

Bernstein Conference 2017

Main Conference
September 13-15, 2017

Satellite Workshops
September 12-13, 2017

PhD Symposium
September 12 & 15, 2017

University of Goettingen
The Central Lecture Hall (ZHG)
Platz der Goettinger Sieben 5, 37073 Goettingen

Program and Abstracts

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Funding

The conference is financially supported by the German Federal Ministry of Education and Research (BMBF) and the German Research Foundation (DFG).



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General Conference Information

Conference Venues

Main Conference, Workshops and PhD Symposium:
University of Goettingen, Central Lecture Hall (ZHG), Platz der Goettinger Sieben 5,
37073 Goettingen

Conference Dinner and Public Lecture:
Alte Mensa, Wilhelmsplatz 3, 37073 Goettingen

Opening Hours Registration Desk

Tuesday, Sept 12	13:00 - 18:30 h
Wednesday, Sept 13	08:30 - 18:30 h
Thursday, Sept. 14	08:30 - 18:30 h
Friday, Sept 15	08:30 - 13:00 h

Opening Hours Exhibition

Wednesday, Sept 13	12:00 - 19:45 h
Thursday, Sept 14	09:00 - 19:00 h
Friday, Sept 15	09:00 - 13:00 h

Poster Sessions

Session I, Wednesday, Sept 13	17:15 - 19:45 h
Session II, Thursday, Sept 14	12:15 - 15:30 h

Mounting and Dismounting Posters

Poster boards are numbered according to the abstract numbers in the poster booklet (W indicates the first poster session on Wednesday and T the second poster session on Thursday). Pins for putting up posters will be provided. Posters can be mounted starting at 13:00 on Wednesday and at 09:00 on Thursday. Please take your poster down after your session otherwise the conference staff will remove it. Removed posters that are not picked up at the registration desk by Friday, September 15, 13:00 will be disposed of.

Abstracts

Conference abstracts including high-resolution versions of figures will be published online at <http://www.g-node.org/abstracts/bc17>.

Name Tags

Official name tags will be required for admission to all conference events. Participants who lose their name tags will have to pay a fee of 10.00 EUR to retain a replacement tag.

Wardrobe

Storage space for wardrobe and luggage will be provided in room 004 on the ground floor. The organizer assumes no liability for lost valuables of the wardrobe at the venue.

Silent Room/Working Space

You are welcome to use room 005 on the ground floor. Working space is also provided on the first floor as shown on the floor plan.

Internet

Wireless web access is provided free of charge. Logins and access instructions are provided on request at the registration desk. This WiFi network is unencrypted. Please feel free to use eduroam if you wish.

Bernstein Conference Dinner

The Bernstein Conference dinner will take place at the Alte Mensa, Wilhelmsplatz 3, which is 15 minutes walking distance from the Central Lecture Hall (ZHG). The dinner will start at 19:30. You need to register in advance and have a voucher for the dinner.

How to get to

Main Conference and Workshops

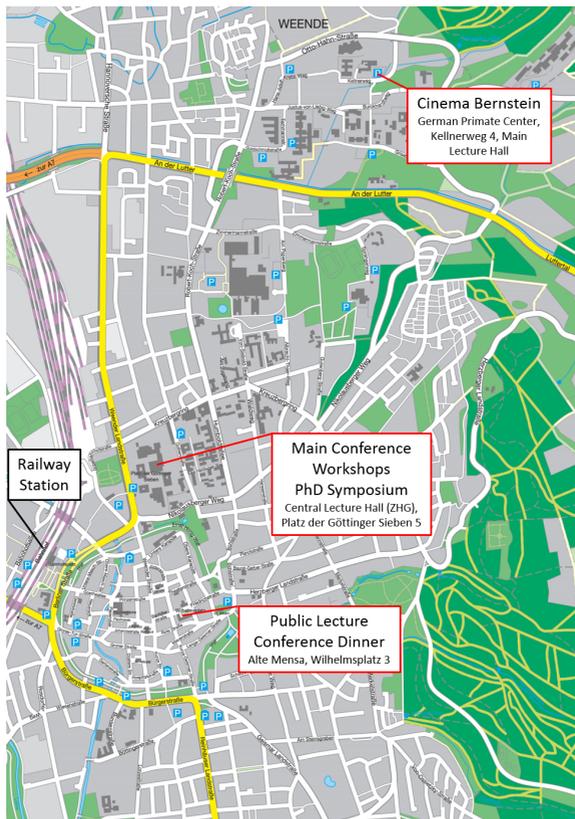
The Central Lecture Hall (ZHG), Platz der Goettinger Sieben 5, is located at the centre of the university complex on the "Platz der Goettinger Sieben". The lecture hall is near the city centre and only ten minutes walk from the railway station.

Public Lecture and Conference Dinner

Both events will take place at Alte Mensa, Wilhelmsplatz 3. Alte Mensa is a historical building of the University of Goettingen and it is located within the city centre.

Cinema Bernstein

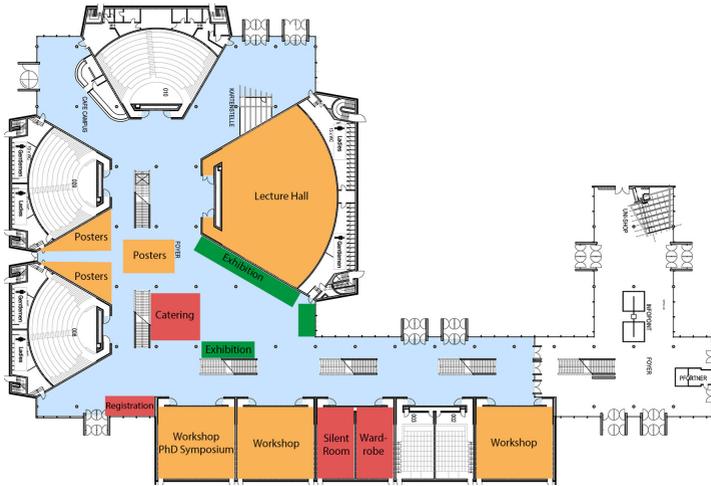
The German Primate Center (DPZ), Kellnerweg 4, Main Lecture Hall can be reached by bus lines 21, 22 and 23 from 'Platz der Göttinger Sieben'. From stop "Kellnerweg" cross the road, go in the direction of the bus. At the mailbox, turn left into the footpath and proceed to the end. Turn right into Kellnerweg. The main entrance of the DPZ is on the left side.



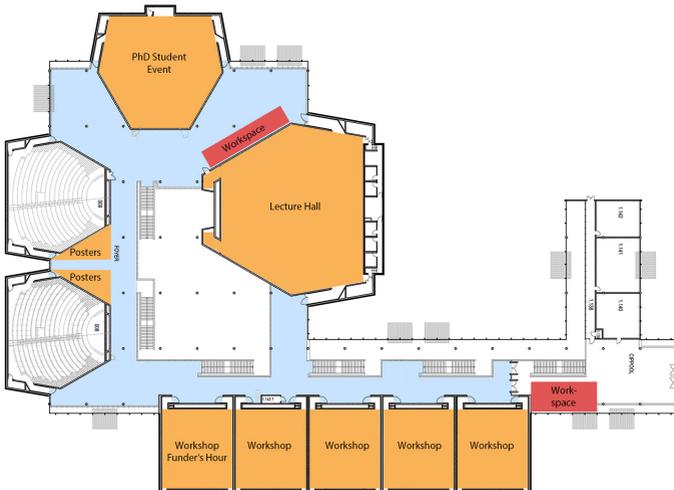
Floor Plan

University of Goettingen, Central Lecture Hall (ZHG)

Ground Floor



First Floor





Schedule

Workshops

The Bernstein Conference features a series of Satellite Workshops on September 12-13. The goal is to provide a forum for the discussion of topical research questions and challenges. Details of the individual workshops are available on the conference webpage (www.bernstein-conference.de)

Half-Day Workshops (Sept 12, 2017, 13:00-18:30)

- WS 1 Topology and dynamic of neuronal networks as guidelines for memristive computing systems (Hermann Kohlstedt, Martin Ziegler)

Half-Day Workshops (Sept 13, 2017, 09:00-12:30)

- WS 2 Information transmission and communication in brain circuits (Claudio Mirasso, Alireza Valizadeh)
- WS 3 Functional network dynamics of the hippocampus (Christian Leibold, Anton Sirota)

Full-Day Workshops (Sept 12, 2017, 13:00-18:30 and Sept 13, 2017, 09:00-12:30)

- WS 4 Connectivity generation, exploration and visualization for large scale neural networks (Wouter Klijn, Sandra Diaz)
- WS 5 Advanced theoretical approaches to collective network phenomena (Moritz Helias, Farzad Farkhooi, David Dahmen)
- WS 6 Multiscale modeling and simulation (Salvador Dura-Bernal, William W. Lytton)
- WS 7 Neural sampling: computations and experimental predictions (Ralf M. Haefner, Gergo Urban)
- WS 8 The neural code: universal grammar or area-specific mechanisms? (Eleonora Russo, Hazem Toutounji)
- WS 9 Deep neural networks tutorial for computational neuroscientists (Matthias Bethge, Jonas Rauber, Wieland Brendel)

Cinema Bernstein

Tuesday, September 12

- 19:00 **Movie and discussion** (Hosts: Julia Fischer and Fred Wolf)
Schläfer / Sleeper (Austrian-German Film 2005, Original with English subtitles)
with **Benjamin Heisenberg** (Artist, Author and Filmmaker)
Venue: German Primate Center, Kellnerweg 4, Main Lecture Hall

Main Conference

Wednesday, September 13

- 13:00 Welcome by **Ulf Diederichsen**,
Vice-President for Research, University of Goettingen
- 13:05 Welcome by **Andreas Herz**,
Chairman of the Bernstein Association for Computational Neuroscience
- 13:15 **Opening Keynote Lecture** (Chair: Fred Wolf)
Kenneth Miller (Columbia University, New York, USA)
The stabilized supralinear network, or, the importance of being loosely balanced
- Session 1: Computational Neuroscience of Learning and Memory**
(Chair: Claudia Clopath)
- 14:00 **Nicolas Brunel** (Duke University, Durham, USA)
Attractor dynamics in networks with learning rules inferred from data
- 14:45 **Adrienne Fairhall** (University of Washington, Seattle, USA)
Control of variability in motor learning
- 15:30 Coffee Break
- 16:00 **Sofia Jativa** (University College London, London, UK)
Effects of short-term plasticity on the memory lifetime of recurrent neural circuits
- 16:15 **Sara Zanon** (Bioengineering, Imperial College London, UK)
Sequential neuromodulation of Hebbian plasticity offers a mechanism for effective reward-based navigation
- 16:30 **Robert Guetig** (MPI for Experimental Medicine, Goettingen, Germany)
Spiking neurons can discover predictive features by aggregate-label learning
- 17:15 - **Poster Session I**
19:45
- 17:30 - **The Funder's Hour** (Chair Fred Wolf, parallel to the poster session I)
18:30 *HFSP: Funding International Research Collaborations in the Life Sciences* with **Barbara Pauly** (HFSP Director of Fellowships) and **J r mie Barral** (NYU, HFSP Long-Term Fellow)
- 20:00 **Public Evening Lecture** (in German) (Host: Marion Silies & Fred Wolf)
Niels Birbaumer
Abolishment of the locked-in state with a Brain-Machine-Interface (BMI)
Gehirn-Maschine-Verbindungen: Wem nutzen sie?

Thursday, September 14

Session 2: Sensory Neuroscience

(Chair: Laura Busse)

- 09:00 **Stephanie Palmer** (University of Chicago, Chicago, USA)
Understanding retinal response through the lens of prediction
- 09:45 **Tom Mrsic-Flogel** (University College London, London, UK)
Principles of local and long-range organisation of cortical circuits
- 10:30 Coffee Break
- 11:00 **Ziad M. Hafed** (Werner Reichardt Centre for Integrative Neuroscience, Tuebingen, Germany)
The foveal visual representation of the primate superior colliculus
- 11:15 **David E Whitney** (Max Planck Florida Institute for Neuroscience, Jupiter, USA)
High cellular and columnar variability underlies the absence of early orientation selectivity
- 11:30 **Judith Hirsch** (University of Southern California, Los Angeles, USA)
Neural circuits for visual processing in thalamus

12:15 Poster Session II

Session 3: Circuit Neuroscience & Connectomics

(Chairs: Viola Priesemann, Claus C. Hilgetag)

- 15:30 **Moritz Helmstaedter** (MPI for Brain Research, Frankfurt am Main, Germany)
Cerebral cortex connectomics
- 16:15 **Merav Stern** (University of Washington, Seattle, USA)
From connectivity to rate dynamics - successes and failures of the mean-field approach
- 17:00 Coffee Break
- 17:30 **Leonardo L. Gollo** (QIMR Berghofer, Brisbane, Australia)
Economic and topological trade-offs in the human connectome
- 17:45 **Wouter Klijn** (Forschungszentrum Juelich, Juelich, Germany)
Visual exploration and generation of connectivity in neural networks: bridging the gap between empirical data and theoretical model definition
- 18:00 **Siegrid Loewel** (University of Goettingen, Goettingen, Germany)
The dynamic architecture of the adult visual cortex: how to keep my brain young?
- 19:30 Conference Dinner at the Alte Mensa, Wilhelmsplatz 3, Goettingen

Friday, September 15

- Session 4: Primate Cognition / Computational Psychology**
(Chairs: Julia Fischer, Alexander Gail)
- 09:00 **Matthew Rushworth** (University of Oxford, UK)
The anterior cingulate cortex in learning and changing behaviour
- 09:45 **Andreas Nieder** (University of Tuebingen, Tuebingen, Germany)
The magical number zero
- 10:30 Coffee Break
- 11:00 **Presentation of the Brains4Brains Award 2017 and lecture by the awardee Elise Genevieve Rowe** (Monash University, Melbourne, Australia)
Bayesian mapping reveals that attention boosts neural responses to predicted and unpredicted stimuli
- 11:30 **Hazem Toutounji** (Central Institute of Mental Health and Bernstein Center Heidelberg-Mannheim, Mannheim, Germany)
A state space model for change point detection in multivariate spike count data
- 11:45 **Benjamin Dann** (German Primate Center, Goettingen, Germany)
Single trial population activity of the fronto-parietal grasping network evolves through three independent subspaces
- 12:00 **Anne Churchland** (Cold Spring Harbor Laboratory, New York, USA)
Single-trial decisions are accurately predicted by inhibitory neural population activity
- 12:45 Closing remarks by **Fred Wolf**
- 13:00 Announcement of the Bernstein Conference 2018 in Berlin

PhD Symposium

Venue: The Central Lecture Hall (ZHG), Platz der Goettinger Sieben 5, 37073 Goettingen

Tuesday, September 12

- 18:00 **PechaKucha presentations Part 1**
- 19:00 **Coffee break**
- 19:15 **PechaKucha presentations Part 2**
- 20:15 **PhD Social (Dinner tba)**

Friday, September 15

- 15:15 **Tatjana Tchumatchenko**, Max Planck Institute for Brain Research, Frankfurt, Germany
- 15:30 **Marion Silies**, European Neuroscience Institute, Goettingen, Germany
- 16:00 **Panel Discussion: Tatjana Tchumatchenko & Marion Silies**
- 18:00 **PhD Social (Dinner tba) & Farewell**



Special Events

Cinema Bernstein

Tuesday, September 12, 19:00

Venue: German Primate Center, Kellnerweg 4, Main Lecture Hall
with **Benjamin Heisenberg** (Artist, Author and Filmmaker)

Schläfer / Sleeper (Austrian-German Film 2005, Original with English subtitles)

Benjamin Heisenberg's 2005 film *Schläfer / Sleeper* - giving some of the most accurate cinematic representations of laboratory life - shows actors Bastian Trost, Mehdi Nebbou and Loretta Pflaum in a story of rivalry and treachery set in a triangle of love, science and politics. Conceived in the aftermath of 9/11 and the ensuing security legislation, it paints an intimate picture of an unsettled society undermined by a silent epidemic of suspicion. Johannes, a new assistant at the university, is asked to provide reports on Farid, an Algerian Postdoc - who is suspected of being a sleeper. He refuses, but a seed of doubt has been planted. A fragile friendship overshadowed by competition in the lab and rivalry in their relationship with Beate, a joint friend, eventually leads to betrayal. „My approach to the story was to interweave the political level in a delicate way with the characters' private conflicts in order to make the subtle corruption of the characters perceptible.“ (B. Heisenberg, 2005) *Schläfer* received numerous awards, among them the Midas Prize, EuroPAWS, for the best fiction drama set in science and technology, and was screened at the Cannes Film Festival 2005. The Cinema Bernstein screening of the film, open to the general public, will feature a discussion with artist, author and filmmaker Benjamin Heisenberg. Born 1974 in Tübingen, Heisenberg grew up near Würzburg and studied sculpting (1993-1999) and filmmaking (1997-2005) in Munich. He has directed three feature films, numerous short films and is a founding editor of the film magazine *Revolver*. Most recently he co-designed the public artwork at the Munich Documentation Centre for the History of National Socialism.

The Funder's Hour

Wednesday, September 13, 17:30

Venue: Central Lecture Hall (ZHG), first floor, room 105

with **Barbara Pauly** (HFSP Director of Fellowships) and

Jérémie Barral (NYU, Long-Term Fellow)

HFSP: Funding International Research Collaborations in the Life Sciences

Computational and theoretical neuroscience can open up novel avenues towards understanding the nervous system by integrating methods and concepts of natural sciences, life science and engineering. This year's Bernstein Conference features The Funder's Hour to provide an exchange forum for funding agencies, which support such trans-disciplinary frontier research and the computational neuroscience community. The Funder's Hour 2017 will present the Human Frontier Science Program (HFSP). HFSP has been supporting frontier research on the complex mechanisms of living organisms since 1990. HFSP Director of Fellowships Barbara Pauly will describe HFSP's funding aims and principles (HFSP: Funding International Research Collaborations in the Life Sciences). HFSP Long-Term Fellow Jérémie Barral (NYU) will report about his HFSP experience and show some highlights of his HFSP funded research (Synaptic scaling to maintain neuronal dynamics and propagate information). The short presentations will be followed by a moderated questions and answers session.

Public Lecture (in German)

Wednesday, September 13, 20:00

Venue: Alte Mensa, Wilhelmsplatz 3, 37073 Goettingen

Niels Birbaumer

Abolishment of the locked-in state with a Brain-Machine-Interface (BMI)

Gehirn-Maschine-Verbindungen: Wem nutzen sie?

Completely locked-in patients (CLIS) cannot communicate with any motor response despite intact cognitive and emotional response systems. Four ALS (amyotrophic lateral sclerosis) patients in CLIS learned to respond with a brain oxygenation and deoxygenation change of frontal brain areas using portable NIRS (near infrared spectroscopy) to short questions requiring a yes or no response presented auditorily within 15 seconds. CLIS duration in the four patients has lasted from 4 months to eight years and was validated with EOG measurement during all sessions. Each session contained 20 to 60 questions (half with yes and half with no answers). All experiments take place at the home of patients. Questions with known answers were used to train a support vector machine classifier (SVM). After achieving 70% correct answers open questions were asked and feedback of the classified answer was provided to the patients. EEG from 6 electrodes served to control sleep and vigilance decrement: questions were interrupted if sleep-like patterns appeared. 16 to 60 sessions over several months assured stability of communication with an average correct response rate of more than 70% to known and 90% correct answers to open questions. Among open questions quality of life questions were asked on a weekly basis to three of the patients with longer CLIS duration, all patients report good quality of life as previously reported by our group. Open questions answers are validated by stability over time, information of family and care takers, sentences with semantic errors and face validity (i.e. pain questions during periods of

intense pain due to decubitus and other illness related problems). These results suggest that brain machine interfaces using metabolic brain signals may end the unbearable silence of CLIS.

Supported by the Deutsche Forschungsgemeinschaft (DFG), The Eva and Horst Koehler-Stiftung, Bundesministerium fuer Bildung und Forschung (BMBF, Motor-Bic), Stiftung Volkswagenwerk (VW), Wyss Center for Bio and Neuroengineering, EU Horizon 2020: LUMINOUS. The lecture is brought to you with friendly support of the Otto Bock HealthCare GmbH.

Brains for Brains Award 2017

The Brains for Brains Award is an initiative of the Bernstein Association for Computational Neuroscience. Since 2010, the Brains for Brains Award honors outstanding young international scientists who achieved a peer-reviewed scientific publication before starting their doctoral thesis. It consists of a travel fellowship of 2,000 € covering their trip to Germany, participation in the Bernstein Conference and two individually planned visits to selected Computational Neuroscience labs in Germany. This year's award will go to Elise Genevieve Rowe (Monash University, Melbourne, Australia). She will present a poster during the poster session on Thursday, September 14. Additionally, she will give a short talk during the award ceremony on Friday, September 15.

Bernstein Network Computational Neuroscience Information Booth

The Bernstein Network is a research network in the field of computational neuroscience; this field brings together experimental approaches in neurobiology with theoretical models and computer simulations. The network started in 2004 with a funding initiative of the Federal Ministry of Education and Research to promote the transfer of theoretical insight into clinical and technical applications. Today, after 10 years of funding by the Federal Ministry, the Bernstein Network has over 200 research groups. It is named after the German physiologist and biophysicist Julius Bernstein (1839-1917).

The Bernstein Network comprises three central facilities, which are represented at the information booth:

Bernstein Coordination Site (BCOS)

BCOS is the connecting link to partners in science, industry, politics and the general public. It oversees a large information pool and can provide valuable networking resources. BCOS activities are aimed at facilitating scientific encounter, supporting young researchers and public outreach. The **SMARTSTART** Joint Training Program in Computational Neuroscience, which is coordinated by BCOS, prepares young researchers for a career in computational neuroscience. Information on all services can be gained during poster sessions.

German Neuroinformatics Node (G-Node)

During the conference, the G-Node will present their new data management service GIN. GIN is a free service for managing, sharing, and publishing research data. It features versioned storage, multi-user access for collaboration, and publishing of data using persistent identifiers (DOI). The G-Node invites all conference participants to try out the new service: bring your data, discuss your data management needs, hear about upcoming features. Demos will be given during poster sessions and coffee breaks. Walk-ins are welcome any time during the conference.

Bernstein Facility for Simulation and Database Technology (SimLab Neuroscience)

High Performance Computing (HPC) can help your neuroscience projects. SimLab Neuroscience, at the Juelich Supercomputing Centre, provides advanced support in the fields of data analysis, modeling, simulation, HPC methods and visualization. It also offers tutorials, workshops, and courses to help you make the transition to HPC. Come and meet us at the booth and find out what the Bernstein Facilities can do for you.

Further events at the Bernstein Network Information Booth:

Wednesday, September 13

15:30 BCOS Travel Grants: Award ceremony and group picture

Bernstein Association for Computational Neuroscience

The Bernstein Association for Computational Neuroscience supports research and education in computational neuroscience and reaches out to the general public to bring across research topics and current findings. The Bernstein Association was founded in 2009 by members of the Bernstein Network and is recognized as a non-profit organization. Everyone who is active in the field of computational neuroscience or related subjects can become a member of the Bernstein Association. The general assembly usually takes place at the annual Bernstein Conference. More information can be found at the Bernstein Network Information Booth or on the website (www.nncn.de/en/bernstein-association)

Thursday, September 14

14:15 – General Assembly 2017 Bernstein Association for Computational Neuroscience (Room 005) – for members only
15:15



Invited Talks

[1 1] The Stabilized Supralinear Network, or, The Importance of Being Loosely Balanced

Kenneth Miller¹

1. Center for Theoretical Neuroscience, Columbia University, 3227 Broadway, L6-070, Mail Code 9864, New York, NY 10027, USA

I will describe the Stabilized Supralinear Network mechanism and its application to understanding sensory cortical behavior. The mechanism is based on a network of excitatory (E) and inhibitory (I) neurons with very simple assumptions: (1) Individual neurons have an expansive or supralinear input/output functions, e.g. a power law with power >1 ; (2) Feedback inhibition is sufficiently strong to drive the network to a stable fixed point for a given stable input. The expansive input/output function leads effective synaptic strengths to grow with network activation. The network then transitions, with increasing external input strength, between two regimes: (1) For weak activation, the network is very weakly coupled, with neuronal input dominated by external input (from outside the network) rather than network input. In this regime, responses to different stimuli sum supralinearly, and hence different stimuli tend to facilitate one another's response. (2) For stronger activation, the network becomes strongly coupled, with input dominated by network input; and recurrent excitation becomes strong enough to potentially create network instability, but the network is stabilized by feedback inhibition. With increasing activation the network input is more and more dominated by inhibitory input. In this strongly-coupled regime, the network is loosely balanced: the E/I dynamics lead network input to partially cancel external input, so that the net input grows sublinearly as a function of the external input. This turns out to be equivalent to the "balanced network" of Van Vreeswijk and Sompolinsky, but in a regime in which the net input remaining after cancellation is comparable to the components that are cancelled ("loose balance") rather than being negligibly small in comparison ("tight balance"). This makes all the difference for network behavior: whereas tight balance yields network response that is a linear function of external input, loose balance yields nonlinear behaviors that look strikingly like sensory cortical behaviors. These behaviors include "normalization" or sublinear summation of responses to multiple stimuli that becomes linear for weak stimuli and becomes "winner-take-all" for stimuli of strongly unequal strength; and surround suppression that becomes surround facilitation for a weak center stimulus. In addition, whereas tight balance creates an asynchronous regime (without correlations), loose balance allows correlated variability that is gradually

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Cite as: Miller K (2017) The Stabilized Supralinear Network, or, The Importance of Being Loosely Balanced. *Bernstein Conference 2017 Abstract*. doi: [10.12751/nncn.bc2017.0001](https://doi.org/10.12751/nncn.bc2017.0001)

[1 2] Attractor dynamics in networks with learning rules inferred from data

Nicolas Brunel¹

1. *Departments of Statistics and Neurobiology, The University of Chicago, 947 E. 58th St., MC0926, Chicago, IL 60637, USA*

The attractor neural network (ANN) scenario is a popular scenario for memory storage in association cortex, but there is still a large gap between these models and experimental data. In particular, the distributions of the learned patterns and the learning rules are typically not constrained by data. In primate IT cortex, the distribution of neuronal responses is close to lognormal, at odds with bimodal distributions of firing rates used in the vast majority of theoretical studies. Furthermore, we recently showed that differences between the statistics of responses to novel and familiar stimuli are consistent with a Hebbian learning rule whose dependence on post-synaptic firing rate is non-linear and dominated by depression. We investigated the dynamics of a network model in which both distributions of the learned patterns and the learning rules are inferred from data. Using both mean field theory and simulations, we show that this network exhibits attractor dynamics. Furthermore, we show that the storage capacity of networks with learning rules inferred from data is close to the optimal capacity, in the space of unsupervised Hebbian rules. These networks lead to unimodal distributions of firing rates during the delay period, consistent with data from delay match to sample experiments. Finally, we show there is a transition to a chaotic phase at strong coupling strength, with a extensive number of chaotic attractor states correlated with the stored patterns.

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Cite as: Brunel N (2017) Attractor dynamics in networks with learning rules inferred from data. *Bernstein Conference 2017* Abstract. doi: [10.12751/nncn.bc2017.0002](https://doi.org/10.12751/nncn.bc2017.0002)

[1 3] Control of variability in motor learning

Adrienne Fairhall¹

1. *Department of Physiology and Biophysics, University of Washington, HSB Rm G311, Box 357290, Seattle WA 98195-7290, USA*

The birdsong learning circuit presents an outstanding opportunity to explore the explicit realization of the computational algorithms of learning in a biological network at the level of detailed anatomy, circuit dynamics and neuronal biophysics. We will discuss biological mechanisms of variability generation and control that may underlie the bird's capacity to learn, monitor and maintain song quality.

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Cite as: Fairhall A (2017) Control of variability in motor learning. *Bernstein Conference 2017* Abstract. doi: [10.12751/nncn.bc2017.0003](https://doi.org/10.12751/nncn.bc2017.0003)

[I 4] Spiking neurons can discover predictive features by aggregate-label learning

Robert Guetig¹

1. Max Planck Research Group Theoretical Neuroscience, MPI for Experimental Medicine, Hermann-Rein-Straße 3, 37075 Göttingen, Germany

The brain routinely discovers sensory clues that predict opportunities or dangers. However, it is unclear how neural learning processes can bridge the typically long delays between sensory clues and behavioral outcomes. Here, I introduce a learning concept, aggregate-label learning, that enables biologically plausible model neurons to solve this temporal credit assignment problem. Aggregate-label learning matches a neuron's number of output spikes to a feedback signal that is proportional to the number of clues but carries no information about their timing. Aggregate-label learning outperforms stochastic reinforcement learning at identifying predictive clues and is able to solve unsegmented speech-recognition tasks. Furthermore, it allows unsupervised neural networks to discover reoccurring constellations of sensory features even when they are widely dispersed across space and time.

