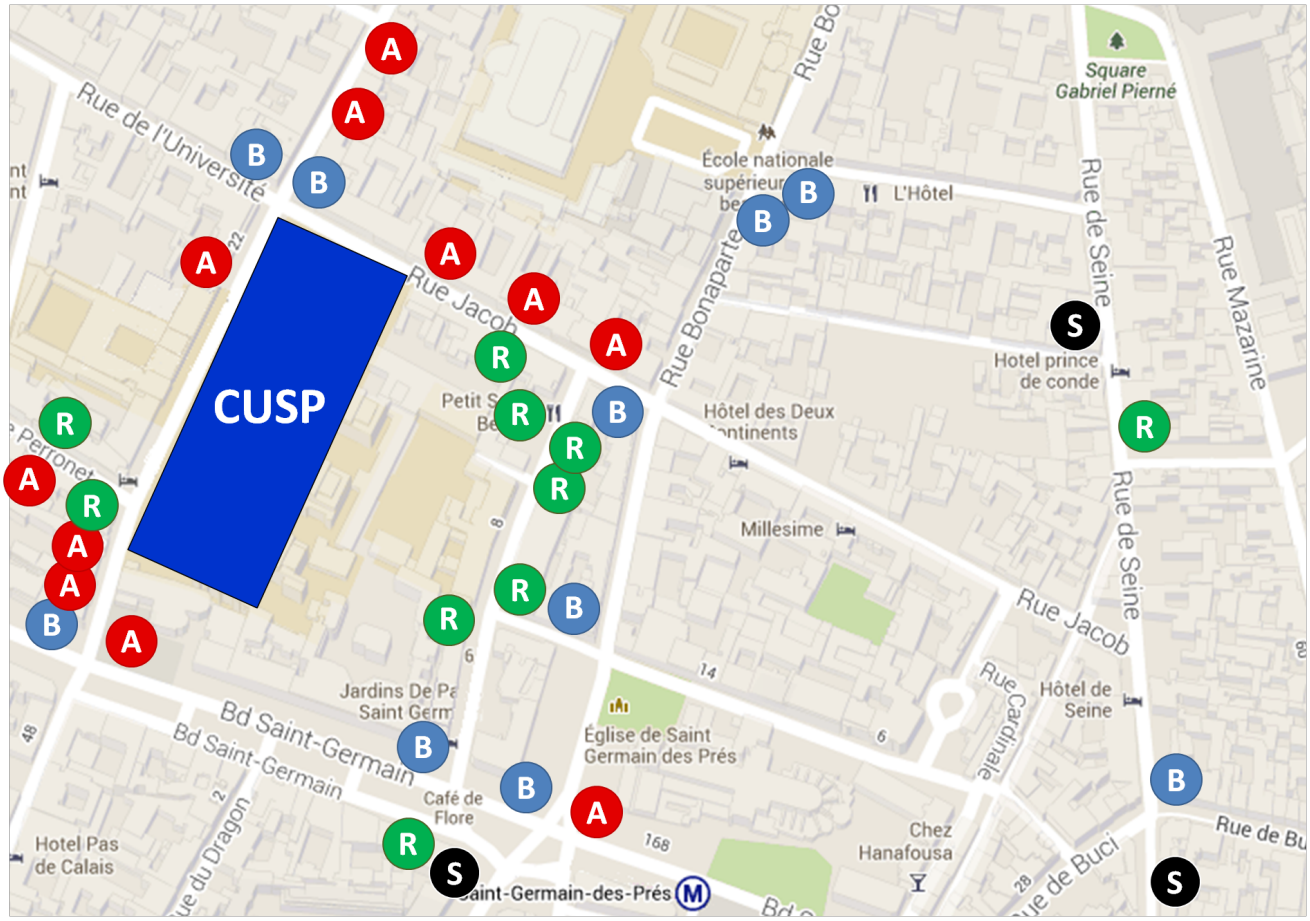
Map 2: **A** - Meeting venue; **B** - W2 location.

What	**Where (When)**
Tutorials	Grignard and Curie (4th floor) – July 13
Keynote Lectures	Binet – K1: July 13, 5.15pm; K2: July 14, 9.10am; K3: July 15, 9.10am; K4: July 16, 4.10pm
Welcome Reception	Hall – July 13, 6.15pm-8pm
Registration	Hall – July 13-18
Oral Sessions	Binet – July 14-16
Poster Sessions	Hall and Hall 2 – July 14-16
Workshops	Grignard, Curie (4th floor); Nicolas (5th floor) – July 17-18 W2 at Salle du Conseil (see Map 2) – July 17-18
Coffee Breaks	Hall 2
OCNS meetings	LNP Meeting Room (H335) 3rd floor – July 13-16
Gala Dinner	July 15, 7pm – see page 19 for location and directions
Exhibits	Hall 2 – July 13-18

Around the Meeting Venue

The Centre Universitaire des Saints-Pères is centrally located in the lively Saint Germain-des-Prés neighborhood. The neighborhood is well-furnished with *boulangeries* (bakeries), *brasseries* (literally breweries, they are small restaurants serving simple food besides beer, of course) and restaurants. Prices range from reasonably cheap to unreasonably expensive. In the map below you will find a small selection of places you can reach within a 5 minutes walking. Note, however, that on July 14, which is both a Sunday and the French National Day, some of these places will be closed. No worries, you can simply walk to the Saint-Michel neighborhood (15 minutes) where everything will be open.

If you want to eat your sandwich at banks of the Seine, go out of the Centre Universitaire and follow the Rue des Saints-Pères rightward for 5 minutes.



CUSP – Centre Universitaire des Saints-Pères
A – Take Away
B – Brasserie
R – Restaurant
S – Supermarket

Local Information

Since a list of all tourist attractions, restaurants and interesting things in Paris would triple the size of this book, we decided to collect just a few suggestions from people that live in Paris. You probably got you a guide book anyway. (And you wouldn't read four pages of restaurant suggestions, would you?)

Restaurants:

- **Le Procope**, 13 Rue de l'Ancienne Comédie. Tel: +33 1 40 46 79 00
  The oldest restaurant of Paris in continuous operation since 1686. Traditional french food for around 30€, but there is a perfectly fine menu for 21€.

- **Au P'tit Grec**, 62 Rue Mouffetard
  Here you can eat delicious crepes for a reasonable price (around 5€). There is often a queue, but it is worth waiting for.

- **Les Pipos**, 2 Rue de l'École Polytechnique. Tel: +33 1 43 54 11 40
  An often very crowded but atmospheric bar á vins. The menu is filled of meat dishes and delicacies from the french countryside tradition. Prices around 35€.

- **Le Jardin d'Ivy**, 75 rue Mouffetard. Tel : 01.47.07.19.29
  A restaurant with cozy and romantic atmosphere, with lots of candles and a flower garden as background. With the 28€menu you will get delicious food. Highly recommended.

- **Leon**, 131 Boulevard Saint-Germain. Tel: +33 1 43 26 45 95
  A little more than a bistro, but certainly not a fancy restaurant. Mussels, served in a thousand different ways and flavors for prices starting from around 20€.

- **Brasserie Lipp**, 151 Boulevard Saint-Germain. Tel: +33 1 45 48 53 91
  A Paris classic, the atmosphere is vintage and the service not always perfect. Try out the choucroute Lipp, sauerkraut, sausages and pork knuckle, apart from the mash, beer demi Lipp blonde. Rather expensive, though: 50-60€ for a full meal.

- **La Coupole**, 102 Boulevard du Montparnasse. Tel: +33 1 43 20 14 20
  Beautiful brasserie, which was also a ballroom in the early 20th century. It is best to book in advance since it is always very crowded. Prices are above average (60-70€), but the huge plateau dishes of fresh seafood are certainly worth a try.

- **Polidor**, 41 Rue Monsieur le Prince. Tel: +33 1 43 26 95 34
  5 stars for the traditional and popular ambience. 4 stars for the kitchen with many traditional French dishes as the roast duck with cabbage. No reservations taken: go there at 7.30pm or earlier. Prices 30-35€.

- **Han Lim**, 6 Rue Blainville. Tel: +33 1 43 54 62 74
  One of the best korean restaurant in Paris. Quiet ambiance, sincerely hospitable service. Lunch menu at 13€, a key specialty Korean BBQ around 16€.

- **Chez Gladines**, 30 Rue des 5 Diamants. Tel: +33 1 45 80 70 10.
  A very popular Basque Restaurant, exellent hearty food, great rate quality/price. You cannot reserve, make sure you show up early, otherwise you have to wait. Around 15€.

- **Pietro Pizzeria**, 65 Boulevard de Vaugirard. Tel: +33 1 43 22 61 46.
  The authentic napolitan pizzas are very good, the staff is friendly and the desserts are awesome. Around 15€.

- **Pizza Vesuvio**, 1 rue Gozelin. Tel: +33 1 83 76 16 54.
  Very good Pizzeria close to the main meeting venue. Around 14€.

Bars and Clubs:

- **Académie de la Bière**, 88B Boulevard de Port-Royal
  Small bar with lots of beers, mainly from Belgium.

- **Chez Prune**, 36 Rue Beaurepaire
  Situated on the Canal Saint-Martin, a nice bar to sit outside.

- **Truskel**, 12 Rue Feydeau
  Close to Grands Boulevards. A small, dark bar with rock, alternative and pop from the 80s and 90s.

- **L'assasin**, 99 rue Jean-Pierre Timbaud
  Alternative bar with live music. You can get great food here as well.

- **Brasserie La Maison**, 65 Boulevard de la Villette
  A french bar. People start to dance around midnight.

- **Favela Chic**, 18 Rue du Faubourg du Temple
  Club with brasilian atmosphere.

- **Café Oz**, 3, place Denfert-Rochereau
  Australian bar/club, open every day. Sunday to Tuesday there are usually concerts in the evening. Wednesday to Saturday party at night.

- **Rex Club**, 5 Boulevard Poissonnière
  Electro Club.

- **In general**, you can find a lot of nice places around **Rue Mouffetard** (close to the Panthéon), in the area of the **Grands Boulevards** (Boulevard Montmartre and Rue Montmartre) and around **Rue Oberkampf**.

Some Things to Do:

- The 14th of July is the **French National Day**. There will be **fireworks** at around 11pm at Trocadero (close to the Eiffel Tower).

- **Bals des pompiers**: On the 13th and 14th of July there will parties at many firestations all around Paris organized by the firefighters.

- The **Musée du quai Branly** features indigenous art, cultures and civilizations from Africa, Asia, Oceania, and the Americas.

- The **Musée d'Orsay** is very close to the main meeting venue. So is the **Louvre**.

- Take a **bike** from one of the bike-sharing stations and have a tour **at night**, when there is no traffic. (See below for the bikesharing.)

- You have a nice 360°-view over Paris on the **Tour Montparnasse** (13,5 €, 10,5 €for students).
- Visit the **Parc des Buttes-Chaumont**. It is probably the most beautiful park in Paris.
- Have a walk on the **Coulée verte**, an elevated parkway which allows you to walk over the roofs of Paris. It can be accessed close to the Opera Bastille.
- Visit the **Canal Saint-Martin**. It is very nice to have a picnic on its banks, but there are also a number of popular restaurants and bars along it.

Other Information

Money:

- Visa and Mastercard are accepted in almost all stores.
- Banks are generally open from 9am to 5pm, or 6pm, from Monday to Friday, sometimes from Tuesday to Saturday. Certain branches may close at lunchtime, between 12.30pm and 2pm.
- All prices generally include taxes.

Airports:

Paris has two airports: Charles De Gaulle Airport (CDG) and Orly Airport (ORY). The simplest, cheapest, and fastest way to get to Paris from its airports is to take the RER B. From CDG, this will bring you directly to the *Saint-Michel* station (which is within walking distance from the meeting venue) or to the *Châtelet station*, from where you can change into Metro line 4 (direction *Porte d'Orléans*) and get off at the *Saint-Germain-des-Prés* station. From ORY, there is a shuttle service, called *Orlyval*, that will bring you to the RER B station *Anthony*. From there you can go to the *Saint-Michel* station or you can change into Metro line 4 (direction *Porte de Clignancourt*) at the *Denfert-Rochereau* station, and get off at the *Saint-Germain-des-Prés* station. Taxi from CDG costs 50-60 €, from ORY 30-40 €.
If you are landing at Beauvais Airport, you will find a bus shuttle service that will bring you to Paris *Porte Maillot*. From *Porte Maillot* you can take Metro line 1 (direction *Château de Vincennes*) and get off at the *Palais Royal (Musée du Louvre)* station. From there, it is only a short walk to the meeting venue.

Local Transport:

- **Metro/Bus/RER**: Tickets for Metro, Bus and RER (a kind of regional Metro) can be bought from all vending machines in the Metro stations, either individually (1,70 €) or as a *carnet*, that is, in a packet of 10 (13,30 €). If you plan to use public transportation frequently during your stay, you can buy a 'Paris Visite' ticket which grants you unlimited access to almost all transportation systems for up to 5 days (10,55 €- 33,70 €). The **last Metro** runs at around 0:45 am, except on Fridays, Saturdays and on nights before a holiday, when the service ends at around 1:45 am.
- **Taxi**: The common telephone number for all taxi companies is: 01 45 30 30 30.
- **Bike**: You can find 'velib' bike-sharing stations everywhere in Paris. A 1-day ticket costs 1,70 €, a 7-day ticket costs 8 €. A credit card is required to use the service.

Useful Numbers:

- Ambulance: 15
- Police (emergencies): 17
- European emergency number: 112
- Fire brigade: 18

Hospitals:

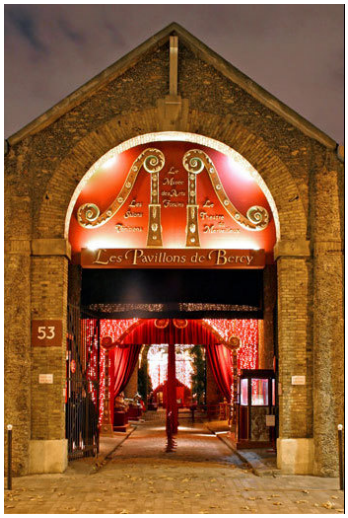
- The closest to the meeting venue is Hotel-Dieu. It is situated on the Île de la Cité, very close to the Notre Dame Cathedral (the famous one). Phone: 01 42 34 82 34.
- For paediatric emergencies: Hôpital Necker. Phone: 01 44 49 42 90.

Below is a map with main meeting, W2 and Gala dinner locations, as well as the locations of the main train stations in Paris.

MM	–	Meeting venue
W2	–	W2 location
BQ	–	Gala dinner
GN	–	Gare du Nord
GE	–	Gare de l'Est
GL	–	Gare de Lyon
GM	–	Gare de Montparnasse

Gala Diner

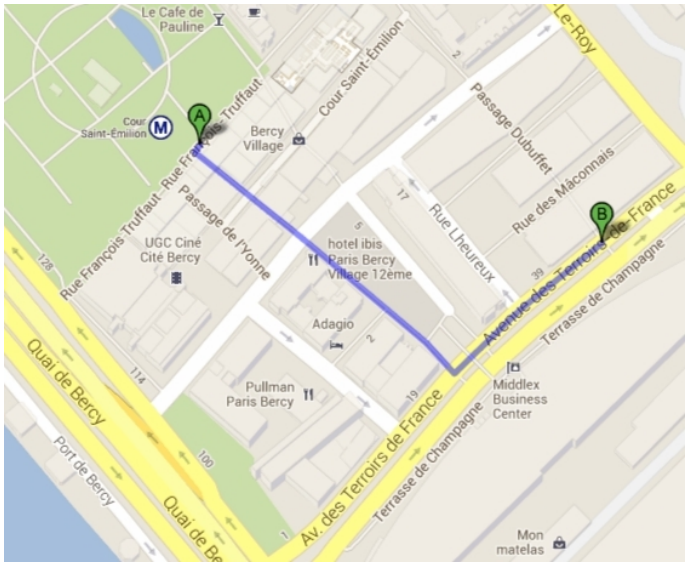
The gala diner will take place on Monday evening at 7pm in the 'Musée des Arts Forains' in the Bercy Quarter. A buffet will be served in the ambience of a vintage funfair museum.



Buses from the meeting venue to the museum will be provided on 1st come 1st served basis.

If you don't make it to the bus, take the **Metro**, it's very easy:

- Take line 4 at Saint Germain-des-Prés in direction Porte de Clignancourt.
- In Chatelet, change to line 14 in direction Olympiades.
- Get out at Cour Saint-Émilion.
- Follow the map below:



Program

Tutorials

	Saturday, July 13	
Space	**Morning (9:00 - 12:20)**	**Afternoon (13:30 - 16:50)**
Curie	**T1 -** Neural-mass and neural-field models	
Curie	**T2 -** Theory of correlation transfer and correlation structure in recurrent networks	
Grignard	**T3 -** Modeling and interpretation of extracellular potentials	
Grignard	**T5 -** Brain activity at rest: Dynamics and structure of the brain in health and disease	**T4 -** Probabilistic inference as a neural-computing paradigm
Grignard	**T6 -** Developing neuron and synapse models for NEST	
Curie	**T7 -** Advanced modelling of spiking neural networks with BRIAN	
Curie	**T8 -** Managing complex workflows in neural simulation and data analysis	
Grignard	**T9 -** Massively parallel time encoding and channel identification machines	

T1 **Neural-mass and neural-field models**

Space Curie, (9.00-12.20; 13.30-16.50)

Axel Hutt, INRIA Nancy, France
Viktor Jirsa, Institut de Neuroscience de Systemes, France
John Terry, University Exeter, UK
Wolfram Erlhagen, University Minho, Portugal

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

Space Curie, (9.00-12.20; 13.30-16.50)

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

T3 **Modeling and interpretation of extracellular potentials**

Space Grignard, (9.00-12.20; 13.30-16.50)

Gaute T Einevoll, Norwegian University of Life Sciences, Ås, Norway
Szymon Leski, Nencki Institute of Experimental Biology, Warsaw, Poland
Espen Hagen, Norwegian University of Life Sciences, Ås, Norway

T4 **Probabilistic inference as a neural-computing paradigm**

Space Grignard, (13.30-16.50)

Dejan Pecevski, Graz University of Technology, Graz, Austria

T5 **Brain activity at rest: Dynamics and structure of the brain in health and disease**
Space Grignard, (9.00-12.20)

Gustavo Deco, Universitat Pompeu Fabra, Spain

T6 **Developing neuron and synapse models for NEST**
Space Grignard, (9.00-12.20; 13.30-16.50)

Abigail Morrison, Research Center Jülich, Germany
Jochen M Eppler, Research Center Jülich, Germany

T7 **Advanced modelling of spiking neural networks with BRIAN**
Space Curie, (9.00-12.20; 13.30-16.50)

Romain Brette, École Normale Supérieure, Paris, France
Marcel Stimberg, École Normale Supérieure, Paris, France
Victor Benichoux, École Normale Supérieure, Paris, France
Cyrille Rossant, University College London, UK
Nelson Cortés Hernández, École Normale Supérieure, Paris, France
Dan Goodman, Massachusetts Eye and Ear Infirmary, Boston, USA
Bertrand Fontaine, Albert Einstein College of Medicine, New York, USA

T8 **Managing complex workflows in neural simulation and data analysis**
Space Curie, (9.00-12.20; 13.30-16.50)

Andrew P Davison, UNIC, CNRS, Gif sur Yvette
Sonja Grün, Research Center Jülich, Germany
Michael Denker, Research Center Jülich, Germany

T9 **Massively parallel time encoding and channel identification machines**
Space Grignard, (9.00-12.20; 13.30-16.50)

Aurel A. Lazar, Columbia University, New York, US

Main Meeting

Saturday July 13

9:00 – 16:30 **Tutorials**

17:00 – 17:15 **Welcome and announcements**

17.15 – 18:15 K1 **Keynote 1:**
Studying Large-Scale Brain Networks: Electrical Stimulation & Neural-Event-Triggered fMRI
Nikos K Logothetis

18:15 – 20:00 **Welcome reception (appetizers and buffet)**

Sunday July 14

9:00 – 9:10 **Announcements**

9:10 – 10:10 K2 **Keynote 2:**
The influence of metabolic energy on neural computation
Simon Laughlin

10:10 – 10:40 **Break**

Oral session I: Synapses and plasticity

10:40 – 11:00 O1 ***Endocannabinoids mediate spike-timing dependent potentiation and depression: a model-based experimental approach***
Yihui Cui, Vincent Paille, Bruno Delord, Stéphane Genet, Elodie Fino, Laurent Venance, and Hugues Berry*

11:00 – 11:20 O2 ***Trial-to-trial tracking of excitatory and inhibitory synaptic conductance using Gaussian-mixture Kalman filtering***
Milad Lankarany*, Wei. Ping Zhu, M. N. S. Swamy, and Taro Toyoizumi

11:20 – 11:40 O3 ***Inhibitory STDP generates inverse models through detailed balance***
Maren Westkott, Christian Albers, and Klaus Pawelzik*

11:40 – 12:00 O4 ***A cerebellar learning model that reproduces the behavior of vestibulo-ocular reflex adaptation in wild-type and knock-out mice***
Claudia Clopath*, Aleksandra Badura, Chris De Zeeuw, and Nicolas Brunel

12:00 – 14:00 **Break for lunch**

Oral session II: Visual system

14:00 – 14:20 O5 ***We now know what fly photoreceptors compute***
Uwe Friderich, S. A. Billings, Mikko Juusola, and Daniel Coca*

14:20 – 14:40 O6 ***Spontaneous emergence of simple and complex receptive fields in a spiking model of V1***
Eugene M. Izhikevich, Filip Piekniewski, Jayram Nageswaran, Csaba Petre, Micah Richert*, Sach Sokol, Philip Meier, Marius Buibas, Dimitry Fisher, and Botond Szatmary

14:40 – 15:00 O7 ***How efficient coding of binocular disparity statistics in the primary visual cortex influences eye rotation strategy***
Sarah Marzen*, Joel Zylberberg, and Michael Deweese

15:00 – 15:20 O8 ***Propagating waves structure spatiotemporal activity in visual cortex of the awake monkey***
Lyle Muller*, Alexandre Reynaud, Frederic Chavane, and Alain Destexhe

15:20 – 15:50 **Break**

Oral session III: Correlations

15:50 – 16:10 O9 ***Non-renewal spiking and neural dynamics – A simple theory of inter-spike interval correlations in adapting neurons***
Tilo Schwalger*, Benjamin Lindner

16:10 – 16:50 F1 **Featured oral 1:**
Consistency requirements determine optimal noise correlations in neural populations
Joel Zylberberg*, Maxwell Turner, Yu Hu, Jon Cafaro, Greg Schwartz, Fred Rieke, and Eric Shea-Brown

16:50 – 17:10 O10 ***The role of neural correlations in a decision-making task***
Federico Carnevale, Victor De Lafuente, Ranulfo Romo, and Néstor Parga*

17:10 – 20:10 **Poster session I: Posters P1 – P145**

Monday July 15

9:00 – 9:10 **Announcements**

9:10 – 10:10 K3 **Keynote 3:**
Rescuing the spike
Sophie Deneve

10:10 – 10:40 **Break**

Oral session IV: Sensory mechanisms

10:40 – 11:00 O11 ***Cellular temperature compensation of sensory receptor neuron responses***
Frederic Roemschied*, Monika Eberhard, Jan-Hendrik Schleimer, Bernhard Ronacher, and Susanne Schreiber

11:00 – 11:20 O12 ***Self-organized lateral inhibition improves odor classification in an olfac-tion-inspired network***
Bahadir Kasap, Michael Schmuker*

11:40 – 12:00 F2 **Featured oral 2:**
Sensory dynamics transformation into effective motor behavior
Roberto Latorre, Rafael Levi, and Pablo Varona*

12:00 – 14:00 **Break for lunch**

12:30 – 14:00 ***Informal information session: Funding and other opportunities related to computational neuroscience***
Jeannette Kotaleski, Mathieu Girerd, and Kenneth Whang

Oral session V: Hippocampus

14:00 – 14:20 O13 ***Mechanisms of sharp wave-ripple generation and autonomous replay in a hippocampal network model***
Szabolcs Káli*, Eszter Vértes, Dávid G Nagy, Tamás F. Freund, and Attila Gulyás

14:20 – 14:40 O14 ***Extracellular field signatures of CA1 spiking cell assemblies during sharp wave-ripple complexes***
Jiannis Taxidis*, Kamran Diba, Costas Anastassiou, György Buzsáki, and Christof Koch

14:40 – 15:00 O15 ***Cross-talk and transitions between multiple environments in an attractor neural network model of the hippocampus***
Sophie Rosay*, Remi Monasson

15:00 – 18:00 **Poster session II: Posters 146 – P290, coffee will be served**

18:00 – 19:00 **Travel to gala dinner (Metro is recommended)**

19:00 **Gala dinner**

Tuesday July 16

9:00 – 9:10 **Announcements**

Oral session VI: Motor control

9:10 – 9:50 F3 **Featured oral 3:**
Nonlinear dynamics of mechanosensory flight control in flies
Martin Zapotocky*, Jan Bartussek, and Steven N Fry

9:50 – 10:10 O16 ***Model-based prediction of fusimotor activity during active wrist movements***
Bernard Grandjean, Marc Maier*

10:10 – 10:40 **Break**

10:40 – 11:00 O17 ***Operant conditioning of single units in rat motor cortex allows graded control of a prosthetic device***
Valerie Ego-Stengel*, Pierre-Jean Arduin, Yves Fregnac, and Daniel Shulz

11:00 – 12:00 **OCNS Member Meeting**

12:00 – 14:00 **Break for lunch**

Oral session VII: Network structure and dynamics

14:00 – 14:20 O18 ***Structural Features Beneath Neuronal Avalanches***
Leonardo Maia*, Thiago Mosqueiro

14:20 – 14:40 O19 ***The Inhibitory Network of the Striatum at the Edge of Chaos***
Adam Ponzi*, Jeffery Wickens

14:40 – 15:00 O20 ***Inferred network from prefrontal cortex activity of rats unveils cell assemblies***
Gaia Tavoni*, Ulisse Ferrari, Francesco P Battaglia, Simona Cocco, and Remi Monasson

15:00 – 15:20 O21 ***Multiscale modeling of cortical information flow in Parkinson's disease***
Cliff Kerr*, Sacha J van Albada, Samuel Neymotin, George Chadderdon, Peter Robinson, and William Lytton

15:20 – 15:50 **Break**

15:50 – 16:10 O22 ***HCN1-mediated interactions of ketamine and propofol in a mean field model of the EEG***
Ingo Bojak*, Harry Day, and David Liley

16:10 – 16:15 **Brain Corporation $10k Prize in Computational Neuroscience - Announcement of Winners**

16:15 – 17:15 K4 **Keynote 4:**
The Brain Activity Map: Imaging the Activity of Entire Neural Circuits
Rafael Yuste

17:15 – 20:15 **Poster session III: Posters P291 – P435**

Wednesday July 17

9:00 – 19:00 **Workshops**

Thursday July 18

9:00 – 19:00 **Workshops**

Workshops

Wednesday, July 17		
Space	**Morning**	**Afternoon**
Grignard	**W1 -** Advances in Activity-Dependent Synaptic Plasticity	
Salle du Conseil*	**W2 -** Network neurosciences: structure and dynamics	
Nicolas	**W3 -** Computations in the cerebellar circuit: advances on the modeling front	
Curie	**W4 -** Methods of Information Theory in Computational Neuroscience	
Grignard	**W5 -** Neural mechanisms of working memory limits	
Curie	**W6 -** Methods of Systems Identification for Studying Information Processing in Sensory Systems	
Curie	**W7 -** Early touch: from neural coding to haptic space geometry	
Grignard	**W8 -** Dendrite function and wiring: experiments and theory	
Curie	**W9 -** New developments in decoding the encoding of chemical senses	
Nicolas	**W10 -** New approaches to spike train analysis and neuronal coding	
Grignard	**W11 -** Metastable Dynamics of Neural Ensembles	
Curie	**W12 -** Advances in neural mass modeling	

Thursday, July 18		
Space	**Morning**	**Afternoon**
Grignard	**W1 -** Advances in Activity-Dependent Synaptic Plasticity	
Salle du Conseil*	**W2 -** Network neurosciences: structure and dynamics	
Nicolas	**W3 -** Computations in the cerebellar circuit: advances on the modeling front	
Curie	**W4 -** Methods of Information Theory in Computational Neuroscience	
Grignard	**W5 -** Neural mechanisms of working memory limits	
Nicolas	**W13 -** Spike-based computation	
Curie	**W14 -** Modeling general anesthesia: from theory to experiments	
Grignard	**W15 -** Recent advances in experimental and computational characterization of neural assemblies	
Curie	**W16 -** Computational properties of inhibitory synapses	
Curie	**W17 -** Functional role of correlations: theory and experiment	
Grignard	**W18 -** Relevance of Synaptic Plasticity for Multistable Behaviour in Neural Systems	
Grignard	**W19 -** Full Brain Network Dynamics - modeling, analysis, experiments	
Curie	**W20 -** Validating Neuro-computational Models of Neurological and Psychiatric Disorders	

Note: *W21 will be held Thursday evening from 18:00 to 20:00 in the Space Curie*

W1 Advances in activity-dependent synaptic plasticity

Space Grignard, Wed & Thu, 9:00-17:30

Paul Munro, University of Pittsburgh, Pittsburgh, PA, USA
Claudia Clopath, Columbia University, New York, NY, USA

W2 Network neuroscience: structure and dynamics

Salle du Conseil, Wed & Thu, 9:00-17:30

Michele Giugliano, University of Antwerp, Antwerp, Belgium
Daniele Marinazzo, University of Ghent, Ghent, Belgium

W3 Computations in the cerebellar circuit: advances on the modeling front

Space Nicolas, Wed & Thu, 9:00-17:30

Egidio d'Angelo, University of Pavia, Pavia, Italy
John Porrill, University of Sheffield, Sheffield, UK
Paul Dean, University of Sheffield, Sheffield, UK
Sergio Solinas, University of Pavia, Pavia, Italy

W4 **Methods of information theory in computational neuroscience**

Space Curie, Wed & Thu, 9:00-17:30

Alexander G. Dimitrov, Washington State University, Vancouver, WA, USA
Michael Gastpar, EPFL, Lausanne, Switzerland
Conor Houghton, University of Bristol, Bristol, UK
Aurel A. Lazar, Columbia University, New York, NY, USA
Tatyana Sharpee, The Salk Institute, La Jolla, CA, USA
Simon R Schultz, Imperial College, London, UK

W5 **Neural mechanisms of working memory limits**

Space Grignard, Wed & Thu, 9:00-17:30

Albert Compte, IDIBAPS, Barcelona, Spain
Zachary Kilpatrick, University of Houston, Houston, TX, USA

W6 **Methods of systems identification for studying information processing in sensory systems**

Space Curie, Wed, 9:00-17:30

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

W7 **Early touch: from neural coding to haptic space geometry**

Space Curie, Wed, 9:00-17:30

Jonathan Platkiewicz, Université Pierre et Marie Curie, Paris, France
Vincent Hayward, Université Pierre et Marie Curie, Paris, France

W8 **Dendrite function and wiring: experiments and theory**

Space Grignard, Wed, 9:00-17:30

Hermann Cuntz, Goethe University, Frankfurt, Germany
Michiel Remme, Humboldt University, Berlin, Germany
Ben Torben-Nielsen, EPFL, Lausanne, Switzerland

W9 **New developments in decoding the encoding of chemical senses**

Space Curie, Wed, 9:00-17:30

Maxim Bazhenov, University of California, Riverside, CA, USA
Mark Stopfer, NIH, Bethesda, MD, USA

W10 New approaches to spike train analysis and neuronal coding
Space Nicolas, Wed, 9:00-17:30

Conor Houghton, University of Bristol, Bristol, UK
Thomas Kreuz, ISC-CNR, Florence, Italy

W11 Metastable dynamics of neural ensembles
Space Grignard, Wed, 9:00-17:30

Gustavo Deco, University Pompeu Fabra, Barcelona, Spain
Emili Balaguer Ballester, Bournemouth University, Bournemouth, UK

W12 Advances in neural mass modeling
Space Curie, Wed, 9:00-17:30

Ingo Bojak, University of Birmingham, Birmingham, UK
Stephan van Gils, University of Twente, Enschede, The Netherlands
Sid Visser, University of Twente, Enschede, The Netherlands

W13 Spike-based computation
Space Nicolas, Thu, 9:00-17:30

Romain Brette, Ecole Normale Supérieure, Paris, France
Sophie Denève, Ecole Normale Supérieure, Paris, France

W14 Modeling general anesthesia: from theory to experiments
Space Curie, Thu, 9:00-17:30

Axel Hutt, INRIA, Nancy, France

W15 Recent advances in experimental and computational characterization of neural assemblies
Space Grignard, Thu, 9:00-17:30

Adrien Peyrache, New York University Medical Center, New York, NY, USA
Sami El Boustani, MIT, Cambridge, MA, USA

W16 Computational properties of inhibitory synapses
Space Curie, Thu, 9:00-17:30

Fidel Santamaria, University of Texas San Antonio, San Antonio, TX, USA

W17 Functional role of correlations: theory and experiments
Space Curie, Thu, 9:00-17:30

Tatjana Tchumatchenko, Columbia University, New York, NY, USA
Srdan Ostojic, Ecole Normale Supérieure, Paris, France

W18 Relevance of synaptic plasticity for multistable behaviour in neural systems
Space Grignard, Thu, 9:00-17:30

Alessandro Torcini, Institute of Complex Systems, Florence, Italy
Christian Hauptmann, Research Center Juelich, Juelich, Germany

W19 Full brain network dynamics - modeling, analyses, experiments
Space Grignard, Thu, 9:00-17:30

Victor Jirsa, Institut de Neurosciences des Systèmes INSERM, Marseilles, France
Gustavo Deco, University Pompeu Fabra, Barcelona, Spain

W20 Validating neuro-computational models of neurological and psychiatric disorders
Space Curie, Thu, 9:00-17:30

Basabdatta Sen Bhattacharya, University of Lincoln, Lincoln, UK
Fahmida Chowdhury, NSF, Arlington, VA, USA

W21 Postdoc and student career strategy workshop
Space Curie, Thu, 18:00-20:00

Jorge Mejias, University of Ottawa, Ottawa, Ontario, Canada

Abstracts

Tutorials

T1 Neural-mass and neural-field models

Space Curie, (9.00-12.20; 13.30-16.50)

Axel Hutt, INRIA Nancy, France
Viktor Jirsa, Institut de Neuroscience de Systemes, France
John Terry, University Exeter, UK
Wolfram Erlhagen, University Minho, Portugal

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

Fundamentals of neural-mass and neural-field models: Physiological motivation and mathematical descriptions (Axel Hutt)

The talk will motivate physiologically the mathematical description of neural populations based on microscopic neural properties. Several different mathematical models will be discussed, explained and compared. The major aim of this presentation is to explain the standard models found in the literature in terms of mathematical elements and physiological assumptions.

Pattern formation in neural network systems and applications to movement dynamics (Viktor Jirsa)

We will introduce and systematically evaluate the mechanisms underlying pattern formation in neuronal networks. A particular focus will be given to the contribution of network connectivity. The conditions will be derived aiding in the emergence of low-dimensional sub-spaces, in which nonlinear dynamic flows are constrained to manifolds. Applications of these concepts and experimental examples will be provided from the field of human movement sciences.

Brain networks in epilepsy: Fusing models and clinical data (John Terry)

We will explore how both physiological and phenomenological mathematical models can be developed to understand the mechanisms that underpin transitions in clinically recorded EEG between activity corresponding to normal function and the pathological activity associated with epilepsy. We present evidence that the bifurcation sequences of physiologically inspired models give rise to dynamical sequences that precisely map those observed in both focal and generalised seizures. We further demonstrate that seizure frequency can be understood through the relationship between the dynamics of brain regions and the network structures that connect them. Our mathematical models help to explain previously counterintuitive experimental studies demonstrating loss of connectivity paradoxically

makes generalised seizures more likely.

The dynamic neural-field approach to cognitive robotics (Wolfram Erlhagen)

In recent years, there has been an increased interest by part of the robotics community in using the theoretical framework of dynamic neural fields to develop neuro-inspired control architectures for autonomous agents. The formation of self-sustained activity patterns in neural populations explained by the theory offers a systematic way to endow robots with cognitive functions such as working memory, decision making, prediction and anticipation. In the tutorial, I will present a Dynamic Neural-Field architecture for natural human-robot collaboration that is heavily inspired by neuro-cognitive mechanisms supporting joint action in humans and other primates. The model is formalized as a large-scale network of reciprocally connected neural populations, each governed by a classical field dynamics of Amari type.

T2 Theory of correlation transfer and correlation structure in recurrent networks

Space Curie, (9.00-12.20; 13.30-16.50)

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

In the first part, we will study correlations arising from pairs of neurons sharing common fluctuations and/or inputs. Using integrate-and-fire neurons, we will show how to compute the firing rate, auto-correlation and cross-correlation functions of the output spike trains. The transfer function of the output correlations given the inputs correlations will be discussed. We will show that the output correlations are generally weaker than the input correlations [Moreno-Bote and Parga, 2006], that the shape of the cross-correlation functions depends on the working regime of the neuron [Ostojic et al., 2009; Helias et al., 2013], and that the output correlations strongly depend on the output firing rate of the neurons [de la Rocha et al, 2007]. We will study generalizations of these results when the pair of neurons is reciprocally connected.
In the second part, we will consider correlations in recurrent random networks. Using a binary neuron model [Ginzburg & Sompolinsky, 1994], we explain how mean-field theory determines the stationary state and how network-generated noise linearizes the single-neuron response. The resulting linear equation for the fluctuations in recurrent networks is then solved to obtain the correlation structure in balanced random networks. We discuss two different points of view of the recently reported active suppression of correlations in balanced networks by fast tracking [Renart et al., 2010] and by negative feedback [Tetzlaff et al., 2012]. Finally, we consider extensions of the theory of correlations of linear Poisson spiking models [Hawkes, 1971] to the leaky integrate-and-fire model and present a unifying view of linearized theories of correlations [Helias et al, 2011].
At last, we will revisit the important question of how correlations affect information and vice-versa [Zohary et al, 1994] in neuronal circuits, showing novel results about information content in recurrent networks of integrate-and-fire neurons [Moreno-Bote and Pouget, Cosyne abstracts, 2011].

References:

- de la Rocha et al. (2007), Correlation between neural spike trains increases with firing rate, Nature 448:802–6
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50:3171–3190

- Hawkes (1971), Point Spectra of Some Mutually Exciting Point Processes, Journal of the Royal Statistical Society Series B 33(3):438–443
- Helias et al. (2011), Towards a unified theory of correlations in recurrent neural networks, BMC Neuroscience 12(Suppl 1):P73
- Helias et al. (2013), Echoes in correlated neural systems, New Journal of Physics 15(2):023002
- Moreno-Bote & Parga (2006), Auto- and crosscorrelograms for the spike response of leaky integrate-and-fire neurons with slow synapses, PRL 96:028101
- Ostojic et al. (2009), How Connectivity, Background Activity, and Synaptic Properties Shape the Cross-Correlation between Spike Trains, J Neurosci 29(33):10234–10253
- Renart et al. (2010), The Asynchronous State in Cortical Circuits, Science 327(5965):587–590
- Shadlen & Newsome (1998), The variable discharge of cortical neurons: implications for connectivity, computation, and information coding, J Neurosci 18:3870–96
- Tetzlaff et al. (2012), Decorrelation of neural-network activity by inhibitory feedback, PLoS Comp Biol 8(8):e1002596, doi:10.1371/journal.pcbi.1002596
- Zohary et al. (1994), Correlated Neuronal Discharge Rate and Its Implications for Psychophysical Performance, Nature 370:140–143

T3 Modeling and interpretation of extracellular potentials

Space Grignard, (9.00-12.20; 13.30-16.50)

Gaute T Einevoll, Norwegian University of Life Sciences, Ås, Norway
Szymon Leski, Nencki Institute of Experimental Biology, Warsaw, Poland
Espen Hagen, Norwegian University of Life Sciences, Ås, Norway

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

In the first part of this tutorial, we will go through

- the biophysics of extracellular recordings in the brain,

- a scheme for biophysically detailed modeling of extracellular potentials and the application to modeling single spikes [1-3], MUA [4] and LFP, both from single neurons [5] and populations of neurons [4,6], and
- methods for
  - estimation of current-source density [7] from LFP data, such as the iCSD [8-10] and kCSD methods [11], and
  - decomposition of recorded signals in cortex into contributions from various laminar populations, i.e., (i) laminar population analysis (LPA) [12] based on joint modeling of LFP and MUA, and (ii) a novel scheme using LFP and known constraints on the synaptic connections [13]

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

- LFPy (compneuro.umb.no/LFPy), a versatile tool based on Python and the simulation program NEURON [14] (www.neuron.yale.edu) for calculation of extracellular potentials around neurons, and
- tools for iCSD analysis, in particular,
  - CSDplotter (for linear multielectrodes [8]) (software.incf.org/software/csdplotter)
  - iCSD 2D (for 2D multishank electrodes [10]) (software.incf.org/software/icsd-2d)

References:

[1] Holt & Koch (1999), J Comp Neurosci 6:169

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

[3] Pettersen & Einevoll (2008), Biophys J 94:784

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

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

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

[7] Nicholson & Freeman (1975), J Neurophsyiol 38:356

[8] Pettersen et al. (2006), J Neurosci Meth 154:116

[9] Łęski et al. (2007), Neuroinform 5:207

[10] Łęski et al. (2011), Neuroinform 9:401

[11] Potworowski et al. (2012), Neural Comp 24:541

[12] Einevoll et al. (2007), J Neurophysiol 97:2174

[13] Gratiy et al. (2011), Front Neuroinf 5:32

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

T4 Probabilistic inference as a neural-computing paradigm

Space Grignard, (13.30-16.50)

Dejan Pecevski, Graz University of Technology, Graz, Austria

Probabilistic inference has been proven to be a very suitable framework for explaining many of the computations that the brain performs in face of great amount of uncertainty present in the sensory inputs and its internal representations of the world [Rao et al., 2002; Fiser et al., 2010; Tenenbaum et al., 2011; Kording et al., 2004]. However, it still remains an open question how these probabilistic inference computations are implemented in the neural circuits of the brain. In this tutorial we will present recent results that give new perspectives on how probabilistic inference and learning could be carried out by networks of spiking neurons.

The tutorial is organized in two parts. In the first part we will briefly overview several basic topics from probabilistic inference, including graphical models, belief propagation, Markov chain Monte Carlo methods and Gibbs sampling. In the second part we will start by describing a recently developed framework for probabilistic inference with stochastic networks of spiking neurons that performs Markov chain Monte Carlo sampling, called neural sampling [Buesing et al., 2011]. We will further show that by introducing specific network motifs or dendritic computation in the spiking neural networks, they can be made to perform neural sampling in general graphical models that exhibit also higher-order relations between the random variables [Pecevski et al., 2011]. We will then continue discussing results about learning probabilistic models, in particular a study where it was shown that STDP in a stochastic winner-take-all network structure implements the expectation-maximization algorithm, a powerful machine learning algorithm for unsupervised learning [Nessler et al., 2009]. In [Habenschuss et al., 2012], the model from [Nessler et al., 2009] was extended with homeostatic plasticity of the neuronal excitabilities, which improved the performance and robustness of learning. It was also demonstrated theoretically that this extended model can be understood as performing expectation-maximization under posterior constraints. Finally, we will show how many winner-take-all network motifs as in [Habenschuss et al., 2012] can be combined together in a larger recurrent spiking neural network which is capable of solving a generic learning task: to learn a probabilistic model from input data streams, where the dependencies in the probabilistic model can be a priori based on any arbitrary graphical model structure.

References:

- Rao et al. (2002), Probabilistic models of the brain: Perception and neural function, Mit Press
- Fiser et al. (2010), Statistically optimal perception and learning: from behavior to neural representations, Trends Cogn Sci 14:119–130
- Tenenbaum et al. (2011), How to Grow a Mind: Statistics, Structure, and Abstraction, Science 331:1279–1285
- Kording & Wolpert (2004), Bayesian integration in sensorimotor learning, Nature 427:244–247
- Buesing et al. (2011), Neural Dynamics as Sampling: A Model for Stochastic Computation in Recurrent Networks of Spiking Neurons, PLoS Comput Biol 7:e1002211

- Pecevski et al. (2011), Probabilistic Inference in General Graphical Models through Sampling in Stochastic Networks of Spiking Neurons, PLoS Comput Biol 7:e1002294
- Nessler et al. (2009), STDP enables spiking neurons to detect hidden causes of their inputs, Advances in Neural Information Processing Systems 22:1357–1365
- Habenschuss et al. (2012), Homeostatic plasticity in Bayesian spiking networks as Expectation Maximization with posterior constraints, Advances in Neural Information Processing Systems 25:782–790

T5 Brain activity at rest: Dynamics and structure of the brain in health and disease

Space Grignard, (9.00-12.20)

Gustavo Deco, Universitat Pompeu Fabra, Spain

Perceptions, memories, emotions, and everything that makes us human, demand the flexible integration of information represented and computed in a distributed manner. The human brain is structured into a large number of areas in which information and computation is highly segregated. Normal brain function requires the integration of functionally specialized but widely distributed brain areas. We contend that the functional and encoding roles of diverse neuronal populations across areas are subject to the intra- and inter-cortical dynamics. In this tutorial, we try to elucidate precisely the interplay and mutual entrainment between local brain area dynamics and global network dynamics, in order to understand how segregated distributed information and processing is integrated. We can deepen our understanding of the mechanisms underlying brain functions by complementing structural and activation based analysis with dynamics. In particular, a large body of fMRI, MEG, EEG, and optical imaging experiments reveal that the ongoing brain activity of the brain at rest is not trivial but highly structured in very specific spatio-temporal patterns known as Resting State Networks (RSN). Indeed, the Functional Connectivity (FC) at rest, i.e. the spatial correlation matrix between the temporal signals reflecting spontaneous brain activity at different positions, is topologically very well structured according to the underlying RSNs. A profound understanding of these operations will help to elucidate the computational principles underlying higher brain functions and their breakdown in brain diseases. Thus, we will discuss the effects on the resting state of lesions and different type of damage in neuropsychiatric disorder.

T6 Developing neuron and synapse models for NEST

Space Grignard, (9.00-12.20; 13.30-16.50)

Abigail Morrison, Research Center Jülich, Germany
Jochen M Eppler, Research Center Jülich, Germany

The neural simulation tool NEST [1] is a simulator for heterogeneous networks of point neurons or neurons with a small number of electrical compartments aiming at simulations of large neural systems. It is implemented in C++ and runs on a large range of architectures from single-processor desktop computers to large clusters with thousands of processor cores. This tutorial is an extension course for anybody who is already working with either the neural simulation tool NEST, or has other experience

with the simulation of networks of point neuron models. Some programming background in C++ is helpful but not required. We will start with a refresher on setting up networks of neurons focussing on customising the neuronal and synaptic parameters before, during and after creation of the network elements. In the second part we will provide a hands-on demonstration of how to develop a new neuron or synapse model for NEST. We will start from a skeleton and follow the process through to using the new model in a network simulation.

References:

[1] Gewaltig & Diesmann (2007), NEST (Neural Simulation Tool), Scholarpedia 2(4):1430

T7 Advanced modelling of spiking neural networks with BRIAN

Space Curie, (9.00-12.20; 13.30-16.50)

Romain Brette, École Normale Supérieure, Paris, France
Marcel Stimberg, École Normale Supérieure, Paris, France
Victor Benichoux, École Normale Supérieure, Paris, France
Cyrille Rossant, University College London, UK
Nelson Cortés Hernández, École Normale Supérieure, Paris, France
Dan Goodman, Massachusetts Eye and Ear Infirmary, Boston, USA
Bertrand Fontaine, Albert Einstein College of Medicine, New York, USA

BRIAN [1,2] is a simulator for spiking neural networks, written in the Python programming language. It focuses on making the writing of simulation code as quick as possible and on flexibility: new and non-standard models can be readily defined using mathematical notation. This tutorial will present the current state of development of BRIAN and will enable participants to adapt and extend Brian to their needs. It will also cover existing Brian extensions (brian hears [3], model fitting toolbox [4], compartmental modelling) and introduce "best practices" for complex simulations. Furthermore, it will present strategies for improving the speed of simulations by using Brian's C code generation mechanism [5].
We strongly encourage interested participants to mail us further suggestions or comments beforehand: marcel.stimberg{at}ens.fr. Note that this tutorial is not meant as a beginner's introduction to BRIAN, participants should either already use BRIAN in their research or be at least familiar with it (e.g. by working through the tutorials available at [1]) before the tutorial starts.

References:

[1] http://briansimulator.org

[2] Goodman & Brette (2009), The Brian simulator, Front Neurosci, doi:10.3389/neuro.01.026.2009.

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T8 Managing complex workflows in neural simulation and data analysis

Space Curie, (9.00-12.20; 13.30-16.50)

Andrew P Davison, UNIC, CNRS, Gif sur Yvette
Sonja Grün, Research Center Jülich, Germany
Michael Denker, Research Center Jülich, Germany

In our attempts to uncover the mechanisms that govern brain processing on the level of interacting neurons, neuroscientists have taken on the challenge of tackling the sheer complexity exhibited by neuronal networks. Neuronal simulations are nowadays performed with a high degree of detail, covering large, heterogeneous networks. Experimentally, electrophysiologists can simultaneously record from hundreds of neurons in complicated behavioral paradigms. The data streams of simulation and experiment are thus highly complex; moreover, their analysis becomes most interesting when considering their intricate correlative structure.

The increases in data volume, parameter complexity, and analysis difficulty represent a large burden for researchers in several respects. Experimenters, who traditionally need to cope with various sources of variability, require efficient ways to record the wealth of details of their experiment ("meta data") in a concise and machine-readable way. Moreover, to facilitate collaborations between simulation, experiment and analysis there is a need for common interfaces for data and software tool chains, and clearly defined terminologies. Most importantly, however, neuroscientists have increasing difficulties in reliably repeating previous work, one of the cornerstones of the scientific method. At first sight this ought to be an easy task in simulation or data analysis, given that computers are deterministic and do not suffer from the problems of biological variability. In practice, however, the complexity of the subject matter and the long time scales of typical projects require a level of disciplined book-keeping and detailed organization that is difficult to keep up.

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

In this tutorial we will identify the reasons for the difficulties often encountered in organizing and handling data, sharing work in a collaboration, and performing manageable, reproducible yet complex computational experiments and data analyses. We will also discuss best practices for making our work more reliable and more easily reproducible by ourselves and others – without adding a huge burden to either our day-to-day research or the publication process.