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Cite as: Guetig R (2017) Spiking neurons can discover predictive features by aggregate-label learning. *Bernstein Conference 2017 Abstract*. doi: [10.12751/nncn.bc2017.0004](https://doi.org/10.12751/nncn.bc2017.0004)

[I 5] Understanding retinal response through the lens of prediction

Stephanie E. Palmer¹

1. Department of Organismal Biology and Anatomy, University of Chicago, 1027 E 57th St, Chicago, IL 60637, USA

Prediction is necessary for overcoming short timescale sensory and motor delays present in all neural systems. In order to interact appropriately with a changing environment, the brain must respond not only to the current state of sensory inputs but must also make rapid predictions of these inputs' future state. To test whether the visual system performs optimal predictive compression and computation, we compute the past and future stimulus information in populations of retinal ganglion cells, the output cells of the retina, in salamanders and rats. For some simple stimuli with mixtures of predictive and random components to their motion, we can derive the optimal tradeoff between compressing information about the past stimulus while retaining as much information as possible about the future stimulus. By changing parameters in the input motion, we can explore qualitatively different motion prediction problems. This allows us to begin to ask which prediction problems the retina has evolved to solve optimally. Furthermore, we explore the tradeoffs between optimally representing predictive information in the stimulus, and decorrelating neural responses in time.

©(2017) Palmer SE

Cite as: Palmer SE (2017) Understanding retinal response through the lens of prediction. *Bernstein Conference 2017 Abstract*. doi: [10.12751/nncn.bc2017.0005](https://doi.org/10.12751/nncn.bc2017.0005)

[1 6] Principles of local and long-range organisation of cortical circuits

Thomas Mrsic-Flogel¹

1. Biozentrum, University of Basel, Klingelbergstrasse 50 / 70, CH - 4056 Basel, Switzerland

The rules by which neurons in neocortex choose their synaptic partners are not fully understood. In sensory cortex, intermingled neurons encode different attributes of sensory inputs and relay them to different long-range targets. The first part of the talk will discuss how local connectivity is constrained by response similarity and long-range projection target in mouse primary visual cortex (V1). The second part of the talk will focus on the organisation of long-range connectivity patterns of V1 neurons, revealing that most neurons innervate multiple intracortical targets and thus act to coordinate activity across subsets of cortical areas.

©(2017) Mrsic-Flogel T

Cite as: Mrsic-Flogel T (2017) Principles of local and long-range organisation of cortical circuits. Bernstein Conference 2017 Abstract. doi: [10.12751/nncn.bc2017.0006](https://doi.org/10.12751/nncn.bc2017.0006)

[1 7] Neural circuits for visual processing in thalamus

Judith Hirsch¹

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The thalamus is often viewed as a gatekeeper, relaying sensory signals to the cortex during waking and halting their flow during sleep. While true, this is an impoverished description. Our work explores how thalamic circuits contribute to sensory processing *per se*. We focus on the lateral geniculate nucleus, which conveys information from the eye downstream. Relay cells in the geniculate make few connections with each other but are embedded in two dense inhibitory networks. First, local interneurons supply feedforward inhibition; second, neurons in the visual sector of thalamic reticular nucleus (a thin sheet of gabaergic cells that cloak the thalamus) provide feedback inhibition. The initial part of the presentation explores how retinogeniculate divergence and convergence, coupled with feedforward inhibition, might facilitate signal detection and enhance perceptual acuity. The later part explores the role of the reticular nucleus, from the perspectives of feature detection and spatial attention.

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[I 8] Cerebral Cortex Connectomics

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The mapping of neuronal connectivity is one of the main challenges in neuroscience. Only with the knowledge of wiring diagrams is it possible to understand the computational capacities of neuronal networks, both in the sensory periphery, and especially in the mammalian cerebral cortex. Our methods for dense circuit mapping are based on 3-dimensional electron microscopy (EM) imaging of tissue, which allows imaging nerve tissue at nanometer-scale resolution across substantial volumes (typically hundreds of micrometers per spatial dimension) using Serial Block-Face Scanning Electron Microscopy (SBEM). The most time-consuming aspect of circuit mapping, however, is image analysis; analysis time far exceeds the time needed to acquire the data. Therefore, we developed methods to make circuit reconstruction feasible by increasing analysis speed and accuracy, using a combination of crowd sourcing and machine learning. We have applied these methods to circuits in the mouse retina and improved them for the application to much larger neuronal circuits in the cerebral cortex. We are currently mapping the local circuit structure of different cortices in various species. The goal is to measure the invariants of circuit structure between individuals and across mammalian species, in search for the algorithms of sensory perception. Future work will include the search for engrams of sensory experience in the cerebral cortex, and for alterations in neuronal network structure in psychiatric disease.

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[1 9] From Connectivity to Rate Dynamics - Successes and Failures of the Mean-Field Approach

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Mean-field theory is commonly used to analyze the dynamics of large neural network models. In this approach, the interactions of the original network are replaced by appropriately structured noise driving uncoupled units in a self-consistent manner. This allows properties of the network dynamics to be predicted and the behavior of the network to be understood as a whole. Results in random matrix theory have been used to relate the structure of the connectivity of neural networks to their mean-field dynamics. In my talk I will explain the mean-field approach, discuss its relation to random matrix theory, and analyze how the dynamics of neural network models are related to their connectivity structure. I will provide examples of networks that the mean-field theory describes accurately as well as examples, analyzed with the use of matrix theory, in which small modifications in the connectivity matrix can result in large deviations from mean-field predictions.

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[1 10] The dynamic architecture of the adult visual cortex: how to keep my brain young?

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My lab is focused on understanding the development and plasticity of neuronal circuits in the mammalian cortex. We use a combination of techniques, including various imaging/physiological techniques and behavioural analyses to explore how experience and learning influence the structure and function of nerve cell networks and how activity patterns and genetic factors influence these processes. We hope that answering these key questions not only helps to understand the rules underlying brain development, functioning and learning but additionally will open up new avenues to develop clinically relevant concepts to promote regeneration and rehabilitation for diseased and injured brains. In my lecture, I will focus on recent experiments about the role of various stimulating environments on visual cortical plasticity in mice. In addition, I will present a completely new molecular mechanism governing both the duration of a critical period in early postnatal development and the maturation of nerve cell contacts.

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[I 11] The anterior cingulate cortex in learning and changing behaviour

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The dorsal anterior cingulate cortex (dACC) is one of the brain areas that has received most attention from cognitive neuroscientists using techniques from EEG to fMRI. Activity in dACC has been linked with a variety of processes such as error detection and cognitive control. dACC is often linked to the most sophisticated features of human behaviour even though dACC is present in many species. I will argue that dACC can be related to basic computations that many animals including humans perform to decide whether to maintain or change their current course of action. It carries activity relating to the average value of alternative courses of action available in the environment and it tracks how successful recent behaviour has been over multiple time scales. This means that it can track whether current behaviour is more or less successful than it has been recently and whether a change in behaviour is likely to be more successful. Discerning such trends makes predicting the value of future behaviour possible. Time-scale specific interactions occur with representations with similar characteristics in a limited number of other brain areas. Individual variation in neurotransmitter levels in dACC (glutamate and GABA) can be related to individual variation in the way in which experience is used to influence behaviour.

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[I 12] The magical number zero

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Zero stands for emptiness, for nothing - and yet it is considered to be one of the greatest achievements of humankind. However, zero is a most abstract and difficult concept. It took a long stretch of human history for zero to be recognized. Children show a delayed understanding of numerosity zero, long after they comprehend positive integers. Only advanced animals with which we share a nonverbal quantification system exhibit rudiments of a grasp of zero numerosity. For a brain that has evolved to process sensory stimuli, conceiving of empty sets ("nothing") as a meaningful category ("something") demands high-level abstraction. Our single-neuron data recorded in nonhuman primates suggest a parieto-frontal processing hierarchy along which empty sets are steadily detached from visual properties and gradually positioned in a numerical continuum. Based on converging evidence from different disciplines like history of mathematics, developmental psychology, animal cognition, and neurophysiology, I will argue that the emergence of zero passes through four corresponding representations in all of these interrelated realms: first sensory "nothing", next categorical "something", then quantitative empty sets, and finally the number zero. The concept of zero shows

how the brain, originally evolved to represent stimuli, detaches from empirical properties to achieve ultimate abstract thinking.

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[I 13] **Single-trial decisions are accurately predicted by inhibitory neural population activity**

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Decisions are driven by the coordinated activity of diverse neural populations in multiple structures. Inhibitory neurons play a critical role in many models of decision-making, but the difficulty in measuring large inhibitory populations in behaving animals has left their *in vivo* role mysterious. To understand the contributions of excitatory and inhibitory neural populations to perceptual decision-making, we measured neural responses in transgenic mice expressing tdTomato in inhibitory neurons (GAD2-Cre crossed with Ai14 reporter line). To record neural activity, mice were injected with AAV9-Synapsin-GCaMP6f in the posterior parietal cortex (PPC). Mice were then presented with a series of multisensory “events” (clicks and flashes), the rate of which fluctuated stochastically over a 1000 ms period. Mice were trained to lick to a right (left) spout to report that event rates were judged above (below) an abstract category boundary (16 Hz). 2-photon imaging was used to measure single-neuron responses during these decisions. In each session, 600 neurons were simultaneously recorded while mice performed 400 trials.

To evaluate the relationship between neural activity and decision-making, we trained linear classifiers to distinguish activity preceding left vs. right choices on single trials. In keeping with previous work, we observed that overall population activity could reliably predict the animal's choice. To understand how specific cell types shape this population activity, we evaluated excitatory and inhibitory neurons separately. Surprisingly, inhibitory population activity alone could reliably predict the animal's choice at a level that was indistinguishable from excitatory neurons. Importantly, the distributions of weights assigned by the classifiers were similar for excitatory and inhibitory populations. This argues that for both cell types, the pooled activity of many neurons can effectively distinguish decision outcomes. These findings argue in favor of decision-making models in which pools of inhibitory neurons are specifically targeted by populations of excitatory neurons in favor of a particular choice.

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Contributed Talks

[C 1] Effects of short-term plasticity on the memory lifetime of recurrent neural circuits

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See also Poster: W 33

It is a substantial open challenge to understand how recurrent neural circuits can act as a memory buffer, despite fast forgetting by individual neurons. Previous work has primarily focused on understanding memory lifetimes of recurrent circuits with conventional static synapses. However, synapses are rich dynamical systems in their own right, and recent experimental findings [1,2] along with previous theoretical proposals [3,4] implicate these characteristics as supporting short-term memory. Recently, an information-theoretic upper bound for memory lifetime was derived for linear recurrent networks. Specifically, it was shown that any linear network can, at most, achieve a memory life time proportional to the number of neurons in the network. Furthermore, it was shown that only a delay line, or any network that is equivalent to a delay line up to a unitary transformation, can saturate this bound [5]. Here, we extend this information-theoretic analysis to understand the role of dynamic synapses with short-term plasticity on memory performance. By linearizing a non-linear network, we study how short term plasticity modifies the effective connectivity matrix of the network to change the memory performance. We tested this framework in different architectures, concentrating on networks with very poor memory performance i.e. normal networks. We show that dynamical synapses modify the internal structure of these networks and improve their memory performance. We will analyze the conditions under which memory performance is improved and under which the effective connectivity matrix approximates an effective delay line. We expect that the short-term plasticity of synapses might be key for understanding how recurrent neural circuits buffer temporal signals during cognitive processing, and furthermore it suggests a different way for synapses to be considered as a neural substrate of working memory.

Acknowledgements

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[C 2] Sequential neuromodulation of Hebbian plasticity offers a mechanism for effective reward-based navigation

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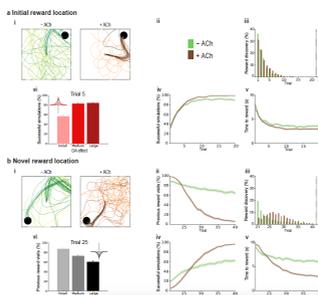
See also Poster: T 38

Neuromodulation is thought to act as a supervisory or modulatory signal by providing feedback about the environment. It has been shown to play a fundamental role in acquiring new behaviours. However, it is still unclear how neuromodulated learning is attained at the synaptic level. We decided to investigate this by combining experimental and computational methods.

Our experimental data on mouse hippocampal slices show that acetylcholine bias spike-timing dependent plasticity (STDP) towards depression (Brzosko et al., in submission), while the subsequent application of dopamine retroactively converts synaptic depression into potentiation, effectively acting as an eligibility trace (Brzosko et al., eLife, 2015; Brzosko et al., in submission).

We therefore set out to explore the computational and behavioural potential consequences of these findings. We incorporated our novel sequentially-modulated plasticity rule in a spiking network model of a reward-based navigation task (Fremaux et al., 2013). In agreement with previous network models of reinforcement learning, our results indicate that dopamine-modulated STDP enables learning of associations between actions and delayed rewards. Our modelled agent can successfully navigate to the reward location. Furthermore, the addition of cholinergic depression enables learning from negative outcomes. This allows for flexible learning and relearning, especially useful in a real-world, changing environment.

Thus, temporally sequenced neuromodulation of STDP not only enables associations to be made between actions and outcomes, but also provides a possible mechanism for aligning the timescales of cellular and behavioural learning .



Temporally sequenced cholinergic and dopaminergic modulation of STDP yields effective navigation towards changing reward locations.

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[C 3] The foveal visual representation of the primate superior colliculus

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A primary reason for using non-human primates in visual neuroscience is their foveated retina. However, neurophysiological investigations of foveal representations are not common because of difficulties with eye movements and small response field (RF) sizes. This creates a pressing need to study foveal representations, especially given that foveal processing is a mode of operation that we rely on heavily in our daily life. Here we recorded from the foveal visual representation of the superior colliculus (SC) in 2 awake and 2 anesthetized monkeys. In the awake animals, we recorded from 121 neurons with foveal preferred eccentricities and compared their visual RF characteristics to those of >200 more eccentric neurons. We corrected for eye position during fixation to obtain better estimates of RF shapes and sizes. In the anesthetized animals, we densely mapped preferred RF locations and related them to SC anatomy, mapping 66 foveal sites and comparing them to >100 more eccentric ones. We systematically moved our electrodes by 100, 250, or 500 micrometer steps along the two-dimensional SC surface. Foveal SC neurons' RF's were strongly skewed and lateralized, having sharp cutoffs at the "foveal edge" of the visual representation. RF skew decreased progressively with increasing eccentricity, along with an exponential increase in RF size. Such increase also happened within the central foveola region (<0.5 deg radius), suggesting non-uniform sampling of visual space by the SC even within the smallest eccentricities. Foveal visual neural sensitivity was also as strong as, if not marginally stronger, than peripheral neural sensitivity. Our dense mappings of SC surface topography revealed a highly orderly foveal representation, which was continuous with peripheral topography. We used our mappings to develop a 3-D model of the SC's topographic foveal visual representation, demonstrating more than twice the foveal magnification factor predicted by classic models that extrapolated peripheral measurements. In all, our results demonstrate strong laterality of visual representations in the foveal SC, non-uniform sampling of space, high neural sensitivity, and a surprisingly large foveal magnification factor. The magnification and continuity of foveal topography at this level of detail have implications on the potential impacts of small eye movements on visual coding, and might also explain certain characteristics of microsaccade amplitude distributions.

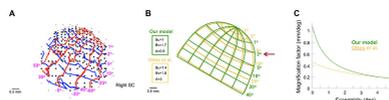


Illustration of increased foveal magnification in SC. (A) Iso-eccentricity (blue) and iso-direction (red) lines from data. (B, C) Classic model (Ottes et al., 1986; yellow) and our model (green). The classic model, based on extrapolation from periphery, grossly underestimates foveal magnification.

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[C 4] High cellular and columnar variability underlies the absence of early orientation selectivity

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See also Poster: T 97

Selectivity for stimulus orientation is a fundamental property of primary visual cortex in primates and carnivores, where it is organized into a smoothly varying columnar map that emerges in an activity-dependent manner during early postnatal life. Despite extensive experimental and theoretical work, it remains unclear what factors limit the emergence of orientation selectivity, such as weak responsiveness to visual stimuli, high trial-to-trial variability, and/or an intermixed 'salt-and-pepper' organization of orientation preferences at the cellular level. To distinguish between these potential factors, we visualized population activity in the visual cortex of developing ferrets with longitudinal imaging of GCAMP6s at both cellular resolution with two-photon calcium imaging and columnar resolution with widefield epifluorescence imaging. Prior to eye opening, we show that cellular and population responses evoked by single presentations of a grating stimulus surprisingly exhibit robust, modular patterns of network activity resembling activity patterns evoked by gratings in mature animals. However, the spatial location and pattern of domains activated by presentation of the identical stimulus orientation varies substantially across trials, a variability that accounts for the low orientation selectivity of individual neurons and the inability to visualize coherent maps of orientation preference. Yet variability in network activity patterns is not a general feature of the developing cortex, as the modular patterns of network activity evoked by uniform luminance steps are already selective at these ages. Furthermore, we show that trial-averaged activity patterns evoked by gratings show similarity to the mature orientation map as early as 1-2 days prior to eye opening. We conclude that the early disassociation between stimulus orientation and consistent patterns of modular network activity is a major factor underlying the absence of orientation selectivity in a developing cortical network already exhibiting highly modular functional organization.

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[C 5] Economic and topological trade-offs in the human connectome

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See also Poster: T 8

The brain resides in an evolutionary landscape that pits the costs of its anatomical wiring against the computational advantages conferred by its complexity. The processes shaping this exchange remain poorly understood. We address this problem by studying random variants of the connectome that introduce subtle perturbations to network topology while preserving the geometrical embedding and wiring length of the brain. We first show that the presence of hubs widely distributed throughout cortical regions confers a wiring cost that the human brain minimizes. Although slight perturbations of brain networks reduce the wiring length of inter-hub connections, these perturbations quickly disconnect inter-hemispheric links to prefrontal hubs and yield daughter networks that substantially differ from one another. If the variation in structure is permitted to accumulate, strong peripheral connections progressively connect to central nodes and hubs shift toward the middle of the brain. Progressive randomization of brain networks also leads to a topologically unstable intermediate regime consistent with a phase transition in complex systems. Intriguingly, the fragility of hubs to disconnections shows a significant association with the acceleration of grey matter loss in schizophrenia that is stronger than the association with hub strength. Together with effects on wiring cost, we suggest that fragile prefrontal hub connections and topological instabilities act as evolutionary influences on complex brain networks whose set point may be perturbed in neurological and psychiatric disorders.

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[C 6] Visual exploration and generation of connectivity in neural networks: bridging the gap between empirical data and theoretical model definition.

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See also Poster: T 9

The study of connectivity is central in the diverse disciplines of neuroscience. On one hand, the structured definition of network connectivity is an essential step in network simulations. On the other hand, we can derive connectivity information from

experimental data and various theoretical models at multiple scales. However, the connectivity information in these two contexts is represented differently. This results in a language gap limiting the flow of knowledge learned at different levels of abstraction. In this work, we present a first step in the creation of a shared visual language to bridge this gap between model based and empirical neuroscience, allowing us to work towards a single integrated representation of the brain.

We have developed a visual and source-agnostic interactive interface to generate connectivity in neural networks at various scales. Based on NeuroScheme [1] and the Connection Set Algebra (CSA)[2], we can generate connectivity and use it in simulator-specific scripts to later perform simulations of the dynamics of the network. Our approach allows us to interactively create, explore and visualize connectivity even for large scale networks where probability based connections are used to describe the synapse generation. Here we show initial results of the tool applied to Potjan's and Diesmann microcircuit model as an initial use case for describing and exploring the connectivity.

With this approach, we offer the neuroscientific community a generic tool for the easy generation and exploration of connectivity. The lack of dependency on a specific simulator makes this tool a good starting point for validation of complex neural network models using many simulation and emulation platforms, particularly when coupled. Our future applications involve incorporating this tool to complete workflows consisting of raw data processing, interactive exploration, creation and visualization of abstract connectivity models, simulation, analysis and validation.

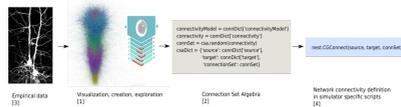


Diagram showing our approach to bridge empirical and theoretical connectivity representations

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[C 7] A state space model for change point detection in multivariate spike count data

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Neural activity from higher cortical areas in awake, behaving animals has highly dynamic and strongly nonstationary population-wide response properties. Nonstationary events in the form of changes in the firing rate statistics of recorded spike count time series may arise from a variety of sources. They may encode features of the experimental paradigm and could, potentially, correspond to the neural computations associated with the performance of a given behavioural task. In order to identify nonstationary events, or change points, in neural data, and to relate these events to behaviour, a model-based approach to change point detection may bear certain advantages not shared by model-free techniques. Here, we develop such an approach for detecting and parametrising multiple changes in multivariate spike count data within the statistical framework of State Space Models (SSM). The model assumes a nonlinear, nonstationary, autoregressive Gaussian process, parametrised by the onset of change and its time scale, that captures relevant features of the underlying latent neural dynamics. Given their discrete, nonnegative nature, high-dimensional spike count time series are generated from the low-dimensional latent states through a Poisson observation model. We devised an initialisation algorithm, a model selection method and an estimation procedure that makes for a practical and efficient solution to change point identification from large data sets, such as recordings from developmental studies. Model parameters are constrained in a way that assures model identifiability, which we demonstrate by estimating latent states and model parameters from synthetic data where the ground truth is known. We also show that population-wide change points and their time scales can be reliably estimated, even when this information is lost by the averaging effect of classical methods, such as summarising data in peristimulus time histograms. As a real data example, the model is fitted to multiple single unit recordings from rat medial prefrontal cortex neurons during an operant rule switching task. The resulting reconstruction of the underlying nonstationary dynamics allows matching the neural correlates of learning to their behavioural counterpart by relating behavioural changes to population-wide change points, as estimated by the model.

Acknowledgements

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[C 8] Single trial population activity of the fronto-parietal grasping network evolves through three independent subspaces

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See also Poster: W 2

Neurons in the fronto-parietal grasping network are known to be modulated for perceptual processing, movement preparation, and movement control^{1,2}. Due to neuronal presence of all three processes in the same network it is considered to be involved in the transformation between the different kinds of information as well as decision making. Yet, how this information is encoded and transformed in the system is still in debate. We addressed this question using parallel recordings from many neurons in the fronto-parietal grasping network (AIP and F5) of the macaque monkey while animals were either visually instructed or freely chose to grasp a handle with one of two grip types. Classical tuning analyses revealed all neurons in both areas to be significantly modulated by task parameters. However, single-neuron response patterns were complex and dynamically over time, and were heterogeneously distributed with no sign of categories. In contrast, when we considered the whole neuronal population as one strongly interconnected network, in which neural population activity evolves dynamically through space-space over time and conditions, a clear low dimensional structure became apparent. Nearly all task specific single trial activity could be explained by an evolution of just three independent informational subspaces representing visual, preparatory, and movement activity (Figure 1). Interestingly, for free-choice trials, where no specific visual information was given, all task specific activity during the decision process was explained by the preparatory space, suggesting that preparatory activity explains all decision related activity in this task. Crucially, contributions to all three informational subspaces were randomly distributed across the whole fronto-parietal neuronal population with no significant category structure. Furthermore, a regularized recurrent neuronal network trained to produce muscle activity could well reproduce the neuronal dynamics both at the single unit and the population level. The fact that nearly all task specific single trial neuronal variance across all neurons and areas can be understood as a dynamical process evolving through just three information subspaces offers a new perspective on the fronto-parietal grasp network. The independence of the visual, preparatory, and movement subspaces even allows to disentangle and analyze their information separately, which could be of great importance for decoding approaches.

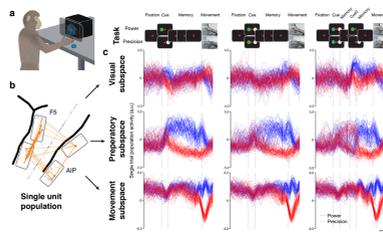


Figure 1 (a) Schematic view of the behavioral task. (b) Anatomical network of all parallel recorded neurons of one dataset³. (c) Single trial population dynamics of fronto-parietal single units for the visual, preparatory, and movement subspace.

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Posters Wednesday

Attention, reward, decision making

[W 1] **Gamma oscillations organize top-down signaling to hypothalamus and enable food seeking**

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Lateral hypothalamus (LH) is crucial for regulation of feeding, yet little is known about the regulation of LH by top-down inputs from cognitive control regions. Top-down forebrain innervation of LH is provided, to a large extent, by inhibitory inputs from the lateral septum (LS), a key region for governing innate behaviors according to environmental context; LS is connected, in turn, with cortical networks. Here we combined optogenetics and computational modeling-guided high-density unitary recordings from these regions in mice during spontaneous behavior in a free-access feeding paradigm (Carus-Cadavieco et al., *Nature*, 2017). We found that food-seeking behavior relies on gamma (30-90 Hz) oscillations, coordinated between LH and upstream brain regions. When mice engaged in approach to the food zone, the gamma power in LS and LH matched the time required to reach the food zone, but not the drinking zone. Gamma-rhythmic input to LH from somatostatin-positive LS cells evoked food approach without affecting food intake. LS inhibitory input enabled separate signaling by LH neurons according to their feeding-related activity, making them fire at distinct phases of the gamma oscillation. Using CLARITY, *in vivo* electrophysiology and computational modeling, we identified medial prefrontal cortex projections providing gamma-rhythmic inputs to LS, leading to improved performance in a food-rewarded learning task. Conversely, LS and its major afferent region, hippocampus, displayed coordination by theta rather than gamma oscillations (Bender et al., *Nature Communications*, 2015). Overall, our study identifies a novel top-down pathway, which utilizes gamma synchronization to guide activity of subcortical networks and to regulate feeding behavior by dynamic reorganization of functional cell groups in hypothalamus.

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[W 3] Effects of cognitive biases and imperfect reward predictions on perceptual learning

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Roving is a random task-sequencing paradigm, in perceptual learning, whereby multiple tasks are learned in a randomly interleaved sequence. For certain experiments, such as bisection tasks, human subjects appear to be unable to learn the individual tasks under roving conditions. In general, theoretical descriptions of perceptual learning experiments have resorted to approaches involving tuning of inputs, using either recurrence or suppression. However, these approaches have exhibited only partial success in tackling roving. In 2012, Herzog et al. proposed a theoretically inspired explanation involving a constant drift in synaptic efficacies in the system (unsupervised bias), due to an inability to maintain accurate task specific estimates of performance. This leads to a failure to learn using feedback. We update this approach, adding additional features, which though adding realism tend to counteract the action of the unsupervised bias. We then use this model to examine whether the unsupervised bias is sufficient to explain roving or not.

The proof-of-concept model proposed in Herzog et al. does indeed lead to a failure to correctly learn during roving but, while it fails due to the mooted unsupervised bias in the learning rule, the implementation relies on unbounded weight growth, an unrealistic phenomenon. We introduce a simple weight normalisation term, to counteract the unbounded weight growth, and implement a cognitive bias, often observed in human subjects, towards 50:50 presentation ratios. We thus discover a more appropriate model of human perceptual learning performance. Our model (i) learns correctly on a single bisection or vernier task, (ii) fails to learn during roving of multiple tasks, (iii) exhibits the human tendency towards 50:50 ratios of choice, thus failing when a 75:25 ratio is used, and (iv) correctly learns when informed of the altered presentation ratio, similarly to human subjects (unpublished data). A further extension to the original model, operating on a much slower timescale, allows the task critic system to learn over time to separately identify the tasks. This ultimately leads to learning of the initially unlearnable tasks, as seen in much longer timescale experiments.

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[W 4] Optimal Deployment of Feature-based Attentional Gain in Macaque Visual Cortex

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Attending to spatial locations or to non-spatial features has been shown to enhance the gain of those neurons in primate visual cortex that preferentially respond to stimuli matching these attended locations and features. Here we investigated how attention affects the population responses of neurons in the middle temporal visual area of rhesus monkeys to bi-directional movement inside the receptive field. The monkeys were trained to detect a direction or speed change in the target motion direction, ignoring the distractor motion. In control trials, monkeys instead focused their attention onto the fixation spot. Population activity profiles for these two conditions were determined by systematically varying the patterns' directions while maintaining a constant angle between them. As expected, the response profiles show two peaks representing the strong response of the two groups of neurons preferring one of the two motion directions. Switching spatial attention from the fixation spot into the receptive field resulted in an enhanced activity of the neurons representing the attended stimulus and suppression of the activity from the neurons representing the distractor in the neuronal population. Furthermore, the population data indicated a direction-dependent attentional modulation that does not peak at the target feature, but rather along the slopes of the activity profile representing the target direction. Our results suggest that attentional gains are differentially allocated to neuronal populations to optimize the discriminability of the target direction, in line with an optimal gain mechanism (Navalpakkam and Itti, 2007).

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[W 5] Perceptual confirmation bias as a result of approximate hierarchical inference

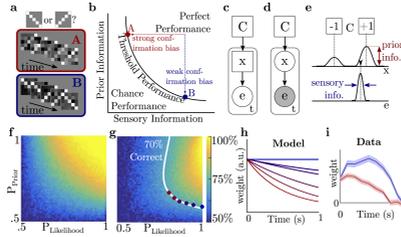
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When subjects have to make decisions based on a temporal sequence of weak pieces of evidence, they often show a bias towards overweighting early evidence compared to late evidence [1]. In perceptual decision-making experiments, such a 'confirmation bias' (CB) is observed in some studies [2,3] but not all [4,5]. Since these studies differed in species (monkey, human) and modality (vision, auditory) the cause of this difference is unclear. Here, we present an intuitive computational framework that can account for the differences reported in the literature, and that makes psychophysical predictions which are confirmed by preliminary data. Our framework extends the traditional ideal observer model by explicitly accounting for an intermediate sensory representation stage between external stimulus and behavior. This allows us to partition the information in the stimulus about the decision into the information between stimulus and sensory representation, and between sensory representation and decision. We show that existing studies which find a CB have low sensory information and high decision-related information, with the opposite for those studies that do not find a CB. While exact inference in such a hierarchical system shows no biases, approximate inference algorithms may. We show that performing probabilistic inference using a neural sampling based approximation [6,7] leads to the same pattern of bias as in the data. Finally, we present supporting psychophysical data from a human experiment in which we compare biases for threshold stimuli that only differ in how information is partitioned with respect to the sensory representation.