We will cover a number of tools that can facilitate a reproducible workflow and allow tracking the provenance of results from a published article back through intermediate analysis stages to the original data, models, and/or simulations. The tools that will be covered include Git [2], Mercurial [3], Sumatra [4], VisTrails [5], odML [6], Neo [7]. Furthermore, we will highlight strategies to validate the correctness, reliability and limits of novel concepts and codes when designing computational analysis approaches (e.g., [8,9,10]).

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T9 Massively parallel time encoding and channel identification machines

Space Grignard, (9.00-12.20; 13.30-16.50)

Aurel A. Lazar, Columbia University, New York, US

This two part tutorial focusses on Time Encoding Machines (part I) and Channel Identification Machines (part II). The tutorial will give an overview of (i) nonlinear decoding of stimuli encoded with neural circuits with biophysical neuron models, (ii) functional identification of biophysical neural circuits, and (iii) the duality between the two. Scaling to massively parallel neural circuits for both encoding and functional identification will be discussed throughout. The tutorial will provide numerous examples of neural decoding and functional identification using the Time Encoding Machines Toolbox and the Channel Identification Machines Toolbox. Tutorial material, programming code and demonstrations will be provided.

Part I: Time Encoding Machines

The nature of the neural code is fundamental to theoretical and systems neuroscience [1]. Can information about the sensory world be faithfully represented by a population of sensory neurons? What features of the stimulus are encoded by a multidimensional spike train? How can these features be decoded? Why does the cochlear nerve carry some 30,000 fibers and the optic nerve some 1,000,000? We will discuss these questions using a class of neural encoding circuits called Time Encoding Machines (TEMs) [2]. TEMs model the encoding of stimuli in early sensory systems with neural circuits with arbitrary connectivity and feedback. These circuits are realized with temporal, spectro-temporal and/or spatio-temporal receptive fields, and biophysical neuron models with stochastic conductances (Hodgkin-Huxley, Morris-Lecar, etc.) [3,4,5,6]. The tutorial will

review key theoretical results and provide numerous examples of massively parallel neural encoding circuits and stimulus decoding algorithms with the Time Encoding Machines Toolbox (http://www.bionet.ee.columbia.edu/code/ted.html).

Part II: Channel Identification Machines

Parameter estimation is at the core of functional identification of neural circuits. How are estimates of model parameters affected by the stimuli employed in neurophysiology? What is a suitable metric to assess the faithfulness of identified parameters and the goodness of model performance? These are key open questions that are of relevance to both theoretical and experimental neuroscientists. We will discuss these questions using a class of algorithms called Channel Identification Machines (CIMs) [7,8,9] and give an overview of the functional identification of massively parallel neural circuit models of sensory systems arising in olfaction, audition and vision. These circuits are built with temporal, spectro-temporal and non-separable spatio-temporal receptive fields, and biophysical spiking neuron models. The tutorial will demonstrate how CIMs achieve the efficient identification of neural circuit models and will provide numerous examples of functional identification using the Channel Identification Machines Toolbox (http://www.bionet.ee.columbia.edu/code/cim.html).

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Invited Presentations

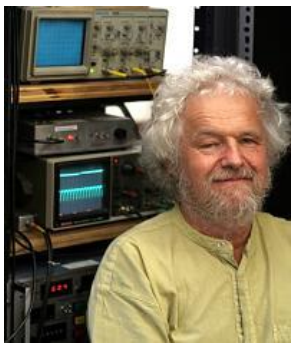
Nikos K Logothetis
Max Planck Institute
for Biological Cybernetics,
Tübingen, Germany.

K1 – Studying Large-Scale Brain Networks: Electrical Stimulation & Neural-Event-Triggered fMRI

The brain is 'the' example of an adaptive, complex system. It is characterized by ultra-high structural complexity and massive connectivity, both of which change and evolve in response to experience. Information related to sensors and effectors is processed in both a parallel and a hierarchical fashion. The connectivity between different hierarchical levels is bidirectional, and its effectiveness is continuously controlled by specific associational and neuromodulatory centers. In the study of such systems one major problem is the adequate definition for an elementary operational unit (often called an 'agent'), because any such module can be a complex system in its own right and may be recursively decomposed into other sets of units. A second difficulty arises from the synergistic organization of complex systems and of the brain in particular. Synergy here refers to the fact that the behavior of an integral, aggregate, whole system cannot be trivially reduced to, or predicted from, the components themselves. Localizing and comprehending the neural mechanisms underlying our cognitive capacities demands the combination of multimodal methodologies, i.e. it demands concurrent study of components and networks; one way of doing this, is to combine invasive methods which afford us direct access to the brain's electrical activity at the microcircuit level with global imaging technologies such as magnetic resonance imaging (MRI). In my talk, I'll discuss two such methodologies: Direct Electrical Stimulation and fMRI (DES-fMRI) and Neural-Event-Triggered fMRI (NET-fMRI).

DES-fMRI can be used in hopes of gaining insight into the functional or effective connectivity underlying DES-induced behaviors. Yet, our first findings suggest that DES has an important limitation: It clearly demarcates all monosynaptic targets of a stimulated site, but it largely fails to reveal polysynaptic cortico-cortical connectivity.

NET-fMRI, on the other hand, appears to offer great potential for mapping whole-brain activity that is associated with individual local events. In the second part of my talk, I'll describe the characteristic states of widespread cortical and subcortical networks that are associated with the occurrence of hippocampal sharp waves and ripples; the brief aperiodic episodes associated with memory consolidation.



Simon Laughlin
Department of Zoology,
University of Cambridge,
Cambridge, United Kingdom.

K2 – The influence of metabolic energy on neural computation

Computational Neuroscience is a vital part of the brave effort to reverse engineer brains, ultimately our own. Our efforts are confounded by an embarrassment of riches. Brains' winning technology, cell and molecular biology, enables neurons to connect and perform a huge variety of operations and adapt them with unparalleled ingenuity and subtly. Faced with so much that can be done, how do we discover what is done? Three constraints can guide us. One is what has to be done, the nature of the task and the operations that must be performed to generate the behaviour that is observed. Another is data (usually incomplete) about what is being done. I will talk about the third constraint, physical, chemical and biological limits to what can be done and, in particular, energy consumption.
Beyond the realms of quantum computers, computation dissipates energy. Consequently energy supply and heat loss ultimately limit processing power. Here the brain is severely limited by its winning technology; neurons are low energy density devices and this restricts bandwidth and noise. I will discuss how brains attempt to operate effectively with feeble neurons influences its unique style of computation, by considering chemical and electrical protein circuits, matching and adapting components, hybrid processing, redundancy reduction and its opposite, sparsification. I will propose that the efficient brain behaves like the Physics PhD Student from Hell, who does everything as slowly as possible, as inaccurately as possible and, wherever possible, uses chemistry. But, like many clever students, the brain is charmingly adaptable.

Sophie Deneve
Group for Neural Theory,
LNC, DEC, ENS,
Paris, France.

K3 – Rescuing the spike

Sensory and motor variables are represented by large populations of neurons. We hypothesized that these representations are constrained such that they can be read-out linearly (synaptic integration)

while limiting the metabolic cost. Such framework can predict many aspects of neural tuning, robustness and adaptation. Moreover, spiking networks with balanced excitation and inhibition naturally produce such efficient codes. In such balanced networks, each membrane potential monitors the coding error, e.g. the mismatch between the feed-forward inputs and the prediction of these inputs by lateral connections. Each spike implements a greedy minimization of this error. Most importantly, any network of integrate and fire neurons will self-organize into this optimal regime with a simple Hebbian plasticity rule enforcing the balance between excitation and inhibition. Through learning, initially regular, highly correlated spike trains evolve towards Poisson-distributed, asynchronous spike trains with much lower firing rates. Importantly, single unit variability is a consequence of degeneracy in the code, not noise: the population as a whole tracks the signal perfectly. This suggests that balanced spiking networks are not equivalent to rate models, but in fact orders of magnitude more reliable.

Rafael Yuste
Howard Hughes Medical Institute,
Columbia University,
New York, USA.

K4 – The Brain Activity Map: Imaging the Activity of Entire Neural Circuits

The function of neural circuits is an emergent property that arises from the coordinated activity of large numbers of neurons. To capture this, we propose launching a large-scale, international public effort, the Brain Activity Map Project, aimed at imaging the full record of neural activity across complete neural circuits. This technological challenge could prove to be an invaluable step toward understanding fundamental and pathological brain processes.

Contributed Talks

F1 Consistency requirements determine optimal noise correlations in neural populations

Joel Zylberberg[1*], Maxwell Turner[2], Yu Hu[1], Jon Cafaro[2], Greg Schwartz[2], Fred Rieke[2,3], and Eric Shea-Brown[1]

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A key challenge in population coding is to understand the role of correlations between the activities of different neurons. While the existence of correlations in primary visual cortex (for example), is somewhat controversial, retina presents a relatively clean story, with many studies observing that correlations exist and are important in shaping the population activity distribution. Given the retina's role in conveying visual information to the brain, and the relative clarity of the experimental data, retina offers a unique opportunity to study how correlations affect neural function. This question has received much attention, and previous work emphasizes that we must distinguish between two important types of correlations. First, there are the signal correlations, which describe how the mean (averaged over trials of the same stimulus) responses of two cells co-vary as the stimulus is changed. The noise correlations, on the other hand, describe how two neurons' responses co-vary over the repeat trials of the same stimulus. How do signal- and noise- correlations inter-relate with respect to population coding? Several theoretical principles have emerged. For example, Averbeck et al. [1] showed that, for optimal discriminability of two different stimuli, a pair of neurons should have opposite signs for their signal- and noise- correlations: positive signal correlations demand negative noise correlations, and vice versa. This 'opponent signal and noise correlations' situation yields better discriminability than occurs with uncorrelated noise, and these results do extend to larger populations. For heterogeneous populations, subsequent works indicates that the situation is more nuanced.

To experimentally test these theoretical ideas, we measured the noise correlations for a population of direction selective retinal ganglion cells with different signal correlations for different pairs, and observed that, regardless of the signal correlations (positive for some cell pairs, and negative for others), all neural pairs had small positive noise correlations. This is in contrast with the notion of opponent signal and noise correlations. To understand this discrepancy, we created a simple mathematical model of our experimental system, in which the overlap between two neurons' tuning curves dictates their signal correlations; the signal correlations differed between cell pairs, and belong to the set $\{0, 1, -1\}$. The noise correlations are the independent variable for our numerical experiments. Note that our model population has 8 (>2) neurons in it. In our model, optimal coding performance (measured, for example, using linear Fisher information) occurs when the noise covariance matrices lie on a boundary of the space of allowed covariances. Recall that only positive definite covariance (and correlation) matrices are possible, which means that correlations between pairs need to be consistent across the population; this requirement shapes the boundary. For example, if neurons A and B have a perfect noise correlation ($\rho_{AB} = 1$), and so do neurons B and C ($\rho_{BC} = 1$) then neurons A and C must also have a perfect noise correlation ($\rho_{AC} = 1$). One cannot choose ($\rho_{AB} = 1$, $\rho_{BC} = 1$, $\rho_{AC} = 1$) = (1,1,-1), for example, and pair-by-pair arguments about what the noise correlations should be will necessarily miss this restriction. We have further proven mathematically that, regardless of the encoder details, the optimal encoder, using either OLE performance, or linear Fisher information as a metric,

must lie on a boundary of the space of allowed noise covariance matrices. The small all-positive noise correlations we observed yielded near-optimal performance in our model population. Considering one pair at a time, it might be better to choose negative noise correlations for cell pairs with positive signal correlations and vice versa, but such choices may be impossible, due to the requirement that the correlations be consistent across the population. Since the optimal solutions must lie on the boundary of the allowed space of correlation matrices, the consistency requirement is a critical factor in determining the noise correlation structure that optimizes population coding.

Acknowledgements
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F2 Sensory dynamics transformation into effective motor behavior

Roberto Latorre[1], Rafael Levi[1,2], and Pablo Varona[1⋆]

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How sensory information is transformed into effective motor action is one of the most fundamental questions in neuroscience. The intrinsic dynamics of sensory networks can play an important role in the sensory-motor transformation. However, it is difficult to experimentally assess the study of all the stages present in the processing of a sensory-motor transformation. Biophysical models of sensory, central and motor systems can largely contribute to understand the information processing mechanisms involved in this transformation. Nevertheless, because of the lack of experimental results, there are very few models including all these stages to address the transformation of sensory dynamics into a motor program.

Complex intrinsic sensory dynamics can be related to multifunctionality in the sensory-motor transformation. Multifunctionality of neural systems has only been partially addressed in neuroscience research. One remarkable example of relationship between intrinsic sensory dynamics and multifunctionality is the gravimetric organ of the mollusk *Clione limacina* [1,2]. In this work we used conductance based models of sensory, central and motor circuits and electrophysiological recordings to address the study of the dual role of a sensory network to organize two different context-dependent motor programs. Our experimental and modeling results indicate that the sensory signals are modified to fit the changing behavioral context, and they are readily interpreted by the rest of the nervous system to produce the correct motor output. We show that a winner-take all dynamics in the gravimetric sensory network drives the repetitive rhythm of *Clione*'s wing CPG model during routine swimming [3]. On the other hand, a winnerless competition dynamics in the same sensory network organizes the irregular pattern observed in the wing CPG during hunting behavior [1]. These two dynamics are interpreted by the wing CPG to generate the characteristic rhythmic motion during routine swimming and the fast irregular motion that is observed during hunting behavior. Our modeling results also indicate that specific

activation phase locks in the sensory network dynamics are transformed into specific motor events in the wing CPG. The activation phase locks can play an important role in motor coordination.

These results support the view that the dual dynamics of the statocyst network by itself can explain the two motor programs observed during routine swimming and during hunting behavior in *Clione* [4]. In other words, the motor program could be generated right at the sensory network fitting the changing behavioral context in the sensory signals. In this way, the rest of the neurons in the sensory-motor transformation can just react normally to this signaling.

Acknowledgements
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F3 Nonlinear dynamics of mechanosensory flight control in flies

Martin Zapotocky[1⋆], Jan Bartussek[1,2], and Steven N Fry[3]

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In most animals, rhythmic motion is governed by central pattern generators (CPGs) – neural circuits that generate periodic patterned output. Sensory input to the CPG is not necessary to maintain the periodic neural activity, but is known to strongly influence it. For example, proprioceptive afferent input may entrain the neural CPG rhythm to the mechanical resonance frequency of a limb [1]. CPGs have been shown to govern the locomotor rhythms in vertebrates as well as in insects. A CPG governing the flight rhythm, however, has not been identified in flies, which are known for their remarkable flight maneuverability. In flies, the wingbeat rhythm is generated myogenically by stretch-activated power muscles and hence is not directly controlled by neural input. It is therefore unclear if the insights gained for proprioceptive feedback in CPG-based motor systems [1] likewise apply to flight control in flies.

In our study, we investigated if and how proprioceptive feedback influences the rhythm of the myogenic wing beat oscillator. We concentrated on mechanosensory input from the halteres – specialized 'gyroscopic' sensory organs of flies. The halteres are known to activate the motor neurons of miniscule steering muscles, which in turn modulate the motion of the wings. In our experiments [2], tethered flying fruit flies (*Drosophila melanogaster*) were vibrated by a piezoelectric actuator to stimulate the haltere mechanosensory pathways. We used a laser Doppler interferometer to measure the vibrations of the tether resulting from the superposition of the piezo-delivered stimulus and the fly's wing beat.

We determined the phase relationship between the wing motion and the mechanical stimulus in each wing stroke and applied an automated synchrogram analysis [3] to detect entrainment and higher-order synchronization. The flies synchronized with the stimulus for specific ranges of stimulus amplitude and frequency, revealing the characteristic Arnol'd tongues of a forced limit cycle oscillator. Our analysis shows that the steering muscles (activated by proprioceptive input) act as a *mechanical* forcing of the central power muscle oscillator. We propose that the mechanical forcing of a myogenic limit cycle oscillator permits flies to avoid the comparatively slow control based on a neural central pattern generator.

In the entrainment study described above, flies were attached to tethers with high resonance frequency and damping coefficient. While experimenting with tethers of varying mechanical properties, however, we observed that the fly's behavior was significantly influenced by the properties of the tether. To systematically explore this influence for a given fly, we altered the tether's resonance frequency by clamping the tether at a different point in each distinct flight test. In these experiments, no piezo stimulus was applied. Yet, when the tether resonance frequency was comparable to the wing beat frequency, the forces from the fly lead to large cumulative motion of the tether and activation of the haltere mechanosensors. The fly therefore received delayed feedback dependent on its previous activity. This lead to a variety of observed dynamical regimes, including the locking of the wing beat frequency to the tether resonance frequency when their initial difference was sufficiently small (similar to limb entrainment in [1]). We were able to reproduce most of the observed dynamical features in a simplified mathematical model of two mutually coupled oscillators: a phase-reduced nonlinear oscillator representing the wing beat and a linear oscillator representing the tether dynamics.

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O1 Endocannabinoids mediate spike-timing dependent potentiation and depression: a model-based experimental approach

Yihui Cui[1,2,3], Vincent Paille[1,2,3], Bruno Delord[3,4], Stéphane Genet[3,4], Elodie Fino[1,2,3], Laurent Venance[1,2,3], and Hugues Berry[5,6⋆]

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Activity-dependent long-term potentiation (LTP) and depression (LTD) of synaptic strength underlie multiple forms of learning and memory. Endocannabinoids (eCBs) have consistently been described as mediators of short- or long-term synaptic depression through the activation of the endocannabinoid-type-1 receptor (CB1R) or the transient receptor potential vanilloid-type-1 (TRPV1). Here we inves-

tigated whether eCBs could also promote long-term potentiation, an essential requirement for eCBs to be a genuine bidirectional system and to fully encode for learning and memory.

To this aim, we combined in vitro spike timing-dependent plasticity (STDP) protocols in rodents and a biophysical model of the signaling pathways likely to be involved. The model describes the temporal dynamics of three main signaling systems: the postsynaptic NMDAR-CaMKII pathway (adapted from [1]), the postsynaptic mGluR-PLCβ system (adapted from [2]) as well as postsynaptic eCB synthesis and subsequent activation of postsynaptic TRPV1 and presynaptic CB1R in a retrograde fashion. Using the model to drive the experiments, we uncovered the existence of an eCB-mediated spike-timing dependent potentiation (eCB-LTP). This eCB-LTP is homosynaptic, astrocyte-independent and expressed in young and adult animals and across various brain regions (cortex and striatum), supporting its role as a widespread signaling system for spike-based plasticity. We deciphered the signaling pathways (pre- and postsynaptic receptors and enzymes) involved in this new form of plasticity and demonstrated that eCB plasticity has a postynaptic induction and a presynaptic maintenance. On the postsynaptic side, our results show that the dynamics of free cytosolic calcium is a key element for eCB-LTP induction. eCB-LTP is triggered when eCB transients reach sufficiently high levels. Since the enzymes that synthetize eCBs are calcium-activated, eCB-LTP induction requires large levels of cytosolic calcium. On the presynaptic side, eCBs encode for bidirectional plasticity via a triad composed of eCB levels, presynaptic PKA and presynaptic CaN: intermediate eCB levels promote presynaptic CaN activity, that yields eCB-LTD, whereas large eCB amplitudes favor presynaptic PKA activity, which leads to eCB-LTP. Both effects are predicted to rely on the inhibition exerted by activated CB1R on presynaptic adenylate cyclase and P/Q-type voltage-gated calcium channels. Moreover, we show that eCB-LTP and eCB-LTD can be induced sequentially in the same neuron, depending on the cellular conditioning paradigm. Therefore, our results demonstrate that eCBs, just like glutamatergic or GABAergic signaling, form a generic system able to encode for bidirectional plasticity and capable of genuine homeostasis.

Lastly, we found that eCB-LTP is triggered by very few paired spikes (5 to 10 post-pre spikes at 1 Hz are enough). Thus, eCB-LTP provides synapses with a mechanism able to react to the very first occurrences of incoming activity. This ability strongly contrasts with NMDAR-dependent LTP which, in a classical (1 Hz) STDP context, requires the iteration of at least 75-100 paired stimulations to be expressed, at odds with the observations that new associative memories and behavioral rules can be learned within few or even a single trials in mammals (e.g. [3]). Our results suggest that eCB-LTP may represent a neuronal substrate for such rapid learning abilities.

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O2 Trial-to-trial tracking of excitatory and inhibitory synaptic conductance using Gaussian-mixture Kalman filtering

Milad Lankarany[1⋆], Wei. Ping Zhu[1], M. N. S. Swamy[1], and Taro Toyoizumi[2]

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Interaction of the excitatory and inhibitory synaptic inputs constructs the shape of the receptive fields and can elucidate the synaptic mechanism underlying the functional activities of neurons. Estimating trial-to-trial excitatory and inhibitory synaptic conductance from noisy observation of membrane potential or input current can reveal drivers of neurons and play an important role in our understanding of information processing in neuronal circuits. Although recent studies introduced statistical methods that estimate trial-to-trial variation of synaptic conductance [1, 2], most previous works use the well-known least square (LS) method to estimate the excitatory and inhibitory synaptic conductance from the trial-mean of recorded traces of membrane potential or input current [3-5]. We first analytically show that the LS method is not only incompetent to capture trial-to-trial variation of synaptic conductance but also provide biased estimation of synaptic conductance and excitatory/inhibitory covariance if fluctuation of synaptic conductance and membrane potential is correlated. Next, we propose a novel method based on Gaussian mixture Kalman filtering (GMKF) that not only overcomes the aforementioned limitations of the LS method but also gives the opportunity of trial-to-trial estimation of the excitatory and inhibitory synaptic conductance. We show that our proposal requires fewer assumptions than the recent proposals [1, 2] that also provide trial-to-trial estimation of synaptic conductance. In particular, the proposed technique outperforms [1] by providing the ability of estimating an unknown synaptic distribution using Gaussian mixture model (GMM). We believe that our findings have a significant influence on our understanding of the balance of excitatory and inhibitory synaptic input and the underlying cortical circuitry.

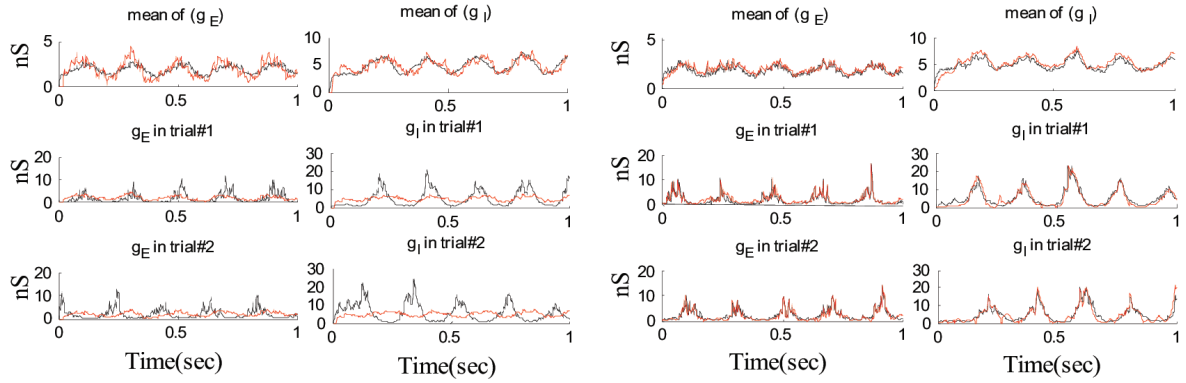

Figure 1. Estimating excitatory and inhibitory synaptic conductance using LS Voltage-clamp (left) and GMKF (right). LS method cannot estimate trial-to-trial synaptic conductances (10 trials each lasted 1 sec is used to provide data for both methods). True values (black solid line) and estimated ones (red dashed lines). See supplementary for more details.

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O3 Inhibitory STDP generates inverse models through detailed balance

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In the song bird ('backward') mappings from sensory representations to motor areas recently were proposed that would 'postdict' the motor activations during singing. Such a sensor-motor mapping represents an inverse model of the motor-sensor-loop passing through the world and thereby can explain the impressive imitation capabilities of song birds [1].
The neurobiological mechanisms that might generate, fine tune and continuously adapt such inverse models, however, are not known. Here we show that spike timing dependent plasticity (STDP) of the inhibitory synapses is sufficient for the self-organisation of the inverse model in a simple closed loop motor-sensor-motor system [2]. Similar to the case of forward models, where predictable, self-generated inputs become suppressed [3], the proposed mechanism generates sparse motor activities by cancelling predictable fluctuations of the neurons' excitabilities. Our results show that inverse mappings can be learned with an elementary and biologically plausible learning rule and thus could underly imitation learning. In our presentation we will discuss also the potential relevance of this mechanism for the operation state of recurrent networks as e.g. cortex [4].

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O4 A cerebellar learning model that reproduces the behavior of vestibulo-ocular reflex adaptation in wild-type and knock-out mice

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The cerebellum is crucial for different types of motor learning. Established theories of cerebellar learning posit that the cerebellum learns by adjusting the weights of Parallel Fiber (PF) to Purkinje cells (PC) synapses, thanks to teaching signals provided by Climbing Fiber inputs. While these theories are consistent with a large body of experimental data, in particular on synaptic plasticity in PF to PC synapses, they cannot easily explain a growing body of experimental work, which seems to indicate a significant role of other sites of plasticity. Recent advances in the development of a large number in transgenic animals, as well as behavioral and electrophysiogical comparative studies between these animals and wild-type animals, have opened an unprecedented window into the mechanisms underlying learning in this structure. In particular, it has been shown that specific knock-outs are impaired selectively on difficult variants of the vestibulo-ocular reflex (VOR) adaptation task, one of the most studied cerebellar-dependent motor learning tasks. These impairments can occur even though the classical plasticity mechanisms are left untouched. These data pose significant new challenges for established models of cerebellar learning.