Figure caption: (a) Example stimuli for coarse orientation task: A: low sensory information but high prior information, B: high sensory information but low prior information. (b) Decomposition of stimulus information. Black line indicates stimuli with identical information (at threshold) trading off sensory and prior information. (c-e) Formalization of hierarchical inference model. (f-g) %correct for exact inference (f) and sampling-based model (g). (h-i) Weighting of evidence over time in model (h) and our preliminary psychophysical data.

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Caption in main abstract

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[W 6] Reciprocity of social influence

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It has been shown that humans use advice from other humans in order to improve their decisions. To this end they integrate their own and their partner’s evidence by taking into account the reliability of their information. At the same time, social interactions are subject to reciprocity, for example in the case of trust, people are more likely to trust those who trust them. Yet, whether social influence and advice taking is reciprocal remains an open question. To address this question, we designed an experiment in which human subjects needed to solve a perceptual decision making task together with a virtual partner. Both the human player as well as the virtual partner first made an initial decision independently and then were allowed to revise their initial decision on the basis of the initial decision of the other player. Finally, the revised decisions of both players were revealed. Participants were made to believe that the virtual partner was another human subject participating in the experiment. We manipulated the amount of influence that the virtual partner took from the participants. Our result show that humans were more strongly influenced by their partner, if they had reciprocally more influence on the decisions of their partner. We then repeated the experiment telling subjects that their partner is a computer. This time the reciprocity of influence on the decision disappeared. These findings can be interpreted in the sense that humans use reciprocity to communicate in social decision making.

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Brain disease, network dysfunction and intervention

[W 7] Pathological phase-amplitude coupling in the subthalamic nucleus cannot be explained by non-sinusoidal oscillations

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Deep brain stimulation in the subthalamic nucleus (STN) is an established treatment for patients with Parkinson's disease (PD). A central finding in the analysis of local field potential recordings from the implanted electrodes is an enhanced level of beta band (13-30 Hz) synchronization. Furthermore, clinical symptoms of the disease have been associated with a pathological phase-amplitude coupling (PAC) between beta and high-frequency oscillations (HFO, 150-400 Hz). Recently, a similar pathological PAC between beta and broadband gamma activity in motor cortex in PD was demonstrated to be fully explained by the non-sinusoidal shape of the beta oscillation waveform¹. In contrast to the commonly assumed functional coupling between two separate oscillators, this suggests that a single non-linear oscillator underlies the beta-gamma PAC. We investigated whether this also applies to the pathological beta-HFO PAC in STN. To this end, we examined local field potential recordings of 12 patients with deep brain stimulation electrodes implanted in STN. Non-sinusoidalness of the beta oscillation waveform was quantified using the sharpness, the steepness and the phase distribution of the beta waveform. The three measures were compared with beta-HFO PAC values of the same data². In contrast to the beta-gamma PAC in motor cortex, we found no significant correlations between non-sinusoidalness and PAC values. This indicates that beta-HFO PAC in the STN likely arises from separate beta and HFO generators that become pathologically synchronized in the context of Parkinson's disease.

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[W 8] More mild epileptic bursts indicate reduced susceptibility to seizures.

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Epilepsy is a disease of hyper-synchronized and excessive brain activity. The most common form of epilepsy is mesial temporal lobe epilepsy (MTLE), where a wide range of epileptiform activity (EA) emerges from hippocampal structures. Here we investigate EA in local field potentials of mice with MTLE. We developed a PYTHON-based detection and classification toolbox for EA analysis, that enables us to distinguish several types of EA-bursts and to quantify their dynamics: Severe EA -bursts were often grouped in clusters. Most of these ictal phases were surrounded by transition states which consisted of high densities of mild EA. The most severe events were typically followed by a depression period, the duration of which increased with the severity of the event. Inter-ictal phases with a higher rate of mild EA lasted longer and at session level the rate of severe and mild EA was anti-correlated. Hence we showed that the presence of mild EA-bursts indicates a reduced susceptibility to seizure-like events.

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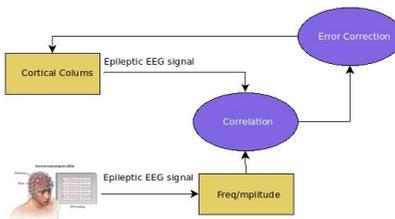
[W 9] A Computational Model To Detect The Occurrence Of Epilepsy Using EEG Signals

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Normally the cortical neurons communicate with each other in a synchronized and systematic spikes, while in epilepsy the spikes will be electrical storm like which spreads all over the cortex with random, excessively higher frequency and voltage. Epilepsy is a very common mental disease which affect about 50 million patient worldwide. One of the suggested treatments for epilepsy is the deep brain stimulation (DBS). Where we can give electrical pulses at certain amount and specific pattern [4] to the internal layers of the brain cortex. However, although this method has a lot of advantages, it has some limitations or disadvantages, other than the surgical operation related problems, which can include: Some abnormalities might been noticed in the speech and ocular functions of the patient. The patient might has some muscle twitching. The feeling of paresthesias. Sometimes the simulation if the stimulation where near emotional related nuclei like the orbital and medial parts of the prefrontal cortex in the brain, this can cause the serious emotional problems, like the feeling of depression, impulsion of even the thinking or desire of suicide. These disadvantages, which are also serious problems, occurs to many reasons, among them are giving the wrong voltage, frequency and pattern of spikes to the patient. In order to optimize the curing process of the patient

we need to improve the DBS method by specifying the value of delivered spikes and their patterns for each patient[6]. So we have first to detect the characteristics of each epileptic EEG signal from several patients. Where the EEG is a medical method to record and track the activity of the brain or part of it, through the electrical signals (spikes) transferred between its parts and neurons. In this project we built a mathematical model to simulate the epileptic seizures, based on Janson's model. Janson's model had proved to be some of the best models to simulate epilepsy. We worked on executing this model using both differential equations and MATLAB SIMULINK, we then justified the obtained (simulated) signal with a real epileptic EEG signals, obtained from physio.net of Yale University, using power spectrum technique. The second step included taking live recorded EEG signals from epileptic patients, and work to change some parameters in our model to make the simulated signals more specific. This can be done by training the simulated signals against real physiological signals using NN.



This figure represents a block diagram for the aim of our project.

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[W 10] Credibility of modeling and simulation for clinical translation

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Multiscale modeling and simulation (M&S) continues to expand the boundaries of biomedical research, and is poised to enter the clinical domain. Direct application of M&S in healthcare is being driven by economic and political pressures, as well as by changes in biomedical and computational sciences. M&S has potential to transform *personalized medicine* – treatments directed to the specific patient, and *precision medicine* – treatments directed to subsets of patients identified by genetic or pathological commonalities. However, clinical application of M&S will require greater credibility and reliability of models than has been needed for basic biological research.

Credibility encompasses model *validation and verification* (V&V). Validation asks to what extent we have developed the right model for the biology that we are tasked with reproducing. Validation is particularly challenging when applying models to human biology and human disease, where model parameters rarely come from humans but instead are gathered using data from tissue culture, *in vivo* animal experiments or *in vitro* preparations. We then must seek human measureables that can be compared to model output. Verification encompasses code verification, where we compare to other instantiations of the model, and solution verification, where we ensure that we developed an adequate numerical solution. Verification of both types can be addressed by looking at model reproducibility, the ability to create an independent implementation.

Modeling of clinical diseases of the nervous system adds additional difficulties that are not seen with modeling of the diseases of other organ systems. One difficulty arises from the greater overlaps of scale embedding in the nervous system. For example, apical dendrites of some neocortical pyramidal cells, which are subcellular in scale, can reach across multiple layers of cortex, engaging the network-scale at different locations with different effects. Additionally, the expression of brain function, and of brain disease, occurs in part at the levels of cognition and behavior which can be difficult to quantify, difficult to monitor and difficult to model. Despite all of these difficulties, computational neuroscience can now begin to meet challenges of clinical credibility through a focus on V&V, replicability, reliability and reproducibility, efforts that are becoming the norm in other branches of computational systems biology.

Acknowledgements

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[W 11] NetPyNE: a Python package to facilitate the development, parallel simulation and analysis of biological neuronal networks in NEURON

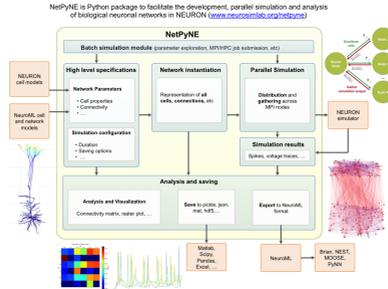
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NEURON is the most widely used multiscale neuronal simulator, as measured by number of publications and available models online. However, building biologically realistic networks and running parallel simulations usually involves a steep learning curve. Additionally, the lack of standardization makes it hard to understand, reproduce and reuse existing models and simulation results. To facilitate these tasks, we developed NetPyNE, a Python package that converts a set of high-level specifications in a standardized, declarative JSON-like format into a parallelized NEURON model. NetPyNE clearly separates model parameters from implementation code. It emphasizes the incorporation of multiscale anatomical and physiological data, including complex spatial distribution of cells, connectivity rules or stimulation patterns. Existing HOC or Python cell models can also be directly imported into NetPyNE. Once the network model is specified, the user can run parallel simulations with a single command which takes care of distributing the workload and gathering data across computing nodes. NetPyNE also facilitates parameter exploration via batch simulations, including pre-defined, configurable setups to automatically submit jobs in multicore machines (Bulletin board) or supercomputers (SLURM/Torque). NetPyNE is available XSEDE supercomputers and the Neuroscience Gateway (NSG). Once the simulation is complete, NetPyNE provides a wide range of visualization analysis functions, including connectivity matrices, 3D representation of network cells, raster plots, spike histograms, power spectra, voltage (or other variables) trace plots, and information measures such as Granger Causality or normalized transfer entropy. To facilitate data sharing, the package saves and loads the specifications, network, and simulation results using common file formats (Pickle, Matlab, JSON or HDF5), and can convert to and from NeuroML, a standard data format for exchanging models in computational neuroscience. The tool website (www.neurosimlab.org/netpyne) includes comprehensive documentation, examples, tutorials, and a Q&A forum. A GUI for the tool is currently under development. NetPyNE is being used in several labs across the world to develop a wide range of multiscale network models including motor cortex, visual cortex, prefrontal cortex, hippocampus, claustrum, or basal ganglia. It is also used by OpenSourceBrain to convert NeuroML models to parallel NEURON and simulate them on supercomputers.



Overview of NetPyNE tool.

Acknowledgements

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[W 12] **Visualization of Neuroscientific Data**

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Neuroscience currently poses many of the most challenging scientific problems. Ambitious initiatives, such as Brain (Jorgenson et al., 2015) or the Human Brain Project (Markram et al., 2011), encourage the collaborative work of multidisciplinary laboratories from different research centres and industry. Technological advances are successfully allowing to obtain experimental data at always higher speed; at the same time, in silico experiments are also generating a huge amount of synthetic data. Therefore, there is a need for new techniques and tools that can help in the exploration and analysis of the generated data, which is already becoming the new bottleneck within the work pipeline due to the overwhelming size of the data to be processed. This poster presents a set of innovative tools for the visualization of neuroscientific data that work in a collaborative fashion (Pastor et al., 2015, Galindo et al., 2016, Garcia-Cantero et al., 2017), creating an ecosystem that provides useful and powerful techniques for the interactive visual exploration and analysis of neuroscientific data.

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[W 13] Deep, generative models of multineuronal activity patterns

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Restricted Boltzmann Machines (RBMs) are energy-based neural network models that perform approximate maximum-likelihood learning of probability distributions, with the learned parameters (or 'weights') of the network taking the form of a generative model for the observed data [1]. With an appropriately chosen network architecture, the hidden units of RBMs easily learn to represent higher-order correlations in the data that are useful as features for downstream applications, such as fine-tuning discriminative models or dimensionality reduction [2, 3]. Inspired by this generative capacity of RBMs, we sought to embed high-dimensional, multineuronal activity patterns recorded by calcium imaging of cortical neurons (from both awake, behaving rodents as well as primary cortical cell culture) into deep autoencoder networks whose weights are initialized with individually-trained RBMs [3]. The resulting low-dimensional codes learned by these deep models capture geometry of the multineuronal 'pattern space' not expressible with linear methods such as Principal Components Analysis (PCA) or Nonnegative Matrix Factorization (NMF). One can also create visualizations of multineuronal activity by either further embedding the weights of the network with other dimensionality reduction algorithms (t-SNE, Isomap, etc.) or by explicitly compressing the bottleneck layer of the network to only 2 or 3 hidden units. We then explore the use of these learned low-dimensional codes and visualizations to elucidate geometric and distributional properties of population patterns with the aim of identifying putative neuronal 'ensembles' (groups of neurons that activate synchronously) [4]. Finally, we relate the geometry and dynamics of the imaged network's ensembles to ongoing behavior and learning, in the case of in vivo recordings, or other biological variables, such as developmental time course of a cell culture (in the in vitro case).

Acknowledgements

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[W 14] The “convis” toolbox: Population Simulation of the Visual System with Automatic Differentiation using Theano

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We developed “convis” [1], a Python toolbox for simulation and efficient fitting of LN-cascade models with large, non-separable spatio-temporal filters and non-linearities of varying complexity. The models are implemented as abstract computational graphs in theano [2] and allow for flexible inspection, optimization and manipulation while computation intensive tasks are executed on a GPU.

Convis is implementing models as a Theano abstract graph of lazy operations, instead of immediate machine instructions, to be able to combine, manipulate and optimize them. By combining layers, complex models can be formulated rapidly. Sub-graphs of any model can be replaced, eg. to exchange a convolution kernel with a reduced parameter version, such as a spline kernel. Also gradients of the output with respect to any of the inputs can be derived by back-propagation and computed as an additional output of the model (automatic differentiation). Optimization modules remove redundancy and simplify the model and decide which operation will be compiled to the CPU and GPU, such that the resulting machine code will run efficiently and numerically stable [2].

Through automatic differentiation and inspectability at run-time, Convis can facilitate parameter exploration, either interactively or by following an optimization method when fitting to data. Computing the first derivative of a parameter is not costly and the penalty of computing second or third derivatives can be justifiable if they speed up the fitting process. In many cases, even very large linear filters can be fitted efficiently to experimental data.

Convis offers a range of pre-build models, such as LN-cascade models with feedback and delays as well as a reimplement of the “VirtualRetina” model [3] including contrast gain control and a spiking mechanism. The models in the toolbox are extensible with additional computational layers, parameterized 3d filter kernels, error functions and optimization routines.

The package is available via Pypi and github [1].

Acknowledgements

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[W 15] Probabilistic Analysis of Two-photon Microscopy Data Using Gaussian Processes

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Two-photon imaging allows light-evoked retinal activity to be measured with a relatively high spatiotemporal precision, and has been used to characterize functional diversity in the mouse retina. It is, nonetheless, constrained: signals are contaminated by optical distortion, photon-shot noise and nonlinear indicator kinetics, and recording sessions are time-limited to avoid bleaching and photo-toxicity, preventing an exhaustive exploration of the stimulus space. The key to advanced downstream analysis or to making predictions to unobserved stimuli is modeling the uncertainty about the signal correctly.

We propose to recover the fluorescence signal using a Bayesian non-parametric method called Gaussian Process (GP) regression, providing a probabilistic estimate of the underlying signal including a model of the uncertainty. Our GP models are fit to calcium and glutamate imaging data in retinal tissue driven by oscillatory stimuli, using a Poisson likelihood function to model photon-shot noise explicitly. We use sparse approximation methods and variational inference to overcome challenges with fitting the GP model parameters to a large number of observations.

The GP models can then be used as the foundation for further statistical analysis: For detecting periods of response differences, one can use GP equality tests which extend inferential statistics from points to functions; effects of stimulus parameters can be tested for in a non-linear ANOVA-like framework; for clustering, the Bhattacharyya distance can be used to describe the similarity of two responses, taking the uncertainty into account. Additional processing steps can be included, accounting for the point spread function of the optical system or the kinetics of the fluorescent indicator.

We wish to use this framework to explore response diversity in the IPL of the mouse retina. In particular, to identify the conditions under which co-stratifying bipolar cell terminals, both on the same and different cells, decorrelate to form distinct functional pathways. Our imaging configuration includes an electrically-tunable lens which allows fast vertical scanning, allowing us to image across multiple layers in the IPL almost simultaneously. The use of parametric stimuli (such as harmonic stimuli with frequency

and contrast parameters) allows us to optimize our stimuli to maximize decorrelation between neurites.

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[W 16] Combining single cell RNA-seq datasets to explore and visualize brain cell variability

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Single cell RNA sequencing is a recently developed technology for obtaining transcriptomes of individual cells from a sample of tissue. It allows to study transcriptomic variability among cells and has become a major tool in cell type discovery, supplementing morphological measurements and electrophysiological recordings. In the last few years, several scRNA-seq datasets of cells sampled from various regions of mouse brains were published, with the number of cells typically ranging from 100s to 1000s in each dataset. Different studies have used different methodologies of single cells isolation, cDNA amplification, and library preparation, making it difficult to combine datasets or to verify to what extent they agree with each other due to the induced “batch effects” between datasets.

Here, we develop computational approaches for combining multiple datasets by removing such “batch effects”, such that the combined data can be used for further dimensionality reduction and/or cell type identification. We found that feature selection can dramatically improve the matching. In particular, we found that selecting only a small number of genes with high expression in a subset of cells and near-zero expression in the remaining cells was a promising strategy, allowing us to obtain a good match of Zeisel et al. 2015, Tasic et al. 2016, and Cadwell et al. 2016 datasets.

However, matching after “batch correction” remains imperfect, and we found that conventional dimensionality reduction methods such as t-SNE can potentially amplify the remaining dissimilarities as they are only aiming at preserving very local information. We are therefore working on adapting t-SNE such that the global arrangement of the resulting clusters is interpretable as well. Our preliminary results show that this can be achieved by using multidimensional scaling to position t-SNE clusters.

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[W 17] A diagrammatic derivation of the TAP-approximation

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Originally invented to describe magnetism, the Ising model has proven to be useful in many other applications, as, for example, inference problems in computer science, socioeconomic physics, the analysis of neural data [1,2,3] and modeling of neural networks (binary neurons). Despite its simplicity, there exists no general solution to the Ising model, i.e. the partition function is unknown in the case of an interacting system. Mean field theory is often used as an approximation being exact in the noninteracting case and for infinite dimensions. A correction term to the mean field approximation of Gibb's free energy (the effective action) of the Ising model was given by Thouless, Anderson and Palmer (TAP) [4] as a "fait accompli" and was later derived by different methods in [5,6,7], where also higher order terms were computed.

We present a diagrammatic derivation (Feynman diagrams) of these correction terms and embed the problem in the language of field theory. Furthermore, we show how the iterative construction of the effective action used in the Ising case generalizes to arbitrary non-Gaussian theories.

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[W 18] **Nonlinear filtering for point emission processes**

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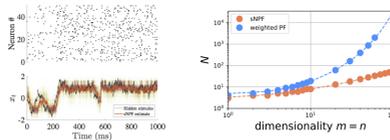
The number of neurons that can be simultaneously recorded doubles every 7 years [1]. This ever increasing number of recorded neurons opens the possibility to address new questions and extract higher dimensional signals from the recordings. It is however unclear how to extract dynamical analog signals from point emission observations when the dimensionality is high. Indeed, traditional particle filter (PF) methods that rely on importance weights can solve this task numerically, but are known to suffer from the curse of dimensionality (COD), i.e. an exponential growth of number of particles with problem dimensionality. Here, we propose the spiking Neural Particle Filter (sNPF), a weight-free PF that extends earlier work [2] to account for point-emission processes and that holds the promise of avoiding the COD.

The decoding problem is formalized as a filtering problem, in which the hidden stimuli $\mathbf{x}_t \in \mathbb{R}^n$ follow an Ito stochastic differential equation (SDE) with nonlinear drift function $\mathbf{f}(\mathbf{x}_t)$, giving rise to m spike trains $s_t = d\mathbf{N}_t/dt$ with instantaneous firing rates $\mathbf{g}(\mathbf{x}_t, t) \in \mathbb{R}^m$. The rate function is kept general, and may reflect biophysical properties such as weighted input summation, or spiking-history dependence via a time-dependent postsynaptic potential. Stochastic filtering is the task of finding the posterior probability density of the hidden stimuli conditioned on the whole spiking history. The solution to this filtering problem is analytically intractable [3].

The sNPF approximately solves this task by propagating equally-weighted particles $\mathbf{x}_t^{(k)}$, representing empirical samples of the posterior, according to the SDE:

$$d\mathbf{x}_t^{(k)} = \mathbf{f}(\mathbf{x}_t^{(k)}) dt + W_t \left(d\mathbf{N}_t - \mathbf{g}(\mathbf{x}_t^{(k)}) dt \right) + \Sigma_x^{1/2} d\mathbf{v}_t^{(k)}.$$

This dynamics is closely related to the dynamics of the first posterior moment [3,4]. The gain W_t determines the emphasis that is laid on the output of the neurons for decoding. We computed W_t as in [4]. We demonstrate that the sNPF can successfully track a hidden stimulus based on spike trains of neurons and that it holds the promise of avoiding the COD (Figure). The favorable scaling with dimensions opens the possibility to accurately estimate high-dimensional signals from a large number of simultaneously recorded spiking neurons.



Left: The filter estimate (orange) tracks a hidden stimulus (black) with nonlinear dynamics, based on a spike train (top left). Shading reflects particle density. Right: Number of particles N for fixed performance scales linearly with dimensionality for the sNPF, and exponentially for a weighted PF.

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[W 19] Measuring the distance of neural morphologies using graph features

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The morphology of neurons is typically considered a defining feature of neural cell types. For example, 14 types of bipolar cells can be discriminated in the mouse retina based on their morphology (Helmstaedter et al. 2013, Kim et al. 2014, Greene et al. 2016), leading to a classification in good agreement with genetic and physiological data (Shekhar et al. 2016, Franke et al. 2017). Similarly, many retinal ganglion cells can be discriminated on morphological terms (Sumbul et al., 2014). Given recent advances in automatic reconstruction and crowd-based tracing techniques, the amount of available data is rapidly increasing (see e.g. www.neuromorpho.org). However, machine learning methods to automatically discriminate or cluster neurons rely on fairly simple representations of neural morphologies, discarding much of the richness of the three-dimensional morphology. Typically, these methods consider the neurite density in three dimensions or low dimensional projections thereof and measure the similarity between two neurons by the euclidean distance of these densities. For retinal neurons, this procedure has been used by analyzing the axon density of bipolar cells or the dendrite density of ganglion cells as a function of IPL depth. However, all fine details contained in the morphological reconstructions such as branching patterns are discarded. Here we propose to measure neuron similarity by retaining the original representation as a tree in the graph theoretical sense and comparing structural differences based on graph statistics. We investigate which similarity measures based on graph features allow reliable discrimination of neural types in the retina and how they can be combined with existing methods to improve discrimination and clustering of neural types.

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[W 20] How to infer distributions in the brain from subsampled observations.

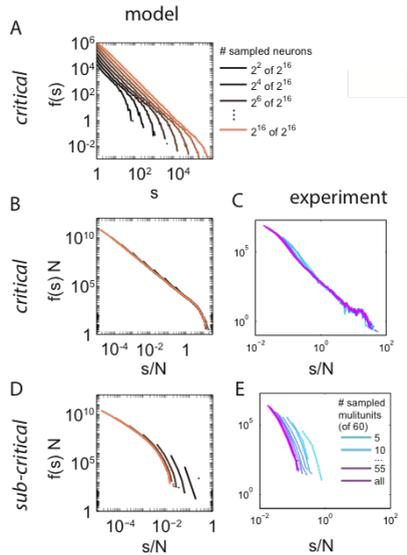
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Inferring the dynamics of a system from observations is a challenge, even if one can observe all system units or components. The same task becomes even more challenging if one can sample only a small fraction of the units at a time. As the prominent example, spiking activity in the brain can be accessed only for a very small fraction of all neurons in parallel. These limitations do not affect our ability to infer single neuron properties, but it influences our understanding of the global network dynamics or connectivity. Subsampling can hamper inferring whether a system shows scale-free topology or scale-free dynamics (criticality) [1,2]. Criticality is a dynamical state that maximizes information processing capacity in models, and therefore is a favorable candidate state for brain function. Experimental approaches to test for criticality extract spatio-temporal clusters of spiking activity, called avalanches, and test whether they followed power laws. These avalanches can propagate over the entire system, thus observations are strongly affected by subsampling. Therefore, we developed a formal ansatz to infer avalanche distributions in the full system from subsampling using both analytical approximation and numerical results.

In the mathematical model subsampling from exponential (or, more generally, negative binomial distribution) does not change the class of distribution, but only its parameters. In contrast, power law distributions, do not manifest as power laws under subsampling [3]. We study changes in distributions to derive “subsampling scaling” that allows to extrapolate the results from subsampling to a full system: $P(s) = p_{sub} P_{sub}(s/p_{sub})$, where $P(s)$ is an original distribution, P_{sub} – distribution in the subsampled system, p_{sub} probability to observe any particular event. In the model of critical avalanches subsampling scaling collapses distributions for all number of sampled units (Figure 1. B). However, for subcritical settings no distribution collapse is observed (Figure 1. D). With the help of this novel discovery we studied dissociated cortical cultures. We artificially subsampled recordings by considering only fraction of all electrodes. We observed that in the first days subsampling scaling does not collapse distributions well, whereas mature (from day 21) allow for a good collapse, indicating development toward criticality (Figure 1. C, E) [4].



Subsampling scaling in model and experiment. Left: model; right: experiments on developing cultures. A: Avalanche size counts $f(s)$, full and subsampled critical models; N - number of sampled neurons. B-C: Subsampling scaling, all $f(s)$ collapse. D, E: No collapse for subcriticality

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[W 21] **Introducing Computer Vision methods to model retina response in Retina Prosthesis applications**

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Retina Prosthesis (RP) is an approach to restore vision, using an implanted device to electrically stimulate the retina. A fundamental problem in RP is to translate the visual scene to retina neural spike patterns, mimicking the computations normally done by retina neural circuits. Starting with an observed scene, in the form of an image, an accordingly devised model is employed, to produce a spatial and temporal pattern of neural activation, or simply put spikes in each implanted electrode.

In the present work, we start from two observations. Firstly, that using raw intensity values has often been insufficient in artificial vision problems, yielding poor results. Currently, implants process the images in such an intensity-based manner, translating the scene raw intensity to stimulation intensity in a proportional fashion.

Secondly, we note the recent congruence of evidence on Retina Ganglion Cell (RGC) functions. We propose to use Computer Vision (CV) methods, to introduce novel visual scene representations, which correspond to RGC functions. Based on recent literature [1, 2, 3, 5, 6] on functional RGC types, DoG filters, edge detection algorithms, optical flow algorithms and statistical measures of spatio-temporal uniformity as entropy and variance are the proper CV methods to model RGC functions. We introduce a novel visual input representation with CV features, using the aforementioned CV methods.

We use CV features as stimulus in a GIF neuron model [4] to reproduce the retina spiking output. A retina simulator [7] provides the stimulus-retina response data to train and test our model. Initial results show that our models achieve significant train set convergence. We evaluate spike train similarity using two measures, Interspike-Interval distance and SPIKE distance. We reject models that sustain a constant firing rate, regardless of the stimulus presented. As the retina simulator used produces different spike trains at each presentation of a specific stimulus, we quantify the simulator's intrinsic variability.

In conclusion, we propose a CV image preprocessing method to model RGC functions and then use the method to reproduce retina output with a standard GIF neuron model. In the future, we aim to further evaluate the preprocessing methods and models presented in this work and pursue to train and test these methods and models on biological retina data.

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Learning, plasticity and memory

[W 22] Storage of memory sequences in the hippocampal circuit

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Despite extensive research, the role of the hippocampus in episodic memory storage and recall is still unclear. We have recently proposed that episodic memories are best represented by temporal sequences of neural activation patterns and that the hippocampal circuit is optimized to store these sequences. Here, we study the possible mechanisms by which memory sequences can be stored and recalled from the cortico-hippocampal circuit, consisting of the EC-CA3-CA1-EC loop. Storing sequence presents entirely different challenges from storing static patterns. During memory encoding, CA3 sequences are hetero-associated with EC sequences, which are driven by sensory inputs. CA3 sequences are generated either intrinsically or via blending the EC inputs and CA3 recurrent inputs. During memory retrieval, CA3 sequences have to be reactivated based on partial, noisy cues, which are provided to EC. The retrieved sequences in CA3 then reactivate the stored patterns in EC via the CA1 layer. We find that memory performance depends on the network's ability to perform pattern completion of individual patterns and robust retrieval of sequences from CA3. These two functions have competing requirements. Modeling CA3 as a fixed randomly connected network facilitates decoding, but sequence retrieval in CA3 fails if any noise is present. On the other hand, using a fixed locally connected network, the stored sequences are retrieved robustly, but the correlations between successive patterns impair pattern completion when decoding the CA3 patterns. Combining the advantages of both models, networks trained on sequences of uncorrelated patterns achieve a good overall memory performance because sequences in CA3 are encoded robustly and do not impair decoding in the feedforward connections to CA1 and EC. In conclusion, the cortico-hippocampal circuit can robustly store and retrieve sequences of patterns, but memory performance critically depends on the network architecture in CA3.

Acknowledgements

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[W 23] Network consolidation during sleep in a spiking neural network

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Sleep is essential for the formation and consolidation of long-term memories in neural circuits. Besides the mechanisms of synaptic and systems consolidation [1], several experimental studies indicate that during sleep several synapses are strengthened while others are weakened resulting to an enhancement of memory traces [2,3]. However, the underlying processes of this enhancement are mainly unknown. Based on our previous work [4], in this study we show that sleep-induced sharp-wave ripples triggers synaptic plasticity and that, in combination with synaptic scaling, synaptic connections are selectively scaled up or down depending on their relevance for long-term storage. In our theoretical model, we use a spiking neural network with calcium-dependent synaptic plasticity [5], which yields LTP and LTD, and activity-dependent homeostatic synaptic scaling [6]. During wake, external stimuli cause synaptic changes forming a feed-forward structure representing a memory trace. Afterwards, during sleep, slow-wave oscillations trigger mainly synaptic scaling inducing downscaling. Furthermore, noise-induced sharp-wave ripples are generated depending on the nonlinear amplification of synchronous inputs in the dendritic trees [7]. These ripples propagate through subparts of the network inducing mainly LTP. Our analyses show that the probability of ripple generation increases significantly if the sub-network contains a memory trace. Furthermore, the strength of the memory trace influences the ripple generation such that synapses being part of strong memories are more strengthened than synapses being in weak memories. Thus, on average, strongly encoded memory traces are enhanced or consolidated, while weaker memories and memory-unrelated synapses are attenuated. In summary, our theoretical model shows how different sleep-dependent activities trigger distinct synaptic mechanisms such that the memory traces encoded in a neural network are actively sorted into memories, which have to be consolidated, and memories, which have to be forgotten.

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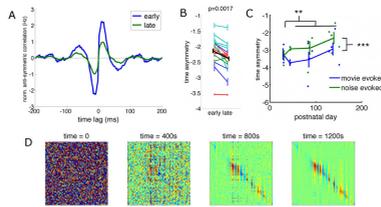
[W 24] The non-sequential state of cortical circuits

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A fundamental task of the brain is learning causal effects between sensory stimuli, by detecting their systematic temporal order. It has been shown that sequential activation of neurons reflects the learned temporal order of experienced stimuli (ref. 1). However, it is not clear how neurons respond to stimuli that have no systematic temporal order, in which forward and backward input sequences balance, and the input is effectively non-sequential. We hypothesize that, after waiting a sufficiently long time, the brain should learn the input statistics and display non-sequential neural activity. Using multi-electrode neural recordings in the primary visual cortex of awake ferrets, here we show that cortical responses actively adapt to produce non-sequential outputs. When subjects are shown a natural movie, to which they are adapted, neural activity is nearly non-sequential; When they are shown unfamiliar artificial noise, neural activity is sequential at first, but then it converges to a nearly non-sequential state within a few minutes (Fig.1a,b). Furthermore, this difference between responses to natural and artificial stimuli was not present at eye opening but developed over several days (Fig.1c). In order to understand the neural mechanisms underlying this adaptation, we studied a computational model of neural circuits dynamics undergoing spike timing-dependent plasticity. We proved that the most celebrated principles of synaptic organization, those of Hebb (ref 2,3) and Dale (ref 4,5), are necessary and sufficient for the maintenance of non-sequential activity (Fig.1d). We show that when these principles are violated, even non-sequential inputs can produce sequential outputs. These results reveal a new functional role of ubiquitous properties of neural circuits in ensuring the preservation of temporal sequentiality of cortical responses and thereby providing a dynamical substrate for the reliable learning of temporal information.



A) Anti-symmetric part of cross correlation is lower after learning in one subject. B) Asymmetry decreases in nearly all 16 subjects. C) Asymmetry is larger for noise vs movie, but no difference at eye opening (day=30). D) Model synaptic matrix converges to a Hebb and Dale structure via STDP.

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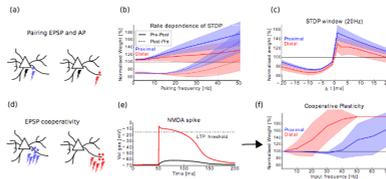
Cite as: Bernacchia A, Fiser J, Hennequin G, Lengyel M (2017) The non-sequential state of cortical circuits. *Bernstein Conference 2017 Abstract*. doi: [10.12751/nncn.bc2017.0044](https://doi.org/10.12751/nncn.bc2017.0044)

[W 25] Beyond spike-timing-dependent plasticity: a computational study of plasticity gradients across basal dendrites

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Synaptic plasticity is thought to be the principal mechanism underlying learning in the brain. Models of plastic networks typically combine point neurons with spike-timing-dependent plasticity (STDP) as the learning rule. However, a point neuron does not capture the complexity of dendrites, which allow non-linear local processing of the synaptic inputs. Furthermore, experimental evidence suggests that STDP is not the only learning rule available to neurons. Implementing biophysically realistic neuron models, we studied how dendrites allow for multiple synaptic plasticity mechanisms to coexist in a single cell. In these models, we compared the conditions for STDP and for the synaptic strengthening by local dendritic spikes. We further explored how the connectivity between two cells is affected by these plasticity rules and the synaptic distributions. Finally, we show how memory retention in associative learning can be prolonged in networks of neurons with dendrites.



Plasticity gradient along basal dendrites.

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[W 26] Recurrent amplification in a grid-cell network

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Grid cells are neurons of the medial entorhinal cortex that are tuned to the animal's position in the environment and whose firing fields form a hexagonal grid pattern in space [1]. Since their discovery, grid cells have been studied extensively, for the striking regularity of their patterns, and because they are believed to support high-level cognitive tasks such as self-location, memory, and navigation [2,3]. Nevertheless, to date, it remains unclear how grid-cell activity is formed and how grid cells interact within the cortical network.

Interestingly, grid cells are organized in discrete functional modules that are characterized by similar spatial scales and orientations [4]. Because cells of the same module tend to respond in concert to external manipulations of the environment [4,5] and their spiking activity is temporally correlated [6], grid cells are thought to be recurrently connected [7]. Yet the functional role of such recurrent connections is still debated. On the one hand, attractor models use structured recurrent connectivity to generate grid fields [8], but it is unclear how such a connectivity could emerge without an anchor to the physical space. On the other hand, feed-forward models can generate grids from spatially-selective inputs [9,10], but they largely neglect the recurrent dynamics within modules.

Here we propose that broadly-tuned grid patterns could first be learned via spatially-irregular feed-forward inputs and then sharpened or amplified by the recurrent connections. To evaluate this hypothesis, we propose a minimal mathematical model of the neural activity within a grid-cell module. Using both analytical and numerical methods, we study the conditions in which grid patterns could be recurrently amplified, and we quantify the amount of amplification that is obtained as a function of the connection strengths and the quality of the input tuning. Finally, we show how a connectivity structure suitable for amplification could spontaneously emerge from the activity correlations already present at the feed-forward input.

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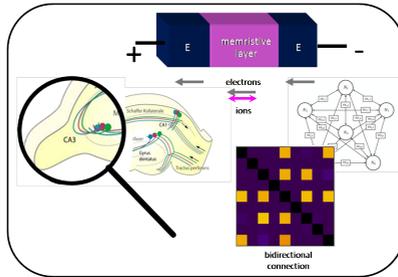
[W 27] Emulation of hippocampal functionalities by memristive Hebbian Plasticity

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Since the early days of Solid State Electronics, there have been considerable attempts to emulate the functioning of mammalian brains by analogue and digital circuits. Recent developments in the research area of solid-state device physics open new perspectives which allow the design of neural circuits that come closer to their biological counterpart. This so-called memristive devices are characterized by a resistance, which depends on the previous applied electrical potentials. Based-on the challenging behaviour of memristive devices, this contribution aims to investigate the possibilities of memristive systems to emulate cognitive functionalities and computations which occur in the cortex and hippocampus of the human brain. Learning and memory processes within hippocampal circuits are regulated by synaptic plasticity mechanisms that rely on variable activity dependent changes in the connection between individual neurons such as spike-timing-dependent plasticity (STDP). The volatility and history dependence of memristive systems are here used to incorporate a voltage-based plasticity rule suitable to account for a variety of experimental data on STDP. The here presented model also enables the formation of bidirectional connections and the formation of local receptive fields in auto-associative networks. The obtained network performance is discussed in the framework of synaptic plasticity and network architecture in the hippocampus underlying distinct cognitive functions.



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[W 28] Bistable activation of CA3 interneurons regulates the generation of sharp wave-ripple events

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Hippocampal sharp wave-ripples (SWR) are spontaneous, oscillatory extracellular events, which reflect the synchronous activation of large neuronal ensembles. They are thought to mediate the consolidation of explicit memories and are known to originate in the CA3 region. However, the mechanisms underlying their occurrence remain to date unclear.

Experiments both *in vivo* and *in vitro* have shown that pyramidal cells and different types of interneurons are involved in the generation of SWR, and that these cells preferentially fire in different parts of the cycle. Pyramidal cells (PYR) have low activity during SWR, and only a small fraction of them fires once during an event. Fast-spiking, parvalbumin-positive basket cells (PV⁺BC) are mainly active during SWR but almost silent outside them, whereas other subgroups of GABAergic interneurons are known to reduce their spiking activity during a SWR event (see e.g. [1]). Recent experimental evidence suggests that a subpopulation of SOM⁺ cells belongs to the latter group. Interestingly, the activation of PV⁺BC triggers a SWR event [2], and the stimulation of a single pyramidal cell activates the interneuronal network which drives the generation of a SWR [3].

To explain the controversial contribution of inhibition to the generation of SWR, we study how a network comprising an excitatory population (PYR) and two types of inhibitory cells (PV⁺BC, and a group of tonically firing interneurons, SOM⁺) can rapidly generate the strong build-up of activity required to initiate a SWR event. In a rate-based model, we derive bistability conditions that enable the system to switch from an 'outside-SWR' state, in which the network is dominated by the active SOM⁺ cells (PV⁺BC and PYR are mostly inactive), to an 'inside-SWR' state, in which the

activation of PV⁺BC triggers the disinhibition of PYR via suppression of SOM⁺ cells. We hypothesize that a synaptic depression mechanism in the connections from PV⁺BC to SOM⁺ cells influences the termination of the 'inside-SWR' state and the occurrence of successive SWR. Furthermore, we compare the behavior of the rate-based model with simulations of a network of leaky integrate-and-fire neurons. The 'outside-SWR' and 'inside-SWR' states are here characterized by a balanced regime of PYR and the active interneuronal populations (SOM⁺ cells and PV⁺BC, respectively).

Acknowledgements

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[W 29] Associative properties of a structural plasticity rule based on firing rate homeostasis

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The interaction between Hebbian and homeostatic plasticity in neuronal networks has recently received a lot of attention. Hebbian synaptic plasticity like STDP, known for its associative properties, leads to instabilities in recurrent networks, and different homeostatic mechanisms have been proposed to stabilize the learning process. While slow homeostatic plasticity has been observed in experiments, a mechanism fast enough to compensate instabilities on short time scales remains to be found [1]. The goal of this work is to contribute another aspect to the understanding of this interaction: could associative properties also emerge from a rule based on homeostatic principles [2]? We consider the maturation of networks in the primary visual cortex (V1) of mice as an example. In contrast to the situation right after eye-opening, neurons in adult V1 are more likely to connect to other neurons that have similar preferred orientations (PO) [3]. We simulate this maturation process in a recurrent network of leaky integrate-and-fire neurons, in which excitatory to excitatory connections are subject to a structural plasticity rule based on the homeostasis of firing rates [4,5]. We found that upon stimulation that emulates early visual experience [6], the connection probability is indeed modulated according to the PO of neurons. Moreover, we could show that this effect is long-lasting and the emerging structure decays only slowly when the specific external stimulation is turned off. Our results demonstrate very clearly that associative properties can also emerge from a plasticity rule that is only based on firing rate homeostasis in single neurons, and that is not explicitly dependent on correlations between the activity of neurons.

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[W 30] Prediction and control of non-linear dynamics by local stable learning in a recurrent spiking neural network

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The brain needs to construct forward or inverse models of the non-linear dynamics of muscles, limbs and the external world for motor control and planning. How spiking neural networks can learn such models is an open problem, despite significant progress via reservoir computing, FORCE learning, and other methods [Abbott et al., 2016, DeWolf et al., 2016, Denève et al., 2017].

We propose Feedback-based Online Local Learning Of Weights (FOLLOW) [Gilra and Gerstner, 2017] which especially draws from function and dynamics approximation theory [Funahashi, 1989, Eliasmith and Anderson, 2004] and adaptive control theory [Ioannou and Sun, 2012]. Using our FOLLOW scheme, a recurrently-connected network of heterogeneous spiking neurons learns its feedforward and recurrent weights, so as to predict or control a low-dimensional non-linear dynamical system $d\vec{x}/dt = \vec{f}(\vec{x}, \vec{u})$, where $\vec{u}(t)$ is the control input and $\vec{x}(t)$ are the state variables. We derive the learning rules showing global uniform (Lyapunov) stability with the error tending to zero asymptotically, under reasonable assumptions and approximations. The learning rules are synaptically local involving the pre-synaptic firing rate and an error feedback current injected into the post-synaptic neuron.

Using a two-link arm as an example, we show that our network learns a forward predictive model for motor planning i.e. it predicts the joint angles and velocities $\vec{x}(t)$ given joint torques $\vec{u}(t)$, or learns an inverse model for motor control i.e. it infers the torque $\vec{u}(t)$ that would generate a desired state trajectory $\vec{x}(t)$. We further use the inverse model to control the arm to draw on a wall.

With FOLLOW learning, we propose a more biologically plausible, specifically synaptically local, scheme of how the brain may learn forward and inverse models to perform motor

planning and control. Extensions like incorporating Dale's law for further biological plausibility, hierarchical coding and control, semi-supervised learning, and applications to neuromorphic computing and neurorobotics are planned for future work.

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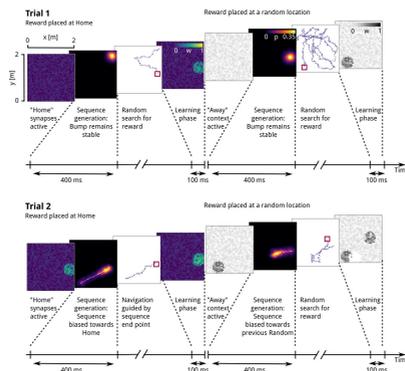
[W 31] Predictive Place-cell Sequences for Goal-finding Emerge from Goal Memory and the Cognitive Map: A Computational Model

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Hippocampal place-cell sequences observed during awake immobility often represent previous experience, suggesting a role in memory processes [1]. However, recent reports of goals being overrepresented in sequential activity suggest a role in short-term planning [2], although a detailed understanding of the origins of hippocampal sequential activity and of its functional role is still lacking. In particular, it is unknown which mechanism could support efficient planning by generating place-cell sequences biased towards known goal locations, in an adaptive and constructive fashion. The hypothesis that certain forms of sequential activity can guide behavior implies specific properties of the sequence-generating mechanism. First, for efficient behavioral guidance, sequence trajectories should be task-dependent, depicting currently relevant trajectories preferentially. Second, trajectories should include novel combinations of start and end points when necessary. These conditions are not easily met by most existing computational models of sequential hippocampal activity. To fill this gap, we present a model of place-cell sequences, implemented in a large-scale spiking network with physiologically interpretable parameters, in which goal learning by reward-based plasticity shapes the sequence generation process, and in which sequential activity guides spatial behavior [3]. In our model, following reward-based potentiation of cortico-hippocampal synapses, prefrontal contextual representations

bias hippocampal recall activity, which progresses sequentially across the cognitive map-like network structure towards a context-specific goal location. Importantly, sequence trajectories neither replicate previous experiences nor follow virtual directional signals, but rather emerge as an effect of intrinsic network dynamics biased by goal-specific inputs. The resulting place-cell sequences are used to guide the behavior of a virtual rat in a memory-guided decision-making task. Furthermore, the implementation as a large spiking network showing ripple-band oscillations allows to employ a Bayesian decoding approach as used in experimental studies [2,4,5]. Simulations show that this model (1) explains the generation of never-experienced sequence trajectories in known environments, (2) accounts for the bias in place-cell sequences towards goal locations, (3) highlights their utility in flexible route planning, and (4) provides specific testable predictions.



Development of synaptic weights, sequential activity and goal-directed behavior. Time course of the first two simulated trials showing the evolution of context-to-DG synaptic weights, place-cell sequences and behavior. From left to right: Context-to-DG weights, decoded sequence, movement trajectory.

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[W 32] Postsynaptic Activity-Dependent Synaptic Scaling Enables the Functional Organization of Memories

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As known from everyday life, humans are permanently exposed to a variety of sensory inputs from their environment. Thereby, the ongoing challenge, humans have to deal with, is to continuously and adaptively respond to these sensory stimulations. On the neuronal level, modification of synapses (interface between two neurons) is a weighty mechanism for adapting the response properties of neurons according to their external stimulation. Hereby, synaptic plasticity is the main mechanism underlying learning [1-4] and, in combination with a homeostatic mechanism, yields the formation of strongly interconnected subgroups of neurons [5-7], so-called Hebbian cell assemblies (CAs) [1]. Such a CA represents the learned memory trace of the corresponding environmental stimulus [1]. Moreover, dependent on the details of the stimuli, humans exhibit the remarkable ability to organize memories (i.e. CAs), thus, to connect, generalize, and discriminate them, which supports the integration of novel stimuli and enables complex behavior [8,9]. How these memory organizations are realized on a neuronal level based on the idea of cell assemblies is still unknown. In a theoretical neuronal network model, we first analyze its respective dynamics in a mean-field model of two interconnected, homogeneous populations of neurons. These populations serve as memory representations on the neuronal level (i.e. CAs; strong synaptic weights). Given different synaptic learning rules for rate coded neurons [6,7,10,11], we analyze their abilities to dynamically organize two memories. Our analyses show that, the learning rule of Hebbian synaptic plasticity in combination with a postsynaptic activity-dependent synaptic scaling mechanism (SPaSS-rule, [6,7]) enables this functional organization of memories. Here, dependent on the stimulation protocol, the CAs can be associated, discriminated, or can form a sequence. Second, to verify our predictions of the mean-field analysis, we simulate the neuronal dynamics of a recurrent neuronal network using the SPaSS learning rule. In doing so, we can reproduce the learning rule's ability to build up the aforementioned different functional organizations of memories. In summary, this work reveals a neuronal network model whose dynamics underlie Hebbian plasticity in combination with a postsynaptic activity-dependent scaling mechanism and that is capable to exhibit different functional organizations of memories observed in human behavior.

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[W 34] Environmental enrichment accelerates ocular dominance plasticity in mouse visual cortex whereas transfer to standard cages resulted in a rapid loss of increased plasticity

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In standard cage (SC) raised mice, experience-dependent ocular dominance (OD) plasticity in the primary visual cortex (V1) rapidly declines with age: in P25-35 (critical period) mice, 4 days of MD are sufficient to induce OD-shifts towards the open eye; thereafter, 7 days of MD are needed. Beyond P110, even 14 days of MD failed to induce OD-plasticity in mouse V1 (Lehmann & Löwel, 2008; Espinosa & Stryker, 2012). In contrast, mice raised in a so-called “enriched environment” (EE), exhibit lifelong OD-plasticity (Greifzu et al., 2014; 2016). EE-mice have more voluntary physical exercise (running wheels), and experience more social interactions (bigger housing groups) and more cognitive stimulation (regularly changed labyrinths or toys). Whether experience-dependent shifts of V1-activation happen faster in EE-mice and how long the plasticity promoting effect would persist after transferring EE-mice back to SCs has not yet been investigated. To this end, we used intrinsic signal optical imaging to visualize V1-activation i) before and after MD in EE-mice of different age groups (critical period: PD24-35, young: PD90-104 and adult: PD117-283 mice) and ii) after transferring mice back to SCs after P130. Already after 2 days of MD, and thus much faster than in SC-mice, EE-mice of all tested age groups displayed a significant OD-shift towards the open eye. Transfer of EE-mice to SCs immediately abolished OD-plasticity: already after 1 week of SC-housing and MD, OD-shifts could no longer be visualized in EE-mice. In an attempt to rescue abolished OD-plasticity of the EEtoSC-mice, we either administered the anti-depressant fluoxetine to the mice (in drinking water) or supplied a running wheel (RW) in the SCs. OD-plasticity after MD was only rescued for the

RW-mice. Altogether our results show that raising mice in less deprived environments like large EE-cages strongly accelerates experience-dependent changes in V1-activation compared to SC-raising. Furthermore, preventing voluntary physical exercise of EE-mice in adulthood immediately precludes OD-shifts in V1.

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[W 35] Respiratory entrainment of memory circuits

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Decades of research have identified neural oscillations as a mechanistic substrate for the formation of cell assemblies and the coordination of information transfer between remote brain regions. During exploratory behavior, the hippocampus and the prefrontal cortex are organized by theta oscillations, known to support memory encoding and retrieval, while during sleep the same structures are dominated by slow oscillations that are believed to underlie the consolidation of recent experiences.

Although most known neural oscillations are generated by intra-cerebral pacemakers and circuits, here we focused our attention to breathing, the most fundamental and ubiquitous rhythmic activity in life. We report respiratory entrainment of limbic circuits, including the prefrontal cortex and hippocampus, two structures critically involved in memory consolidation and retrieval.

Using a combination of extracellular recordings using high-density silicone probes, calcium imaging, photometry, pharmacological and optogenetic manipulations in mice, we identify that a rhythmic oscillation (2-6 Hz and termed respiratory θ rhythm) entrains neuronal activity across structures. We characterize the translaminar and transregional profile of the respiratory entrainment of the prefrontal cortex and hippocampus and demonstrate a causal role of re-afferent respiratory inputs in synchronizing neuronal activity and network dynamics between these structures in a variety of behavioral scenarios in the awake and sleep state. Prefrontal 4Hz oscillations, recently identified as a physiological signature of fear memory in mice, are a manifestation of the differential cortical entrainment by the respiratory θ rhythm during behavior.

Our results highlight respiration, a persistent rhythmic input to the brain, as a novel oscillatory mechanism mediating inter-regional synchronization of limbic memory circuits and contributing to the formation and expression of neuronal ensembles.

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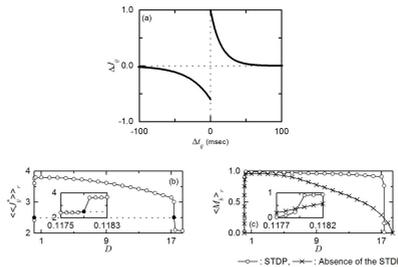
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[W 36] Stochastic Burst Synchronization in A Scale-Free Neural Network with Spike-Timing-Dependent Plasticity

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We consider an excitatory population of subthreshold Izhikevich neurons which cannot fire spontaneously without noise. As the coupling strength passes a threshold, individual neurons exhibit noise-induced burstings. This neuronal population has adaptive dynamic synaptic strengths governed by the spike-timing-dependent plasticity (STDP) [1]. In the absence of STDP, stochastic burst synchronization (SBS) between noise-induced burstings of subthreshold neurons was previously found to occur over a large range of intermediate noise intensities through competition between the constructive and the destructive roles of noise [2]. Here, we study the effect of additive STDP on the SBS by varying the noise intensity D in the Barabasi-Albert scale-free network (SFN) with symmetric preferential attachment with the same in- and out-degrees [3]. This type of SFN exhibits a power-law degree distribution (i.e., scale-free property), and hence it becomes inhomogeneous one with a few "hubs" (i.e., super-connected nodes). Occurrence of a "Matthew effect" in synaptic plasticity is found to occur due to a positive feedback process. Good burst synchronization gets better via long-term potentiation (LTP) of synaptic weights, while bad burst synchronization gets worse via long-term depression (LTD) [see Fig. 1]. Consequently, a step-like rapid transition to SBS occurs by changing D , in contrast to the relatively smooth transition in the absence of STDP. Emergence of LTP and LTD of synaptic weights are investigated in details via microscopic studies based on both the distributions of time delays between the nearest burst onset times of the pre- and the post-synaptic neurons and the pair-correlations between the pre- and the post-synaptic IIBRs (instantaneous individual burst rates). We also investigate the effect of network architecture on SBS for a fixed D in the following two cases: (1) variation in the symmetric attachment degree and (2) asymmetric preferential attachment of new nodes with different in- and out-degrees. Finally, a multiplicative STDP case depending on the states is also investigated in comparison with the above additive STDP case (independent of the states).



(a) Time window for the STDP. (b) Plot of population-averaged limit values of synaptic strengths $\langle \langle J^*_{ij} \rangle_r \rangle$ versus D . (c) Plot of the statistical-mechanical bursting measure $\langle M_b \rangle_r$ versus D .

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[W 37] Bridging structure and function: a model of sequence learning and prediction in primary visual cortex

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In a recent experiment, Xu et al. (2012) have demonstrated that the primary visual cortex of rats engages in spatio-temporal sequence learning and prediction. Specifically, they conditioned rats with a light spot moving repeatedly across a portion of their visual field. They then examined the training effect by briefly flashing the light spot at the starting location of the sequence. Intriguingly, they found that training enhances the similarity of cue-triggered multi-unit spiking to multi-unit spiking during training. The cellular basis underlying the learning in this and similar studies remains unclear, however. Here, we use a recently introduced spiking neural network model (Miner and Triesch 2016) to show that the interaction of spike-timing dependent plasticity (STDP) and homeostatic plasticity mechanisms can explain these experimental results. Our model reproduces the observed changes in stimulus-evoked multi-unit activity. Furthermore, it predicts how training shapes network connectivity to establish its prediction ability. Finally, it predicts that the altered connectivity induces systematic changes in spontaneous network activity. Taken together, our model establishes a new conceptual bridge between the structure and function of cortical circuits in the context of sequence learning and prediction. In addition, it is the first model to explain the sequence learning ability of cortical circuits while also accounting for non-random structural features of cortical wiring, such as a lognormal-like distribution of synaptic efficacies and an overrepresentation of bidirectional connections.

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[W 38] The Different Roles of Synaptic and Intrinsic Plasticities in Sparse and Expansive Sensory Representations

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Processing and categorizing sensory information are fundamental functions of human and animal brains. To do so, different stimuli have to be mapped on distinct memory representations or activity patterns. However, noisy versions of the same stimulus have to be mapped on the same pattern. Across several species [1-4], the involved sensory areas show a low average activity (sparseness) and the input layer has a smaller number of neurons than the representation layer (expansiveness). In a previous theoretical study [5], the authors argue that sparseness and an expansive structure can support the consistent mapping of noisy stimuli to the same activity patterns. This mapping seems to be optimal if the weights of the synapses from the input layer to the representation layer imply activity patterns being attractors of the network's dynamics [5]. However, up to now, it is unclear how these attractors can be formed in an unsupervised manner. Here, we show that the interaction of several well-known synaptic and neuronal plasticity processes - Hebbian synaptic plasticity [6], homeostatic synaptic plasticity [7], and homeostatic intrinsic plasticity [8,9] - forms the optimal mapping of stimuli to activity patterns. For the proper formation of this mapping, each process has a different distinct role: Homeostatic intrinsic plasticity regulates the representation layer's activity keeping it in a sparse state. Hebbian synaptic plasticity increases synaptic weights between representation layer neurons and related input layer neurons thereby linking stimuli to sensory representations. Homeostatic synaptic plasticity, in turn, decreases synaptic weights between non-related input and representation layer neurons minimizing the chances of undesired activation. Moreover, homeostatic synaptic plasticity in combination with homeostatic intrinsic plasticity stabilizes the overall synaptic and neuronal dynamics. Remarkably, after presenting several different stimuli, the interaction of these processes results in synaptic weights which are similar to the optimal structure [5] and yield comparable stimulus-to-pattern mapping.

In summary, our study shows that the combination of several, generic plasticity processes yields the self-organized categorization of sensory stimuli close to optimal. Furthermore, different homeostatic processes appear to have different functional roles providing a potential explanation for the need of several homeostatic processes in neural circuits [10].

Acknowledgements

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[W 39] A computational model for studying structural changes during tDCS

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Background: Transcranial direct current stimulation (tDCS) is a promising approach to treating diseases like depressive disorder or chronic pain. Long lasting aftereffects were observed after tDCS was turned off. This suggests that plastic changes were induced. The mechanism of the emergence and maintenance of these aftereffects, however, remain elusive.

Objective: This study aims at understanding the structural plastic changes caused by tDCS, with the long-term goal to further improve the performance of this approach in clinical applications.

Methods: To represent the cortical tissue underneath the electrodes, we set up a recurrent network of excitatory and inhibitory neurons, in which excitatory-to-excitatory synapses were established according to a structural plasticity model based on firing rate homeostasis (Butz and van Ooyen, 2013; Diaz-Pier et al., 2016; Gallinaro and Rotter, 2017). In our simulations, tDCS induces a weak transient depolarization or hyperpolarization of the membrane potential of excitatory pyramidal neurons.

Results: Weak deflections of membrane potentials ($\Delta U = 0.1\text{mV}$) lead to a moderate change of ongoing neuronal firing rates ($\Delta \nu \approx 1\text{Hz}$). When a certain proportion of excitatory neurons within the network is stimulated with tDCS, due to the structural plasticity rule, firing rate changes induce also local connectivity changes. Depolarizing tDCS leads to local connectivity increase; the increasing magnitude depends on the relative size of the stimulated population. Hyperpolarizing stimulation and repeated stimulation

patterns with proper relaxation time can boost the process of local connectivity increase in depolarizing stimulation.