To better understand the mechanisms of learning in the cerebellum, we built a model that can reproduce the available data on VOR adaptation, in both wild-type and transgenic animals. The model includes some of the main cell types involved in this task: granule cells (GCs), the input layer of cerebellar cortex, that receives vestibular information from the mossy fibers (MFs); Purkinje cells (PCs), as well as molecular layer interneurons (INs); and two cell populations in the medial vestibular nuclei (MVN), one excitatory and one inhibitory, that together control eye movement. The model also includes two sites of learning: the classical GC to PF plasticity site, as well as plasticity in the MF to MVN synapses. We provide a mechanistic understanding on how the system learns VOR adaptation in normal conditions, as well as how the system is impaired by specific knock-outs, which selectively suppress inhibition onto PCs, or increase the excitability of GCs. Finally, we show that our model is consistent with behavioral, as well as in vivo electrophysiological recordings.

O5 We now know what fly photoreceptors compute

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It has been known for some time that photoreceptors transmit more information when driven by stimuli which have 'naturalistic' statistical properties and that processing of naturalistic visual stimuli involves nonlinear transformations of the input signals. Until now it was not clear which statistical features of the stimuli the neurons are selectively 'tuned' to respond to. Another major difficulty was to elucidate the computational mechanisms which explain differences in coding naturalistic and Gaussian stimuli. Here we used a functional model of *Drosophila* photoreceptor to fully characterize their underlying computational capabilities. The model was derived based on photoreceptor responses measured *in vivo* using naturalistic input stimuli, which the system experiences in its natural environment. In order to characterize the nonlinear transformations performed by photoreceptors we analytically computed the higher-order or generalized frequency response functions (GFRFs) of the identified nonlinear photoreceptor model. Using this approach, we demonstrate, for the first time that the observed increase in power of photoreceptor response, and shift towards higher frequencies as the mean light intensity increases, is the result of changes in the operating point of the photoreceptor, which produces a change in the shape of the magnitude of the frequency response functions. This suggests that the photoreceptor adaptation mechanisms are not tuned to fully compensate for the drop in intensity in order to achieve, in an efficient way - by exploiting the photoreceptor nonlinearity - a change in the shape of the magnitude transfer functions, which are optimal for the given mean intensity level.
Furthermore, we examined the significance of both local and non-local high-order phase correlations for fly vision. We simulated the photoreceptor model using synthetic stimuli sequences incorporating local phase correlations (edges) and non-local phase correlations (quadratic phase coupling) superimposed with Gaussian white noise. By decomposing voltage output of photoreceptor somata into linear second- and higher-order responses, we explain the nonlinear mechanisms responsible for coding the local and non-local higher-order statistical features in the stimuli as well as improving their signal-to-noise ratio.
To validate the results, we carried out electrophysiological experiments using a specially designed stimuli sequence, which allows extracting the nonlinear component of the photoreceptor response directly from data, without a model.

Conclusion

The frequency response decomposition employed in this study allowed us to reveal for the first time the quantitative relationship between the higher-order statistical properties of environmental stimuli and processing of these stimuli in fly photoreceptors. In light of the results, we argue that the goal of early sensory coding is to maximize sensitivity to higher-order statistical features of the stimuli that are behaviourally relevant to the animal whilst minimizing sensitivity to non-informative signals, to encode efficiently these features and increase their salience to facilitate further processing. Our framework elegantly explains the differences in coding of naturalistic and white noise signals and how this is achieved efficiently without a change in the response transfer function of the photoreceptor when the mean light intensity is constant. It also explains why and how naturalistic stimuli increase the rate and efficiency of information transmission.

O6 Spontaneous emergence of simple and complex receptive fields in a spiking model of V1

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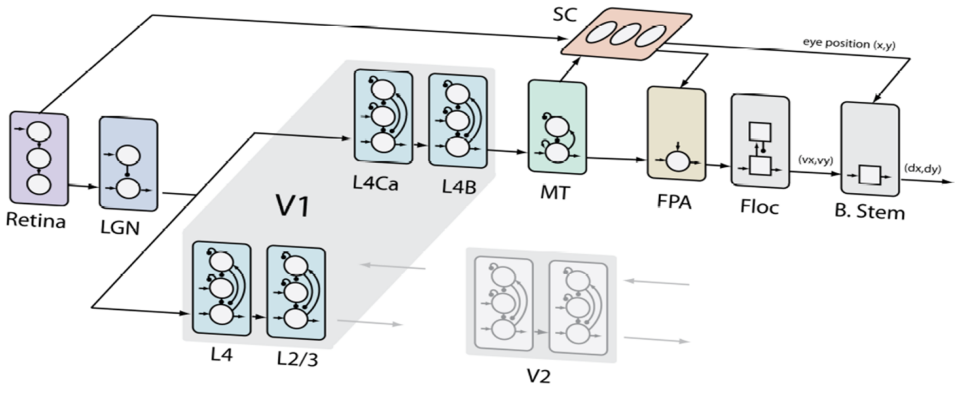
Brain Corporation is engaged in a multi-year project to build a spiking model of vision, paying special attention to the anatomy and physiology of the mammalian visual system. While it is relatively easy to hand-tune V1 to get simple and complex cells, it is not clear how to arrange connectivity in other cortical areas to get appropriate receptive fields, or what the appropriate receptive fields even should be. Instead of pre-wiring cortical connectivity according to a computational theory of how vision should work, we start with a generic "tabula rasa" spiking network having multiple cortical layers and neuronal types (single-compartment RS, FS, LTS cells). The goal is to find the anatomical and physiological parameters so that the appropriate connectivity emerges through STDP and visual experience. Since we know exactly what kind of receptive fields and visual responses are in V1, we build a smaller model of retina-LGN-V1 pathway and tune the STDP parameters so that the expected responses emerge. Admittedly, there are many free parameters that could be tuned to get V1-like responses; our choice is motivated by in-vitro recordings and other published data, whenever possible. Once we trust what we see in V1, we are ready to copy and paste the cortical model to implement V2, V3, V4, and IT areas with the hope that useful connectivity, receptive fields, and visual responses emerge. Our large-scale simulations of the spiking model of the visual system show spontaneous emergence of simple and complex cells, orientation domains, end-stopping receptive fields, extra-classical receptive fields with tuned surround suppression, color opponency that depends on the eccentricity of the receptive field, contrast invariance, and many other features that are routinely recorded in V1. Since the visual model exhibits micro- and full saccades, we observe perceptual behavior, such as the emergence of bottom-up (pop-out) attention. The model underscores the importance of spike-timing dynamics, inhibition, saccadic mechanism, and it imposes important restrictions on the possible types of STDP to model early visual processing.



This presentation is meant to be a flagship introduction to the whole effort by Brain Corporation to build spiking model of vision. There are multiple other submissions elaborating details of our modeling effort. The figure summarizes the gross anatomy of the spiking model of the visual system, including area MT, Superior Colliculus (SC), Frontal Pursuit Area (FPA), Flocculus (Floc), and Brainstem

(B.Stem). The retina covers 10 by 10 degrees of visual field with the density of L, M, and S cones and receptive field sizes of Midget, Parasol, and SBC retino-ganglion cells corresponding to 4 degrees of eccentricity of a primate retina (collaboration with EJ Chichilnisky lab at Salk Institute). The model consists of more than 1M single-compartment neurons of RS, FS, and LTS types (Izhikevich, 2007 "Dynamical Systems in Neuroscience") and 0.25B synapses having AMPA, NMDA, $GABA_A$ and $GABA_B$ conductances, axonal conduction delays, and STDP. In this presentation, we summarize these and other anatomical and physiological assumptions, describe major results and point to other submissions by Brain Corporation scientists for more details.

O7 How efficient coding of binocular disparity statistics in the primary visual cortex influences eye rotation strategy

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Stereopsis, the ability to perceive depth, is crucial for detecting camouflaged objects and for performing tasks that require estimating distance. Understanding how our brain can so quickly estimate depth from the slightly different images from our left and right eyes, the difference of which is called binocular disparity, is still a major unsolved problem in vision research. Several previous computational studies of binocular disparity have found evidence suggesting that various properties of the primary visual cortex (V1) allow V1 to optimally process natural binocular disparity statistics. In particular, the efficient coding hypothesis suggests that the disparity tuning of V1 binocular neurons should reflect the natural range of disparities [1, 2]; the cortical wiring hypothesis suggests that the orientation of ocular dominance stripes should follow the binocular disparity map [3]; and visuomotor optimization theory and the efficient coding hypothesis suggests that eye rotation strategy should be chosen to minimize binocular disparity and motor inefficiency [4, 5].

However, these previous studies all made assumptions about visual environments that are not supported by physiological data, and so we have constucted a more comprehensive simulation of binocular disparity than was used in any of these previous studies. We generate a three-dimensional visual environment with two-point statistics and observer-object distance distribution similar to that of a natural environment; the observer rotates her eyes according to the binocular version of Listing's Law to fixate on either edges of objects, centers of objects, or randomly chosen points on objects; and the resulting binocular disparities are mapped to V1 by a Schwartz conformal map fit to physiological data. The effects of two-point statistics, primary Listing's plane exorotation angle, Listing's Law coefficient, and fixation strategy can be revealed using this simulation in ways that would be very difficult (if not impossible) with experiments. For instance, to measure the effect of two-point statistics on binocular disparity statistics using this simulation, we compare the binocular disparity statistics in our more complicated three-dimensional visual environment to a visual environment with the same observer-object distance distribution but no pixel-pixel correlations.

Our simulations show that the predicted ocular dominance stripe orientations and stereoscopic search zones are largely insensitive to two-point statistics of the visual environment, whereas the joint probability distribution of vertical and horizontal disparities is sensitive to the two-point statistics of the environment. All three are sensitive to interocular distance and eye rotation strategy. Finally, our more

careful treatment of oculomotor strategy and visual environment shows that physiological oculomotor strategy cannot be explained by current visuomotor optimization theory [5].

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O8 Propagating waves structure spatiotemporal activity in visual cortex of the awake monkey

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Propagating waves of activity are seen in many types of excitable media, and in recent years, were found in the neocortex of anesthetized animals [1,2]. To date, however, it still remains unclear whether propagating waves appear during awake and conscious states [3,4]. One possibility is that these waves are systematically missed in trial-averaged data, because of their well-known variability from trial to trial [1].

To test this hypothesis, we developed a phase-based analysis technique, which works on a pixel-by-pixel basis in the unsmoothed data, and provides a quantiative means to distinguish between spatiotemporal forms of the population response. We then applied this to single-trial voltage sensitive dye imaging (VSDI) data, denoised specifically for this purpose [5], and in this work, we show definitively that spontaeous and stimulus-evoked propagating waves occur in the visual cortex of the awake monkey. Furthermore, when looking at the multiple visual areas within the imaging field in these experiments, we observe correlated propagations across primary and secondary visual cortex, illustrating a strong spatiotemporal organization of these waves across cortical areas.

These results demonstrate that propagating waves are systematically and reliably evoked by sensory stimulation, and suggest that they have the potential to affect large-scale information processing by generating a consistent spatiotemporal frame for neuronal interactions. The horizontal fiber network mediating these activity patterns has previously been implicated in active computational roles, as ascending input at a given point in cortex is known to affect the processing of future stimuli across the cortical plane [6,7]. In this work, we implicate these propagations in a specific functional role. These

internally generated propagating waves provide a specific structure for the spatiotemporal activity in visual cortex, uniquely encoding both stimulus identity and time of presentation in the amplitude and phase of the population response [8]. With these results in mind, we go on to discuss the computational paradigms towards which our observations point, elucidating these with numerical models and further analysis.

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O9 Non-renewal spiking and neural dynamics – A simple theory of interspike interval correlations in adapting neurons

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There is accumulating evidence that the spiking of many neurons is not a renewal process but is characterized by correlations between interspike intervals (ISIs) [1]. These correlations are crucial for understanding signal processing in single neurons, however, their origin and structure is still poorly understood theoretically. Here, we present a simple theory of correlations in neural oscillators with spike-triggered adaptation currents, which are a major source of non-renewal spiking. These currents mediate spike-frequency adaptation and are commonly believed to result in negative correlations between adjacent ISIs. For such adapting neurons, we show that the serial correlation coefficient (SCC) is fundamentally related to the neuron's phase response curve (PRC). The relation predicts possible correlation patterns that characterize how correlations depend on the lag between ISIs. Different patterns arise from the specific interplay between nonlinear neural dynamics and adaptation dynamics. In particular, the correlation structure can be determined by a single parameter that includes the shape of the PRC as well as the strength and time scale of the adaptation current (Fig. 1).

For a positive PRC (type I), the SCC is always negative at lag 1. At higher lags, we find either a

monotonically decaying (Fig.1A) or oscillating (Fig.1C) behavior of the SCC depending on the strength of adaptation. Similar correlation structures have been observed in different experimental studies (see e.g. [2]). Despite the distinct patterns, the total correlation as expressed by the sum of the SCC over all lags displays a universal value close to -0.5 at high firing rates and strong adaptation. As an example of a type I neuron, we discuss one-dimensional integrate-and-fire (IF) neurons with adaptation (like e.g. the adaptive exponential IF model [3]) operating in the supra-threshold regime. For these models, the different behaviors of the SCC can be explained by qualitatively different structures of the phase plane spanned by the voltage and the adaptation variables. Our theory also predicts that adapting neurons with a partly negative PRC (type II phase resetting) can additionally exhibit non-negative ISI correlations, which is indeed found in a two-dimensional IF model with adaptation (Fig.1D,E). Thus, adapting neurons can show a richer repertoire of correlation patterns than previously thought.

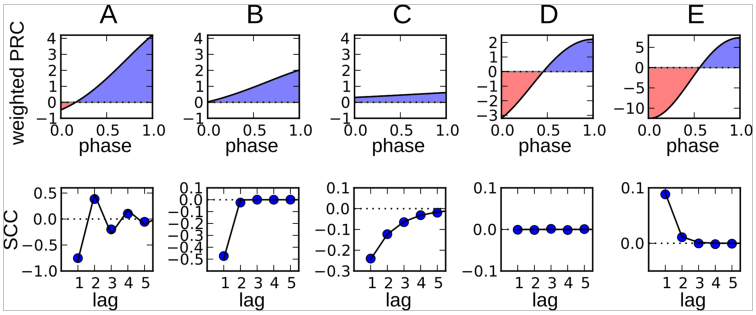

Figure 1. *Possible patterns of ISI correlations of an adapting neuron (bottom). The correlation pattern is determined by the area under the weighted PRCs (top). Shown are data for a two-dimensional noisy resonate-and-fire model with adaptation (simulations:circles, theory: solid line).*

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O10 The role of neural correlations in a decision-making task

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Simultaneous recordings of the firing activity of pairs of cortical neurons have shown that spike-count correlation coefficients (CCs) cover a wide range of values. According to recent theoretical and experimental work [1,2] recurrent cortical networks decorrelate neural activity producing very low CCs. However little is known about the origin of correlations and data analysis based on recordings of cortical activity of awake, behaving animals performing non-trivial tasks are scarce. In this study, we aim to understand the role of neural correlations in perceptual decision-making tasks and its relationship with the covariation between neural firing activity and behavior.

We examine spike-count correlations obtained from pairs of simultaneously recorded premotor cortex (PC) neurons while trained monkeys performed a vibrotactile detection task in which the stimulus was often absent or weak, and the time of its application was variable [3,4]. By analyzing firing rates and correlated variability we show that behavioral outcomes are crucially affected by the state of cortical networks before stimulus onset times.

Our results suggest that sensory detection is partly due to a purely internal signal whereas the stimulus, if finally applied, adds a contribution to this initial processing later on [5]. Noise correlations can be weak; their smallest values are attained at the end of the delay period of the task. Importantly, we found that small CCs are compatible with high firing rates. Although the firing rate in hit trials is higher than in correct rejections, the distributions of CCs over the population of pairs are similar, presenting mean values of 0.06. Moreover, the CCs do not covary with the geometrical mean of the firing rate of the pair.

The observation that single neurons covary with the subject's response (characterized by the choice probability index, CP) is usually explained by the existence of variability correlations among the cells in a neuronal population [6]. Here we show a simple approximate expression that explicitly relates the population-averaged CP index and the CCs. This expression shows that the CP index is different from 0.5 when CCs evaluated using all trials differ from choice-conditioned correlations. Neurons could covary significantly with behavior even if the latter are very small. Thus, we show that there is no contradiction between the correlated activity required for the non-chance CP index and the small correlations produced by decorrelation in recurrent networks.

Although the CP index is useful to study the role of single neurons in decision-making tasks, in reality, the decision is formed through the coordinated action of several pools of neurons. Hence, the relevant quantities to investigate the elaboration of the choice are population variables combining the activity of several pools. By extending the notion of CP from single neurons to neural pools, we defined the $\mathrm{CP_N}$ index to quantify the amount of covariation with behavior of arbitrary linear combinations of oppositely-tuned neural pools. We found that pools of PC neurons exhibiting persistent activity become fully correlated with the subject's choice soon after stimulus onset and during the entire delay period of the task.

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O11 Cellular temperature compensation of sensory receptor neuron responses

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Temperature is known to modulate ion channel kinetics and hence also action-potential generation. This poses a challenge for neural systems that need to retain their functionality also under conditions of varying temperature. Multiple strategies to counterbalance the effects of environmental temperature changes exist: mammals keep their body temperature approximately constant, while poikilothermic species need to implement temperature-compensation at the behavioral, systems, or cellular level. While mechanisms of behavioral and systems level have been identified [1], cellular mechanisms of temperature-compensation as well as their associated metabolic cost remain largely unknown.
We investigated the effect of temperature on auditory processing in the grasshopper. We recorded intracellular responses of auditory receptor neurons to auditory broad-band noise stimuli at different intensities at two distinct behaviorally relevant temperatures. Interestingly, we found that changes in temperature did not have large effects on sound-intensity coding in receptor neurons. These neurons constitute the input layer of a feedforward network and hence do not receive network input. We concluded that the observed temperature robustness of receptor-neuron responses must arise from *intrinsic*, network unrelated effects.
In general, the receptor-neuron response is shaped by two processing steps: mechanosensory transduction and spike generation. Both can contribute to temperature compensation. Either both transduction and spike generation are compensated (hypothesis I), or alternatively, their temperature dependencies can cancel each other (hypothesis II).
To test hypothesis I we assumed a temperature-invariant transduction and asked, first, whether temperature-compensation could be achieved for a spike-generating mechanism with realistic temperature dependencies of the ionic conductances. The latter refers, in particular, to increases of gating kinetics by a factor of 2-4 with temperature increments of 10°C (defining a Q10 value of 2-4) as well as modest increases of peak conductances. Second, we explored whether temperature compensation, if achieved cell-intrinsically, compromises the neuronal energy budget. In other words, is temperature robustness metabolically expensive? To address these questions, we varied the temperature dependence of ionic conductances in a conductance-based neuron model. Based on the spike frequency vs. input current (f-I) relation, we estimated the ability of the model neurons to keep a robust firing rate despite changing temperature. Moreover, we computed the average energetic cost per action potential [2]. Using a database modeling approach [3], we performed a systematic sensitivity analysis for firing-rate changes and energetic cost as a function of the temperature dependence of conductance parameters (i.e. Q10 values of transition rates and peak conductances). Our analysis shows that the key parameters determining the robustness of spike generation relate to the temperature-dependence of the model's *potassium* conductances. In contrast, energy consumption is governed by the temperature dependence of the *sodium* conductance. Consequently, a neuron can achieve temperature-compensation of its firing rate without compromising the energy budget.
To constrain hypothesis II, we used the experimentally observed f-I curves in an objective function and inferred the corresponding transduction process for each spike generation in our sensitivity analysis.

Our results predict that thermosensitive Transient Receptor Potential (TRP) channels have a role in mechanosensory transduction at the grasshopper tympanum, and therefore motivate further experiments.

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O12 Self-organized lateral inhibition improves odor classification in an olfac-tion-inspired network

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The insect olfactory system is capable of classifying odorants by encoding and processing the neural representations of chemical stimuli. Odors are transformed into a neuronal representation by a number of receptor classes, each of which encodes a certain combination of chemical features. Those representations resemble a multivariate representation of the stimulus space [1]. The insect olfactory system thus provides an efficient basis for bio-inspired computational methods to process and classify multivariate data.
Olfactory receptors typically have broad receptive fields, and the odor spectra of individual receptor classes overlap. From the viewpoint of multivariate data processing, overlapping receptive fields cause correlation between input variables (*channel correlation*). In previous work, we demonstrated how lateral inhibition in an olfaction-inspired network reduced channel correlation [2,3]. Decorrelation was achieved by setting the strength of lateral inhibition between two channels according to their correlation, which we pre-computed from the input data.
Here, we propose unsupervised learning of the lateral inhibition structure. The lateral inhibition synapses support inhibitory spike-timing dependent plasticity (iSTDP) [4,5]. After exposing the network to a sufficient number of input samples, the inhibitory connectivity self-organizes to reflect the correlation between input channels. We show that this biologically realistic, local learning rule produces an inhibitory connectivity that effectively reduces channel correlation and yields superior network performance in a multivariate scent recognition scenario.

Acknowledgements
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O13 Mechanisms of sharp wave-ripple generation and autonomous replay in a hippocampal network model

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Several distinct patterns of population activity can be recorded in the hippocampus in vivo. These neural activity patterns depend on the behavioral state of the animal, and include theta-modulated gamma oscillations as well as low-level irregular activity with periodically occurring large-amplitude sharp wave-ripple (SWR) events. During SWRs, neuronal populations in the hippocampus have been found to "replay", on a faster time scale, activity recorded during theta-gamma activity in the exploring animal. Such replay may be important for the establishment, maintenance and consolidation of long-term memory. Our aim was to develop a mechanistic understanding of cellular and network mechanisms underlying the generation of SWRs in general, and spatio-temporal sequence replay during SWRs in particular, based on in vitro and in vivo experimental observations.

A recently developed hippocampal slice preparation, in which SWRs arise spontaneously, has allowed the collection of a large and diverse set of data regarding the properties of SWRs, as well as the characterization of several cell types and synapses which are critical in their generation. Based on these data, combined with phase-plane analysis of the network dynamics, we developed a large-scale network model of the hippocampal CA3 region. We found that our model based on measured cellular and synaptic parameters could faithfully reproduce the experimentally observed SWR activity as long as we included an appropriate slow feedback mechanism which was responsible for the termination of SWR bursts. Our model allowed us to rule out several potential candidates (such as neuronal adaptation) for the slow feedback process, suggesting that either slowly activating interneuronal feedback or short-term synaptic plasticity of connections within CA3 might terminate SWRs. Statistical analysis and fitting of the inter-event interval distribution of SWRs in vitro suggested that, following an initial 'refractory period' after each SWR, the next SWR is initiated stochastically, requiring the simultaneous activation of a threshold number of pyramidal cells. When we implemented the changes in cellular and synaptic properties measured in the slice following the activation of cholinergic receptors, our model replicated the experimentally observed transition from SWR activity to gamma oscillations. Finally, applying a spike-timing-dependent plasticity rule to the recurrent excitatory weights during simulated exploration, the emerging weight structure led to the spontaneous replay of sequences of place cell representations during simulated SWRs. We also found that using structured rather than random weights substantially altered the global network dynamics, resulting in a sparse participation of pyramidal neurons in individual SWR events, and allowing our model to match more closely the

corresponding experimental observations.

Acknowledgements
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O14 Extracellular field signatures of CA1 spiking cell assemblies during sharp wave-ripple complexes

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Although postsynaptic and transmembrane currents over local neuronal populations are considered the main factors for shaping local field potential (LFP) and current source density (CSD) fluctuations [1], high-frequency oscillatory LFPs can also be shaped by extracellular action potentials of pyramidal cell populations [2]. Sharp wave-ripple complexes (SWRs) are typical examples of such high-frequency oscillatory events, observed in hippocampal LFPs during deep sleep and awake immobility. They consist of an extensive depolarization in the CA1 dendritic layer (sharp wave) arising from population bursts in CA3, accompanied by a ∼150-200 Hz LFP oscillation in the CA1 pyramidal layer (ripple). During SWRs, temporal firing patterns of correlated place cells, acquired during wakeful exploration, are replayed in fast-scale, providing a strong indication for the participation of SWRs in memory consolidation. Yet the particular effects of these pattern replays on the hippocampal extracellular field are largely unknown. How are the different ensembles of spiking cells encoded in the emerging ripple-LFPs? Here, we study this association through both a modeling and an experimental approach.

Firstly, we employ a spiking network model of the CA3 and CA1 hippocampal areas that reproduces key features of SWRs based on synchronous CA3 population bursts and strong, fast-decaying CA1 recurrent inhibition [3]. The synaptic input on CA1 pyramidal cells is implemented in a population of morphologically realistic, multi-compartmental models of CA1 pyramidal neurons, based on reconstructed cells [4]. The emerging extracellular fields accurately reproduce properties of SWR LFPs. By developing different spatial distributions of sets of CA1 cells that fire during ripples and others that remain silent, we explore in a systematic fashion the influence of spiking cell assemblies on the spatiotemporal characteristics of emerging extracellular fields during SWRs. In particular, we show how the different spatial arrangements of spiking cells give rise to differences in the depth-profile and CSD characteristics of raw and filtered LFPs.