Conclusions: Our results demonstrate that cell assemblies form in a recurrent network of spiking neurons subject to structural plasticity and transcranial stimulation that perturbs the homeostatic equilibrium. Repeated stimulation or stimulation with opposite currents can enhance connectivity of the new cell assemblies by influencing the rate with which new synapses are formed. We propose the use of this framework as a tool to study structural changes in neuronal networks caused by transcranial electrical stimulation.

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Motor control, movement, navigation

[W 40] Inbound and Outbound Spikes of Grid Fields

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When a rodent moves, grid cells in its medial entorhinal cortex fire at the vertices of a hexagonal lattice spanning the environment [Hafting et al. 2005]. Runs through these firing fields have been studied by De Almeida et al. [2012]. Based on the spike number on trajectory segments pointing towards or away from the firing field centers, these authors argued that grid cells have prospective and retrospective modes. This finding motivated us to carry out a detailed analysis of the inbound and outbound spikes using the dataset from Latuske et al. [2015]. We found that there are more inbound spikes than outbound spikes. At first sight, this suggests that grid cells mostly code prospectively, similar to findings from hippocampal place cells [Mueller & Kubie 1989; Sharp 1999]. There is, however, also an alternative interpretation as the body position inferred from the tracking system may not coincide with the perceived self-center of the animal. Assuming that the "true" firing fields are ideally suited to guide the animal, we shifted spikes either in space or in time such that field size, spatial information or spatial coherence were optimized. When we then analyzed the spikes, their angular distribution

was almost equalized when spatial shifts were applied, but not for temporal shifts. The size of optimal shifts suggests that the animal's perceived self-center is located directly in front of its head which may support the integration of sensory information.

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[W 41] Investigation of spiking activity in monkey motor cortex during resting state and spontaneous movement

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Modeling studies of cortical network dynamics aim to include realistic assumptions on the neuronal properties [1,2]. However, such models are typically bound to neglect functional aspects that relate to behavior. Rather, they describe the “ground” or “resting state” [3] of cortical networks typically characterized as asynchronous irregular spiking [2]. For model validation, i.e., for a concrete comparison of experimental versus model data, we designed a resting state experiment. We recorded the spiking activity for 15min from macaque monkey (pre)motor cortex during rest, i.e. without any task, using a chronically implanted 4x4mm² 100 electrode Utah Array (Blackrock Microsystems). Based on a video recording of this experiment, we differentiate between “resting” intervals and intervals of spontaneous movements.

In this study we thoroughly characterize the spiking activity during resting and movement state. We identify 146 single units and subdivide them into putative excitatory and inhibitory neurons based on their spike shapes [4]. We estimate their firing rates, (local) coefficients of variation, and their pairwise fine temporal correlations. Comparing the distributions of these measures we find only small differences between our two behavioral states: during movement temporal correlations are more broadly distributed with a lower average value compared to resting periods. Furthermore, we find that putative inhibitory neurons fire faster and more regular compared to putative excitatory neurons in both states. When focusing on single units, we notice that several neurons increase their firing rates systematically when the monkey moves compared to rest, whereas others decrease or do not change their rates. We use cross-correlation histograms (CCH) to identify possible monosynaptic connections and groups of intracorrelated units. In the averaged CCH we observe oscillations of about 30Hz that are especially pronounced in putative inhibitory neurons and during rest. In conclusion, there is relatively little difference between behavioral states on the population level, but clear differences on the level of individual neurons.

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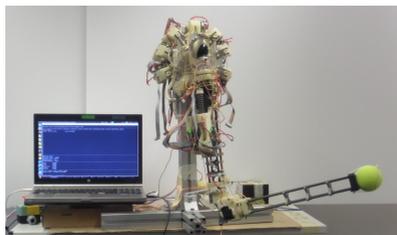
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[W 42] Hebbian Learning Based Sensorimotor Association in a Closed-Loop Neurobotic Experiment

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Learning in the human brain is heavily based on non-supervised, associative learning. Here, Hebbian learning rules [1] can be observed as a generalization of concepts which can be refined in terms of signals types and occurrences e.g. towards Spike-Timing Dependent Plasticity. While on higher layers complex sensory spaces are processed to execute (deliberative) behaviors, on a lower level sensor stimuli inputs directly result into muscle activities. An association between sensor and motor signals in a spatial and temporal relation is learned, or motor behaviors associated to sensor events. This association process is demonstrated in a closed-loop experiment where the sensory space of a biomimetic robotic arm based on [2] is connected to motor commands with a neural network that applies variations of Hebbian learning [3]. In interactive experiments with human users we observe synaptic strengthening between neurons that process occurring sensory and motor events in a spatial as well as temporal context. Learned correlations are stored distributed in synaptic connections between neuron units and represent observed event types, variations and time of occurrences. The internal representation is robust against noise in sensor signal amplitudes. The executed experiments emphasize the potential of Hebbian like learning rules in unsupervised closed loop learning scenarios. While we demonstrate a short circuit experiment with few sensory inputs only, we see the potential of up-scaling to higher neural layers including a variety of sensory domains and hereby sophisticated reactions on complex sensory spaces.



Experiment Platform: A Myrobotics Anthropomimetic Arm

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[W 43] Biophysical Foundation and Function of the Depolarizing Afterpotential in Principal Cells of the Medial Entorhinal Cortex

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Neurons in layer II of the rodent medial entorhinal cortex (mEC) encode spatial information. One particular group - grid cells - tend to fire at specific spatial locations that form hexagonal lattices covering the explored environment. In addition, grid cells show frequent and highly irregular burst episodes within these firing fields. Such burst episodes have received little attention but may contribute substantially to encoding spatial information. In vitro recordings of mEC principal cells have revealed that the action potential is followed by a prominent depolarizing after-potential (DAP). Its biological function, biophysical foundation, and relation to other electrophysiological features are, however, poorly understood. Using a paired-pulse paradigm, we therefore investigated the function of DAPs in vitro and studied how they influence the generation of further action potentials. The intensity of the first current pulse was chosen to elicit an action potential; the intensity of the second pulse was adjusted until a 2nd spike was fired. During the DAP the current needed for generating the 2nd spike was strongly reduced. To investigate the biophysical foundation of DAPs, we modeled mEC principal cells using single-compartment models with Hodgkin-Huxley like ion channels. The model reproduced key cell characteristics: Sag potentials during hyperpolarizing steps, membrane-potential resonance during ZAP stimuli, and the main DAP characteristic: Facilitation of spike generation during the DAP. Visualizing the current kinetics, we found that all currents in the model (Inat, Inap, Ikdr, Ipas, Ih) contribute to the DAP. A reopening of the fast sodium current seems to be necessary for DAP generation. Many layer II neurons show DAPs. However, it is not clear whether these neurons belong to distinct neural subpopulations. Therefore, we performed both supervised and unsupervised statistical data analyses to understand the parameter variation across the population. Our analysis suggests that the existence of a DAP can be predicted by a set of electrophysiological parameters. Yet, our results do not support the idea that the absence or presence of DAPs alone allows for classifications into specific principal cell types. Taken together our results indicate that DAPs facilitate bursting, are not specific

to a certain type of mEC cells, and their origin cannot be attributed to a single ion channel.

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[W 44] The vestibular neural code is driven by robustness rather than efficiency

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Sparse representations of natural stimuli have been shown to be useful models of neuronal population codes in various sensory systems [1]. Here we investigate whether sparse coding of head motion yields efficient representations and whether there is physiological evidence for this coding principle. To this end, we learned a sparse code from recordings of linear acceleration and angular velocity of the human head. The code consists of a set of signal-adapted basis functions, or kernels, that represent temporal patterns of all six degrees of freedom of movement. We found that, although the sparse code could reduce the redundancy in the measured motion data, we could not find highly efficient representations in the same way as in other modalities such as vision and audition. Furthermore, comparing the properties of the learned kernels with those of convergent neurons in the vestibular nucleus (VN) yielded only weak similarities. Based on these results, we argue that efficient encoding of natural stimuli is not the primary focus of the vestibular neural code.

We recorded linear acceleration and angular velocity of head motion from 10 different subjects performing 7 different activities each (running, biking, walking on grass, walking on pavement, playing soccer, walking upstairs, walking downstairs) using an inertial measurement unit. Furthermore, we recorded a second set of data from 3 different subjects over the course of one whole day. Both datasets were used to learn a sparse code with different previously published sparse coding algorithms. The coding capacity was evaluated by encoding a distinct set of test data with the learned kernels and measuring the error of reconstruction from the coded data.

We found that the coding capacity of the learned kernels was significantly lower when compared to basis functions learned from images or sound. Furthermore, we developed several experiments that compare the kernels' properties to those of VN neurons (spatiotemporal tuning, sensitivity to linear acceleration vs angular velocity, sensitivity to gravitational vs translational acceleration), showing only limited similarities.

We argue that there are different reasons for the observed results: the inherent non-linear topological structure of motion cannot be faithfully represented by the linear sparse-coding model and the vestibular system has no behavioral motivation to efficiently encode natural head motion [3].

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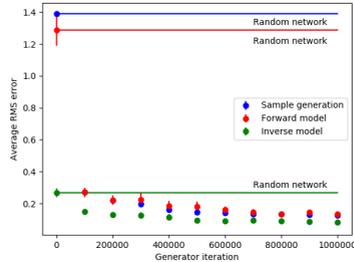
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[W 45] Generative adversarial networks as integrated forward and inverse models for motor control

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Previous studies in computational motor control support the notion that the human brain uses internal models to control voluntary movements. The forward model predicts the future state, while the inverse model computes the control command to achieve a desired state. Everyday human motor behaviors involve the coordination of many limbs and muscles. It remains unclear what kind of algorithm the brain uses for high dimensional internal models. Generative adversarial networks (GAN) were proven successful in generating novel samples from a learned high-dimensional distribution [1]. Moreover, GANs were able to successfully complete missing information in high-dimension samples [2]. Here, we test a GAN as an integrated forward and inverse model for motor control. We used a two-link arm environment as a toy model. The state of each of the two links is defined by an angle and an angular velocity. Control commands affect the angular velocities. First, we trained a deep network \mathbb{G} to encode the dynamics of the system. The arm's pre-act-post states (pre-action state, action and post-action state) were sampled by random motor babbling. In an adversarial training process, \mathbb{G} was trained to generate novel pre-act-posts samples closer to the true distribution, compared to the initial untrained network. Next, we tested whether \mathbb{G} can behave as a forward and inverse model. We provided only partial information to \mathbb{G} (pre-act for forward and pre-post for inverse) to investigate whether \mathbb{G} can complete the missing information. We found that our trained network could more accurately predict the missing variables, compared to untrained networks: post for forward and act for inverse, as required for the internal models. The current study demonstrates that GANs can be used as an integrated forward and inverse model [3] in a simple motor control problem (\mathbb{R}^{14}). As GANs have previously been successfully applied in generating high dimensional data samples, we expect that our method can be extended into control problems in higher dimensions, however, this remains to be shown in future investigations .



Training reduced the error between the true and the generated samples

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Neurons, networks, dynamical systems

[W 46] Analyzing the competition of gamma rhythms with delayed pulse-coupled oscillators in phase representation

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A prominent type of oscillatory activity are gamma rhythms, which may play an important role in neuronal information processing. Two mechanisms have mainly been proposed for their generation: Interneuron Network Gamma (ING) and Pyramidal-Interneuron Network Gamma (PING). Experiments have shown that both mechanisms can exist in the same cortical circuits. This raises the question: how do ING and PING interact when both can in principle occur? Are the network dynamics a linear superposition, or do ING and PING interact in a nonlinear way and if so, how?

To address these questions, we first generalize the phase representation for nonlinear one-dimensional pulse coupled oscillators as introduced by Mirollo and Strogatz to type II oscillators with a phase response curve with zero crossings. We then give a full theoretical analysis for the regular gamma-like oscillations of simple networks consisting of two neural oscillators, an "E-neuron", mimicking a synchronized group of pyramidal cells, and an "I-neuron" representing such a group of interneurons. The phase representation

allows to define in simple manner scenarios of interaction between the two neurons, which are independent of the details of the neuron models. We analytically derive the relevant scenarios and characterize their occurrence.

The networks can be tuned to oscillate in ING or PING mode. We focus particularly on the transition region, where both rhythms compete to govern the network dynamics and compare with oscillations in reduced networks, which can only generate either ING or PING. Our analytically derived oscillation frequency diagrams indicate that except for small coexistence regions, the networks generate ING, if the oscillation frequency of the reduced ING network exceeds that of the reduced PING network, and vice versa. For type I I-neurons the network oscillation frequency slightly exceeds the frequencies of corresponding reduced networks; it lies between them for type II I-neurons. In networks oscillating in ING (PING) mode, the oscillation frequency responds faster to changes in the drive to the I (E)-neuron than to changes in the drive to the E (I)-neuron. The finding suggests a method to analyze which mechanism governs an observed network oscillation. Notably, also when the network operates in ING mode, the E-neuron can spike before the I-neuron such that relative spike times of the pyramidal cells and the interneurons alone are not conclusive for distinguishing ING and PING.

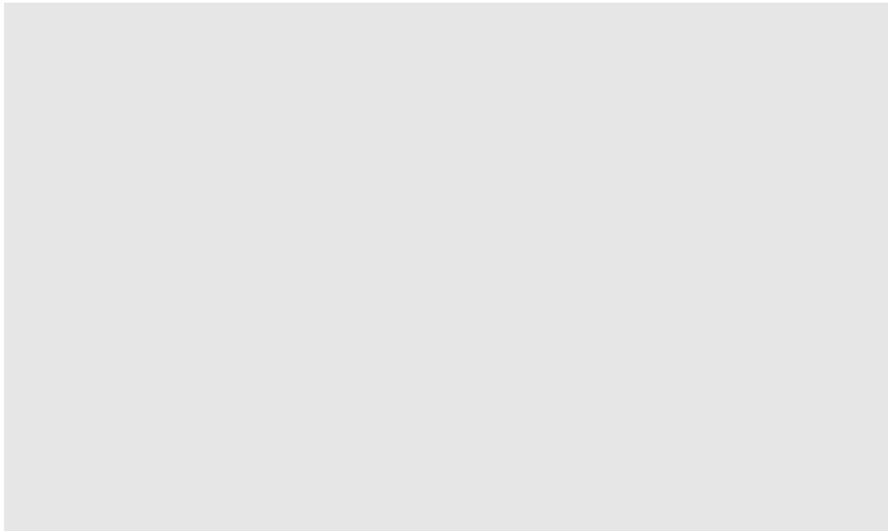
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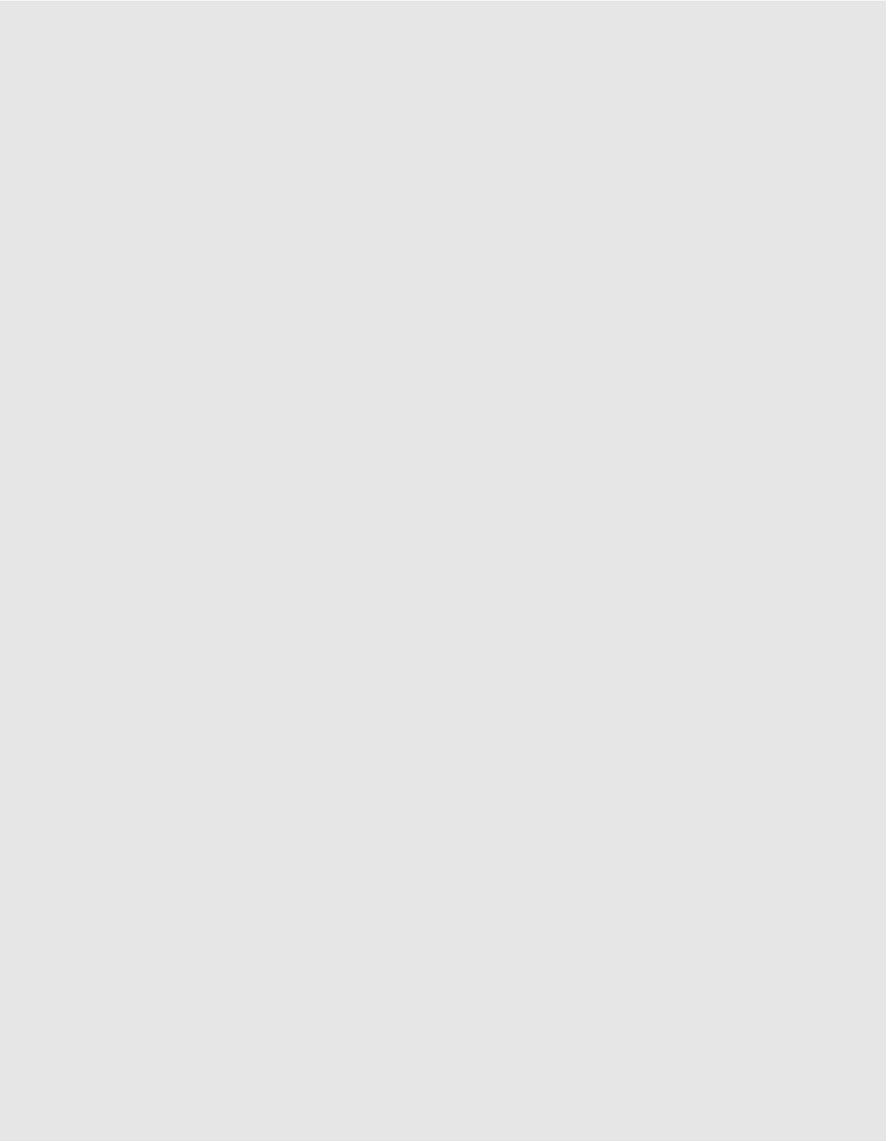
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[W 47] **Withdrawn**





[W 48] Spike rate models derived from recurrent networks of adaptive neurons

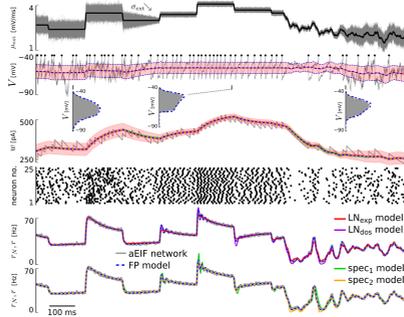
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The spiking activity of single neurons can be well described by a nonlinear integrate-and-fire model that includes somatic adaptation. When exposed to fluctuating inputs sparsely coupled populations of these model neurons exhibit stochastic collective dynamics that can be effectively characterized using the Fokker-Planck equation. This approach, however, leads to a model with an infinite-dimensional state space and non-standard boundary conditions. Here we derive from that description four simple models for the spike rate dynamics in terms of low-dimensional ordinary differential equations using two different reduction techniques: one uses the spectral decomposition of the Fokker-Planck operator, the other is based on a cascade of two linear filters and a nonlinearity, which are determined from the Fokker-Planck equation and semi-analytically approximated. We evaluate the reduced models for a wide range of biologically plausible input statistics and find that both approximation approaches lead to spike rate models that accurately reproduce the spiking behavior of the underlying adaptive integrate-and-fire population. Particularly the cascade-based models are overall most accurate and robust, especially in the sensitive region of rapidly changing input. For the mean-driven regime, when input fluctuations are not too strong and fast, however, the best performing model is based on the spectral decomposition. The low-dimensional models also well reproduce stable oscillatory spike rate dynamics that are generated either by recurrent synaptic excitation and neuronal adaptation or through delayed inhibitory synaptic feedback. The computational demands of the reduced models are very low but the implementation complexity differs between the different model variants. Therefore we have made available implementations that allow to numerically integrate the low-dimensional spike rate models as well as the Fokker-Planck partial differential equation in efficient ways for arbitrary model parametrizations as open source software. The derived spike rate descriptions retain a direct link to the properties of single neurons, allow for convenient mathematical analyses of network states, and are well suited for application in neural mass/mean-field based brain network models.



Network of adaptive exponential integrate-and-fire neurons, mean-field Fokker-Planck model and derived low-dimensional spike rate models. From top to bottom: fluctuating external input, membrane voltage (one neuron and the population), adaptation current, spike times, spike rate of all models (2x).

Acknowledgements

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[W 49] Unstructured Network Topology Begets Privileged Neurons and Stimulus-dependent Sequential Recruitment

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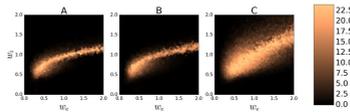
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A perennial question in computational neuroscience is the ‘neural code’ employed by spiking assemblies. A convenient model system are assemblies with self-organized instability expressed as all-or-none synchronization events (‘network spikes’). We simulated and analyzed assemblies with random (unstructured) connectivity, synapses with short-term plasticity, with and without external stimulation. Here we show that unstructured connectivity begets a class of privileged ‘pioneer’ neurons that herald network spikes (i.e., by discharging reliably during the incipient phase) and that, by means of the rank-order of their firing, encode the site of any external stimulation. We also demonstrate that existence of pioneers is strongly enhanced by a topological heterogeneity.

Firstly, we show how pioneers arise from an interaction between sensitivity and influentialness, in a manner reminiscent of an amplifier. This clarifies the mechanisms that produce pioneers and their distinctive behavior. Secondly, the rank-order of pioneer discharge reliably encodes the site of any external stimulation, in stark contrast to rate-based encoding schemes. We demonstrate this by stimulating the network at one of five alternative locations and by seeking to decode the stimulated location from different measures of activity (both rate- and time-based). Thirdly, by mapping the number of

'pioneers' as a function of recurrent excitation, inhibition, and type of topology, we show that an unstructured and broadly heterogeneous connectivity begets more pioneers than scale-free or homogeneously random connectivity (Figure 1). (Analysis based on interval from neuron discharge to peak population activity. Pioneer neurons exhibit mean larger than standard deviation.) Thus, a robust fraction of pioneers requires more than mere presence of 'hubs' (e.g., scale-free topology).

We conclude that random assemblies with self-organized instability offer valuable insights bearing on the issue of 'neural coding'. Finally, we propose such assemblies as a minimal model for the privileged 'pioneer neurons' that reliably predict network spikes in mature cortical neuron assemblies in vitro [1,2].



Fraction of pioneer neurons (in %) and E/I balance, for various unstructured connection topologies: (A) homogeneous random, (B) scale-free random, (C) heterogeneous random. In A and B, pioneers are restricted to a comparatively narrow regime. In C, the domain of pioneers is greatly enlarged.

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[W 50] Detecting single-cell stimulation in large neural networks

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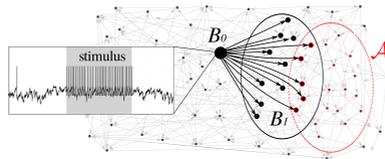
Over the last decade, evidence is accumulating that single neurons can have an impact on the activity of large neural networks [1]. One particularly striking example is that awake rats can be trained to report the transient stimulation of a single cortical cell [2]. This finding seems to be in contrast with studies suggesting that cortical networks are chaotic and therefore only averages on large populations encode information [3].

As a first take on this still unanswered theoretical problem, we test the "null hypothesis": we study whether the stimulation of a single cell can be detected in one of the simplest models capable of reproducing the asynchronous irregular spiking of the cortex: a random network of excitatory and inhibitory leaky integrate-and-fire neurons [4]. To

mimic the long-tailed distribution of postsynaptic potentials measured in the cortex [5], synaptic couplings are drawn here from an exponential distribution [6].

We propose a simple readout mechanism to detect the occurrence of the stimulus. We show with numerical simulations and analytical estimates that detection rates are comparable to the experimental results if the readout is slightly biased toward specific neurons, a proxy for the training of the experimental subjects [7].

Furthermore, we observe that a second network acting as readout requires a smaller bias to detect the occurrence of the single-cell stimulation. This improvement in the detection performance is due to inhibitory neurons in the readout removing input cross-correlations [8], which are the main source of detection-limiting fluctuations.



A randomly selected neuron (B_0) is transiently stimulated, mimicking [2]. The readout population \mathcal{A} is a random selection of neurons with a bias towards B_0 , the direct postsynaptic targets of B_0 . The activity of \mathcal{A} is fed to the readout.

Acknowledgements

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[W 51] Dissecting the Sholl intersection profile

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Sholl analysis has been an important technique in dendritic morphometry for over sixty years [1]. In counting the number of dendritic branches at a given distance from the soma, the Sholl intersection profile collapses three-dimensional neuronal structure into a one-dimensional representation: dendritic complexity as a function of distance. The collapsed representation facilitates comparison of different morphologies; be they similar neurons from different brain regions[2] or those displaying the effects of pathology[3]. We have investigated how far Sholl intersection profiles can be predicted by other dendritic measures. This allows us to directly interpret differences between Sholl profiles as differences in the functionality of a neuron.

The two measures needed to predict a Sholl profile are the domain spanned by the dendritic arbor and the angular distribution of how far dendritic segments deviate from a direct centripetal path to the soma. The first measure is principally determined by axon location and hence microcircuit structure; the second arises from optimal wiring considerations whereby dendrites with a stronger centripetal bias will typically have shorter path lengths between afferent synapses and the soma. The latter measure is quantified by a new metric: the root angle. These two factors predict the Sholl profiles of large numbers of neurons taken from the NeuroMorpho[4] database.

We have reinterpreted a widely-used morphometric measure to have a functional meaning, allowing differences and changes in Sholl profiles to be analysed in terms of their consequences for the cell.

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[W 52] Spike-Patterns in a relaxation-type oscillator comprising finite time delay

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Mutual phase synchronization of coupled nonlinear oscillators encompasses a broad range of phenomena in science and engineering [1]. Most of the theoretical and experimental work assumes instantaneous coupling schemes, where the signal propagation speed between individual oscillators is considered as infinitely fast. Nonetheless, over the last decades it became clear that signal time delay can play a significant role in complex systems such as gene-regulator networks or coupled neuronal oscillator networks. Interestingly, it turned out that time delay can lead to counterintuitive results. For example, delay can induce or suppress instabilities in otherwise stable and non-stable complex dynamical systems, respectively [2]. Here we present our results on relaxation-type oscillators comprising a finite time delay during pulse coupling. The oscillators are based on programmable unijunction transistor circuits, which offer a simple read out of the pulse trains (spike patterns) as well as the realization of excitatory and inhibitory coupling schemes. A Field Programmable Gate Array (FPGA, Cyclon V) was applied to generate adjustable signal time-delays. For both, the oscillator period and the time delay, biological relevant times in the order of several tenth 's of milliseconds were used. The spikes of a relaxation oscillator were delayed and feed back to the excitatory and inhibitory input of the same oscillator [3]. This self-projected system led to different patterns and can be considered as the simplest system to study reentry mechanisms [4,5].

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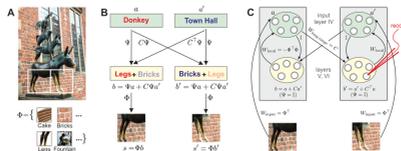
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[W 53] **Generative models of visual cortex with short and long range horizontal interactions**

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In V1, neuronal responses are sensitive to context: responses to stimuli presented within the classical receptive field (cRF) are modulated by stimuli in the surround. Recently, sparse coding models [1] have been successful in explaining part of these modulatory effects [2]: Their dynamics implements an inference process to seek an optimal (w.r.t. accuracy and sparseness) representation of a visual input in terms of fundamental features. This is achieved through a competition between similarly tuned neurons with overlapping input fields, which also mediates contextual modulation. However, this connection scheme implies that neurons with non-overlapping input fields do not interact. Therefore, the proposed mechanism cannot explain the majority of contextual modulations since these are usually caused by surround stimuli positioned far from the cRF. To overcome this limitation, we propose an extension of the classical framework [2] by defining a new generative model for visual scenes that includes dependencies among different features in spatially well-separated locations (Fig.1AB). To perform inference in this model, we also derive a biologically inspired dynamics and a lateral connection scheme for optimally processing local and contextual information. The result can be interpreted as a neural network where units are linked by short range horizontal connections within the same hypercolumn and by long range connections between different hypercolumns (Fig. 1C). Each hypercolumn contains units that receive input from a localized region of the visual field and builds a sparse representation of its input as if it was presented in isolation. In parallel, these local representations are combined by providing contextual information to each other. In our simulations connections are learned from natural images. Long-range connections reflect the co-occurrence of features in different visual field locations: this predicts a connectivity structure linking neurons with similar orientation and spatial frequency preferences, which is similar to the typical patterns found for long-ranging (3-4mm) horizontal axons in visual cortex [4]. Subjected to contextual stimuli typically used in empirical studies, our model replicates several hallmark effects of contextual processing, part of which [3] were not explained by [2]. In summary, our model provides a novel framework for contextual processing in the visual system proposing a well-defined functional role for horizontal axons.