Next, we apply our analysis to a set of LFPs and unit activity, recorded *in vivo*, from multiple locations in areas CA3 and CA1 of the rat hippocampus while animals run on a linear track with resting areas at both ends. The spiking activity of detected place cells, firing in sequence during the running sessions, is replayed in either forward or reverse order during SWRs occurring at the resting areas [5]. We trace the differences in the depth profile and CSDs of SWR LFPs between such forward and reverse replays. Based on our modeling results, we provide a link between systematic changes in the spatiotemporal features of the LFPs, with the corresponding ensembles that gave rise to each ripple episode.

This work provides a deeper understanding of the nature of extracellular fields and offers a new approach to the decoding of ongoing cell assemblies based on extracellular current flows. Differences in

the emerging SWR field activity may play an important role in information processing during memory consolidation.

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O15 Cross-talk and transitions between multiple environments in an attractor neural network model of the hippocampus

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Place cells are neurons in the hippocampus whose activity depends on the animal's location in space and are therefore thought to be crucial for spatial representation [1]. Based on the assumption that CA3 works as an attractor neural network [2] models have shown that spatially-localized attractors, corresponding to different 'environments' or 'spatial maps', can be encoded in one network [2-3]. Transitions and cross-talks between attractors coding for different maps remain, however, poorly understood.

Motivated by a recent experiment showing bistability between competing spatial representations, paced by theta waves [4] we propose a recurrent model network, whose synaptic connections J_{ij} sum up contributions coming from all the environments according to: 1. The contribution to J_{ij} due to an environment vanish when the centers of the place fields of cells i & j are further away than some cut-off distance w; 2. Place fields are randomly remapped from one environment to the other. Using tools and concepts from the statistical physics of disordered systems we have solved the model and show that the network can be in one of three regimes, depending on the level of noise in the neural dynamics, T, and the number of environments, L (Figure 1**A**). In particular, we have found the maximal values of T and L (given the other parameters of the model e.g. the number of place cells, the average firing rate,...) such that, spatially-localized and environment-specific activity is possible. In addition we have observed the presence of spontaneous, i.e. in the absence of external input, dynamical transitions from the activity localized in one map to the activity representative of another environment (Figure 1**B**). Those transitions are strongly reminiscent of those experimentally observed in [4]. The statistical features of the transitions and their dependence on the parameters of the model can be understood in great analytical details.

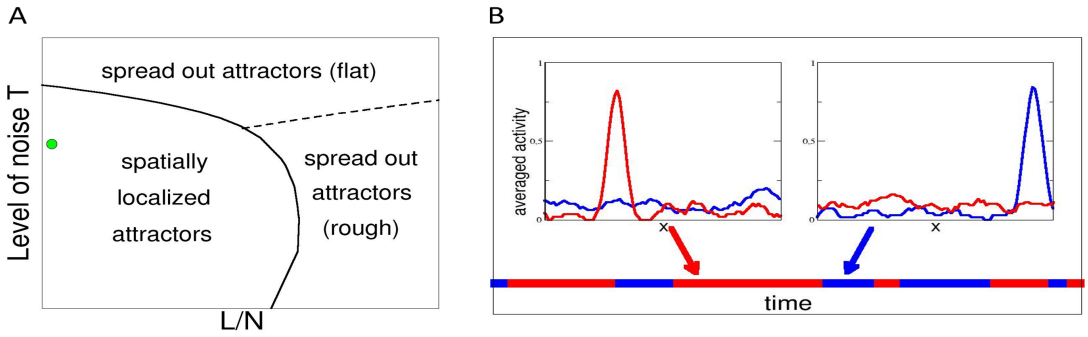

Figure 1: *Properties of the model (1D-space with w=0.05, 10% of active neurons).* ***A.*** *Sketch of the phase diagram. For moderate T and L the network activity is spatially localized in one of the stored maps and encode a specific location in space. For high T (strong noise) or high L (strong map interference) the activity profile spreads over the whole space, either uniformly or with rough, interference-induced ripples.* ***B.*** *Transitions between two stored maps (symbolized with blue and red colors) during a Monte Carlo simulation with N=1000 neurons; values of T and L correspond to the green spot in panel A. Insets: place-cell activity vs. place-field center locations in blue and red maps, for two instants indicated by the arrows.*

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O16 Model-based prediction of fusimotor activity during active wrist movements

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Introduction

Muscle spindles, whose activity is determined by muscle length changes and by fusimotor drive (i.e. γ-drive), provide critical information about movement position and velocity [1]. However, task-dependent fusimotor drive remains largely unknown [2], since no fusimotor neurons have ever been recorded during active, voluntary upper limb movements, whether in animals nor in humans. So far an estimation of γ-drive could only be obtained through an indirect inference of fusimotor activity from observed muscle spindle activity. Our aim was to model the effect of γ-drive on muscle spindles and to simulate voluntary wrist movements for which the

Methods

Our conceptually simple computational model (an adaptation of [3]) allows for a direct quantification of γ-drive. A forward calculation predicts spindle responses based on time-varying γ-drive and muscle length changes. This computational model thus links a biomechanical (musculo-tendon) wrist model

to length- and γ-drive-dependent transfer functions of group Ia and group II muscle spindles. These transfer functions were calibrated (Fig. 1A) with extant data from passive movements in the cat [4].

Results

Our simulations suggest that (i) empirically observed muscle spindle activity profiles can to a large part be explained by a strongly task-dependent γ-drive (Fig. 1B), (ii) observed differences between individual muscle spindle response profiles can be explained by a corresponding variability in the γ-drive (Fig. 1B), and (iii) observed phase advance of spindle responses can to a large part be explained by appropriate γ-drive.

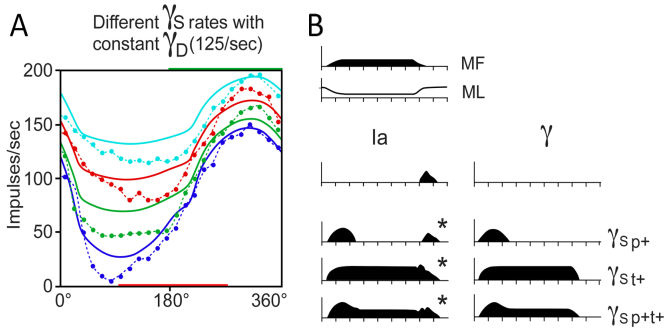

Figure 1. ***A.*** *Fit between passive [4] (dotted lines) and simulated (lines) Ia responses during sinusoidal stretch under constant γ_D-drive (125 Hz) and 4 different rates of γ_S-drive (top to bottom: 125, 75, 50, 0 Hz).* ***B.*** *Simulated Ia responses (left column) during active muscle contraction for 4 different γ_S-drives (right column): no, phasic, tonic and phasic-tonic drive. The asterisk indicates simulated responses similar to empirically observed Ia responses [5].*

Conclusion

Our simulation predicts that γ-drive is strongly modulated and task-dependent and that appropriate γ-drive can explain many empirically observed aspects of group Ia and II muscle spindle responses during active movements.

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O17 Operant conditioning of single units in rat motor cortex allows graded control of a prosthetic device

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Operant control of a prosthesis by neuronal cortical activity is one of the successful strategies for implementing brain-machine interfaces, by which the subject learns to exert a volitional control of

goal-directed movements.

Here, several motor cortex neurons were recorded simultaneously in head-fixed awake rats and were trained, one at a time, to modulate their firing in order to control the speed and direction of a 1D actuator carrying a water bottle. In the first phase of the experiment, the bottle could only move in one direction and this was triggered by an increase in firing rate. Most neurons submitted to this conditioning successfully increased their activity during trials, and this effect was enhanced across sessions. Once trained, the neuron chosen to control the operant behavior reacted consistently more rapidly than the other recorded neurons after trial onset. We observed also that the firing rate variability increased in an anticipatory way before trial onset, specifically for the neurons that could be conditioned successfully. However, this effect was observed only in the initial phases of the conditioning.

In the second phase of the experiment, neurons modulated their firing rate up or down in order to control the direction and speed of the water bottle. The bottle could thus move bilaterally, and the goal was to maintain the bottle in front of the rat'mouth in order to allow drinking. All conditioned neurons adapted their firing rate to the instantaneous bottle position so that the drinking time was increased relative to chance. The mean firing rate averaged over all trajectories depended on position, so that the mouth position operated as an attractor (at least for the bottle starting side). Again, the conditioned neuron reacted on average faster than the other neurons and led to a better bottle control than if trajectories were simulated using the activity of simultaneously recorded neurons.

Overall, our results demonstrate that conditioning single neurons is a suitable approach to control a prosthesis in real-time, and that these neurons occupy a lead position after learning, acting as "master" neurons in the network.

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O18 Structural Features Beneath Neuronal Avalanches

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Recently, Friedman *et al* performed high-resolution measurements [1] that strongly supported an universal critical character (in the sense of statistical physics) of neuronal avalanches, despite the abnormally high frequency of large events ("bumps" in the distributions of size and lifetime of avalanches) deforming the pure power-law behavior expected from analogies to equilibrium critical phenomena that became manifest only for smaller events.

Based on simulations of the Kinouchi-Copelli (KC) model, we have shown [2] that such bumps may not be experimental artifacts and really be typical at criticality when the topology of the neural network is the Barabási-Albert (BA) model, leading to a scale-free degree distribution of exponent -3.0. On the other hand, those simulations could not reproduce the exponents of the power-law region of the avalanche distributions in [1] (namely, -1.7 for the size distribution and -1.9 for the lifetime distribution). Besides that, the KC dynamics on BA topology revealed that the information capacity (entropy of avalanche size distribution) did not exhibit critical optimization, in contrast with an earlier experiment [3].

In this study we investigate the KC dynamics on the Uncorrelated Configuration Model (UCM) [4]. The UCM is a kind of "wiring procedure" of a neural topology that can lead to scale-free degree distributions with tunable exponents and can be "matched" to BA model. However, even so their avalanches are not identical. While the UCM also allows the appearance of bumps on the avalanche distributions, it both shows that the information capacity may be critically optimal and exhibits quantitatively accurate values of the critical exponents for small avalanches, suggesting the UCM may be descriptive of some structural features in the systems claimed to exhibit critical dynamics.

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O19 The Inhibitory Network of the Striatum at the Edge of Chaos

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The striatum forms the main input structure to the Basal Ganglia (BG), a subcortical structure involved in the selection and reinforcement learning of action sequences. It is 90% composed of medium spiny neurons (MSNs) which inhibit each other through collaterals, receive excitatory projections from cortex and are the only cells projecting outside the striatum. Because of its inhibitory structure the MSN network is often thought to act selectively, transmitting the most active cortical inputs downstream in the BG while suppressing others. However studies show that local MSN network connections are too sparse and weak to perform global selection and their function remains puzzling. Here we suggest that rather than generating a static stimulus dependent activity pattern the MSN network is optimized to generate stimulus dependent dynamical population activity patterns for extended time periods after variations in cortical excitation. Indeed MSNs form cell assemblies whose population firing rates vary coherently on slow behaviourally relevant timescales [1]. Furthermore individual MSNs display diverse response profiles locked to task and reward predicting events [2,3] with phasic activity peaks broadly distributed across the whole spectrum of delays after task events [4-6]. We have previously shown [7,8] that such activity emerges in a model of a spiking MSN network but only at realistic connectivities of $\sim$15% and only when MSN generated inhibitory post-synaptic potentials (IPSPs) are realistically sized. Here we suggest a reason why the MSN network generates such activity. We investigate how network generated population activity interacts with temporally varying cortical driving activity, as would occur in a behavioural task. We find [9] that at unrealistically high connectivity a stable winners-take-all regime is found where network activity separates into fixed stimulus dependent regularly firing and quiescent components. In this regime only a small number of population firing rate components interact with cortical stimulus variations. Around 15% connectivity a transition to a more dynamically active regime occurs where all cells constantly switch between activity and qui-

escence. In the low connectivity regime MSN population components wander randomly and here too are independent of variations in cortical driving. Only in the striatally realistic transition regime do weak changes in cortical driving interact with many population components so that sequential cell assemblies are reproducibly activated for many hundreds of msecs after stimulus onset and PSTH display strong stimulus and temporal specificity. We show that this activity is maximized at striatally realistic connectivities and IPSP sizes and cortical stimuli are also maximally distinguished at striatally realistic parameter settings. In fact Lyapunov exponent computations show that, quite remarkably, the MSN network sits precisely at a marginally stable point, the edge of chaos. Thus we suggest the local MSN network has optimal characteristics - it is neither too stable to respond in a dynamically complex temporally extended way to cortical variations, nor is it too unstable to respond in a consistent repeatable way. We discuss how these properties may be utilized in temporally delayed reinforcement learning tasks strongly recruiting the striatum.

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O20 Inferred network from prefrontal cortex activity of rats unveils cell assemblies

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We analyzed recordings of prefrontal cortex activity of a rat in three different phases: while the animal faces a task in which a rule has to be learned and during the previous and subsequent sleep phases. We inferred an Ising model (characterized by binary variables and local fields and couplings as parameters) from the recorded spiking frequencies and pairwise correlations between neurons. We have shown how the inferred model can be used to deepen the analysis of the recordings, unveiling the presence of highly coordinated groups of neurons (cell assemblies), that is neurons that are activated together and synchronously inhibit the activity of other specific neurons.

To identify the coactivated groups, we found the maxima of the log-likelihood of a configuration of neurons (stable states), defined as the sum of all the fields and couplings relative to the active neurons, performing an ascent dynamics on the energy landscape. When the model is inferred from the activity binned into 10ms time bins, the only stable state is the one with all silent neurons. By adding an external input into the model and slowly increasing its value, stable states with more and more active neurons appear (see fig.1A), starting from the neurons with higher spiking frequency. Remarkably, the curves in fig.1A show large jumps at specific values of the input strength, corresponding to the co-activation of strongly interconnected neurons, which not necessarily have high average activity. These highly synchronized neurons have been found from the models of both the awake and sleep epochs (see fig.1B), and are partially shared between different phases.

We investigated the meaning of the external input parameter, discovering that it carries information on the time scale at which we observe correlations between neurons, namely the time bin width. In fact, the two curves of fig.1A, which refer to the model inferred from the neuronal activity binned into two different time bins (10ms and 30ms), overlap by applying a translation of log(30ms/10ms) in the input strength. The fact that at Δt=30ms the fit co-activated group appears for a small input strength $H \sim 1$ means that the group is likely to be co-activated in a 30ms time scale.

Neurons found in activated and inhibited groups extracted from our model correspond to large entries in the two principal eigenvectors of the Pearson correlation matrix obtained from the recorded activity. In particular the 1st component has large and positive entries on both activated groups, and the 2nd component shows negative entries on the 1st group, and positive ones on the 2nd group which entails that the groups can activate together or not. Moreover the activation of a group causes the inhibition of another group, which has also large entries on the 1st and 2nd components but with opposite signs. The sign of the components therefore refects in an intricate manner the activation-inhibition relationships between different groups.

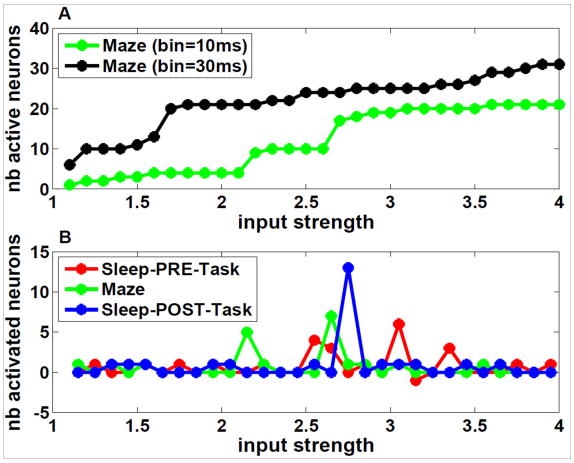

Figure 1: Co-activation of neurons as a function of the input strength and of the time bin. A. *Number of active neurons $A(H)$ in the stable states vs. external input H. Parameters (fields and couplings) of the Ising model were inferred from the activity of the Maze epoch, binned into time windows of Δt_1=10ms (green) and Δt_2=30ms (black). The two curves can be superimposed upon translation of the input strength by $\log(\Delta t_2/\Delta t_1)$.* ***B.*** *Number of newly activated neurons, defined through $A(H+0.1) - A(H)$, in the stable configurations for the three epochs vs. input strength H; the fields and couplings of the Ising model have been inferred for each epoch.*

O21 Multiscale modeling of cortical information flow in Parkinson's disease

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The basal ganglia play a crucial role in the execution of movements, as demonstrated by the severe motor deficits that accompany the neuronal degeneration underlying Parkinson's disease (PD). Since motor commands originate from the cortex, an important functional question is how the basal ganglia influence cortical information flow, and how this influence becomes pathological in PD. In contrast, previous models of PD have focused on either global brain dynamics or on local information flow, since developing a model that is valid on both scales presents a major technical challenge.

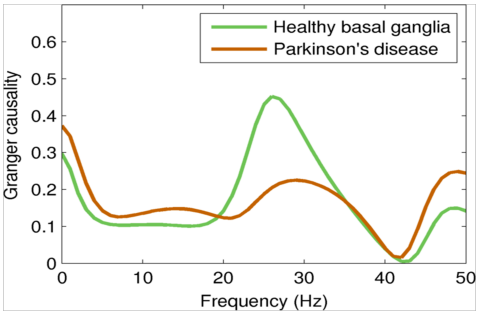

Figure 1: *Spectral Granger causality from layer 4 to layer 5, a major feedforward pathway in the cortex. The healthy model shows strong causality in the high-beta/low-gamma (20-35 Hz) band; this is almost entirely lost in the PD-driven model, resulting in substantially lower total causality.*

To address this issue, we developed a composite neuronal network/neural field model. The neuronal network consisted of 4950 event-driven rule-based neurons, divided into 15 excitatory and inhibitory cell populations in the thalamus and cortex. This model was then embedded in a neural field model of the basal ganglia-thalamocortical system, including the cortex, thalamus, striatum, subthalamic nucleus, and globus pallidus. Both network and field models have been separately validated in previous work [1-3], with both shown to produce realistic firing rates and spectra. Two field models were explored: one with parameters based on data from healthy individuals, and one based on data from individuals with PD. Spikes generated by these field models (which represent inputs from distant brain areas) were then used to drive the network model (which represents a small region of association cortex). We then explored the effects that these drives had on the information flow and dynamics of the network.

Compared to the network driven by the healthy field model, the PD-driven network had lower firing rates and increased power at low frequencies, consistent with clinical PET and EEG findings; it also had more spike bursts, indicating pathologically increased intracortical coherence. The PD-driven network showed significant reductions in Granger causality (see Figure 1). In particular, the reduction in Granger causality from the main "input" layer of the cortex (layer 4) to the main "output" layer (layer 5) represents a possible explanation for some of the characteristics of parkinsonism, such as bradykinesia. These results demonstrate that the brain's large-scale oscillatory environment, represented here

by the field model, strongly influences the information processing that occurs within its subnetworks.

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3. Van Albada SJ, Gray RT, Drysdale PM, Robinson PA: **Mean-field modeling of the basal ganglia-thalamocortical system. II. Dynamics of parkinsonian oscillations**. *J Theor Biol* 2009, **257**:664-88.

O22 HCN1-mediated interactions of ketamine and propofol in a mean field model of the EEG

Ingo Bojak[1,2,3⋆], Harry Day[2], and David Liley[4,5]

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[2] *School of Psychology (CNCR), University of Birmingham, Edgbaston, Birmingham B15 2TT, UK*

[3] *Donders Institute, Radboud University Nijmegen (Medical Centre), 6500 HB Nijmegen, The Netherlands*

[4] *Brain & Psychological Sciences Research Centre, Swinburne Uni. of Tech., Hawthorn, Victoria 3122, Australia*

[5] *Cortical Dynamics Ltd., Suite 4, 462 Burwood Road, Hawthorn, Victoria 3122, Australia*

Ketamine and propofol, two popular anesthetic agents, are generally believed to operate via disparate primary mechanisms: ketamine through NMDA antagonism and propofol through the potentiation of $GABA_A$-gated receptor currents. However, surprisingly the effect of ketamine on the EEG is markedly altered in the presence of propofol. Specifically, while ketamine alone results in a downshift of the peak frequency of the alpha rhythm, and propofol keeps it roughly constant - when administered together, they increase the alpha peak frequency [1]. Recently it has been found that both ketamine and propofol inhibit the hyperpolarization-activated cyclic nucleotide-gated potassium channel form 1 (HCN1) subunits, which induces neuronal membrane hyperpolarization [2]. Furthermore, HCN1 knockout mice are significantly less susceptible to hypnosis with these agents; but equally affected by HCN1-neutral etomidate [2]. We show here [3] that an established mean field model of electrocortical activity can predict the EEG changes induced by combining ketamine and propofol by taking into account merely the HCN1-mediated hyperpolarisations, but neglecting their supposed main mechanisms of action (NMDA and $GABA_A$, respectively). See Figure 1. Our results suggest that ketamine and propofol are infra-additive in their HCN1-mediated actions. This is consistent with independent experimental

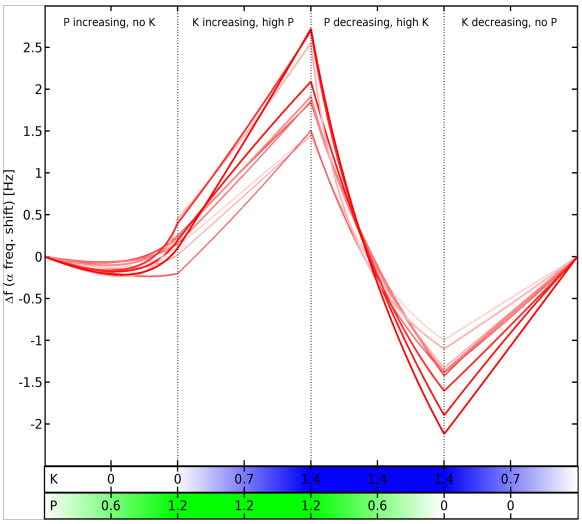

Figure 1. *Predicted shift of the alpha peak frequency of ten parameter sets during four phases of linear change to the normalized ketamine (K) and propofol (P) concentrations, respectively.*

evidence [4]. We show here that the HCN1-mediated actions of ketamine and propofol, hitherto neglected by models of anaesthetic action, can not only explain a range of counterintuitive induced EEG changes but also predicts the infra-additivity of these drugs.

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Workshops

W1 Advances in activity-dependent synaptic plasticity

Space Grignard, Wed & Thu, 9:00-17:30

Paul Munro, University of Pittsburgh, Pittsburgh, PA, USA
Claudia Clopath, Columbia University, New York, NY, USA

Since Hebb articulated his Neurophysiological Postulate in 1949 up to the present day, the relationship between synapse modification and neuronal activity has been the subject of enormous interest. Laboratory studies have revealed phenomena such as LTP, LTD, and STDP. Theoretical developments have both inspired studies and been inspired by them in an effort to reveal the biological principles that underlie learning and memory. The intent of the proposed workshop is to foster communication among researchers in this field. The workshop is intended to be of interest to experimentalists and modelers studying plasticity from the neurobiological level to the cognitive level. The workshop is targeted toward researchers in this area, hopefully drawing a 50/50 mix of experimental results and theoretical ideas. Another goal is to bring together established researchers with grad students and postdocs.

The proposed workshop will be held for two days (July 17-18), with four 45 minute talks in the morning sessions, and three 45 minute talks in the afternoons. We will organize a lunchtime poster session for students and postdocs if there is enough interests. Let us know by e-mail if you want to present a poster, or give a talk, since we have a couple of slots still available.

Invited Speakers:
Harel Shouval, Mayank Mehta, Walter Senn, Julie Haas, Eugene Izhikevich, Ole Paulsen, Karl Kandler, Alison Barth, Claudia Clopath, Paul Munro, Jenia Jitsev, Eilif Muller, and Anne Warlaumont.

W2 Network neuroscience: structure and dynamics

Salle du Conseil, Wed & Thu, 9:00-17:30

Michele Giugliano, University of Antwerp, Antwerp, Belgium
Daniele Marinazzo, University of Ghent, Ghent, Belgium

This workshop will gather leading experts in the field of network neurosciences, to discuss how the information processed and stored by neural populations depends on their structure. The workshop specifically aims at discussing how one can efficiently enlighten the two-way road, linking structure and function. Through its 8 invited talks, the workshop focuses on five major key issues:

- Which connectivity measures and complex network properties are most appropriate to map function and behavior of groups of neurons, at multiple scales?
- Connectivity motifs have been identified as characteristic networks building blocks: do they find their raison d'être in structure itself, or in their capability to store and process information, or, as suggested by recent evidence, also in short-term dynamics of excitatory synaptic transmission?

- How can one distinguish and study the innate (intrinsic and unsupervised properties of neuronal circuits) and experience-related structure/function properties (i.e. learning and the interplay between dynamics and circuit rewiring/re-tuning)?

- Given the spiking history, how many neurons one needs to record to infer their connectivity, and other features of the network?

- What can one learn on neuron-network (micro-to-macro) interactions in both dynamics and structural aspects? To what extent structure and/or activity at the network and single neuron level constrain each other?

Invited Speakers:

Demian Battaglia, Max Planck For Dynamics and Self Organization, BCCN, Germany
Paolo Bonifazi, School of Physics and Astronomy, Tel Aviv University, Israel
Nicolas Brunel, Dept. Statistics, Neurobiology, Univ Chicago, USA
Michele Giugliano, Dept. Biomedical Sciences, Univ. Antwerpen, Belgium
John Hertz, Nordita, Stockholm, Sweden
Viktor Jirsa, System Neuroscience Institute, Unimed, Marseille, France
Asaf Gal, Network Biology Research Laboratories, Technion, Israel
Stefan Rotter, Bernstein Centre Freiburg, Germany

Contributed Talks:

J. Cortes (Ikerbasque-Biocruces)
B. de Sancristóbal (Universitat Pompeu Fabra)
M. Dhamala (Georgia State University)
G. Einevoll (Norwegian Univ. Life Sci.)
L. Gollo (Queensland Inst Med Res.)
R. Hindriks (Universitat Pompeu Fabra)
F. IsikKarahanoglu (EPFL & Univ. Geneva)
J. Lizier (CSIRO)
T. Mäki-Marttunen (Tampere Univ. Technol.)
J. Mejias (University of Ottawa)
D. Pinotsis (Univ. College London)
V. Priesemann (Max Planck Institute for Brain Research)
D. Van De Ville (EPFL & Univ. Geneva)

W3 Computations in the cerebellar circuit: advances on the modeling front

Space Nicolas, Wed & Thu, 9:00-17:30

Egidio d'Angelo, University of Pavia, Pavia, Italy
John Porrill, University of Sheffield, Sheffield, UK
Paul Dean, University of Sheffield, Sheffield, UK
Sergio Solinas, University of Pavia, Pavia, Italy

Investigation of the olivo-cerebellar system has attained a high level of sophistication, leading to the characterization of several structural and functional properties of neurons, synapses and circuits. Research has expanded and deepened in so many directions, and so many theories and models have been proposed, that an ensemble review of the matter is now needed. One major question, here as well as in the other brain circuits, is how single neuron and synaptic properties combine at the circuit level and contribute to circuit computations. A powerful tool for gaining insight into circuit functions is computational modeling. This workshop deals with the state of the art of single neuron and network models of the cerebellum, in order to stimulate future research. The major aspects that will be covered in this Workshop are:

- single cell modeling: granular layer, molecular layer, DCN, IO neurons
- cerebellar network models
- system models, closed-loop models, hybrid models
- robotic control
- theoretical model vs. computational models
- impact of single neuron properties on circuit computation

Invited Speakers:

Guy Billings

Claudia Casellato and Alessandra Pedrocchi: Brain-inspired robotic control: learning in cerebellum-driven movement tasks through a cerebellar realistic model

Egidio D'Angelo

Erik De Schutter

Jesús Garrido: Millisecond-scale regulation of spike timing by distributed synaptic plasticity at the cerebellum input stage

John Porrill

Thierry Nieus: Information transmission at the cerebellar granule cell

Eduardo Ros

Christian Rössert

Angus Silver

Sergio Solinas

Volker Steuber: Multiple computational roles of synaptic plasticity in the cerebellum

Patrick Van Der Smagt

Tadashi Yamazaki: Realtime Cerebellum: GPU-accelerated numerical simulation of a cerebellar spiking

W4 Methods of information theory in computational neuroscience

Space Curie, Wed & Thu, 9:00-17:30

Alexander G. Dimitrov, Washington State University, Vancouver, WA, USA
Michael Gastpar, EPFL, Lausanne, Switzerland
Conor Houghton, University of Bristol, Bristol, UK
Aurel A. Lazar, Columbia University, New York, NY, USA
Tatyana Sharpee, The Salk Institute, La Jolla, CA, USA
Simon R Schultz, Imperial College, London, UK

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.

Invited Speakers:
Dmitri B. Chklovskii, HHMI Janelia Farm, Ashburn, VA.
Pier Luigi Dragotti, Imperial College.
J. Leo van Hammen, Technical University of Munich
Lubomir Kostal, Academy of Sciences of the Czech Republic.
Simon Laughlin, Department of Zoology, University of Cambridge.
Aurel A. Lazar, Department of Electrical Engineering, Columbia University.
Jean-Pierre Nadal, Laboratoire de Physique Statistique, CNRS UMR8550, École Normale Supérieure, Paris.
Alex Pouget, University of Geneva.
Mark van Rossum, University of Edinburgh.
Simon R. Schultz, Department of Bioengineering, Imperial College.
Tatyana O. Sharpee, The Computational Neurobiology Laboratory, Salk Institute.
Lawrence C. Sincich, University of Alabama.
Naftali Tishby, Hebrew University.
Taro Toyoizumi, Riken Brain Sciences Institute.

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W5 Neural mechanisms of working memory limits

Space Grignard, Wed & Thu, 9:00-17:30

Albert Compte, IDIBAPS, Barcelona, Spain
Zachary Kilpatrick, University of Houston, Houston, TX, USA

Working memory can store multiple pieces of transitory information during a delay period. Each item of information can then be used in later neural computations. Electrode recording and fMRI studies have identified several brain areas that encode working memory with persistent activity or based on transient neural dynamics. Importantly, the process appears to have some fundamental limits. First, memory of a single cue degrades in accuracy as a function of the delay time. Second, memory accuracy of multiple objects is limited by the number of objects present. Several conceptual frameworks have been proposed to explain the nature of these limitations (slots vs. resource models). Only recently have the neural mechanisms responsible for working memory limits been explored computationally and neurophysiologically. This session will bring together experimentalists and theorists to discuss how the neural activity that serves working memory affects its accuracy and capacity.

Talks:

Ronald van den Berg, University of Cambridge; Variability in visual working memory

Jaejin Lee, University of Pittsburgh

Christian Machens, Champalimaud

Jonathan Wallis, University of California - Berkeley; Prefrontal mechanisms contributing to working memory

Paul Bays, University College London; Working memory capacity and allocation reflect noise in neural populations

Vladimir Itskov, Univerisity of Nebraska Associative memory encoding in bump attractor networks: switching between dual functions on the same network

Sophie Deneve, École Normale Supérieure; What are the real limits of working memory in balanced spiking networks?

Yuri Dabaghian, Baylor College of Medicine; A topological model of the hippocampal spatial map and spatial learning capacity

Gianluigi Mongillo, Universite Paris Descartes

Tim Buschman, Princeton University; Neural Dynamics of Working Memory Capacity Limits in Prefrontal and Parietal Cortex

Masud Husain, University of Oxford; Forgetting over seconds: attention and the role of the hippocampus

Yoram Burak, Hebrew University of Jerusalem; Fundamental limits on persistent activity in stochastic attractor networks

Rita Almeida, Karolinska Institute; A computational and experimental study of the relations between precision and capacity of visuo-spatial working-memory for several items
Zachary Kilpatrick, University of Houston; Optimizing working memory with heterogeneity of recurrent cortical excitation
Albert Compte, Institut d'investigacions Biomediques August Pi i Sunyer; Bump attractor dynamics in prefrontal cortex underlie behavioral precision in spatial working memory

W6 Methods of systems identification for studying information processing in sensory systems

Space Curie, Wed, 9:00-17:30

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

A functional characterization of an unknown system typically begins by making observations about the response of that system to input signals. The knowledge obtained from such observations can then be used to derive a quantitative model of the system in a process called system identification. The goal of system identification is to use a given input/output data set to derive a function that maps an arbitrary system input into an appropriate output.
In neurobiology, system identification has been applied to a variety of sensory systems, ranging from insects to vertebrates. Depending on the level of abstraction, the identified neural models vary from detailed mechanistic models to purely phenomenological models.
The workshop will provide a state of the art forum for discussing methods of system identification applied to the visual, auditory, olfactory and somatosensory systems in insects and vertebrates.
The lack of a deeper understanding of how sensory systems encode stimulus information has hindered the progress in understanding sensory signal processing in higher brain centers. Evaluations of various systems identification methods and a comparative analysis across insects and vertebrates may reveal common neural encoding principles and future research directions.
The workshop is targeted towards systems, computational and theoretical neuroscientists with interest in the representation and processing of stimuli in sensory systems in insects and vertebrates.

Invited Speakers:
Alex Borst, Max Planck Institute for Neurobiology, Martinsried.
Sonja Gruen, Forschungszentrum Juelich.
Vivek Jayaraman, Janelia Farm, Ashburn, VA.
Mikko I. Juusola, Department of Biomedical Science, University of Sheffield.
Holger G. Krapp, Department of Bioengineering, Imperial College.
Aurel A. Lazar, Department of Electrical Engineering, Columbia University.
Brian D. McCabe, Department of Pathology and Cell Biology, Columbia University.
Karim G. Oweiss, Department of Electrical and Computer Engineering and Department of Neuroscience, Michigan State University.
Jean-Pierre Rospars, INRA Versailles.

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W7 Early touch: from neural coding to haptic space geometry

Space Curie, Wed, 9:00-17:30

Jonathan Platkiewicz, Université Pierre et Marie Curie, Paris, France
Vincent Hayward, Université Pierre et Marie Curie, Paris, France

Research has produced many results regarding the perceptual capabilities of touch, and indiÂcations regarding its underlying mechanisms, but the computational nature of haptic (active touch) perception has been considered only recently. Inspired by research programs conÂducted in computational vision, the workshop will focused on questions related to 'early touch', which can be defined in a broad sense as 'inverse contact mechanics'. In classical contact mechanics, the problem is to determine the mechanical deformation of objects in contact. In touch, the nervous system is confronted with the inverse problem of recovering object attributes from the mechanical deformation induced by the object. Experts of differÂent backgrounds (neurophysiology, psychophysics, biomechanics, robotics, mathematics) will discuss problems such as the computational functions of skin mechanics and afferent neurons organization, the neural basis of texture perception, the notion of tactile saliency maps, and motor-sensory contingencies in haptic exploration.

Speakers:
Ehud Ahissar (Weizmann Institute of Science, Israel).
Angelo Arleo (Université Pierre et Marie Curie, France).
Sliman Bensmaia (University of Chicago, USA).
Vincent Hayward (Université Pierre et Marie Curie, France).
Jan Koenderink (Katholieke Universiteit Leuven, Belgium).
Masashi Nakatani (Columbia University Medical Center, USA / Keio University, Japan).
Tony Prescott (University of Sheffield, UK).
Andrew Pruszynski (University of Umeå, Sweden).
Junji Watanabe (NTT Communication Science Laboratories / Tokyo Institute of Technology, Japan).

W8 Dendrite function and wiring: experiments and theory

Space Grignard, Wed, 9:00-17:30

Hermann Cuntz, Goethe University, Frankfurt, Germany
Michiel Remme, Humboldt University, Berlin, Germany
Ben Torben-Nielsen, EPFL, Lausanne, Switzerland

Neuronal dendritic trees are complex structures that endow the cell with powerful computing capabilities and allow for high neural interconnectivity. Studying the function of dendritic structures has a long tradition in theoretical neuroscience, starting with the pioneering work by Wilfrid Rall in the 1950's. Recent advances in experimental techniques allow us to study dendrites with a new perspective and in

greater detail. For example, dendritic function can now be studied in awake, behaving animals. Also, owing to the precise characterization of neural circuits, the role of the single dendrite can be studied in the context of its connectivity. The goal of the workshop is to provide a resume of the state-of-the-art in experimental, computational and mathematical investigations into the functions of dendrites in a variety of neural systems.

Speakers:
Idan Segev (Hebrew University, Jerusalem) keynote
Matthew Larkum (Charité, Berlin) keynote
Bill Kath (Northwestern University, Evanston)
Yulia Timofeeva (University of Warwick)
Christoph Schmidt-Hieber (University College London)
Henrik Lindén (Royal Institute of Technology (KTH), Stockholm)
Srikanth Ramaswamy (Blue Brain Project, Lausanne)
Hermann Cuntz (Ernst Strüngmann Institute, Frankfurt)
Michiel Remme (Humboldt University, Berlin)
Ben Torben-Nielsen (Blue Brain Project, Lausanne)

W9 New developments in decoding the encoding of chemical senses

Space Curie, Wed, 9:00-17:30

Maxim Bazhenov, University of California, Riverside, CA, USA
Mark Stopfer, NIH, Bethesda, MD, USA

Olfactory and gustatory stimuli are vast in number and defy simple description. In contrast to light or sound, no low dimensional basis suffices to represent chemical stimuli. And yet, the olfactory and gustatory systems map the complex and high-dimensional world of chemical stimuli into unique and reproducible ensembles of neuronal activity. This mapping includes multilevel processing and involves complex strategies for information encoding. Recent developments in the field of chemical senses lead to a need to revisit existing theories and models of how information about chemical stimuli is represented in the brain. In this workshop we will discuss new findings that explain how chemical stimuli are encoded, and how different forms of neural plasticity can optimize sensory representations. The goal of this workshop is to bring together experimental and computational neuroscientists to discuss new developments in the well-characterized forms of sensory processing.

Speakers:
Stijn Cassenaer (Caltech)
Brian Smith (Arizona State University) - confirmed
Thomas Nowotny (University of Sussex) - confirmed
Alex Koulakov (CSHL) - confirmed
Jean-Pierre Rospars (INRA)
Mark Stopfer (NIH-NICHD) - confirmed
Maxim Bazhenov (University of California, Riverside) - confirmed
Rainer Friedrich (Friedrich Miescher Institute for Biomedical Research)
Dmitry Rinberg (NYU)

Nathan Urban (Carnegie Mellon)

W10 New approaches to spike train analysis and neuronal coding

Space Nicolas, Wed, 9:00-17:30

Conor Houghton, University of Bristol, Bristol, UK
Thomas Kreuz, ISC-CNR, Florence, Italy

Spike trains are central to signaling and computation in the brain; they are frequently the data collected in neuroscientific experiments and recent advances in electrophysiological techniques mean that they are now being collected across large neuronal populations and for synaptically connected neurons. As a consequence, describing and analyzing spike trains and quantifying their properties is a common challenge and one that is important in our efforts to understanding how the brain codes, integrates and processes information. Nonetheless, spike train analysis remains difficult and even immediate questions such as the degree to which spike trains carry a temporal or rate code are not only difficult to answer, they are difficult to ask in an unambiguous way. The purpose of this workshop is to discuss how different approaches, such as measures of spike train (dis)similarity and methods from information theory, can be used to define quantitative properties of neuronal signaling. Such properties could be used to analyze the large quantities of experimental data now available in a way that would help specify and address questions about neuronal coding and processing. Contributions will include experimental and theoretical studies, data analysis as well as modeling.

Speakers:
Ralph G. Andrzejak, Barcelona, Spain
Florian Mormann, Bonn, Germany
Il Memming Park, Austin, TX, USA
Friederice Pirschel, Oldenburg, Germany
Jose Principe, Gainesville, FL, USA
Rodrigo Quian Quiroga, Leicester, UK
Jonathan Victor, New York, NY, USA

W11 Metastable dynamics of neural ensembles

Space Grignard, Wed, 9:00-17:30

Gustavo Deco, University Pompeu Fabra, Barcelona, Spain
Emili Balaguer Ballester, Bournemouth University, Bournemouth, UK

Is the traditional view on brain activity dynamics, in which the cognitive flow of information wanders through multiple stable states driven by task–dependent inputs, still a robust model? This picture has been recently challenged both empirically and from the modelling perspective. In this workshop we will address a range of recent complementary views of cortical activity dynamics.
In several contemporary models, intrinsic activity fluctuations drive default transitions between metastable states shaped by anatomical connectivity, even in the absence of external stimuli. Noise enriches the dynamical repertoire of deterministic states; creating flexible "ghost" attractors which permit the

effective processing of task–related cognitive entities. Another proposed metaphor of transient brain dynamics consisted of successions of metastable saddle states; dynamical objects which are particularly reliable but form which neural activity eventually switches among them, even without the intervention of noise.
In this workshop we will have modelling and data analysis contributions which focus on metastable activity dynamics, analyses of non–stationary neural recordings and transient dynamics during cognitive processing. The next topics will be discussed:

- Attracting and transient dynamics of neural ensembles
- Non–stationary neural recordings and data analyses
- Cortical activity dynamics at resting state
- Transient dynamics during perception
- Travelling waves in cortex
- Cognitive processing dynamics.

Speakers:
Daniel Durstewitz: Choice-specific neural trajectories in a multiple-item working memory task
Pablo Varona: Models and novel experimental tools to address information processing in transient neural dynamics
Emili Balaguer-Ballester: Can we Identify Latent Non-Stationary Dynamics in Neural Populations?
Ruben Moreno–Bote: Poisson-like spiking and contrast invariant sampling in multi-stable networks with probabilistic synapses
Marcello Massimini: To be announced
Mavi Sanchez-Vives: Organization of emergent patterns of activity in control and altered cortical networks
Maurzio Matttia: Multiscale nonlinear dynamics of slow oscillations across cortical surface
Gustavo Deco: The relation between structural and functional connectivity in resting state

W12 Advances in neural mass modeling

Space Curie, Wed, 9:00-17:30

Ingo Bojak, University of Birmingham, Birmingham, UK
Stephan van Gils, University of Twente, Enschede, The Netherlands
Sid Visser, University of Twente, Enschede, The Netherlands

Neural mass and mean-field models describe the behavior of a neural network by considering the average behavior of large ensembles of neurons. This allows for a comparison with (macroscopic) experimental measurements such as EEG, fMRI and MEG. Since not all neurons are modeled individually, a distinct advantage of these lumped models is the reduction in dimensionality of both the parameter and variable space, which reduces computation time and makes possible the mathematical analysis of the model's behavior. Topics of ongoing research are, among others, the inclusion of additional neural mechanisms such as spike rate adaptation or bursting, investigating the effects of higher order statistics of the neural masses on the dynamics, the formal and/or computational correspondence between

microscopic and macroscopic models, adapting models for different regions of the brain, describing pathologies and drug effects, and the development of new analytic tools for these systems.
In this workshop we wish to bring together researchers in the field who are excited to discuss the latest advances in the construction, analysis and application of neural mass models.

Speakers:
Steven Schiff
John Terry
Viktor Jirsa
Olivier Faugeras
Stephan van Gils
Stefanos Folias
Sid Visser
Nicolas Brunel
Michael Breakspear
Ingo Bojak
Gustavo Deco
Dimitris Pinotsis
David Liley
Axel Hutt

W13 Spike-based computation

Space Nicolas, Thu, 9:00-17:30

Romain Brette, Ecole Normale Supérieure, Paris, France
Sophie Denève, Ecole Normale Supérieure, Paris, France

According to David Marr, computational systems can be analyzed at three levels: the computational level (what does the system do?), the algorithmic or representational level (how does it do it?) and the physical level (how is it physically realized?). Traditionally, in computational neuroscience, the algorithmic level is described in terms of firing rates, while the physical level is seen as a translation of these algorithms in terms of spikes. For example, spike trains may be produced as realizations of Poisson processes with the underlying firing rates. In other words, rate-based approaches postulate that the algorithmic and physical levels are independent. Spike-based approaches to neural computation have in common that they question the independence of these levels. Instead, algorithms and representations are defined at the level of spikes. The goal of this workshop is to present the various flavors of spike-based computation. It will consist of a series of short presentations covering recent research, with ample time for discussion. This will be a one-day workshop with a series of short talks and ample time for discussion.

Speakers:
Romain Brette, Sophie Deneve, Robert Gütig, Olivier Marre, Eugene Izhikevich, Lars Buesing, Walter Senn, Kenneth Harris

W14 Modeling general anesthesia: from theory to experiments

Space Curie, Thu, 9:00-17:30

Axel Hutt, INRIA, Nancy, France

The neural dynamics during general anesthesia is far from being understood. One reason for this lack of understanding is the complex neural interactions on different spatial and temporal scales. For instance, anesthetic agents act on synaptic receptors essentially evoking a macroscopic change of population activity, such as Local Field Potentials, EEG/MEG or resulting change of cerebral blood flow, and inducing the loss of consciousness in patients. The workshop aims to address recent theoretical and experimental advances in the field and provides a forum for an improved exchange of ideas between different research groups.

Speakers:
Jamie Sleigh, University of Auckland: An explanation for the variation in trajectories during emergence from general anaesthesia
Alain Destexhe, CNRS Paris: Excitatory and inhibitory cell activity in human temporal cortex during wake and sleep states
Anthony Hudetz, Medical College of Wisconsin: Anesthesia shrinks the repertoire of brain states as estimated from electrophysiological and fMRI data
David Liley, Swinburne University of Technology: Mean field modelling of burst suppression in general anaesthesia
Axel Hutt, INRIA Nancy: Effect of synaptic and extra-synaptic GABAergic inhibition on neural population dynamics
Kristin Sellers, University of North Carolina - Chapel Hill: Anesthesia amplifies visual responses through cortical disconnectivity and suppression of spontaneous state dynamics
Alistair Steyn-Ross, University of Wakato: Investigation of inhibitory mechanisms in a mean-field anaesthesia model
UnCheol Lee, University of Michigan: The inhibition of feedback connectivity after anesthetic induced unconsciousness associates with reconfiguration of brain network hub structure: study with empirical EEG data and mathematical modeling.
Rosalyn Moran, Virginia Tech Carilion School of Medicine and Research Institute: tba.
Ingo Bojak, University of Reading: HCN1-mediated interactions of ketamine with propofol and the EEG.

W15 Recent advances in experimental and computational characterization of neural assemblies

Space Grignard, Thu, 9:00-17:30

Adrien Peyrache, New York University Medical Center, New York, NY, USA
Sami El Boustani, MIT, Cambridge, MA, USA

The functional organization of neuronal activity that gives rise to perception, memory storage and,

more generally, every cognitive process remains one of the greatest mysteries of neuroscience. It has been suggested that internal representations, either evoked or recalled, would take the form of a transient coordination between subsets of neurons, the so-called "cell assembly". Some of the proposed mechanisms have awaited experimental confirmations for decades until the recent development of experimental methodologies such as multi-electrode recordings, two-photon imaging or optogenetics. These techniques offer unprecedented opportunities to monitor and interrogate large neural circuits. Existing models are constantly challenged by new insights. These include cell type heterogeneity, the complexity of neuronal oscillations, and the modulation of correlation structures during ongoing states. This workshop aims at confronting the most recent experimental challenges to computational models describing the emergence and functional properties of neuronal ensembles as well as opening a new avenue to theoretical developments unifying our understanding of perception, memory and motor control.

Speakers:
Brice Bathellier (Institute of Molecular Pathology, Vienna): Prediction of behavioral sound categorization by discrete neocortical population dynamics
Yves Fréegnac (CNRS, Gif-sur-Yvette): tba
Aleena Garner (Allen Institute, Seattle): Characterizing cell assemblies in visual cortex
Kenneth Harris (Imperial College, London): Organization of neuronal assemblies in auditory cortex
Rodrigo Quiroga (University of Leicester, Leicester): Concept Cells
Germán Sumbre (ENS, Paris): Ongoing spontaneous activity dynamics of large neural networks in zebrafish larvae
Gassper Tkacik (IST Austria, Klosterneuburg): Recent progress in understanding the retinal neural code
Tim Vogels (EPFL, Lausanne): tba

W16 Computational properties of inhibitory synapses

Space Curie, Thu, 9:00-17:30

Fidel Santamaria, University of Texas San Antonio, San Antonio, TX, USA

Inhibitory synapses not only regulate the propagation of activity throughout the brain, but also are an important element in the processing of information in single cells. Over the last few years, it has become evident that inhibitory synapses are not static; rather, their strength depends on short term and long term ionic changes. Thus, it is important to understand how temporally varying activation of inhibitory synapses might affect the spread of information throughout the cerebellar cortex. This workshop will focus on the biophysical properties of inhibitory synapses, their synaptic and ionic plasticity and the diverse computational contribution to synaptic integration and processing.

Speakers:
Thomas Kuner, Heidelberg: Clomeleon
Peter Jedlicka, Frankfurt: Modeling GABAergic current plasticity Chloride metabolism
Erik De Schutter, Okinawa
Boris Gutkin, Paris
Alain Marty, Paris
Fidel Santamaria, San Antonio: Chloride concentrations across the Purkinje cell dendrite

W17 Functional role of correlations: theory and experiments

Space Curie, Thu, 9:00-17:30

Tatjana Tchumatchenko, Columbia University, New York, NY, USA
Srdan Ostojic, Ecole Normale Supérieure, Paris, France

Understanding neural correlations has been the topic of intense activity in the recent years. While great progress has been made in unraveling the mechanistic origins of correlations in neural networks, their role in neural computations and plasticity is still not clear. The purpose of this workshop is to bring together theorists and experimentalists working on that topic in order to review recent progress and generate ideas for future work. The program will consist of 30-40 minutes talks and a round table discussion. There will also be ample time for informal discussions.

Speakers:
Valentin Dragoi (Houston U)
Alex Ecker (Tuebingen)
Michael Graupner (New York U)
Ilan Lampl (Weizman)
Peter Latham (UCL)
Alex Pouget (Geneva)
Jaime de la Rocha (Barcelona)
Robert Rosenbaum (Pittsburgh)
Stefan Rotter (Freiburg)

W18 Relevance of synaptic plasticity for multistable behaviour in neural systems

Space Grignard, Thu, 9:00-17:30

Alessandro Torcini, Institute of Complex Systems, Florence, Italy
Christian Hauptmann, Research Center Juelich, Juelich, Germany

Fluctuating spontaneous activity has been observed in several areas of the brain. In particular, irregular oscillations among more synchronized and less synchronized states appear in the hyppocampus during slow-wave sleep and quiet wakefulness, due to the massive endogenous activation of large neuronal populations. Furthermore, abnormal synchronization processes characterize several neurological diseases. For instance, under healthy conditions particular neuronal populations located in the thalamus and the basal ganglia fire in an uncorrelated manner. In contrast, abnormal synchronization of these neuronal populations causes Parkinsonian resting tremor.
Recent computational studies have revealed that synaptic plasticity is a fundamental ingredient to ensure multistability in neuronal circuits. In particular, spike timing dependent plasticity (STDP) appears to play a crucial role in promoting the coexistence of states with different level of synchrony. These studies can be extremely useful for the understanding of mechanisms of memory consolidation in the neocortex as well as for the development of new Deep Brain stimulation techniques.