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

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[W 54] Na-K-ATPase-mediated neuronal adaption

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Na-K-ATPases regulate cellular homeostasis by maintaining ionic concentrations on both sides of neuronal membranes within physiological ranges. It has also been suggested that they contribute to neural computation, in particular to mechanisms of adaptation [1, 2]. There is, however, no general agreement on the pumps' computational contribution especially, as there are alternative mechanisms to create spike-frequency adaptation, such as M-type or calcium-activated potassium channels. In this study, we take a mathematical modeling approach to shed further light on the computational relevance of the Na-K-ATPase. To this end, a conductance-based neuronal point model is combined with previously described dynamics of the Na-K-ATPase, allowing intracellular and extracellular ionic concentrations to vary. At first glance, the net current produced by the pump reduces a cell's excitability comparable to the adaptation mediated by slow potassium channels, see also [3, 4]. As we demonstrate, however, pump activity and the associated changes in ionic concentrations also result in a less predictable yet computationally relevant feature of neuronal dynamics. We analyzed the neuron model during stimulation with fast, zero-mean noise on top of a prolonged step current that takes the neuron to a mean-driven firing regime. After the onset of mean-driven firing, pump activity progressively increases (due its dependence on voltage and internal Na, the latter of which accumulates over seconds). As expected, a hyperpolarizing current results and mediates spike-rate adaptation. Consequently, also the neuron's attractor location changes (in terms of effective current threshold) and – provided the stimulus persists – results in a pump-induced shift of neuronal dynamics from an initially mean-driven towards a fluctuation-driven regime (with ensuing consequences for encoding of time-dependent stimulus components, see for example [5]). Interestingly, the pump-induced current eventually (in our case after 5 sec) also induces bistable firing: the cell translates from stimulus-driven irregular firing to highly irregular patterns of stochastic bursting. We found this bursting to persist even for higher stimulus cut-off frequencies, suggesting an intrinsic bistability of the neuron. Pump-induced bursting

may hence be a relevant property of strongly stimulated neurons and its functional relevance for computation in local networks remains to be explored.

Acknowledgements

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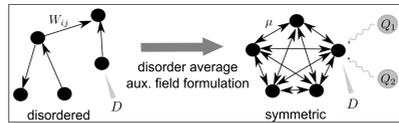
[W 55] Distributed correlations in motor cortex suggest virtually unstable linearized dynamics

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Despite the large amount of shared input between nearby neurons in cortical circuits, massively parallel spiking recordings of various in vivo networks exhibit pairwise covariances in ensembles of neuronal spike trains that are on average close to zero [1]. The low average has been well understood in terms of active decorrelation by inhibitory feedback [2,3] in networks that operate far away from the critical point, which marks the onset of avalanche-like activity [4]. Experiments, however, also show large variability of covariances across pairs of neurons. An explanation for their wide distribution in relation to the static (quenched) disorder of the connectivity in recurrent networks is so far elusive. Here we combine ideas from spin-glass theory [5] with a generating function representation for the joint probability distribution of the network activity [6] to derive a finite-size mean-field theory that reduces a disordered to a highly symmetric network with fluctuating auxiliary fields (Fig. 1). The theory relates the statistics of covariances to the statistics of connections, in particular the largest eigenvalue of the connectivity matrix, and explains the experimentally observed covariance distributions [7]. The analytical expressions expose that both, average and dispersion of the latter, diverge at a critical point which has been studied in terms of a transition from regular to chaotic dynamics [8,9,10]. This critical point does not arise from net excitation, but rather from disorder in networks with balanced excitation and inhibition. Applying these

results to recordings from motor cortex suggests its operation close to this breakdown of linear stability.



Disorder average maps network with frozen variability in connections to highly symmetric network with fluctuating auxiliary fields.

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[W 56] Stochastic computation on spiking neuromorphic hardware

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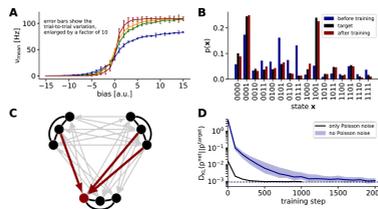
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In order to cope with ambiguous, incomplete and noisy sensory input, the brain is believed to rely on some form of probabilistic computation [1-4]. Within this context of "Bayesian brain", it has been suggested to interpret neural firing activity as sampling from a probability distribution [5-9]. Recently, it was shown that networks of Leaky Integrate-and-Fire (LIF) neurons can approximately sample from Boltzmann distributions with binary random variables when elevated into a high-conductance state via high-frequency Poisson noise which endows LIF neurons with approximately logistic activation

functions [10]. Such spiking Boltzmann machines can then be trained from sensory inputs to perform inference in the corresponding data spaces [11].

Here, we present the first realization of such LIF networks on the BrainScaleS system [12]. BrainScaleS is a mixed-signal neuromorphic device that enables the fast emulation of spiking neural networks with a speedup of 10^4 compared to biological time. We report that on BrainScaleS, sigmoidal response functions can be reliably set up (Fig. 1A), which is a necessary precondition for training such networks on hardware. During training, parameter updates are calculated on a standard computer between consecutive emulations of the network on hardware. After training, the firing activity of the emulated network approximates the desired target distribution (Fig. 1B); the remaining deviations are due to the limited configurability of hardware parameters, as for instance the 4-bit weight resolution.

In addition, since the external bandwidth of the hardware system is limited, the total amount of available Poisson noise is bounded as well, restricting the maximal number of neurons that can be elevated into the high-conductance state. Inspired by the mammalian cortex, where neurons are exposed to the activity of some 10^4 presynaptic partners [13], we demonstrate in simulations that high-frequency Poisson noise can be successfully replaced by the spiking activity of adjacent functional networks (Fig. 1C). This way, networks of networks can be constructed where each neuron only uses the activity from adjacent LIF networks as irregular background input, with zero external Poisson input (Fig. 1D). We believe that this approach will enable the implementation of large-scale networks of deterministic LIF neurons on large-scale neuromorphic systems that can perform stochastic computations without bandwidth-consuming external noise.



(A) Response functions of four different hardware neurons. (B) Sampling on hardware with a network of four neurons. (C) The spiking activity of adjacent networks can be used as irregular input. (D) Networks without external noise are able to reach a similar sampling quality as with Poisson noise.

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[W 57] On the organization of functional subunits in dendrites of pyramidal cells

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Dendrites of many neuron types exhibit complex morphologies and spatially modulated distributions of ionic channels (Major et al., 2013). These cell-type specific characteristics generate non-trivial integrative properties that cannot be described by single compartment models. In an attempt to find simple computational models that mimic key morphological features, a cascade of linear filters followed by a static nonlinearity has turned out to be a promising candidate (Häusser & Mel, 2003, Larkum et al., 2009). This two-layer processing scheme captures the somatic responses of CA1 pyramidal cells to synaptic input to basal and proximal dendrites but sometimes fails for synaptic input to distal dendritic branches in the tuft.

We investigate whether such “anomalous” responses to synaptic input to the tuft can be modeled by cascades that implement additive or multiplicative feedback. To identify such extended cascades, we apply iso-response methods (see Gollisch & Herz, 2012),

where inputs to a dynamical system are varied such that a chosen output measure stays constant. These stimuli define lower-dimensional “iso-response manifolds” whose shape directly reflects the stimulus interaction. We show that all nonlinearities and the feedback of a cascade can be inferred from a few iso-response manifolds.

We apply this approach to a detailed multi-compartment model of a CA1 pyramidal cell (Poirazi et al., 2003) and show that i) feedback improves the prediction of the somatic response to proximal synaptic input but plays only a minor functional role (cascades without feedback have already low prediction error), ii) multiple dendritic branches in the proximal apical dendrites and the tuft form large functional subunits, iii) somatic responses to synaptic input to terminal branches in the tuft cannot be modeled by feed-forward cascades (without feedback), iv) most of these “anomalous” responses can be described by cascades that implement feedback.

Overall, these results demonstrate that iso-response methods are a powerful tool to decipher dendritic function, applicable to multi-electrode or photostimulation techniques.

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[W 58] How to train your $dR_{ago}NN$: teaching neural networks probabilistic inference under biological constraints

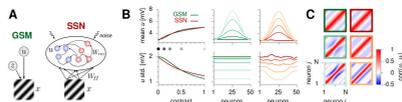
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The study of the dynamics and function of cortical circuits has typically proceeded either in a bottom-up fashion, identifying the biological mechanisms responsible for a variety of experimental findings, without reference to computational function, or by top-down approaches which link features of neural activity to specific computations, without specifying underlying circuit-level mechanisms. Here we bridge these two approaches and study the dynamics and function of cortical circuits in a unifying framework. Specifically, we develop novel methods for training stochastic neural networks with biological constraints, such as Dale’s principle and realistic single-neuron activation functions [Priebe & Ferster, 2008], to match the statistical moments of network activity to full response distributions rather than to deterministic target activities (for the details of the methods, see [Hennequin & Lengyel, 2016]). We show that V1-like dynamics emerge for both trial-averaged activities and across-trial variability in excitatory-inhibitory

networks (Fig. 1A, right) trained for sampling-based inference under the Gaussian Scale Mixture (GSM) model, a widely-used generative model of natural images [Wainwright & Simoncelli, 2000; Orbán et al., 2016] (Fig. 1A, left).

We first show that the GSM posterior mean grows with stimulus contrast z , superlinearly for small z and saturating for large z , while the posterior variance decreases with contrast. These relationships are successfully reproduced by the trained network, which furthermore appropriately generalizes to novel stimuli (Fig. 1B, left) and reproduces the stimulus-dependence of GSM posterior correlations (Fig. 1C). Finally, we show that the network operates in the dynamical regime of stabilized supralinear networks (SSN) that accounts for highly nonlinear properties of both across-trial mean and variability of V1 responses [Rubin et al., 2015; Hennequin et al., 2016]. Thus, our results suggest a generic function for inhibition-stabilized dynamics with a loose excitation-inhibition balance: they are ideal substrates for fast probabilistic inference with recognition models. Conversely, our approach could also be used to infer the brain's internal models based on observed dynamics.



Color code: GSM (green), SSN (red), color intensity indicates contrast level. A) Sketch of both models. B) Mean and std. of the membrane potentials (left: population average); middle & right: shown for each neuron). C) Full membrane potential correlation matrices.

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[W 59] Pinwheel pattern parameterize a manifold of optimized V1 architectures

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It is a long-standing hypothesis that the functional architecture of the primary visual cortex (V1) can be quantitatively understood as the solution of an optimization principle. If this hypothesis is correct, the strong individual variability of the specific layout of functional columns in V1 found experimentally indicates that there cannot be only a single optimal architecture of V1. Indeed the currently best-supported models of V1's functional architecture [1] exhibit multidimensional manifolds of solution families that represent degenerate optima [2]. Any parameterization of this manifold provides a system of effective degrees of freedom of the entire system.

Here we examine whether and how the configuration of pinwheel positions can be used as such a system of effective degrees of freedom. Using methods from the mathematical theory of pattern forming systems we first show that under general symmetry assumptions the phases of individual Fourier components of the pattern of orientation domains are independent and degenerate. Their number is finite, grows linear with the range of nonlocal interactions and sets the dimensionality of a torus of degenerate optima. We examine the influence of moving along this torus and find that only a subset of directions maps to genuine changes of the columnar arrangement. In particular we derive one set of basis vectors that leave the configuration of pinwheel positions invariant and a complementary set that leads to genuinely different pinwheel configurations. The finite dimensionality of these sets demonstrates that in such optima, pinwheel positions are not independent degrees of freedom but are correlated such that a finite subset of pinwheels determines the configuration of all. We then examine the mapping between the experimentally observable pinwheel positions and the abstract phase representation of the manifold. We find that the requirement of minimal rearrangement of the system of orientation domains leads to a simple mapping between phase changes and relative pinwheel positions.

It is currently unknown whether the biologically observable visual cortical architecture in fact represents one point on a continuous steady state manifold. The formalism developed in this work lays the foundations to predict signatures of state manifold topology and dimension in the correlated motion of pinwheel defects.

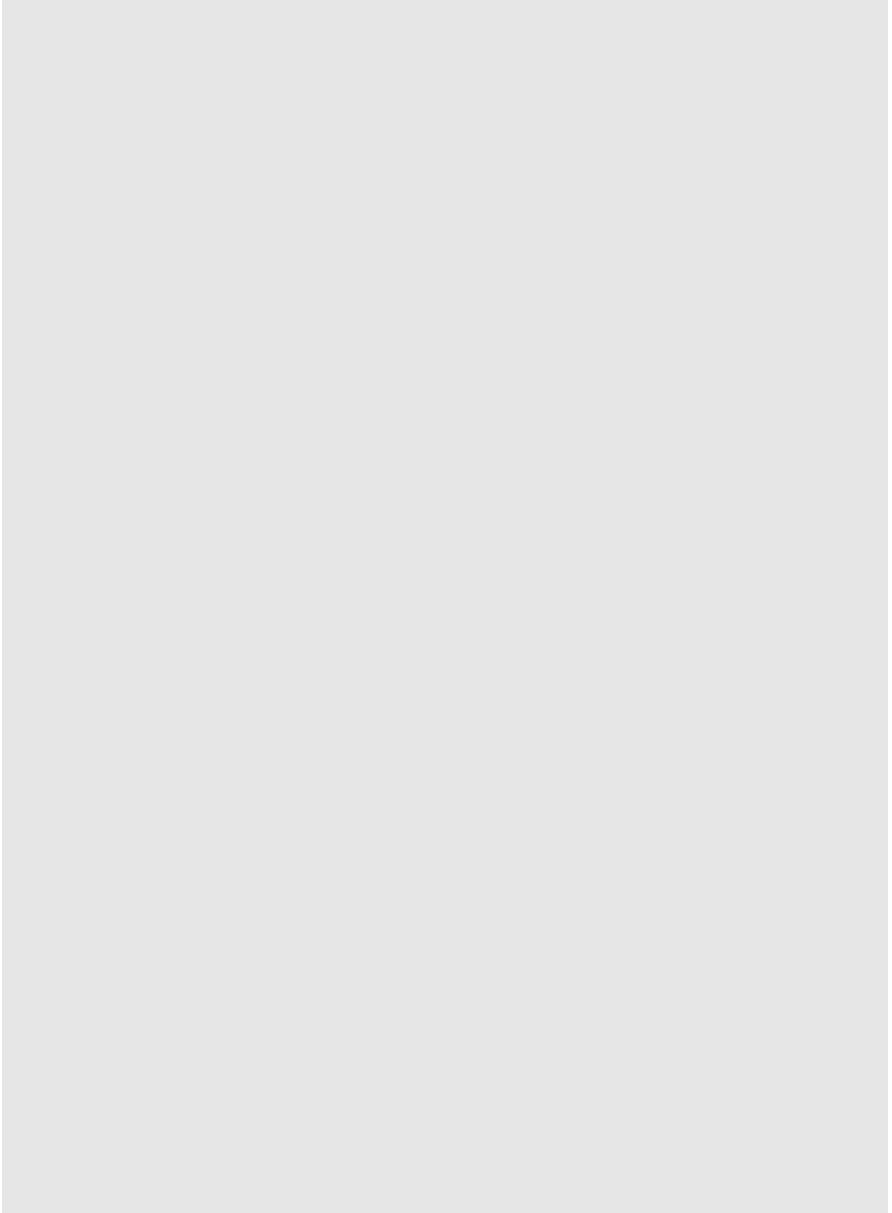
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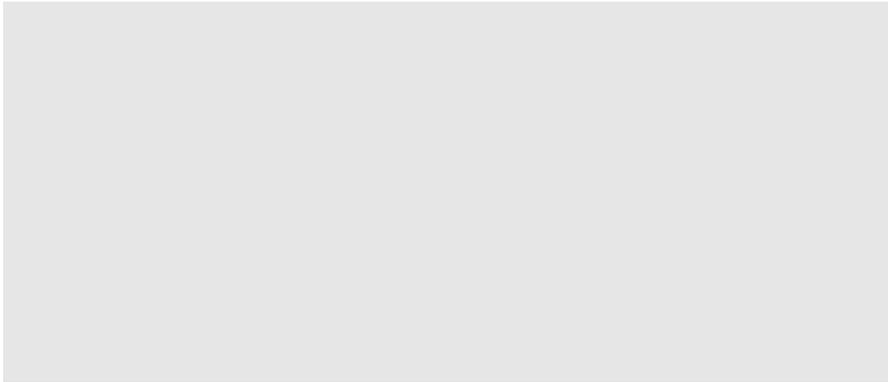
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[W 60] ***Withdrawn***





[W 61] Coexistence of critical sensitivity and subcritical specificity can yield optimal population coding

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The vicinity of phase transitions selectively amplifies weak stimuli, yielding optimal sensitivity to distinguish external input. Along with this enhanced sensitivity, enhanced levels of fluctuations at criticality reduce the specificity of the response. Given that the specificity of the response is largely compromised when the sensitivity is maximal, the overall benefit of criticality for signal processing remains questionable. Here it is shown that this impasse can be solved by heterogeneous systems incorporating *functional diversity*, in which critical and subcritical components coexist. The subnetwork of critical elements has optimal sensitivity, and the subnetwork of subcritical elements has enhanced specificity. Combining these segregated features extracted from the different subgroups, the resulting collective response can maximise the dynamic-range-to-noise-ratio. Although numerous benefits can be observed when the entire system is critical, our results highlight that optimal performance is obtained when only a small subset of the system is at criticality.

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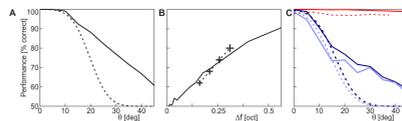
[W 62] Integration of orientation and spatial frequency in a model of visual cortex

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In the visual system complex scenes have to be integrated from simple local features into global and meaningful percepts. Contour integration, a basic process useful for figure-ground segregation and object recognition, is already well understood in terms of orientation alignment. However, there are other features playing a role in this process. Spatial frequency for example has a strong influence on contour visibility. To gain deeper insights into the process of contour integration, we quantified the effect of spatial frequency (SF) on contour visibility as a second cue and investigated if the observed psychophysical effects can be explained by a simple neural mechanism: We hypothesized that interactions are strong between neurons with similar preferred SFs, and that the effective range of the interactions scales with SF. Specifically, we constructed a structurally simplistic cortical model integrating contour integration stimuli consisting of oriented Gabor patches with different orientations and SFs, into which contours of aligned and/or SF-homogeneous patches were embedded. Feature integration in the model is performed by recurrent interactions between populations with receptive fields (RFs) selectively tuned to orientation and spatial frequency of localized stimulus patches. Excitatory connections realize a long-ranging association field with strong links between collinear and co-circularly aligned RFs. Inhibitory interactions provide medium-range normalization and are independent of orientation preference. Interaction strength exponentially decreases with increasing SF difference, and also the range of interaction depends on SF. We were able to quantitatively reproduce the results of psychophysical studies examining the effect of SF on contour integration [1, 2]. For three different experimental paradigms, we show a comparison of model (solid lines) and human psychometric curves (dashed lines) for contour detection in Fig. 1. Thus, we can explain multiple SF-depending effects on contour integration by a simple unifying principle: The more similar the preferred SFs of two neuronal populations, the stronger they are connected. Different magnitudes of effects depending on 'low' or 'high' SFs can be explained by a variable length scaling of the interactions. This mechanisms that we suggest accounts for previously unexplained findings, helping to create a more comprehensive understanding of computation in the visual system.



A: Contour defined by alignment only. B: Contour defined by SF shift only (between contour and background). C: Contour defined by alignment, plus SF jitter on all Gabor patches (light and dark blue). For jitter on contour elements only (red), the target remains visible even for large tilt angles.

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[W 63] Interplay of microscopic and macroscopic dynamics in randomly connected neural networks

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In experimental studies of the animal's brain, dynamics such as oscillation and synchrony have been observed on the macroscopic scales [1]. It has also been suggested that these macroscopic dynamics interact with microscopic dynamics of individual neurons and play parts in brain functions such as learning and memory [2,3,4]. Randomly connected neural networks have been studied numerically and theoretically for the modelling of dynamics in the brain [5,6,7]. However, although there have been pioneering studies of dynamics on multiple scales [8,9,10,11], most of theoretical analyses in previous studies have been restricted to cases in which the neural networks exhibit relatively simple macroscopic dynamics and the microscopic dynamics of individual neurons have only weak and indirect interactions with the macroscopic dynamics. A comprehensive theory predicting interplay of microscopic and macroscopic dynamics is therefore still missing. In the present study, we numerically and theoretically investigate randomly connected neural networks with precisely balanced strong excitation and inhibition. In these networks, microscopic fluctuations of individual neurons serve as driving forces of macroscopic dynamics while macroscopic dynamics largely constrain the microscopic fluctuations. As a result of these interactions, the networks exhibit complicated macroscopic behaviours such as intermittent transitions similar to UP-DOWN states [1], noise-induced oscillation, stochastic resonance and amplification of extrinsic and intrinsic signals due to coherence over the two different scales. A mean-field theory predicts these behaviours of the model in good agreement with numerical results.

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[W 64] The Role of Criticality in Generation of Phase Amplitude Coupling in a Leaky Integrate-and-Fire Neural Network

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Phase Amplitude Coupling (PAC) is a type of Cross-Frequency Coupling where the phase of the low frequency signal modulates the amplitude of the high frequency. A wide range of cognitive functions have been shown to be associated with PAC. However, the mechanistic role and neural substructure of PAC in controlling the brain functions are poorly known. Here, we develop a computational model to study the formation of PAC in the neural system. Using a model that generates complex behaviors, we set out to address if PAC appears near the critical point at which the system shows optimal functionality. The neurons' population in an all-to-all network with Leaky Integrate-and-Fire Model (LIFM) dynamics is considered, and their interaction efficacy is quantified by a control parameter, K . when a neuron fires, it jumps back to its resting potential (zero). As the result of neural interaction, the potential of neurons connected to it make a step ahead if K is positive or backward if K is negative. Changing the control parameters results in different behaviors of the model and the critical point is obtained in a specific control parameter where there is power low behavior in the network. Our model generated low (<20 Hz) and high (30-80 Hz) frequency oscillatory activities intrinsically through constant input. Our results indicate that PAC occurs when the network enters the criticality state, suggesting a tight association between them.

This is the first cue to our knowledge that PAC occurs at the criticality state of the neural network, which helps to better understand the role of PAC in brain. Furthermore, while in previous models an oscillatory input to the modeled network is deployed as a modulating frequency component to generate PAC, our model uses no oscillatory input to drive PAC. This makes our network more biologically plausible compared to other models, since the neural tissue generated PAC regardless of its input. Also, the simplicity of our model (in terms of both its architecture and number of control parameters) makes it a perfect selection compared to many previous models used to model PAC generation.

[W 65] Role of spontaneous activity in the development of orientation preference maps

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A smooth layout of orientation domains is a hallmark of the functional architecture of mammalian visual cortex. It has been shown that these coherent layouts have developed independently at least twice in evolution with quantitatively indistinguishable layout rules arguably implying a strong functional benefit. However, the rules that underlie the formation of these coherent representations within developing cortical circuits remains poorly understood. We propose that correlated spontaneous activity in the developing cortex could be used to establish networks of spatially distributed, but functionally co-tuned neurons that are later evident in the mature cortex. To investigate this possibility, we recorded neural activity in layer 2/3 in the developing ferret visual cortex using in vivo wide-field and 2-photon calcium imaging. Prior to eye-opening and the emergence of orientation maps, we find that spontaneous activity patterns and activity patterns evoked by drifting gratings already exhibit remarkably widespread and specific modular correlation patterns. Furthermore, we show that the spatial layout of evoked and spontaneous activity patterns is highly similar on both a fine-spatial scale and across large ranges (>1mm), suggesting an already well-defined functional architecture constraining the possible activity patterns. Using Principal Component Analysis, we find that the spatial organization of both evoked and spontaneous activity patterns are both highly similar and the population responses reflect overlapping subspaces. But as orientation maps begin to emerge, evoked activity patterns become less diverse and form a subspace contained within the larger space of spontaneous activity patterns. Thus, we conclude that early grating evoked responses are largely drawn from a pool of possible network patterns already evident in spontaneous activity, but as the orientation map develops, only a reduced set of activity patterns becomes associated with stimulus orientation. These results suggest that early activity patterns in the developing cortex may provide a scaffold for orientation map development.

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[W 66] Model-based reconstruction of recurrent neural networks

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During the last years the connectivity of neural networks has become one of the foci of research in both experimental and theoretical neurosciences. Approaches towards elucidating the “connectome” include: (i) anatomical studies, which trace neurites and identify their synaptic connections to neurites of other neurons, (ii) dual and multiple recordings, where neurons are stimulated and the responses of other neurons are directly measured, and (iii) network reconstructions from measured neural activity. Here, we present an approach belonging to the third class. We use a simple spiking “student” network that adapts its synaptic strengths and neural thresholds to match the activity of a recorded recurrent “teacher” network. The current goal of our work is to reconstruct simulated spiking model networks. Future work shall use experimentally recorded spike trains to reconstruct biological networks. The considered teacher networks consist of recurrently connected leaky integrate-and-fire neurons with optional external constant drive and input spikes. The student networks have the same general structure. They are initialized with random weights and thresholds and adapt them according to supervised Finite Precision Learning [1] to match the spiking activity of the teacher. We investigate the conditions that allow a faithful unique reconstruction of the teacher’s recurrent network connectivity and their relation to the reconstruction of single neuron connectivity and the storage capacity of recurrent spiking neural networks. We apply both Recurrent Network and Single Neuron Finite Precision Learning, compare their performance and assess their feasibility for the reconstruction of biological networks.

Acknowledgements

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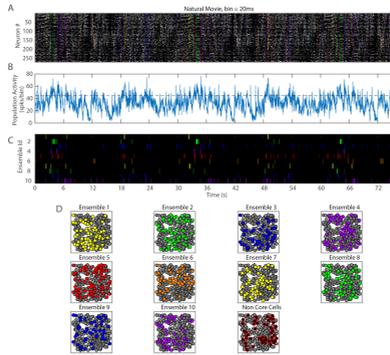
[W 67] On the Search of Retinal Neural Ensembles

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Neural ensembles are a strongly interconnected group of neurons activated by a sequential order of firing (Hebb 1949). However, even when this concept was proposed more than 60 years ago, only recently, the simultaneous recordings and analysis from hundreds of cells has turn to be possible. Moreover, most of the effort for understanding neuronal ensembles have been devoted, so far, to the cortex (Carrillo-Reid et al., 2015; Montijn et al., 2016). However, at our knowledge, little is known about their presence and function at the level of the retina, an accesible part of the central nervous system that communicate directly to the brain. Here we use both a multi-electrode array technique (USB-256 electrodes) for the recording of retinal ganglion cells (RGC) from a diurnal rodent and computational tools (e.g. Carrillo-Reid et al., 2015) to search for the presence and characteristics of retinal neural ensembles under different light conditions: i) spontaneous in dark; ii) full field light stimulation; iii) white noise; iv) natural movie. After we validated our computational tools using synthetic data, we had carried a preliminary assessment to identify the occurrence of neural ensembles in the retina of *Octodon degus*, a diurnal rodent model for aging and neurodegeneration. A preliminary analysis from 270 recorded RGC under a repeated short natural movie as stimulus suggests (Figure 1) the presence of 10 different synchronic neural ensembles. In the Figure 1 A: shows the raster for the 270 RGC under the repeated natural movie (30s per trial); B: population rate and threshold for significant synchronous activity ($P < 0.01$); C: neural ensembles temporal activity (color represent different neural ensembles); D: spatial distribution of detected neural ensembles (color coding similar to C and Non core Cells). The bin used here was 20 ms to favor fast integratiob, however spike timing will also depend on the stimulus nature. In agreement with previous work in the mouse cortex, we found that the number of neural ensembles doesn't significant differ between spontaneous and light evoked conditions . Our results suggest the presence of retinal neural ensembles structures that can be part of a mechanism of population coding. The complete characterisation and understanding of retinal neural ensembles need yet to be establish.



Examples of retinal neural ensembles using a natural movie stimulus.

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[W 68] Axonal potassium channels shape the dynamic gain

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The dynamic gain of a neuron describes how precisely its action potentials are locked to the time course of a fluctuating input. This determines the neurons capacity to encode information about the input. The dynamic gain is shaped by the voltage dependence of sodium channels (Fourcaud-Trocme et al. 2003). This dependence is intuitive, as it is sodium channels that govern the initiation of action potentials. However, the experimentally observed dynamic gain is not readily accounted for by the biophysical properties of sodium channels (Naundorf et al. 2005, 2006).

We use a simple multi-compartment neuron model to study the influence of axonal potassium channels on the neuronal transfer function. In each model, with or without potassium channels, the dynamic gain is obtained in a noise driven regime. The average and the standard deviation of the fluctuating input is chosen to obtain a fixed firing rate and spike statistics (standard deviation of inter-spike-intervals) across models. We find that contrary to simple intuition, the addition of a hyperpolarizing potassium conductance can increase the speed of depolarization away from a fixed point into the

action potential. This is mirrored in an increased bandwidth of the dynamic gain, i.e. an increased cut-off frequency. Potassium channels also introduce a sensitivity for the correlation time of the input. The dynamic gain is increased at intermediate frequencies, if the correlation time is larger, on the order of the activation time constant of the potassium channels.

These results indicate, that the experimentally observed large cut-off frequencies of a few hundred Hertz, as well as the observed sensitivity to the input correlation time might rely on the activation of potassium channels. This introduces the possibility that physiological modulation can dynamically tune the dynamic gain through pathways such as muscarinic innervation that leads to closure of axonal potassium currents.

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[W 69] Gradient Descent for Spiking Neural Networks

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Much of studies on neural computation are based on network models of static neurons that produce analog output, despite the fact that information processing in the brain is predominantly carried out by dynamic neurons that produce discrete pulses called spikes. Research in spike-based computation has been impeded by the lack of efficient supervised learning algorithm for spiking networks. Here, we present a gradient descent method for optimizing spiking network models by introducing a differentiable formulation of spiking networks and deriving the exact gradient calculation. For demonstration, we trained recurrent spiking networks on two dynamic tasks: one that requires optimizing fast (\approx millisecond) spike-based interactions for efficient encoding of information, and a delayed-memory XOR task over extended duration (\approx second). The results show that our method indeed optimizes the spiking network dynamics on the time scale of individual spikes as well as the behavioral time scales. In conclusion, our result offers a general purpose supervised learning algorithm for spiking neural networks, thus advancing further investigations on spike-based computation.