This workshop aims to provide a forum to discuss the relevance of plasticity for the emergence of multistable dynamical behaviours in neuronal populations . The main focus of the workshop will be to understand the relevance of novel numerical findings in the field of computational neuroscience followed by a frank and open discussion with experimental neuroscientists.
The workshop will be organized in maximum 10/12 presentations of 25 minutes each plus 5 minutes for questions/discussion. A final session of 30 minutes is planned for an ample discussion. We plan to leave three/four slots empty in order to allow the participants of CNS*2013 to apply for an oral presentation at this workshop.

Speakers:
1) Maxim Bashenov (URC, USA)
2) Sylvia Daun-Gruhn (University of Cologne, Cologne, Germany)
3) Rowshanak Hashemiyoon (University of Pittsburgh, USA)
4) Stefano Luccioli (ISC-CNR, Firenze, Italy)
5) Gianluigi Mongillo (Descartes University, CNRS, Paris, France)
6) Oleksandr Popovych (Research Center Juelich. Juelich, Germany)
7) Peter Tass (Research Center Juelich. Juelich, Germany)

W19 Full brain network dynamics - modeling, analyses, experiments

Space Grignard, Thu, 9:00-17:30

Victor Jirsa, Institut de Neurosciences des Systèmes INSERM, Marseilles, France
Gustavo Deco, University Pompeu Fabra, Barcelona, Spain

Over the last ten years the information on large scale connectivity composed of the white matter fibers, called the Connectome, has become widely available and lead to many new investigations regarding the theory and modelling, as well as analyses and experimentation of full brain networks. Such brain networks pose novel challenges to theory and computation, since their architecture poses novel constraints upon neuroinformatics platforms (see for instance thevirtualbrain.org). In particular, they are characterized by a complex connectivity matrix, time delays via signal propagation along their many connecting fibers with neural mass models at their network nodes. Even at rest, the full brain networks do not dwell at equilibrium, but show a rich spontaneous dynamics with intermittent coherent fluctuations and characteristic spatial patterns. These spatiotemporal dynamics and its associated cognitive processes are perturbed during brain diseases and disorders such as schizophrenia, autism and epilepsy. For these reasons the resting state dynamics offers itself as an exciting entry point to the study of cognition through the investigation of their associated brain processes in full brain networks. In this workshop we gather researchers and experts in the field discussing the various aspects and challenges related to full brain network dynamics from diverse perspectives including theory and experiments. Each speaker has been allotted a time slot of 45 minutes (30 minutes presentation and 15 minutes discussion), which allows ample time for discussion.

Speakers:
Randy McIntosh, Toronto
Petra Ritter, Berlin
Jean Daunizeau, Paris
Viktor Jirsa, Marseille

Morten Kringelbach, Oxford
Maxime Guye, Marseille
Michael Breakspear, Brisbane
Gustavo Deco, Barcelona

W20 Validating neuro-computational models of neurological and psychiatric disorders

Space Curie, Thu, 9:00-17:30

Basabdatta Sen Bhattacharya, University of Lincoln, Lincoln, UK
Fahmida Chowdhury, NSF, Arlington, VA, USA

Recent years have seen a widespread interest in applying computational models to underpin the neural correlates in neurological and psychiatric disorders, which is essential for drug discovery, disease prediction and better diagnostics. Neuro-computational models are abstractions of highly complex biological circuitry and/or phenomena at a level appropriate to the modeller's target 'problem'. An essential condition for models simulating real world phenomena to be 'usable' is to validate them in order to avoid erroneous understanding and potentially conflicting predictions. In other words, a model can be deemed useful as a tool to aid the understanding and treatment of disease conditions only if it is validated with experimental data. Currently, there is a rich repertoire of computational models, mimicking the functionalities and behaviour of various brain parts. However, the immense diversity in modelling and validation approaches across the globe makes it difficult to compare results, even for similar brain functionality. In addition, validation techniques as well as the experimental data used for validation are not 'homogeneous'. Moreover, being a multidisciplinary field, a structured and co-ordinated approach to benchmark and/or set standards for validation methods and techniques is yet to be initiated. The aim of this workshop is to bring together Engineers and Scientists who work on modelling brain behaviour to discuss

- Potential methods of meaningful validation of neuro-computational models with experimental data.
- Ways of benchmarking validation of different modelling approaches towards a given goal (e.g. modelling anomalies in EEG), so that different models may be compared in meaningful ways.
- Potential new collaborations and leverage existing ones to advance the field.

The expected outcome is a report or white paper written by the group (including the speakers and any interested persons from the audience) to present their findings and a few action items.

Speakers:
Dr. Piotr Suffczynski, Associate Professor, University of Warsaw, Poland
Dr. Romain Brette, Associate Professor, Ecole Normale Superieure, Paris, France
Dr. Claudio Babiloni, Associate Professor, University of Rome "La Sapienza", Rome, Italy
Prof. Ingo Bojak, University of Reading, UK
Dr. Rosalyn Moran, Assistant Professor, Virginia Tech Carilion Research Institute, USA
Dr. Dimitrios Pinotsis, UCL, UK
Dr. Udo Ernst, Institute for Theoretical Physics, University of Bremen, Germany

Dr. Andre Marreiros, Max Planck Institute of Biological Cybernetics, Tubingen, Germany

W21 Postdoc and student career strategy workshop

Space Curie, Thu, 18:00-20:00

Jorge Mejias, University of Ottawa, Ottawa, Ontario, Canada

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

Confirmed Panel Members:
Gary Marsat (West Virginia University, USA)
Daniele Marinazzo (University of Ghent, Belgium)
Astrid Prinz (Emory University, USA)
Walter Senn (University of Bern, Switzerland)
Volker Steuber (University of Hertfordshire, UK)

Posters

Posters

Sunday Posters
Posters P1 – P145

P1 Reduction in inhibitory control is sufficient to generate hyperalgesia in a spiking model of nociceptive integration in the superficial dorsal horn

Mafalda Sousa[1,2⋆], Peter Szucs[1,2], and Paulo C Aguiar[2,3]

[1] *Faculdade de Medicina da Universidade Porto, Porto 4200 -319, Portugal*
[2] *Instituto de Biologia Molecular Celular, Porto 4150-180, Portugal*
[3] *Centro de Matemática da Universidade Porto, Porto 4169-007, Portugal*

P2 A model for grid cells where spatially correlated place cells compete for the grid map nodes

Maria Luisa Castro Guedes[1,2⋆], Paulo C Aguiar[1,2]

[1] *Departamento de Matemática, Faculdade de Ciências da Universidade do Porto, Porto, Portugal*
[2] *Centro de Matemática da Universidade do Porto, Porto, Portugal*

P3 Conversion from spatial patterns of activity to sequences of neuronal activations using gate interneurons

Eduardo Conde-Sousa[1,2⋆], Paulo C Aguiar[1,2]

[1] *Faculty of Sciences, University of Porto, Porto, Portugal*
[2] *Center for Mathematics of University of Porto, Porto, Portugal*

P4 Reorganization of effective network structure with dynamic synapses in cortical circuit and its possible functions

Yuichi Katori[1,2⋆], Kazuhiro Sakamoto[3], Hajime Mushiake[4], and Kazuyuki Aihara[2]

[1] *FIRST, Aihara Innovative Mathematical Modelling Project, JST,*
[2] *Institute of Industrial Science, The University of Tokyo,*
[3] *Research Institute of Electrical Communication, Tohoku University,*
[4] *Department of Physiology, Tohoku University School of Medicine.*

P5 Oscillatory hierarchy in a network of leaky integrate-and-fire neurons with short-term plasticity

Timothee Leleu⋆, Kazuyuki Aihara

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

P6 Impact of the Konio pathway in the thalamocortical visual system: a modeling study

Carlos Carvajal[1,2,3⋆], Thierry Vieville[1], and Frédéric Alexandre[1,2]

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[2] *LaBRI, Université de Bordeaux, Institut des Maladies Neurodégénératives, 33000 Bordeaux, France*
[3] *Université de Lorraine, LORIA UMR 7503, 54600 Villers-lès-Nancy, France*

P7 Neuronal nonlinearity explains greater visual spatial resolution for dark than for light stimuli

Jens Kremkow⋆, Jianzhong Jin, Stanley J Komban, Yushi Wang, Reza Lashgari, Michael Jansen, Xiaobing Li, Qasim Zaidi, and Jose-Manuel Alonso

Graduate Center for Vision Research, State University of New York College of Optometry, NY, 10036, USA

P8 Modeling intracellular silent oscillations and rhythmic discharge in olfactory bulb mitral cells.

Nicolas Fourcaud-Trocme[1,2⋆], Virginie Briffaud[1,2], and Corine Amat[1,2]

[1] *INSERM, U1028; CNRS, UMR5292; Lyon Neuroscience Research Center, 'Olfaction: from coding to memory' Team, Lyon, F-69000, France*
[2] *University Lyon 1, Villeurbanne, F-69000, France*

P9 Optimal pair of hippocampal CA1 phase response curve and spike-timing-dependent plasticity

Ryota Miyata[1,2⋆], Keisuke Ota[3], and Toru Aonishi[1]

[1] *Interdisciplinary Graduate School of Science, Tokyo Institute of Technology, Kanagawa, 226-8502, Japan*
[2] *Research Fellow of the Japan Society for the Promotion of Science, Tokyo, Japan*
[3] *Brain Science Institute, RIKEN, Saitama, 351-0198, Japan*

P10 Modulation of a decision-making process by spatiotemporal spike patterns decoding

Laureline Logiaco[1,4⋆], René Quilodran[2], Wulfram Gerstner[3], Emmanuel Procyk[4], and Angelo Arleo[1]

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P11 The role of inhibition in the generation of reliable spike sequences

Collins Assisi*

Indian Institute of Science Education and Research, Pune, India, 411007

P12 – Withdrawn –

P13 Abatement of epileptic spike-wave discharges through single pulse stimulation

Peter Taylor[1*], Yujiang Wang[2], Justin Dauwels[1], and Gerold Baier[3]

[1] *School of Electrical & Electronic Engineering, Nanyang Technological University. Singapore.*
[2] *Manchester Interdisciplinary Biocentre, University of Manchester, UK.*
[3] *Centre for Organismal Studies, University of Heidelberg, Germany.*

P14 Computational modelling of micro-seizures and focal seizure onset

Yujiang Wang[1*], Peter Taylor[2], and Gerold Baier[3]

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[2] *School of Electrical & Electronic Engineering, Nanyang Technological University, Singapore*
[3] *BioQuant, Centre of Organism Studies, University of Heidelberg, Germany*

P15 Identifying Sources of Non-Stationary Neural Ensemble Dynamics

Emili Balaguer-Ballester[1,2*], Hamid Bouchachia[1], and Christopher C Lapish[3]

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[2] *Bernstein Center for Computational Neuroscience, ZI Mannheim-University of Heidelberg, Mannheim, J5.*
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P16 Natural scene statistics relate to perceptual salience of second-, third-, and fourth-order spatial correlations

Ann Hermundstad[1*], John Briguglio[1], Mary Conte[2], Jonathan Victor[2], Gasper Tkacik[3], and Vijay Balasubramanian[1]

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[2] *Weill Cornell Medical College, New York, NY 10065, USA*
[3] *Institute of Science and Technology Austria, Klosterneuburg, Austria*

P17 Characterizing brain states with Granger causality

Adam B Barrett[1*], Lionel Barnett[1], Paul Chorley[1], Andrea Pigorini[2], Lino Nobili[3], Melanie Boly[4], Marie-Aurelie Bruno[4], Quentin Noirhomme[4], Steven Laureys[4], Marcello Massimini[2], and Anil K Seth[1]

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[4] *Cyclotron Research Centre, Dept. of Neurology, University of Liege, Liege, B30-4031, Belgium*

P18 Combined study of time-series bifurcation and power spectral behaviour of a thalamo-cortico-thalamic neural mass model

Basabdatta Sen-Bhattacharya*

School of Engineering, University of Lincoln, Lincoln, LN6 7TS, UK

P19 Foundations for an Ontology of Brain areas, circuits and functions

Bénédicte M Batrancourt*

CRICM UPMC/INSERM UMR_S975, Paris, 75013, France

P20 Non-multiplicative attentional modulation patterns in area MT

Markus Helmer[1,3*], Vladislav Kozyrev[2,4], Anja Lochte[2,3], Stefan Treue[2,3], Theo Geisel[1,3], and Demian Battaglia[1,3]

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[3] *Bernstein Center for Computational Neuroscience, Göttingen, Germany*
[4] *Institute of Neuroinformatics, Ruhr-University, Bochum, Germany*

P21 Developing orientation maps using realistic patterns of lateral connectivity

Philipp Rudiger[1*], Judith S. Law[1], Jan Antolik[1,2], and James A Bednar[1]

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[2] *Unit of Information and Complexity (UNIC), CNRS, Gif-sur-Yvette, 91198, France*

P22 An automated simulator-independent workflow for reproducible simulation and analysis using Lancet and IPython Notebook

Jean-Luc Stevens[1*], Marco Elver[2], and James A Bednar[1]

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[2] *Institute for Computing Systems Architecture, University of Edinburgh, EH8 9AB, UK*

P23 A modeling study of cortical waves in primary auditory cortex

David Beeman*

Department of Electrical, Computer, and Energy Engineering, University of Colorado, Boulder, CO 80309, USA

P24 Investigating the interplay between spontaneous and evoked activities in cultured neuronal networks by dimensional reduction techniques

Thierry Nieus*, Alessandro Maccione, and Luca Berdondini

Istituto Italiano di Tecnologia (IIT), Neuroscience and Brain Technology Department – Genoa, Italy

P25 Estimating the fraction of falsely detected spikes in high density microelectrode array recordings based on correlations

Oliver Muthmann[1,2⋆], Hayder Amin[3], Alessandro Maccione[3], Evelyne Sernagor[4], Luca Berdondini[3], Matthias Hennig[2], and Upinder Bhalla[1]

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P26 Application of neural mass models to major depressive disorder

Natalia Bielczyk⋆

Donders Centre for Neuroscience, Radboud University, Nijmegen, 6525AJ, the Netherlands

P27 Intrinsic and network mechanisms involved in balanced firing and striatal synchrony during dopamine depletion

Sriraman Damodaran, Kim Avrama Blackwell⋆

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

P28 Biologically Plausible Reinforcement Learning of Continuous Actions

Jaldert Rombouts[1⋆], Pieter Roelfsema[2,3,4], and Sander Bohte[1]

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P29 Modeling Spatio-temporal Effects of Propofol Using a Neural Field Approach

Manh Nguyen Trong[1,2], Thomas Knösche[1⋆], and Ingo Bojak[3,4]

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P30 – Withdrawn –

P31 Influence of Intrinsic and Synaptic Properties on Transmission of Spike Timing Precision

Heather A Brooks⋆, Alla Borisyuk

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

P32 Neural oscillations arising from a linear current with negative conductance

Farzan Nadim[1,2⋆], Yinzheng Guan[1], Jorge Golowasch[1,2], and Amitabha Bose[2]

[1] *Department of Biological Sciences, NJIT-Rutgers University, Newark, NJ 07102, USA*
[2] *Department of Mathematical Sciences, NJIT, Newark, NJ 07102, USA*

P33 Interoperability in the GENESIS 3.0 Software Federation: the NEURON Simulator as an Example

Hugo Cornelis[1], Dimitris Bampasakis[3⋆], Volker Steuber[3], and James Bower[1,2]

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P34 A new method for detecting deception in Event Related Potentials using individual-specific weight templates

Abdulmajeed Alsufyani[1,2⋆], Alexia Zoumpoulaki[1], Marco Filetti[1], and Howard Bowman[1]

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P35 Using spike train distances to identify the most discriminative neuronal subpopulation

Thomas Kreuz⋆, Nebojsa Bozanic

Institute for Complex Systems, CNR, Sesto Fiorentino, Italy

P36 Scale-free dynamics in human neonatal cortex following perinatal hypoxia

James Roberts[1], Kartik Iyer[1,2], Simon Finnigan[2], Sampsa Vanhatalo[2,3], and Michael Breakspear[1⋆]

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[3] *Department of Children's Clinical Neurophysiology, Helsinki University Central Hospital and University of Helsinki, Helsinki, Finland*

P37 Zero-Lag Synchronization in Cortical Motifs

Leonardo Gollo[1,2], Claudio Mirasso[1], Olaf Sporns[3], and Michael Breakspear[2,4,5⋆]

[1] *IFISC, Instituto de Física Interdisciplinar y Sistemas Complejos (CSIC-UIB), Palma de Mallorca, Spain*
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[4] *School of Psychiatry, University of New South Wales and The Black Dog Institute, Sydney, NSW, Australia*
[5] *The Royal Brisbane and Woman's Hospital, Brisbane, QLD, Australia*

P38 Brian 2 – the second coming: spiking neural network simulation in Python with code generation

Marcel Stimberg[1,2⋆], Dan Goodman[3,4], Victor Benichoux[1,2], and Romain Brette[1,2]

[1] *Institut d'Études Cognitives, École Normale Supérieure, Paris, 75005, France*
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P39 A unifying theory of ITD-based sound azimuth localization at the behavioral and neural levels

Victor Benichoux[1,2⋆], Marcel Stimberg[1,2], Bertrand Fontaine[3], and Romain Brette[1,2]

[1] *Equipe Audition, Département d'Etudes Cognitives, Ecole Normale Supérieure, Paris, 75005, France*
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[3] *Dominick P. Purpura Department of Neuroscience, Albert Einstein College of Medicine, Bronx, New York, USA*

P40 An ecological approach to neural computation

Romain Brette[1,2⋆]

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[2] *Laboratoire de Psychologie de la Perception, CNRS and Université Paris Descartes, Paris, 75006, France*

P41 Input dependence of Local Field Potential Spectra: Experiment versus Theory

Francesca Barbieri[1⋆], Alberto Mazzoni[2], Nikos K Logothetis[3], Stefano Panzeri[4], and Nicolas Brunel[5]

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P42 Memory maintenance in calcium-based plastic synapses in the presence of background activity

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P43 Memory capacity of networks with stochastic binary synapses

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P44 What is all the noise about interval timing?

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P45 The influence of network structure on neuronal dynamics

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P46 Microsecond precision of interaural time differences processing in the medial superior olive studied by a computational model

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P47 Neurodynamics of Epilepsy: Synaptic regulation and reversal potential dynamics during seizures in a neural field model with conductance-based synapses

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P48 Requirements for the Robust Operant Conditioning of Neural Firing Rates

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P49 Spatial Shaping of Neural Activity Using Electrical Stimulation

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P50 Perturbations can distinguish underlying dynamics in phase-locked two-neuron networks

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P51 Once more unto the leech: Production of functional motor patterns in leech heart motor neurons

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P52 Self-Organizing 'Harmonic Dominance Stripes' in a Spiking Network Model of the Auditory System

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P53 FitzHugh-Nagumo to Model a Large Number of Diffusive Coupled Neurons

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P54 The emergence of spontaneous activity in neuronal cultures, coherence from noise

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P55 Neuromechanical model of reflexes and locomotor rhythms in the crayfish leg

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P56 Beyond the frontiers of neuronal types: fuzzy classification of interneurons

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P57 EnaS: a new software for neural population analysis in large scale spiking networks

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P58 Dynamics and spike trains statistics in conductance-based Integrate-and-Fire neural networks with chemical and electric synapses.

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P59 A maximum likelihood estimator of neural network synaptic weights

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P60 Beyond dynamical mean-field theory of neural networks

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P61 Neural correlations code for stimulus variance

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P62 Parameter optimization of logistic regression classifiers

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P63 Toward the Drosophila connectome: Structural analysis of the brain network

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P64 Leader neurons drive spontaneous and evoked activation patterns in cortical networks

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P65 Multi-modal novelty and familiarity detection

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P66 A Biophysical Model of Endocannabinoid-Mediated Short Term Depression of Excitation in Hippocampus

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P67 Dynamics of the thalamo-cortical system driven by pulsed sensory stimulation

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P68 A population model of the thalamo-cortical system during deep sleep.

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P69 Exploring minimal models of sensory integration in nematode C. elegans

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P70 Numerical continuation of travelling waves and pulses in neural fields

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P71 The interplay between STDP rules and anticipated synchronization in the organization of neuronal networks

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P72 The behavior of the electric potential across neuronal membranes of spinal ganglion and neuroblastoma cells

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P73 A continuum approach to model neurites/dendrites with emerging subtrees

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P74 Designing Spiking Neural Models of Neurophysiological Recordings using Gene Expression Programming

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P75 Modeling of sodium currents from mesencephalic trigeminal neurons by system identification and sensitivity analysis

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P76 Cellular mechanisms of phase maintenance in the pyloric motif

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P77 Protective role of the half-center oscillator connectivity against external perturbations

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P78 The effects of time delays on synchronization properties in a network of neural mass models

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P79 The mechanisms of late-onset synaptic responses in a realistic model of Unipolar Brush Cells

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P80 The intrinsic and synaptic responsiveness of a new realistic Purkinje cell model

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P81 Distributed synaptic plasticity controls spike-timing: predictions from a cerebellar computational model

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P82 Ensemble neuronal responses in a large-scale realistic model of the cerebellar cortex

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P83 Disruption of transfer entropy and inter-hemispheric brain functional connectivity in patients with disorder of consciousness

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P84 Multistability in large scale models of brain activity

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P85 Computational model of midbrain dopaminergic neuron activity in ageing and obesity

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P86 A neuro-mechanical model containing fast and slow muscle fibres applied to mimic stop and re-start of a stepping leg

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P87 Epileptic spike-wave discharges in a spatially extended thalamocortical model

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P88 Characterising the performance of balanced memory networks

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P89 Recurrence of spatio-temporal patterns of spikes and neural avalanches at the critical point of a non-equilibrium phase transition

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P90 Modeling large populations of spiking neurons with a universal population density solver

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P91 Saccade angle modulates correlation between the local field potential and cerebellar Purkinje neuron activity

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P92 Initial state of single spines affects probability of induction of cerebellar long term depression in a stochastic model

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P93 Exploring the limitations of simulator independence via an implementation of a biophysically detailed cerebellar cortex model in NEURON and NEST

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P94 Challenges of declarative modeling of conductance-based neurons in diverse simulation environments

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P95 Dendritic Volume Mesh Reconstruction for STEPS: How Does Mesh Quality Affect Stochastic Reaction-Diffusion Simulation?

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P96 A biophysical model of cerebellar molecular layer interneuron

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P97 Fast functional imaging of multiple brain regions in intact zebrafish larvae using Selective Plane Illumination Microscopy

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P98 Role of anatomical pathways in shaping posterior alpha oscillations in the resting human brain

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P99 Modeling Alpha-Band Functional Connectivity for MEG Resting State Data: Oscillations and Delays in a Spiking Neuron Model

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P100 Disrupted connectivity in schizophrenia: modelling the impact of structural connectivity changes on the dynamics of spontaneous functional networks.

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P101 Mathematical modeling for resting state functional connectivity of cortical and sub-cortical networks

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P102 A model of perceptual discrimination under sequential sensory evidence

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P103 Sculpting dynamical systems for models of neural computation and memory

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P104 Functional interpretation of biophysical properties of spiking neurons

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P105 Estimating the transfer function of cortical neurons : from simple models to in vitro experiments

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P106 The electrical properties of extracellular media affect cable properties of neurons

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P107 Excitatory and inhibitory contributions to local field potentials in human and monkey

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P108 Shot Noise analysis of subthreshold membrane potential activity in neurons

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P109 Spike timing in rat somatosensory cortex contributes to behavior

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P110 Interactive visualization of brain-scale spiking activity

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P111 Integrating multi-scale data for a network model of macaque visual cortex

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P112 Influence of different types of downscaling on a cortical microcircuit model

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P113 Recurrence and external sources differentially shape network correlations

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P114 Behavioral state differentially regulates input sensitivity and firing rates of motor cortex pyramidal neurons

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P115 Coarse-graining of the dynamics seen in neural networks

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P116 A Computational Model of Prefrontal Cortex Based on Physiologically Derived Cellular Parameter Distributions

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P117 Neuronal Coding in the Rodent Prefrontal Cortex

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P118 Successful prediction of a physiological circuit with known connectivity from spiking activity

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P119 Hybrid scheme for modeling LFPs from spiking cortical network models

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P120 Modeling Extracellular Potentials in Microelectrode Array Recordings

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P121 Dynamics and lifetime of persistent activity states in random networks of spiking neurons with strong synapses

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P122 Electrodiffusive model for neuronal and astrocytic ion concentration dynamics

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P123 How pattern formation in ring networks of excitatory and inhibitory spiking neurons depends on the input current regime

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P124 Dynamics of neural systems in epilepsy

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P125 Interlaminar processing in auditory cortex before and after auditory trauma: spontaneous and evoked responses of independent sources

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P126 Phase transitions in cortical dynamics explain improved information processing under attention

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P127 Sparse somatosensory coding: towards explaining and predicting the response properties of rodent afferent pathway neurons

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P128 Likelihood representation in the owl's sound localization system

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P129 Neural computation with efficient population codes

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P130 A novel recovery algorithm of time encoded signals

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P131 Automated, in-vivo, whole-cell electrophysiology using an integrated patch-clamp amplifier

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P132 Predicting the firing phase of an oscillatory neuron from its impedance profile

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P133 Active inference, eye movements and oculomotor delays

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P134 Inhibitory STDP improves temporal processing in disynaptic circuits

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P135 Pattern separation by neuronal turnover in a feed-forward network

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P136 Scalability properties of multimodular networks with dynamic gating

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P137 Goal conditioning throw mutimodal categorisation in a simulation of rat navigation

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P138 Synaptic boutons sizes are tuned to best fit their physiological performances

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P139 Network reconstruction from calcium imaging data of spontaneously bursting neuronal activity

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P140 Synaptic synergies and their role in integrating distinct synaptic pathways

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P141 Visually guided behavior in freely moving mice

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P142 Optimal spike pattern v.s. noise separation by neurons equipped with STDP

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P143 Learning a sequence of motor responses to attain reward: a speed-accuracy trade-off

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P144 Which Temporal Difference Learning algorithm best reproduces dopamine activity in a multi-choice task?

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P145 Biomimetic stochastic race model in the subcortical saccadic selection processes: a model of the tecto-basal loops

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P146 A Computational Model of the Basal Ganglia Dual Pathways Organization

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P147 Functional Role and Implications of Population Heterogeneity on Vestibular Ocular Reflex Response Fidelity

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P148 Onset-inhibition in the auditory brainstem: A potential mechanism for signal enhancement of speech-like sounds

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P149 Pattern formation in a mean field model of electrocortical activity

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P150 Relating excess spike synchrony to LFP-locked firing rates modulations

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P151 Estimation of neural voltage traces and associated variables in uncertain models

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P152 The Smale Horseshoe Structure in the Firing Rate Model

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P153 Decision-making out of neural events: from discrimination information to psychometric power laws

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P154 Multi-scale modelling with spikes and rate codes: a demonstration in a model of the basal ganglia

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P155 Modulation of dopamine release by alpha7-type nicotinic acetylcholine receptors

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P156 DAergic Neuronal Dynamics: intrinsic properties, receptor dynamics, and network effects

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P157 Dynamic analysis of recurrent phase patterns in spontaneous human EEG

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P158 A Novel 3D Visualization Tool for Large-Scale Neural Networks

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P159 Supervised learning in spiking neurons

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P160 Semi-automatic spike sorting with high-count channel probes

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P161 An asymmetric model of the spinal locomotor central pattern generator: insights from afferent stimulations

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P162 Ideal-observer models of perceptual contrast enhancement

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P163 From laptops to supercomputers: A single highly scalable code base for spiking neuronal network simulations

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P164 Noise decouples covariances from interaction strength

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P165 Impact of inhibition in striatal decorrelation of cortical neuronal avalanches

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P166 Homeostasis in large networks of neurons through the Ising model – do higher order interactions matter?

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P167 Modelling homeostatic control of high-frequency post-synaptic transmission and its effect on metabolic efficiency in the auditory brainstem

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P168 Self-organized criticality in structured neural networks

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P169 Multi-unit response correlations along the tonotopic gradient and within isofrequency laminae of the inferior colliculus

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P170 A potential role for the cerebellar nuclei in absence seizures

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P171 Modeling brain functional connectivity at rest

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P172 A unifying perspective on neuromodulatory effects on signal transmission and plasticity in D1-dominant MSN neurons.

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P173 Single cell neuro-sensory dynamics: Ca2+ chemoreceptor-guided sea urchin sperm motility

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P174 Modelling cerebellar Purkinje cells with simple neuron models of the threshold type

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P175 Non-linear dependency between spiking response and gamma-band power of local field potentials in the human auditory cortex

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P176 Gain control network conditions: the role of the inhibition

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P177 A thalamo-cortical model to explain EEG during anaesthesia

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P178 Effects of tonic inhibition on a cortical neuronal population: implications for general anesthesia under propofol

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P179 Neural Field Simulator: fast computation and 3D-visualization

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P180 Theoretical model for neurovascular coupling via interactions of NO, EET, and 20-HETE

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P181 Prediction was predictable from human brain activity in fronto-parietal cortex

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P182 Pairwise correlation graphs from hippocampal population activity have highly non-random, low-dimensional clique topology

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P183 Emergence of Bottom-up Saliency in a Spiking Model of V1

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P184 Balanced excitation and inhibition in a spiking model of V1

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P185 A Spiking model of Superior Colliculus for Bottom-Up Saliency

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P186 Latency and rate coding in a spiking model of retina

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P187 Beyond inhibition: lateral modulation of plasticity of feedforward synapses in a spiking model of V1

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P188 Temporally evolving surround suppression helps decoding in a spiking model of motion processing

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P189 Self-tuning spike-timing dependent plasticity curves to simplify models and improve learning

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P190 Full-scale structural model of the Inferior Olive and Olivocerebellar projection constructed from constraining meshes and directed growth

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P191 Denervation-induced dendritic reorganization leads to changes in the electrotonic architecture of model dentate granule cells

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P192 The roles of short-term plasticity and synaptic weights in self-organized criticality

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P193 The Virtual Brain: a neuroinformatics platform for simulating large-scale brain network models.

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P194 Modeling epileptic dynamics in the hippocampus using a multiscale approach

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P195 Spatiotemporal dynamics in the human brain during rest: A virtual brain study

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P196 On the spatiotemporal dynamics and couplings across epileptogenic networks

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P197 Variability in brain network model dynamics: comparison of neural mass models and empirical connectivity datasets in the Virtual Brain

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P198 Accelerating the Virtual Brain with code generation and GPU computing

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P199 Functional role of opponent, dopamine modulated D1/D2 plasticity in reinforcement learning

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P200 On the influence of inhibitory STDP on balanced state random networks

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P201 Decoding motor intent from simulated multiple longitudinal intrafascicular electrode recordings

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P202 A flexible software tool for fitting the parameters of neuronal models

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P203 The metabolic cost of maintaining a synapse during development

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P204 Two generic mechanisms for emergence of direction selectivity coexist in recurrent neural networks.

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P205 Behavioral Effects of Disrupted Direct Pathway Signal Flow Caused by Dopamine Depletion

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P206 Coordination of phase precession through feed-forward topologies in the hippocampus

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P207 Phase sequences in a balanced recurrent network

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P208 Modeling ripple oscillations in the hippocampus

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P209 Integration of predictive-corrective incompressible SPH and Hodgkin-Huxley based models in the OpenWorm in silico model of c. elegans

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P210 Learning speech recognition from songbirds

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P211 Neural Population Coding of Movement Direction for Path Integration

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P212 Conductance estimation of a conductance-based neuron model by the differential evolution algorithm

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P213 Estimating synaptic connections from multiple spike trains based on a coupled escape rate model

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P214 Noise-induced anti-correlated slow fluctuations in networks of neural populations

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P215 Novel local learning rule for neural adaptation fits Hopfield memory networks efficiently and optimally

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P216 – Withdrawn –

P217 Information Transmission Efficiency in Neuronal Communication Systems

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P218 Dopamine-NMDA interactions and relevance to gamma band synchrony in schizophrenia

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P219 Maximum penalized likelihood estimation of interspike interval distribution

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P220 Long-term memory stabilized by noise-induced rehearsal

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P221 Information gain on variable neuronal firing rate

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P222 Removing bleaching artifacts from voltage sensitive dye recordings with ICA

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P223 Change Point Detection for Neuronal Data with CUSUM and Classification Methods

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P224 Coding of touch properties by three types of mechanosensory cells of the leech hirudo medicinalis

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P225 SPIKY: A graphical user interface for monitoring spike train synchrony

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P226 Synfire chains and gamma oscillations: two complementary modes of information transmission in cortical networks

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P227 Effect of single neuron firing patterns on network dynamics

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P228 Controlling the Go / No-Go decision threshold in the Striatum

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P229 Investigating dynamical properties of the Caenorhabditis elegans connectome through full-network simulations

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P230 A reaction-diffusion model of cholinergic retinal waves and self-organized criticality

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P231 Axonal injuries and consequences to neuronal computation

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P232 Towards a dynamic clamp for neuro-chemical modalities

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P233 Investigation Of Human Semantic Memory Organization By Hopfield Model: A Consideration From External Data Structure and Intrinsic Memory Organization

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P234 Periodically stimulated piecewise linear adaptive exponential integrate-andfire neuron

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P235 Latency of inhibitory response

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P236 Probabilistic Computation Underlying Sequence Learning in a Spiking Attractor Memory Network

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P237 Neuronal avalanches change from wakefulness to deep sleep – a study of intracranial depth recordings in humans

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P238 Towards a computational model of learning and social interactions of mice in IntelliCage

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P239 Interspike intervals under the constraint of linear synaptic integration and background synaptic activity

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P240 Where are the most informative neurons?

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P241 The influence of multiple firing events on the formation and stability of activity patterns in continuous attractor networks

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P242 Invariance of Visual Operations at the Level of Receptive Fields

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P243 Analytical results for integrate-and-fire neurons driven by dichotomous noise

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P244 Generalized synchronous output of neural populations – does it only encode fast stimulus components?

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P245 Mutual Information Density of Stochastic Integrate-and-Fire Models

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P246 Does noise shift or delete spikes?

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P247 On the effect of network structure and synaptic mechanisms on sustained bursting activity

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P248 Divisive and non-monotonic gain control in open-loop neural circuits

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P249 Learning to perform contrast-invariant cancellation of redundant stimuli

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P250 Positive sparse coding of natural images: a theory for simple cell tuning

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P251 Network oscillations in a neural mass model induced by metabolic modulation are consistent with EEG data of neocortical epileptic seizure onset

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P252 A Large Deviation Principle for Networks of Rate Neurons with Correlated Synaptic Weights

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P253 – Withdrawn –

P254 Motion control of thumb and index finger of an artificial hand for precision grip using asynchronous decoding of CM cell activity

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P255 Inferring network model from local field potentials

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P256 Neuronal chains as processing units of long scale wave information

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P257 The Hopfield-like neural network with governed ground state

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P258 How conductance distributions are shaped by activity-dependent regulation rules

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P259 The rectification of an electrical synapse can change the functional output of a pattern-generating circuit

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P260 Recovering directed networks in neuroimaging datasets using partially conditioned Granger causality

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P261 Maximizing the efficiency of decoding by selecting heterogeneous inputs from a population of electrosensory neurons responding to communication signal

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P262 V1 neurons can distinguish between motion in the world and visual displacements due to eye movements: a microsaccade study

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P263 Binary matrices and simplicial complexes: an algebraic-statistics tool to analyze co-activation of electrophysiological signals in cortical cultures

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P264 Concurrent scale-free and small-world networks support criticality in cortical ensembles

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P265 Modular versus uniform cultured neuronal networks: a modeling study

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P266 Inferring effective dynamics in large-scale networks of cortical neurons

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P267 Non-instantaneous synaptic transmission in spiking neuron networks and equivalence with delay distribution

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P268 Dynamical features of stimulus integration by interacting cortical columns

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P269 Calcium buffering as a mechanism of short-term synaptic plasticity

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P270 Likelihood-free Bayesian Analysis of Neural Network Models

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P271 Temporal sequence learning in reentrantly coupled winner-take-all networks of spiking neurons

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P272 Electrocutaneous stimulus setting for identification of the ascending nociceptive pathway

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P273 Computational optimum of recurrent neural circuits at intermediate numbers of nonlinear dendritic branches

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P274 Locomotor rhythm and pattern generating networks of the human lumbar spinal cord: an electrophysiological and computer modeling study

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P275 Anticipated Synchronization in Neuronal Motifs

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P276 Inferred Ising model unveils potentiation of pairwise neural interactions and replay of rule-learning related neural activity.

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P277 Repetition suppression/enhancement effects as a result of attractor dynamics in local cortical networks

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P278 Bayesian Inference from Single Spikes

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P279 Phase-of-firing coding of dynamical whisker stimuli and the thalamocortical code in barrel cortex

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P280 Trial by trial decoding of decisions in monkey MT cortex from small neuronal populations

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P281 Spontaneous population activity fluctuations boost sensory tuning curves and gate information processing

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P282 Effect of Alzheimer's disease on the dynamical and computational characteristics of recurrent neural networks

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P283 Syntax processing properties of generic cortical circuits

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P284 Aspects of randomness in biological neural graph structures

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P285 Oscillations and chaos in the dynamics of the BCM learning rule

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P286 Discriminating legitimate oscillations from broadband transients

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P287 – Withdrawn –

P288 Categorical perception in monkeys: modeling implicit learning of discrete categories

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P289 Membrane resonance of bursting neuron captured with an ICa/Ih model using multi-objective evolutionary algorithms

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P290 Classification of multivariate data with a spiking neural network on neuromorphic hardware

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Tuesday Posters
Posters P291 – P435

P291 Predicting deep-brain stimulation frequencies to suppress pathological population oscillations in a network model of Parkinson's disease

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P292 Designing Anti-Epileptic Drugs Using Neuronal Dynamics

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P293 Spontaneous Ca++ oscillations in astrocytes initiate high-frequency oscillations in model hippocampal network

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P294 Modeling the development of coarse-to-fine processing in the central visual pathway

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P295 Impact of noise on theta-nested gamma oscillations in a spiking continuous attractor model of grid cells

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P296 In Vivo Connection Imaging Revealed Distinct Feedforward and Intrinsic Neurons in Posterior Inferotemporal Cortex

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P297 A numerical renormalisation group method for the analysis of critical spreading activity in spiking neural networks

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P298 Changes in V1 orientation tuning when blocking astrocytic glutamate transporters: Models for extra- and intrasynaptic mechanisms

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P299 How adaptation currents and synaptic inhibition change threshold, gain and variability of neuronal spiking

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P300 Effects of neuronal adaptation currents on network-based spike rate oscillations

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P301 Predicting the location of the axon initial segment using spike waveform analysis: simulations of retinal ganglion cell physiology

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P302 Estimating invariant dimensions in V2

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P303 Multi-Chimera States in FitzHugh-Nagumo Oscillators

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P304 Natural firing patterns reduce sensitivity of synaptic plasticity to spike-timing

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P305 Phase Transfer Entropy: A novel measure for effective connectivity among neuronal oscillations

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P306 Assessing the role of synchronization and phase coherence in neural communication comparing cortical recordings and integrate-and-fire network models

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P307 How Noise Correlation Impact Population Code in Superior Colliculus: an Information Theoretic Approach

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P308 Long-term effects of weak electrical stimulation on active neuronal networks

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P309 Detection of neuronal signatures by means of data-driven tomography

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P310 A model for selective visual attention predicts information routing

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P311 Role of cell type and synaptic connections during functionally relevant network states in vitro

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P312 Sound envelope extraction in cochlear nucleus neurons: modulation filterbank and cellular mechanism

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P313 Trying hard not to listen: the evolution of information processing in vestibular hair cells

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P314 Motion based prediction and development of response to an "on the way" stimulus

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P315 Olfactory Receptor Neuron Coding in the Turbulent Realm

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P316 Bayesian entropy estimators for spike trains

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P317 Firing-rate models for neurons with a broad repertoire of spiking behaviors

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P318 EEG study of the neural representation and classification of semantic categories of animals vs tools in young and elderly participants

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P319 Dendritic nonlinearities enable PFC microcircuits to serve as predictive modules of persistent activity

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P320 Mathematical model of the thalamo-cortical loop by dysfunction in schizophrenia

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P321 Long-Term Potentiation Through Calcium-Mediated N-Cadherin Interaction is Tightly Controlled by the Three-Dimensional Architecture of the Synapse

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P322 Slow Sodium-Channel Inactivation Underlies Spike Threshold Variability

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P323 The structure of human olfactory space

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P324 A cortical theory of super-efficient probabilistic inference based on sparse distributed representations

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P325 Striatal ensembles continuously represent animals kinematics and limb movement dynamics during execution of a locomotor habit

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P326 A novel anxiety index for the rat behavior in the elevated plus-maze

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P327 Lateral inhibition and odor discrimination in a model of the olfactory bulb

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P328 CPU-GPU hybrid platform for efficient spiking neural-network simulation

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P329 Connection Control Implications in a Distributed Plasticity Cerebellar Model

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P330 Linking neural mass signals and spike train statistics through point process and linear systems theory

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P331 Nonlinear Dynamics of Large-Scale Activity in "Networks of Networks"

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P332 Going Beyond Poisson Processes: A New Statistical Framework in Neuronal Modeling and Data Analysis

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P333 A computational view of area 3b of primary somatosensory cortex

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P334 Sparse coding model captures V1 population response statistics to natural movies

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P335 A sparse coding model of V1 produces surround suppression effects in response to natural scenes

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P336 Fine temporal structure of neural synchronization

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P337 Inferring effective computational connectivity using incrementally conditioned multivariate transfer entropy

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P338 Control of breathing by interacting pontine and pulmonary feedback loops

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P339 Computational modeling of spinal neural circuits involved in transition to hopping pattern in EphA4 knock-out mice

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P340 Method for analyzing spike patterns with Markov transition matrices and Kullback-Leibler divergence

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P341 Cortical origin of Up state onsets and offsets in anesthetized rats

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P342 Network topology and intrinsic excitability of the existing network drive integration patterns in a model of adult neurogenesis.

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P343 Modeling the effects of anomalous diffusion on synaptic plasticity

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P344 Activation-dependent learning rule for GPCR localization - 5ht2AR regulation in Prefrontal Cortical Neurons

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P345 – Withdrawn –

P346 The shaping of the coherence function of resonate-and-fire neuron models

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P347 Influence of biophysical properties on temporal filters in a sensory neuron

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P348 Controlling the neuronal balancing act: Optical coactivation of excitation and inhibition in neuronal subdomains

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P349 Predictions of Energy Efficient Berger-Levy Model Neurons with Constraints

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P350 Hand usage and inter-limb coordination parameters for the bimanual rapid visuo-motor task to quantify sensorimotor dysfunction of participants with str

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P351 Activity dependent modulation of synaptic transmission by presynaptic calcium stores: A dichotomy of short-term depression and facilitation

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P352 Merging dorsal and ventral striatal pathway outputs of basal ganglia circuit in decision making process

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P353 A computational model of striatal neural microcircuit: how dopamine release becomes important to the striatal functions

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P354 First spike latency sensitivity of spiking neuron models

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P355 Complexity and specificity of experimentally induced expectations in motion perception

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P356 Encoding the Laplace transform of stimulus history using mechanisms for persistent firing

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P357 Neuronal morphology as an instrument for information coding: studying the influence of axonal radius and branching points

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P358 Large-scale segmentation and tracing for neurons in Drosophila brain by Fast Automatically Structural Tracing Algorithm (FASTA)

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P359 Difference in modes of firing rate modulation between cortical areas

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P360 Plasticity of Network Dynamics as Observed Experimentally Requires Heterogeneity of the Network Connectivity Pattern

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P361 Neuronal network information processing through heterogeneities and resonance frequency shifts

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P362 pMIIND-An MPI-based Population Density Simulation Framework

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P363 Advanced 3D visualisation of detailed neuronal models using the Open Source Brain repository and interaction with other neuroinformatics resources

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P364 Spikelets and Bursts in Axonless Retinal AII Amacrine Cells Coupled by Gap Junctions

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P365 Sharp transitions of gamma coherence in inhibitory networks occur when a biological context and constraints are imposed

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P366 The effect of neural synchronization on information transmission

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P367 Identification of nonlinear-nonlinear neuron models and stimulus decoding

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P368 – Withdrawn –

P369 Hybridization of multi-objective evolutionary algorithms and fuzzy control for automated construction, tuning, and analysis of neuronal models

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P370 Multi-objective evolutionary algorithms for analysis of conductance correlations involved in recovery of bursting after neuromodulator deprivation

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P371 A Complete Dynamical Study of Time-Varying and Interconnected Networks of Pulse-Coupled Theta Neurons

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P372 Dynamic feature selectivity in the thalamus of the rat whisker system

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P373 The implications of evolutionary changes in the dendritic morphology of cerebellar Purkinje cells for information processing

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P374 Short-term depression of inhibitory Purkinje cell synapses enhances gain modulation in the cerebellar nuclei

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P375 Filamentous actin binding enables ßCaMKII to regulate bidirectional plasticity in cerebellar Purkinje cells

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P376 Information transfer of an Ising model on a brain network

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P377 Novelty Detection in Long-Term Attentional Habituation Processes Using a Bayesian Change Point Algorithm

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P378 Data-driven honeybee antennal lobe model demonstrates how stimulus-onset asynchrony can aid odor segregation

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P379 Validation of an emotional model by EEG recordings of neural responses

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P380 Decoding movement direction using optical imaging of the motor cortex

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P381 Virtual Neurophysiology Laboratories for Life Science Education: Action Potentials and Voltage-/Patch-Clamp Recordings

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P382 Solving dynamical systems in neuromorphic hardware: simulation studies using balanced spiking networks

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P383 Modeling pathological brain rhythms: constructing a neural mass model from single cell dynamics

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P384 – Withdrawn –

P385 Decoding spiking activity in V4, but not V1, correlates with behavioural performance in perceptual learning task

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P386 Attractor dynamics in local neuronal networks

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P387 Synchronization and phase between model cortical areas determine information transfer

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P388 Dynamic changes in direction and frequency range of inter-areal cortical interactions revealed by non-parametric Granger causality

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P389 When less is more: Non-monotonic spike processing in neurons

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P390 Oscillation induced propagation of synchrony in structured neural networks

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P391 Heterogeneous connectivity can positively and negatively modulate the correlation between neural representations

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P392 Distinct roles of inotropic and metabotropic glutamate receptors in rhythmic activity generated by pre-Botzinger complex

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P393 Evolutionary algorithm search for network connectivities conducive to periodic behavior at sub-spiking frequencies

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P394 Critical connectivity for emergence of collective oscillations in strongly diluted neural networks

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P395 The role of environmental feedback in a brain state switch from passive to active sensing

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P396 A MODEL FOR GRID CELLS IN 3-D ENVIRONMENTS

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P397 CONTINUOUS OR DISCRETE? ATTRACTOR DYNAMICS AND SPATIAL REPRESENTATIONS IN A MODEL OF THE HIPPOCAMPAL NETWORK

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P398 Why are all phase resetting curves bimodal?

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P399 Local activity in dendrites controls STDP by altering NMDA receptor kinetics

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P400 Observations of dynamical behavior in a stochastic Wilson-Cowan population with plasticity

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P401 Spike-timing-dependent plasticity steers new Parkinson-stimulation protocols

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P402 A Model of Hippocampal Cell Assembly Dynamics Based on Single-Cell Theta Phase Precession

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P403 Probabilistic Inference of Synaptic Dynamics in Neocortical Microcircuits

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P404 Impact of orientation specific surround modulation and tuning curve shape on population coding and tilt illusion in V1

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P405 Behavioral driving through on line monitoring and activity-dependent stimulation in weakly electric fish

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P406 Assisted Closed-Loops for Brain-Computer Interfaces

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P407 The dynamic connectome: Towards large-scale 3D reconstruction of brain activity in real-time

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P408 Biomechanical costs of reaching movements bias perceptual decisions

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P409 Acquisition and execution of motor sequences by a computational model of the cerebellum

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P410 Prefrontal cortical modulation of information flow in a large-scale model of the cortico-thalamic circuit

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P411 Self-sustained activity in neural networks: influence of network topology and cell types

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P412 Functional consequences of age-related morphologic changes in pyramidal neurons of the rhesus monkey prefrontal cortex

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P413 Graphical analyses in delay interaction networks

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P414 Learning more by sampling less: Subsampling effects are model specific

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P415 Synaptic Scaling enables Dynamically Distinct Short- and Long-Term Memory Formation

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P416 Modelling the interaction of structural and synaptic plasticity

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P417 Realistic simulations of local field potentials in a slice

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P418 Effect of cortex inactivation on spontaneous activity of cells in perigeniculate and dorsal lateral geniculate nuclei

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P419 How do channel densities and various time constants affect the dynamic gain of a detailed model of a pyramidal neuron?

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P420 Modeling inner hair cell ribbon synapses: response heterogeneity and efficiency of sound encoding in an idealized biophysical model

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P421 Dynamical entropy production in cortical circuits with different network topologies

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P422 Olfactory bulb network dynamics as a pattern reservoir for adaptive cortical representations

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P423 From evolving artificial gene regulatory networks to evolving spiking neural networks for pattern recognition

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P424 Exploring the crystal structures of orientation maps in a scalable computational model of visual cortical maps

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P425 An actor-critic model of saccade adaptation

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P426 The Convallis learning rule for unsupervised learning in spiking neuronal networks

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P427 Deep brain stimulation induced effects in a network of ventral intermediate neurons

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P428 A mathematical modeling perspective on the advantages of single receptor type convergence in the olfactory glomeruli

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P429 Independent components of wing kinematics in the fruit fly Drosophila

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P430 Orexinergic Neurotransmission in Temperature Responses to Amphetamines

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P431 Functional identification and evaluation of massively parallel neural circuits

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P432 Neuronal signatures of network transition into bursting

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P433 Dynamics of two-process astrocyte networks

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P434 ERP latency contrasts using Dynamic Time Warping algorithm

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P435 Center-Surround Interactions in a Network Model of Layer 4Cα of Primary Visual Cortex

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