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[W 70] Structured synaptic interactions underlying sharp wave-ripples and the associated replay of neural activity sequences in a network model of area CA3 of the hippocampus

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Population activity patterns recorded in the hippocampus in vivo include theta-modulated gamma oscillations and sharp wave-ripple (SWR) events. During SWRs, neuronal populations in the hippocampus “replay” activity recorded during theta-gamma activity in the exploring animal. Our aim was to develop a mechanistic understanding of cellular and network mechanisms underlying the generation of SWRs, sequence replay during SWRs, and the observed switching to other types of population dynamics such as gamma oscillations. We built a large-scale network model of area CA3 of the hippocampus, and set single-cell and synaptic parameters according to in vitro data. When we used uniform or randomly varying synaptic conductances for all types of connection, there was no sequential activity, and sharp waves with moderate pyramidal cell firing rates and accompanying ripple oscillations were never observed. When recurrent excitatory weights were set by applying an additive spike timing-dependent plasticity (STDP) rule during simulated runs in a circular maze, sharp wave-like activity with ripple oscillations, physiological rates, and accelerated sequential replay of learned activity patterns emerged spontaneously. All of these features of neural activity were robust to scaling the synaptic conductances in a relatively broad range. Application of the recently described symmetric STDP rule enabled both forward and reverse replay as seen experimentally. We then used systematic perturbations of the synaptic weight matrix to explore the links between these different aspects of the neural dynamics and the underlying functional connectivity. After shuffling the weights of individual neurons, sequential activity disappeared, and neither moderate rates nor ripple oscillations could be robustly maintained. On the other hand, binarizing the weights by replacing the strongest weights by their average, and the rest of the weights by their average, did not lead to any fundamental change in the dynamics. These results demonstrate that the distribution of synaptic weights is neither necessary nor sufficient for the physiological activity of the original network. Manipulations which destroyed embedded convergent paths in the weight matrix invariably led to the disappearance of both sequential activity patterns and SWR population dynamics, demonstrating a fundamental link between temporal representations (coding) and population dynamics in structured cortical networks.

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[W 71] Growing critical: Self-organized criticality in a developing neural system

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A variety of neural systems in different species generate neuronal avalanches: bursts of network activity that have a power-law size distribution [1]. An individual avalanche may involve any number of neurons, from one to the entire network; avalanches of all sizes occur with non-negligible frequency. Such behavior is typical for critical systems, that is systems at the transition point between two states with qualitatively different characteristic scales of activity. A possible explanation for the occurrence of neuronal avalanches is that the underlying networks organize themselves into the critical state (self-organized criticality [2]). Here we propose a simple spiking model for developing neural networks. It shows how networks may “grow into” the critical state during development. The network growth and spiking dynamics are adapted from established models: Neurons are inhomogeneous, coupled Poisson processes without refractoriness (for a static network: Hawkes processes) [3, 4]. The extents of neurites are represented by discs, with synaptic coupling strengths proportional to the discs' overlap. Inputs increase the Poisson spike rate of a neuron. Neurites grow if a neuron is silent and shrink when spikes are generated [5 - 7]. Both processes balance at some spike rate, such that the network becomes stationary. Our analysis and numerical simulations show that the larger the stationary state's spike rate is compared to the spontaneous spike rate of isolated neurons, the nearer the stationary network is to the critical point. The avalanche size distribution approaches a power law distribution with exponent $-3/2$. We also derive the avalanche duration distribution analytically and show that its tail approaches a power law with exponent -2 . Both exponents have been reported in experiments. Refractoriness of neurons can be included, but makes analytical considerations difficult and tends to render the system subcritical. Our model can be viewed as a self-exciting Hawkes process with exponential kernel, a process frequently used in life sciences, finance and social sciences to model clustering phenomena. Our derivation of the avalanche duration distribution may prove useful in these fields to analytically describe the durations of phenomena such as criminal gang violence, corporate defaults and disease outbreaks.

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[W 72] Following stage II retinal waves during development with a biophysical model

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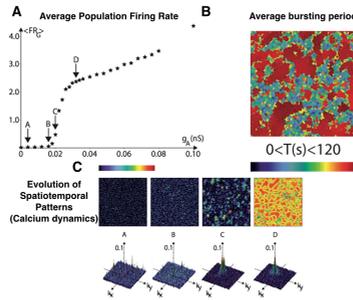
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Retinal waves are spontaneous bursts of activity propagating in the developing retina and playing a central role in shaping the visual system. They appear early in development and gradually disappear upon maturation. They are classified in 3 consecutive stages (I,II,III), mainly characterized by different synaptic transmissions and transient networks of specific cells [1]. However, in order to elucidate the dominant mechanisms shaping retinal waves within a specific time window of maturation, as well as the transition between them, it is important to investigate the continuous variations of biophysical characteristics due to development within each stage. Focusing on stage II, we propose a biophysical model, grounded on experiments, and accessible to dynamical systems analysis, featuring a network of cholinergic-coupled Starburst Amacrine Cells (SAC) with a calcium-controlled slow After HyperPolarization current (sAHP) [2]. In agreement with biophysics SACs burst spontaneously and their interaction via acetylcholine gives rise to waves. A bifurcation analysis exhibits 3 key biophysical parameters having a big impact on SACs spatiotemporal activity: the fast K⁺ conductance, the acetylcholine synaptic strength (varies upon maturation), and the rest potential (varies upon pharmacology). This analysis leads us to reproduce a bench of experimental results [3]. It allows us to explain the wide variability in interwave intervals observed across species with a unique generic mechanism (figure not shown). More generally, the nonlinear dynamics generates heterogeneous local spatial structures inside which retinal waves propagate (Fig 1B). This induces a wide variability in waves characteristics (size, duration) even though our network is perfectly homogeneous. Therefore, we show that although variability is de facto present in biological systems, it is not necessary to explain the appearance of spatial structures and waves, as well as their variability (Fig 1). We analyze how the evolution of cholinergic conductance due to the maturation of nicotinic receptors dramatically changes the retinal wave characteristics. Especially, there is a very narrow interval of acetylcholine conductance where retinal waves size obey a power law distribution, reported first also in [4], suggesting a specific (homeostatic) mechanism

stabilizing temporarily the SACs network in this specific range. Finally, we discuss several experimental predictions of our model.



A. Average population firing rate exhibits a sharp transition when varying the cholinergic conductance g_A . B. Heat map of the average bursting period T (sec) of the network showing patterns where waves do emerge (red) and where they do not (blue). C. Evolution of the patterns when increasing g_A .

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[W 73] Neuronal Dynamic Routing Model through Coherent Oscillations in Complex Network of Electrical Synapses

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The membrane potentials of neurons propagate across electric synapses without directionality, threshold, and time delay in contrast to chemical synapses. Several experimental evidences and computational models have shown that electrical synapses are required to implement not only the movement of the subthreshold oscillation but also the emergence of both the rhythmicity in the global neural network and the spiking synchrony in the mammalian interneurons. How the complex network of neurons executes the simultaneous and coherent propagation of neuronal signals is the center of fundamental issues

in Connectomics, yet is still far away from our systematic understanding theoretically and computationally. Also there are still debates on another issue, called 'Dynamic routing'; how the outgoing signal of the sensory-neurons, regulated by the modulation of firing-frequency, chooses the specific propagating path in various possible ways while the synaptic network is comparatively static.

In this work, we aim to suggest a physical mechanism on how 'Dynamic routing' operates with the propagation of coherent membrane potential oscillations in complex network of electrical synapses. Provided that the coherence length of oscillatory signals is longer than our system size, we employed the quantum mechanical formulation in order to describe the propagation of the subthreshold membrane potential across the complex network of electrical synapses with oscillatory incoming/outgoing signals. We could envisage the interference effect of all possible paths between input and output neurons, and investigated the propagation of coherent oscillatory signals among neurons in the complex network. We demonstrated the existence of allowed or forbidden propagation and the dynamic modulation of the propagation path depending on the wave-number of oscillatory signals in both the virtual network formed like square lattice and the real electrical synaptic network of *C. elegans*.

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[W 74] Exponential Approximations To The First Passage Time Distribution Of Time-Inhomogeneous Stochastic Leaky-Integrate-And-Fire Neuron Model

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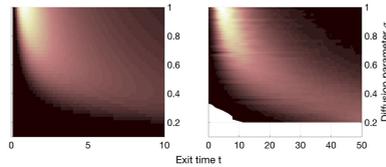
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To investigate the complex dynamics of a biological neuron that is subject to small random perturbations we can use Ito diffusion processes as stochastic neuron models:

$$dV^\sigma(t) = [-g(t)V^\sigma(t) + I(t)] dt + \sqrt{\sigma}dW(t)$$

While many techniques have already been developed to study properties of such models [1], especially the analysis of the first-passage time (FPT) distribution $T^\sigma = \inf(t > 0, V^\sigma(t) \geq V_{th} > V_0)$ remains difficult. In this work I apply the large deviation theory (LDT), which is already well-established in physics and finance [2], to the problem of determining asymptotic estimates of exit times of the mean-reverting Ornstein-Uhlenbeck (OU) process in the small noise limit $\sigma \rightarrow 0$. The OU process instantiates the Stochastic Leaky Integrate and Fire (SLIF) model and thus serves as an example of a biologically inspired mathematical neuron model. Taking the seminal work of Paninski [3] as a starting point, I extend his results on FPT densities to time-inhomogeneous models and I provide explicit LDT approximations for interesting examples like the model proposed by Stevens & Zador [4]. Among the results is a LDT argument for when stochastic neuron

models can be approximated by Poisson processes, a Laplace approximation of the Siegert formula and a connection to extreme value theory and the Fréchet distribution (compare [5], [6] and [7]). Finally I performed several simulations to verify and to reveal systematic biases of these results.



An example of a time-dependent conductance is $g(t) = \frac{a}{1+bt} + c$ which introduces a relative refractory period into the model. Left: LDT prediction of the FPT density for varying values of σ . Right: Numerical simulation of the same density. Note the different scalings of the x axes.

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[W 75] Assessing dynamics from fMRI time series with a piecewise linear recurrent neural network model

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Cognitive functions arise through the coordinated activity between many interconnected brain regions and are thought to be implemented in terms of the stochastic and dynamical properties of the underlying network (Izhikevich, 2007). For example, non-linearly connected networks foster a multitude of stochastic dynamical phenomena such as multi-stability and attractor-hopping, giving rise to cognitive computations related to memory and decision making. By adjusting their internal connections, neural networks may further change their dynamical properties over time, enabling adaptive behavior and learning. Reconstructing stochastic network dynamics from neural time series is therefore highly compelling to advance our understanding of cognitive mechanisms, and could benefit research particularly in the context of psychiatric disorders. From this perspective, psychiatric symptoms and cognitive dysfunctions may be rooted in changes of the underlying dynamical system properties, and the inability to successfully adapt these properties in a given context or during learning. One prominent approach to the reconstruction of stochastic network dynamics from experimental recordings is the use of state space models (SSMs) (see e.g. Smith & Brown, 2003). SSMs treat the often high-dimensional noisy experimental recordings, such as may be obtained from electrophysiology or neuroimaging techniques, as being generated by an underlying (usually much lower dimensional) latent dynamical system subject to process noise. In this way, they may yield essential and compact information about the underlying system's trajectories as well as its governing dynamics. However, so far only few of the proposed models capture non-linear dynamics, essential to emulate many crucial dynamical phenomena related to cognition, and hardly any models capture non-stationary processes which are key to learning and adaptive behavior. Building on a previous model developed in our group (Durstewitz, 2017), here we advance a piecewise linear state space model with non-stationary latent process designed to assess dynamics from functional magnetic resonance imaging (fMRI) recordings, and thus directly applicable to psychiatric data sets. We test the model on fMRI data obtained during a Working Memory (WM) task, and show how the model is capable of capturing meaningful task-related information in its state trajectories, as well as accounting for behavioral variability in its governing dynamics.

Acknowledgements

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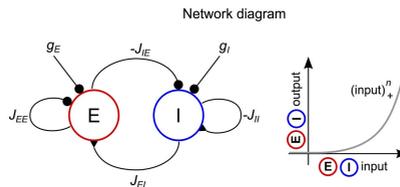
[W 76] **Connectivity regimes of the stabilized supralinear network corresponding to bistable, persistent and oscillatory activity**

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The neural circuits perform many nonlinear computations such as sub- and superlinear summation of inputs, memory storage and oscillations. All these fundamental computations are thought to be generated by nonlinear interactions in recurrently connected networks of excitatory and inhibitory neurons. Therefore, it is plausible to assume that a single recurrent network model could reproduce many of these computations. In the presented work we consider a 2D stabilized supralinear network (SSN) model [1,2]. Recently the SSN model has been shown to reproduce a variety of nonlinear computations such as normalization, surround suppression [3] and stimulus induced variability suppression [4]. In its simplest form the SSN model is a set of two coupled nonlinear differential equations that describe the activity of excitatory and inhibitory populations. The positive constants J_{EE} , J_{EI} , J_{IE} and J_{II} represent the strength of synaptic connections between the populations, g_E and g_I are constant inputs to the populations and the activation function of the populations is captured by the power-law with the exponent $n \geq 2$, Fig. 1. The power-law activation function is motivated by the experimental studies [5] as well as theoretical evidence showing that the power-law is the only function consistent with contrast invariance [6,7]. Even though the SSN is one of the simplest nonlinear network models, it turns out few methods are available to systematically predict what type of steady state solutions to expect for all connectivity matrices and inputs to the network.

We present a new method that allows to map 2D steady states of the SSN model to the zero crossings of a 1D characteristic function. This method allowed us to derive a number of new computational insights. First, we have shown that at most two stable steady states can coexist in the SSN model and derived corresponding connectivity and input constants. Second, we have outlined exact connectivity and time scale regimes for the emergence of a persistent state in the SSN model. Third, we have proven that the SSN model can undergo a Hopf bifurcation and lead to stable oscillatory attractors.



Circuit architecture and neural current-to-firing rate transformation.

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[W 77] Weak electric fields promote resonance of neuronal spiking activity: analytical results from single cell and network models

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Transcranial brain stimulation and evidence of ephaptic coupling have sparked strong interests in understanding the effects of weak electric fields on the dynamics of neuronal populations. While subthreshold effects in single neurons can be explained using multi-compartment models or the cable equation, these models are not well suited for mechanistic analyses of modulation effects in neuronal spiking and network activity. Here, we employ a two-compartment integrate-and-fire neuron model that accounts for an applied or self-generated extracellular field. We first efficiently calibrate its parameters via the cable equation to describe the dynamics of pyramidal neurons. Using the Fokker-Planck equation and a moment closure dimension reduction method we then derive analytical results for the spike rate dynamics of (i) single neurons subject to fluctuating inputs, and (ii) a sparsely coupled two-population network. We show that applied oscillatory weak fields, which effectively mimic anticorrelated inputs at the soma and dendrite, strongly modulate neuronal spiking activity in a narrow frequency band. Self-generated fields also promote spike rate resonance in the same frequency band, but their effect is much weaker. The effect of an applied field carries over to coupled populations of pyramidal cells and inhibitory interneurons, boosting network-induced resonance in the beta and gamma frequency bands. This work provides insights on how extracellular electric fields modulate neuronal spiking activity due to the morphology of pyramidal cells, and contributes a useful theoretical framework to analyze their spiking dynamics.

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[W 78] When extended time window does not improve the accuracy of rate coding

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Since Adrian's work in 1928 [1], it is widely accepted that neuronal firing rates contain a significant

amount of information about external stimulation. As neurons fire spikes irregularly, it is usual to determine their firing

rate by counting the number of spikes in a predefined time window [2]. Nevertheless, the accuracy of rate coding is only rarely inferred from exact spike counts. In our recent work [3], we analysed the distribution of the counts of spikes from a single neuron assuming the stochastic perfect integrate-and-fire model. The main aim of our analysis was to evaluate the maximum possible accuracy of decoding based on observed counts of spikes using the Fisher information about the stimulus intensity, which is a common measure of ultimate decoding accuracy [4].

We investigated effects of several aspects of the neuronal model, especially the influence of the time window duration. Intuitively, one would expect that a longer time window must result in an estimate of the stimulus intensity that has at least the same or better quality. By contrast, our results show that the Fisher information is nonmonotonic with respect to the length of the observation period, which means that extending the time window might in some cases deteriorate the decoding accuracy. We demonstrate that this phenomenon is caused by the discrete nature of the count of spikes, which also suggests that similar results might be obtained for other neuronal models.

Besides the time window duration, we also investigated the role of the presynaptic spontaneous activity. It may also affect the decoding accuracy in a nontrivial way. In a situation when shortening the time window would lower the decoding accuracy, an increased level of spontaneous activity may partially reduce this loss.

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[W 79] Encoding of memories: effective connectivity on the hippocampus and the role of inhibition in the information flow.

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Networks containing a huge number of neurons and synapses confer the brain an immense computational capability. Learning how activity propagates in these intricate networks would help us understand how information is globally integrated. This communication is determined by the structural connections (wiring diagram) linking the different nodes in the network and their functional interaction. These interactions are highly dynamic processes that mostly rely on changes in synaptic efficacy and the differential recruitment of excitatory and inhibitory elements. The combination of both factors determines the effective connectivity of the system in a particular state. Here we have used a computational model and causality measurements to study activity propagation in the hippocampal formation, a brain region critical for the formation of episodic memories. It is composed by the hippocampus proper (areas CA1 and CA3), the dentate gyrus (DG) and the entorhinal cortex (EC). While extensive literature on the connectivity of the first regions exists, the connectivity of the EC remains poorly investigated.

To better understand how the internal structure of EC affects the causality in the hippocampal formation, we implemented a model containing all the above areas. We assumed the EC was formed by 3 layers (II, III and V). We fixed all connections in the model between DG, CA3 and CA1, while the EC connectivity was systematically varied. The causality was estimated using Granger Causality (GC) and Partial Transfer Entropy (PTE). For these measurements, we assumed that only information from DG, CA3 and CA1 was available. We also introduced interneurons in our circuit, considering inhibitory projections from CA1 to CA3. With this new ingredient, we addressed different "causality" measures, such as information flow and synchronization between populations, respectively.

Our procedure revealed that different EC internal connectivity patterns give rise to very distinct causality results in the hippocampus, despite its fixed connectivity. Moreover, different results were obtained for the two methods (GC, PTE), highlighting the importance of the analysis and revealing potential misinterpretations when only partial information is available. Our method allowed us to analyze the differences of causality when excitatory and inhibitory projections are considered and identified the most probable EC configuration to explain the known connectivity between the DG, CA3 and CA1.

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[W 80] Dynamical stability and local phase space structure of networks containing neurons with negative dissipation

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We study the dynamical properties of inhibitorily coupled networks of integrate-and-fire neurons with infinitesimally short synaptic currents. In addition to conventional leaky integrate-and-fire neurons with positive dissipation, the networks contain neurons where the sign of the leak current is switched, leading to negative dissipation. Such mixed networks can exhibit a balanced state of asynchronous irregular spiking activity, independent of the proportions of the two neuron types.

Although the balanced state appears similarly in many different neural network models, its dynamical properties such as chaoticity can vary considerably. In particular, inhibitory networks consisting of integrate-and-fire neurons with positive dissipation and infinitesimally short synaptic currents are stable, non-chaotic despite their irregular dynamics. This property is robust against introducing a number of excitatory connections. Here we show that introducing a single neuron with negative dissipation already renders the network dynamics unstable. For this we compute the largest Lyapunov exponent, which measures the exponential growth rate of generic small perturbations.

To further characterize the dynamics we consider the full spectrum of Lyapunov exponents, which describes the rates of growth or shrinkage of small perturbations in different directions of phase space. Interestingly, the number of negative (positive) Lyapunov exponents approximately equals the number of neurons with positive (negative) dissipation. We analytically explain this using a mean-field approach for the growth or shrinkage of perturbations of the individual neurons: the effect of inhibitory input from the network reduces to increasing the time between the resets of the neurons; this already results in nonzero perturbation growth rates, which reasonably approximate large parts of the Lyapunov spectrum.

The mean field approach raises the question whether single neuron perturbations are perturbations that grow according to a single Lyapunov exponent. The precise directions for the latter are given by the covariant Lyapunov vectors, which specify in particular the directions of the stable and unstable manifolds of a trajectory. It turns out that the Lyapunov vectors consist of mixtures of perturbations to different neurons. We also compute the Lyapunov vectors of small mixed networks, where their directions can be predicted and interpreted in terms of the network architecture.

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Neurotechnology and brain-machine-interfaces

[W 81] Decoding of EEG Brain Signals using Spiking Neural Networks on SpiNNaker Neuromorphic Hardware

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Non-invasive brain machine interfaces (BMIs) on motor imagery movements have been widely studied and used for many years to take advantage of the intuitive link between imagined motor tasks and natural actions. This type of BMI has been widely used as an alternate mode of communication and environmental control for the disabled, such as patients suffering from amyotrophic lateral sclerosis, brainstem stroke and spinal cord injury [1]. Together with recent advancements in neuromorphic computing, which allow real-time and low power implementations of large scale spiking models for data processing, BMI applications could profit from this symbiosis [2].

Taking inspiration from the architecture of the olfactory system of insects [3], we advance and implement a spiking neural network model to decode and predict imaginary movements from EEG signals. The network runs on SpiNNaker, a neuromorphic hardware platform containing 4 chips with 64 cores. Our work provides a proof of concept for a successful implementation of a functional spiking neural network (SNN) for decoding two motor imagery (MI) movements on the SpiNNaker system. With a mean accuracy of 75%, SNN presents a valid alternative to classical machine learning algorithms deployed in BMIs. This approach can be extended in the future to classify more complex MI movements on larger SpiNNaker systems.

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[W 82] A Cooperative Stereo Matching Neural Network on TrueNorth

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Many tasks in nature and robotics require orientation and self-localization within the environment. Fast and reliable depth perception plays a crucial role in these tasks. A prominent solution nature has found to estimate distances is stereo vision. Here, we propose a neural network implementation of a cooperative stereo matching network[1] on TrueNorth[2] using dynamic vision sensors (DVS,[3]) to solve the depth estimation problem for robots. TrueNorth is a neuromorphic chip that comprises one million silicon neurons to facilitate massively parallel computation in large neural nets, while DVS were inspired by mammal eyes and work analogously to retinal ganglion cells. Using these bio-inspired technologies we overcome the main limiting factors of current stereo computation: high-latency frame-based cameras and computationally costly data-processing in a sequential way on standard computers. The network's architecture is based on a network developed for SpiNNaker[4] and the purpose of the network is to estimate depth information using event-based vision streams from a stereo rig of two DVS. The use of TrueNorth allows to leverage currently unmatched capability of low power-consuming real-time processing of large neural networks letting the incoming vision information be processed fast and with low latency. In order to let the DVS communicate directly with TrueNorth, a software interface to stream live DVS data to the chip was developed.

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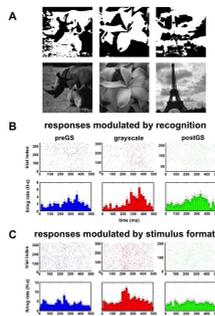
Sensory processing and perception

[W 83] Correlates of rapid learning and object recognition in the spiking activity of human temporal lobe neurons

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One of the greatest feats in human cognition is the ability to rapidly acquire new information. A prominent example of this ability is manifested during learning to identify new objects, even after single trial exposures. The neural mechanisms subserving this behavior, however, are largely unknown. Here, we studied neuronal responses to instances of single shot learning using Mooney images. Mooney images render objects in binary black and white in such a way that they can be extremely difficult to recognize. After exposure to the corresponding grayscale image, it becomes significantly easier to recognize the objects in the original Mooney image. We recorded single unit responses in the human brain, mostly from the medial temporal lobe, from 13 epilepsy patients implanted with electrodes for clinical purposes [1]. The experiment began with presentation of Mooney images. Subjects learned the identity of these initially unrecognized Mooney images via paired viewing of their grayscale counterparts. Finally, the Mooney images were presented again alone. We compared the neuronal responses of 1118 unit clusters in response to three main conditions: (i) Mooney images that were not recognized (preGS), (ii) identical Mooney images that were recognized (postGS) and (iii) corresponding grayscale images (GS). About 20% of them showed significant modulation of firing rates computed in the 0-500 ms interval across conditions. Of those units, 12% showed firing rate modulation dependent on changes in recognition with similar responses to postGS and GS and different responses to preGS and postGS. Additionally, 32% of those units showed similar responses to preGS and postGS and different responses to GS and postGS. These results demonstrate a single unit signature of rapid learning in the human medial temporal lobe and provide initial steps to understand the mechanisms by which top-down inputs can rapidly orchestrate plastic changes in neuronal circuitry. Looking in detail at the neuronal responses for different image motifs shows the complexity of these mechanisms: For one motif a certain neuron may show different responses when comparing grey to black/white image format, with similar preGS and postGS responses. For another motif the same neuron may show different responses when comparing preGS to postGS black/white image format, with similar grey and postGS responses. And for a third motif all three conditions may elicit similar responses in this neuron.



(A) Sample Mooney (top) and grayscale (bottom) stimuli. (B) unit with responses modulated by recognition. GS and postGS condition show similar, preGS different (lower) firing rates. (C) unit with responses modulated by stimulus format: preGS and postGS similar, GS different (higher) firing rates.

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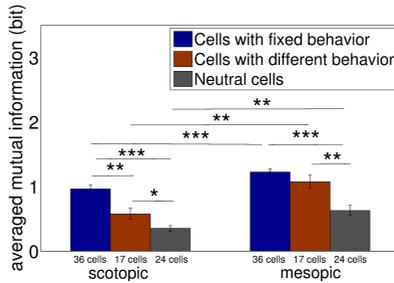
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[W 84] Effects of ambient luminance on retinal information coding

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It is well known that retinal ganglion cell response properties vary with changes in ambient light [1]. For example, a cell that behaves as an ON cell at one luminance level might behave as an OFF cell at another [1,2]. However, the consequences of these changes for information processing in the retina are still largely unknown. Here, we investigated how retinal ganglion cell response type changes due to different ambient luminance levels are related to stimulus information carried by these cells. We used multi electrode arrays to record spiking activity from a total of 86 ganglion cells of two isolated mouse retinas during visual stimulation. Our visual stimuli consisted of homogeneous contrast steps of positive and negative contrast at different ambient light levels, covering the scotopic (dark) to mesopic (bright) regimes. To quantify information carried by the ganglion cell responses, we first applied temporal non-negative matrix factorization (temporal NMF) to decompose each retinal ganglion cell's spike trains into a set of trial-independent non-negative temporal firing patterns and trial-dependent non-negative activation coefficients that represent the strength of temporal firing profiles within a given trial. This factorization yielded a robust low-dimensional representation of the neural responses that captures efficiently a ganglion cell's temporal information [3]. We then decoded stimuli from this low-dimensional representation using multi class linear discriminant analysis (LDA) and used cross-validated decoding performance to estimate mutual information between stimuli and spike trains. Confirming earlier studies [1], we found that a significant number of retinal ganglion cells changed their response type when the ambient light level changed from scotopic to mesopic. Our quantification of stimulus information showed that ganglion cells that kept their response type carried significantly more stimulus information than ganglion cells that changed their response type from scotopic to mesopic vision. Moreover, we found that ganglion cells that clearly behave as ON or OFF cells in at least one ambient light level carried significantly more information than cells without a clear type in any of the ambient light levels (Fig 1). Our results suggest that ambient luminance dependent response type changes cannot be attributed to efficient coding at the single-cell level but do not exclude the possibility that these type changes aid population codes.



Information carried by retinal ganglion cells with different types. Comparison of stimulus information encoded by cells with fixed response type (either ON or OFF) with stimulus information encoded by cells that have an ambient luminance dependent or no clear type in scotopic and mesopic vision.

Acknowledgements

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[W 85] The structure of visual object representations in rat lateral extrastriate cortex

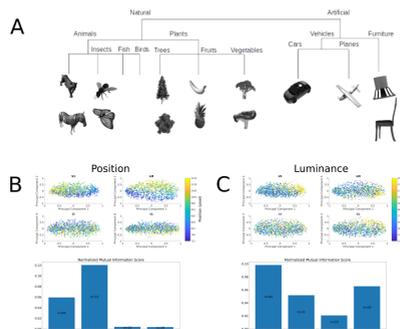
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Rodents are emerging as interesting models to study the mammalian visual system, mainly because of their accessibility to a wide range of experimental techniques. Yet, it remains unclear to what extent rodent visual cortex is capable of supporting higher-order visual functions typical of primate vision. Recent studies have shown that rats are able to perform invariant visual object recognition [1], and that in rat lateral extrastriate cortex low-level visual information is progressively discarded in favor of higher-order information, while the representation of visual objects becomes more invariant [2]. In our study we further investigate the nature and complexity of visual object representations in rat primary visual cortex and lateral extrastriate areas. To this aim, we built a rich stimulus set, consisting of 40 different objects (organized in a semantic hierarchy), each presented under 36 different views (Fig. 1A). As a result, the stimuli spanned

a wide range of low-level, mid-level, and high-level visual features. We recorded neuronal responses from anesthetized animals passively exposed to the stimuli, and we characterized the representation of the stimuli in the population vector space, using a number of multivariate approaches such as dimensionality reduction and clustering analysis [3-4]. In the latter case, the partitions resulting from clustering the objects' neuronal representations were compared, using the normalized mutual information, to stimulus categories based on several visual features. We also performed single neuron analyses, computing the mutual information between the responses of single neurons and different stimulus parameters [2]. Our results suggest that, in accordance with [2], rat visual areas progressively discard low-level visual information. For instance, using Principal Component Analysis it can be seen how, in low-level areas (V1, LM), the first principal components correlate nicely with stimulus luminance and position, while such correlation is lost in higher-level areas (LI, LL) (Fig. 1B and C, top). Similarly, the matching between the clustering of the stimuli in the population vector space and their binning along the luminance and position axes becomes gradually lower along the areas' progression (Fig. 1B and C, bottom). Taken together, these results reinforce the attribution of rat lateral extrastriate areas to an object-processing pathway, where the information about low-level visual properties is gradually pruned.



(A) Object samples taken from the stimulus set, and their underlying categorical structure. (B and C) Top: scatter plot of the principal components of the stimuli (parameter value is color-coded). Bottom: NMI between population clusters and feature based categories for the different visual areas.

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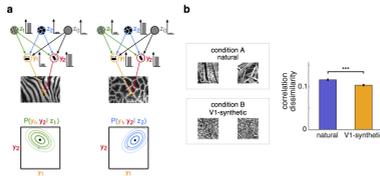
[W 86] Top-down perceptual effects in cortical response statistics

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The visual cortex is characterized by a hierarchical organization, which reflects the compositional nature of natural stimuli. Hierarchical models of perception predict that population codes in the visual system, including the primary visual cortex, integrate bottom-up information about the stimulus with top-down feedback from downstream areas, and this top-down influence conveys information about previously acquired knowledge (Lee & Mumford, 2003). Furthermore, the magnitude of top-down effects was shown to vary based on the presence of higher-order statistical structure in the stimulus (Singer & Gray, 1995). While bottom-up influences have been characterized by a multitude of studies, much less is known about the way top-down influences shape population activity. Here we identify signatures of hierarchical computation in the population activity of V1 and use this to predict stimulus structure dependence of the fine structure of spike count correlations. To test the prediction that spike count correlations are specific to stimuli and their variability is dependent on stimulus structure, we recorded from the V1 of macaques while performing a visual attention task. First, we showed that spike count correlation patterns evoked by natural images are stimulus-specific. To achieve this, we developed contrastive rate matching to control for the confounding effect of stimulus-specific firing rates on correlations. Second, for each natural stimulus, we synthesized images that retained statistical structure corresponding to the V1-level representation of visual input, a linear combination of oriented edges, but lacked any higher-order structure. We demonstrated that spike count correlation patterns are less similar to each other in response to different natural stimuli than in response to different synthetic stimuli, indicating that the signature of hierarchical computation is present in the co-activation structure of V1 populations. Third, we synthesised stimuli retaining second-order structure between the edges corresponding to a texture pattern, which is suggested to be represented in V2 by experimental results (Freeman et al, 2013). In line with the predictions of hierarchical computations, we showed that responses to synthetic textures showed more stimulus-specific spike count correlations than responses to V1-level synthetic images. Our results provide evidence that hierarchical computations account for the rich structure of spike count correlations in V1.



(a) Inferred distributions for a latent feature depend on the stimulus in a hierarchical model of images. (b) Recorded spike count correlations are more specific in response to natural images than to stimuli with no higher-level statistical structure.

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[W 87] Functional characterization of the signal processing chain in the mouse early visual system

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More than 30 types of retinal ganglion cell (RGC) represent parallel channels transmitting different aspects of visual information from the retina to various parts in the brain. Retinal output is most directly conveyed to the cortex via the retino-geniculo-cortical pathway, comprised of RGCs, relay cells in the dorsolateral geniculate nucleus (dLGN) of the thalamus, and the primary visual cortex (V1). It has long been known that this pathway is not homogeneous but consists of parallel channels, each carrying specific information. However, it is still debated which RGC types project to the dLGN and how their output is transformed in the dLGN. Here, we characterized, in the mouse model,

the functional properties of dLGN-projecting RGCs and assessed dLGN responses to the same set of stimuli by extracting elementary weighted response components.

We selectively labelled and physiologically characterized dLGN-projecting RGCs by injecting a Cre-expressing retrograde herpes simplex virus (HSV) into the dLGN of a Cre/loxP reporter mouse line carrying the genetically-encoded calcium indicator GCaMP6f. The transfection of RGC terminals enabled us to perform light-evoked two-photon Ca²⁺ imaging selectively in dLGN-projecting RGCs. Visual stimuli matched those in a previously published survey of mouse functional RGC types (Baden et al., 2016) (frequency/contrast modulated full-field flicker, dense noise, moving bar). We then assigned each dLGN-projecting cell to the best-matching RGC cluster based on the correlation of the GCaMP6f mean deconvolved cell response to the deconvolved OGB-1 cluster mean. Our results showed that a large number of RGC clusters had been assigned dLGN-projecting RGCs.

In a separate set of experiments, we characterized the responses of dLGN neurons to the same visual stimuli using in vivo extracellular multi-electrode recordings in the dLGN of awake, head-fixed mice. We applied sparse non-negative matrix factorization (NNMF) on the dLGN dataset to extract visual response features. The resulting features contained a high degree of variability in contrast and frequency, and multiple features were typically needed to reconstruct dLGN neuron responses.

In conclusion, this study provides a functional characterization of the population of dLGN-projecting RGCs and dLGN neurons, suggesting that the precortical basis of vision displays an unexpectedly rich functional diversity of retino-geniculate projections and thalamic features.

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[W 88] Integrating anatomy, connectivity and biophysics to understand horizontal cell computations in the mouse retina

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In the outer plexiform layer of the mouse retina, two types of cone photoreceptors (cones) provide input to 13 types of cone bipolar cells (CBC). At this first synapse of the visual system, one type of horizontal cells (HC) provides feedback and feedforward input to cones and BCs, respectively. The full computational role of HCs is still unclear: First, recent studies suggest that - in addition to providing lateral (global) feedback to cones - HCs may also provide local, cone-specific feedback (Jackman et al., 2012, Chapot et al., 2017). However, it is unclear how the signal is kept locally isolated in the large HC dendritic tree. In addition, if and how HCs provide direct feedforward input to the different BC types is unknown. To understand the connectivity of HCs with cones and bipolar cells, we reconstructed the cone axon terminals as well as the dendritic trees of three HCs in a serial block-face electron microscopy volume (Helmstaedter et al., 2013), and quantified the contacts (Behrens et al, 2016). Horizontal cells contacted cones and ON-CBCs with their distal varicosities in the invaginating cleft of the cone axon terminal. In addition, HCs contacted ON- and OFF-CBCs with dendritic “thickenings”, short segments of increased dendritic diameter (“bulbs”) on their main dendrites, suggesting that horizontal cells contact CBCs in a separate synaptic strata in the outer plexiform layer. To better understand the conditions under which local signaling is possible in HCs, we built a biophysically realistic model of a HC dendritic branch based on the detailed morphology and connectivity. The model contains AMPA-type glutamate receptors, voltage-gated calcium and potassium channels, as well as intracellular calcium buffers and pumps. Preliminary simulations suggest that distinct features of the morphological structure of HCs support local processing as inputs from different cones remain well isolated in the very fine dendritic tips just as in physiological measurements. As a next step, we will use the model to explore the computational role of the “bulbs” and explore the effect of active conductances commonly found in HCs for switching between local and global information processing.

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[W 89] Probing oxytocin neurons activity in socially interacting rats

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The hypothalamic neuropeptide oxytocin (OT) exerts prominent pro-social effects [3] and hence considered as potential drug for treatment of psychosocial diseases in human patients [5]. Despite numerous publications focused on pro-social effects of OT, it is still unknown how social interaction affects electrical activity of OT neurons.

Recent development of cell-type specific opto- [4] and pharmacogenetic [2] viral vectors allows us to identify and manipulate OT neurons in freely moving rats. Using these vectors combined with optoelectrode technique [1, 2] we recorded single OT neuron activity in the paraventricular (PVN) in rat hypothalamus during rest, exploration, and social interaction with unfamiliar conspecifics. Simultaneously we monitored animal behavior by an automated video tracking system (Noldus EthoVision® XT) coupled to recording of ultrasound vocalizations. Our results show that social interactions induce an increase in theta rhythmicity (4-10 Hz) and in firing rate of individual OT neurons which correlates with the distance between interacting rats.

To dissect the sensory information modalities that preferably triggers OT neurons during social interaction, we recorded their activity when the animals were only able either to see, smell or hear each other. None of this sensory input was sufficient to trigger OT neurons activity for itself. In order to measure the contribution of somatosensory inputs to OT neurons activity in absence of other sensory inputs, we recorded OT cells response to tactile stimulations (i.e. air-puffs) from anaesthetized rats in various regions of the animal body. PSTH (peri-stimulus time histogram) analysis revealed an increase of OT neurons spiking activity with a delay of 0.5-1 seconds from the onset of the stimulus.

In conclusion, the evaluation of intrinsic properties of OT neurons during social interaction might help to dissect sensory pathways controlling OT neuron activity and opens perspectives for translational studies of human psychosocial diseases.

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[W 90] Optimal inference with bistable assemblies: basic theory and simulations

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Psychophysical and physiological evidence suggests that cortical representation of sensory information may rely on bistable assemblies that transition spontaneously between active and inactive states [1-3]. Here we show that such a probabilistic population code belongs to a class of codes known to support Bayesian inference [4-6], using Fisher information to clarify whether response summation preserves or loses information. Typically, lossless summation of non-identically distributed responses requires Poisson variability and stable tuning [4-6]. A population of bistable assemblies satisfies both requirements and also sums sensory signals over time. Simulations confirm that populations of bistable assemblies perform continuous inference [7], in that they integrate sensory signals nearly optimally over time. Moreover, given two (or more) such populations with disparate tuning, performance is robust in the face of time-varying signal quality: when inference is 'ignorant' (as to instantaneous signal quality) performance is nearly as good as when it is 'knowledgeable' (as to signal quality) at each point in time.

In conclusion, we report a compelling functional rationale, in terms of optimal continuous inference, for bistable assemblies with spontaneous transitions between active and inactive states, as supported by recent experimental evidence [1-3].

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[W 91] Cortical state modulates stimulus evoked oscillations in barrel cortex

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Cortical state modulates both background activity and stimulus driven responses in cortical area. State-dependency of sensory responses has been frequently reported, predominantly with a focus on spiking activity. Here, we investigated the link between cortical state and stimulus evoked oscillations in the cortex. We recorded local field potentials (LFP) from barrel cortex along with pre-frontal electroencephalogram (EEG) while presenting brief whisker deflections under urethane anesthesia. Deflections were delivered to a principal whisker using a piezoelectric device. Stimuli were centered at neurometric threshold (0, 1/2T, T, 3/2T, and 2T) and were presented in a pseudorandom order with 5second inter-stimulus interval. Cortical states were identified based on the power of low to high-frequency components of EEG (referred to as the L/H ratio) $\$1$. Each trial was then classified as being in "synchronized" or "desynchronized" state based on the L/H ratio at the time of stimulus. Short time Fourier transform (STFT) was used to calculate time frequency domain of LFPs. We observed that cortical state prominently modulated 7-12 Hz oscillations following the early response of the LFP in a stimulus dependent manner; the power of 7-12 Hz components was significantly higher in desynchronized state at 3/2T and 2T stimulus intensities. Moreover, we observed a significant modulation in gamma range frequencies (30-80). Our results suggest that post-stimulus oscillations may communicate information regarding the sensory input in a state-dependent manner.

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[W 92] Cooperative coding of moving edges in the superior colliculus

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The visual system of vertebrates is adept at extracting salient features of a visual scene such as spatial orientations and motion direction. The retina is the first stage of visual processing and targets brain areas including the superior colliculus. Here the visual scene is represented by a set of overlapping, retinotopically organized feature maps. Orientation selective neurons in the mouse superior colliculus form an inhomogeneous map where, unlike in other visual areas, neurons with the same orientation selectivity cluster together such that a single orientation is represented at each retinotopic location. However, the topographical organization of other feature maps, and their relationship to each other remains unknown. Using two-photon calcium imaging, we recorded the activity of neurons spanning more than half of the superior colliculus, while simultaneously measuring their receptive field and determining their orientation and direction selectivity. We found that the preferred axis of motion of direction selective neurons is dependent on their retinotopic position. When comparing preferred directions with orientation in the same retinotopic location, direction selective neurons showed a strong preference for directions of movement orthogonal to the preferred orientation of nearby orientation selective neurons. These findings uncover a second inhomogeneous map accounting for motion detection that can be superimposed with the pre-established spatial orientation map. Such maps appear to underlie the structure of the superior colliculus, and understanding their relationships will allow us to understand how the colliculus contributes to innate visually guided orientating behaviours.

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[W 93] Response properties of neurons in the binocular visual cortex of PSD95 knockout mice in vivo

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Our labs have recently shown that adult PSD-95 knockout (KO) mice have 9x more AMPA-silent synapses in the primary visual cortex (V1) than wildtype littermates (WT) and retain a lifelong juvenile-like ocular dominance plasticity (Huang et al., 2015, PNAS 112 (24): E3131-E3140). Together with the impaired synapse maturation of layer 4 inputs to excitatory pyramidal cells in layers 2/3 these data raised the

question how V1-neurons respond to visual stimuli in vivo. To this end, we used extracellular multielectrode recordings to investigate response properties of neurons in the binocular part of V1 of isoflurane-anesthetized PSD-95 KO and WT mice. Visual stimuli consisted of moving sine wave gratings of 8 different orientations (2 directions each) and seven spatial frequencies (0.05-0.32 cycles/degree), presented with 2 Hz temporal frequency at full contrast (42.7 cd/m² maximum luminance) to either the ipsi- or contralateral eye. We recorded both evoked and spontaneous spike rates, and quantified the orientation selectivity of the evoked responses (calculated as the orientation selectivity index = OSI), the preferred spatial frequency, and preferred direction of all recorded units. Two of the investigated response properties yielded significant differences between WT and KO mice: Compared to the WT mice, PSD-95 KO mice showed elevated response rates to contralaterally presented gratings of preferred orientation and spatial frequency (mean±SEM: WT=26±2 spikes/sec, KO=38±4 sp/s, Man Whitney rank sum test: p=0.035). In addition, the orientation selectivity index of PSD-95 KO mice was less well matched between contralateral and ipsilateral eye responses (orientation selectivity difference between ipsi and contra eye stimulation, mean±SEM: WT=0.13±0.04, KO=0.20±0.03, Man Whitney rank sum test: p=0.02). Ongoing behavioral experiments investigate what consequences these differences might have for visual perception.

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[W 94] How does structural volatility affect cortical representations?

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Even without any explicit learning paradigm, ongoing synaptic changes can be found in auditory cortex (e.g. [1]). How does this volatility in structural connections affect the functional properties of cortical circuits? We address this issue in a parallel effort of model and experiment, using a firing rate model to interpret population activity in awake mouse auditory cortex recorded via chronic two-photon calcium imaging. Previous experiments in mouse auditory cortex have shown that responses to complex sounds typically cluster into a near discrete set of activity patterns [2]. In a parameter regime where recurrent connections are sufficiently heterogeneous and the network is dominated by inhibition our model can reproduce key features of experimental data such as this type of clustering and a near log-normal activity distribution. Here we use this model to study the impact of synaptic turnover on collective response properties. Changes in synaptic strength are assumed to follow a random process matching empirical rules derived for spine size changes observed in mouse auditory cortex [1]. The model shows that gradual changes in the circuitry can induce rich dynamics in sensory representations: often,

representations remain fairly stable over extended periods of time, interrupted by abrupt and strong transitions that can affect the responses to several stimuli simultaneously, a behavior which appears consistent with our experimental data. Moreover, the overall degree of stability of stimulus responses depends more sensitively on the rate of change of inhibitory than excitatory connections. The model predicts a several-fold slower rate of change of inhibitory synapses to account for the degree of stability of representations that we observe in the data. We conclude that even subtle and random ongoing changes in synaptic connections can have a significant and highly nonlinear effect on the stability of sensory representations.

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[W 95] Visual cortical circuits in a state of flux

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The cerebral cortex is a primary learning center in the mammalian brain. To assimilate the stream of incoming information substantial circuit-turnover is therefore expected, even in primary sensory cortices. It is a long-standing theoretical prediction that visual cortical circuits are in a "state of flux", such that e.g. the preferred orientation of the neurons represents a non-equilibrium steady state of circuit turnover [1-2]. Neurons in the primary visual cortex are selective for the orientation of light/dark edges in their receptive fields and in primates and carnivores these neurons form iso-orientation domains ordered around point-like topological defects called pinwheel centers. If the circuit-turnover prediction is true and multiple steady states coexist, then signatures of circuit-turnover should be observable as a rearrangement of the domains over time. We experimentally tested this by a large-scale screen for the largest conceivable change in the arrangement of orientation domains: the generation and annihilation of pinwheel pairs [1-3]. We conducted acute high-accuracy large-scale intrinsic signal imaging experiments in 30 ferrets. In 29 of the ferrets we further employed an adapted pairing protocol [4] between imaging sessions to drive the cortical circuits out of a potential stationary state. To harvest significant topological changes we quantified measurement precision using re-sampling methods, the probability of pinwheel existence by tracking their position between bootstrap samples, and the accuracy orientation preference estimation from inter-sample tuning distributions. We analyzed the dynamics of 1590 pinwheels by comparing the measured layouts in 4 subsequent imaging sessions of 42 minutes each. Using this extensive data set we found rare but conclusive examples of pinwheel rearrangement. The rate of these events was increased when the pairing paradigm was

used. Our results demonstrate for the first time dynamical changes of visual cortical architecture establishing conclusively that visual cortex networks are in a persistent state of flux.

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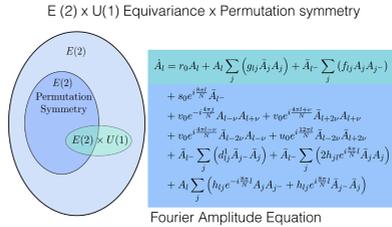
[W 96] A symmetry based parametrization of candidate optimization principles for functional architecture of the primary visual cortex

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Grey matter represents one of the energetically most expensive tissues in the mammalian body. It is thus generally expected that evolutionary optimization minimizes the allocation of grey matter to processing tasks and systems and favors functionally optimized network structures. This should especially apply to the primary visual cortex (V1), the largest areas in the neocortical visual system and one of its oldest parts. The so called common design of orientation preference maps (OPM) found in different taxa which separated since more than 65 million years ago during the basal radiation of placental mammals indicates such an underlying process of evolutionary optimization [1]. However the utility functions underlying these biological optimization processes are not well understood. Since the visual world exhibits Euclidean geometry any candidate utility functions for the architecture of the primary visual cortex is likely to be symmetric under the Euclidean group. Here we use this principle to derive a general parameterization of all symmetric utility functions for the design of the system of orientation domains and patchy horizontal connections in V1. We further subdivide the space of optimization principles by higher symmetries up to $E(2) \times U(1) \times$ the so called permutation symmetry. Manifolds of solutions called essentially complex planforms (ECP) were found to be model independent ground states of the fully symmetric model [2]. These solutions also are currently the best explanation of the common design of V1 orientation columns that independently evolved in primates and carnivores [1,3]. We find that another family of solution, the circular phase progressive solution (CPP), can also match the biologically observed common design. While CPPs are typically unstable in fully symmetric models this finding suggests to search through a wider space of utility functions for candidates that have CPPs as genuine ground states.

Overall our study for the first time provides a comprehensive parameterization and classification of utility functions for V1. This parameterization will enable to systematically screen the space of candidate optimization principles in a data-driven approach and to probe whether their solutions fulfill the benchmark of the common design.



Hierarchy of E(2) optimization theories.

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[W 98] **Inhibitory mechanisms shape visual motion processing in Drosophila**

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Many animals use motion cues to extract relevant information about their environment which they use for navigation. The neuronal network behind motion vision has been investigated for more than 60 years and is still far from being solved. To compute motion, the brain needs to detect luminance changes over space and time. Two classical models describe how to compute direction selective signals, the Hassenstein-Reichardt-Correlator (HRC) and the Barlow-Lewick model (BL). The HRC relies on a non-linear amplification of motion in the preferred direction. In contrast, the BL model suppresses signals that move in the null direction. Recent studies propose a combination of these two mechanisms that lead to direction-selective (DS) responses in the visual system (Fisher et al., 2015, Leong et al., 2016, Haag et al., 2016). The cellular components that implement null-direction suppression have not been revealed. Using in vivo 2 photon calcium imaging of the first DS cells of the fly visual system, T4 and T5, we showed that direction selectivity and orientation tuning require the contribution of inhibitory GABAergic circuits (Fisher et al., 2015). While a pharmacological block of GABAA receptors leads to a loss of DS responses as well as orientation tuning in T4 and T5 neurons, T4/T5 specific knockdown of the GABAA receptor Rdl does not effect direction-selectivity. This suggests that GABAergic inhibition does not act on T4 and T5 directly but likely takes place in upstream circuitry. Therefore, we aimed to identify

upstream inhibitory cells that are required for DS responses in T4 and T5. Reasoning that a loss of DS responses would also lead to a loss of motion guided behaviors, we used data from a forward genetic screen in which we identified InSITE Gal4 driver lines that lead to a deficit in behavioural responses to moving ON and OFF stimuli, when synaptic activity was blocked in the Gal4 pattern (Gohl et al., 2011; Silies et al., 2013). We tested if the Gal4 expression patterns contained GABA-positive neurons and identified cell types that might be required for motion vision. Additionally, we established a new cell type specific reporter strategy termed 'FlpFlag' that intersects cell type specificity with exon trapping and allows to visualize the endogenous expression of a protein in a cell type specific way. We are currently testing the role of identified GABAergic cell types for shaping DS responses and orientation tuning in the fly visual system.

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[W 99] Causal evidence for retina dependent and independent visual motion computations in mouse cortex

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How neuronal computations in the sensory periphery contribute to computations in the cortex is not well understood. We examined this question in the context of visual-motion processing in the retina and primary visual cortex (V1) of mice. We disrupted retinal direction selectivity – either exclusively along the horizontal axis using FRMD7 mutants or along all directions by ablating starburst amacrine cells – and monitored neuronal activity in layer 2/3 of V1 during stimulation with visual motion. In control mice, we found an overrepresentation of cortical cells preferring posterior visual motion, the dominant motion direction an animal experiences when it moves forward. In mice with disrupted retinal direction selectivity, the overrepresentation of posterior-motion-preferring cortical cells disappeared, and their response at higher stimulus speeds was reduced. This work reveals the existence of two functionally distinct, sensory-periphery-dependent and independent computations of visual motion in the cortex.

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[W 100] Synergistic decoding of complex texture motion from populations of direction-selective ganglion cells

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In the retina, motion direction is processed by direction-selective (DS) ganglion cells that preferably respond to a certain direction of drifting motion but are suppressed by motion into the opposite direction. A subset of these cells is thought to report the direction of global image motion, as induced by body, head and eye movements, to downstream brain areas to guide compensatory eye movements. Although direction and velocity of the projected image are constantly changing during natural viewing, the directional preference of DS ganglion cells is usually probed with uniformly drifting bars or gratings. Here, we report from experiments in the isolated salamander retina that also during complex texture motion, the directional preference of DS cells is preserved. But in complex visual scenes the encoding of motion direction is ambiguous due to the cells' simultaneous encoding of local contrast changes. These ambiguities in the encoding of motion direction of the individual cells can be resolved by reading from a population of DS cells with different preferred directions. Here, the joint population response provides more information about the global motion trajectory than would be expected by summing the individual contributions, resulting in a synergistic trajectory readout. Strong positive response correlations between DS cells enhance this synergy. This serves as an example of how population codes synergistically improve the extraction of single features from neurons that encode multiple features simultaneously from complex visual scenes.

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[W 101] Differential tuning of the low and high-frequency components of the neurophonic spectrum reveals the spike contribution of barn owl's nucleus laminaris neurons

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Extracellular field potentials are often challenging to interpret due to thousands of contributing transmembrane current sources. We aim at revealing the neural sources of the “neurophonic”, which is a frequency-following extracellular potential that can be recorded in the network formed by the nucleus magnocellularis (NM) and the nucleus laminaris (NL) in the brainstem of the barn owl. NL anatomy is well understood, and putative generators of the neurophonic are the activity of afferent axons from NM, the synaptic activation onto NL neurons, and spikes of NL neurons.

We recorded the neurophonic in response to binaural high-frequency tones (3-7 kHz) close to the recording site's best frequency, and we varied the interaural time difference (ITD). The mean activity of the monaural inputs to NL does not change with ITD. However, their relative phase does, causing cancellation or summation of input signals. The activity of the binaurally sensitive output of NL, i.e., firing rate of NL neurons, strongly depends on ITD. Our recordings contained both of these signals, and we analysed the broadband power spectrum of the response (0-18 kHz).

The low-frequency component (LFc, 200-700 Hz) of the neurophonic spectrum depended on ITD. The spectrum of extracellularly recorded NL neurons' action potentials closely resembled this component. Thus, the LFc reflects the contribution of action potentials initiated in NL neurons. The spectral component at the stimulus frequency (SFc) was much stronger than the LFc. The SFc also depended on ITD, reflecting the activity of the inputs and their relative phase change with ITD. The power spectrum at other frequencies did not depend on ITD. We used the LFc as a proxy for NL neurons' local population activity, and the SFc as a proxy for NM axons' local population activity. We compared the ITD and frequency tunings of these proxies at each recording site. The best ITDs of the LFc and the SFc were independent. Also the tuning to stimulus frequency was different: LFc's showed typically a 400 Hz lower best frequency than SFc's. Both findings indicate that the LFc might originate from NL neurons' axons in the vicinity of the electrode. Related NL neurons can be located tens to hundreds of micrometers away. The findings are consistent with the known anatomy of NL. Our analysis thus reveals the small contribution of NL neurons to the neurophonic, improving our understanding of the extracellular field potentials.

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Posters Thursday

Attention, reward, decision making

[T 1] Studying the role of dopamine in action and perception with 'active inference' and a hierarchical gaussian filter in a social decision-making task with different environmental volatilities

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Computational psychiatry aims to enable mapping of mental processes to their neural substrates for accurate diagnosis and treatment of mental disorders. To achieve that end, more sophisticated biologically plausible normative models are needed that allow for precise phenotyping of individuals based on their behavior in tasks.

In most computational models in psychiatry, perception and action are modeled as separate processes. In recent years however, it has become evident that neurotransmitters such as dopamine are involved in both action and perception, suggesting that one should be studied in light of the other. To unify these two processes, 'active inference', a Bayesian framework for studying brain function has been proposed that formulates action and perception as integral parts of the same inferential process [1]. This framework provides a normative account of decision-making that prescribes behavior by bestowing agents with a set of prior beliefs about how they should behave and the assumption that they minimize their free-energy (or surprise). Conditions that are beneficial are encoded as not surprising and minimizing free-energy naturally leads to pursuing actions that lead to 'rewards' and avoiding others. These prior beliefs are established at different time scales, for example, some are gained through evolution. We combined this framework with a hierarchical Gaussian filter that can capture subjects' ability to learn in conditions with different levels of volatility [2].

We focused on studying the role of dopamine using our model. In active inference, dopamine is hypothesized to encode the confidence in actions (i.e., the certainty that choosing a policy would lead to the desired outcomes). Dopamine has also been shown to be involved in learning in volatile environments but hypothesized to affect social learning differently [3]. To study these hypotheses, a decision-making task with social and non-social aspects was modeled that required learning under different levels of volatility. We collected data from human subjects under the influence of L-DOPA (a precursor of dopamine) or a placebo. We studied the group differences and our hypotheses by analyzing the model parameters estimated from inverting our model based on the decisions subjects made on the task. Finally, we showed the advantages of our model by comparing it to a simpler model from the reinforcement learning paradigm commonly used in behavioral modeling.

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[T 2] Environmental change events: neural encoding, attentional modulation, and perceptual correlates

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Direction-selective MT neurons play a key role in the accurate encoding and perception of visual motion. Allocation of spatial attention to the receptive field (RF) of MT neurons enhances their responses. Although most studies have focused on the encoding of constant visual motion and its attentional modulation, here, we address how MT encodes a change in the direction of visual motion and investigate how attending to such a stimulus influences its neuronal representation. We recorded the activity of 52 MT neurons from two hemispheres of two monkeys while they performed a direction change detection task. While the animal kept its gaze on a fixation point, a static random dot pattern (RDP) was shown either inside or outside the RF, cueing the animal to the location of an upcoming target. After a short time period, two moving RDPs were concurrently displayed. At a random time point a direction change of 25° occurred in the RDP inside the RF. The monkey was instructed to detect the target change and to ignore similar distractor changes to receive a fluid reward. Our data show that the MT population response precisely encodes the motion direction, however, it encodes a direction change that is larger than the physical change. For the distractor stimulus, the overestimation was 8° and 13° for the target stimulus. We show this overestimation and its attentional enhancement can be accounted for by normalization models of adaptation. Using a multiplicative model based on the normalization model of adaptation, we were able to largely capture the characteristics of the neuronal response to the direction change. We also determined the perception of such direction changes in a human psychophysical experiment, similar to that used with the monkey. Our results show that a physical direction change of 25° is perceived as 32°, an overestimation of about 7°. Applying a linking model to our electrophysiological results support the notion that an overestimated encoded direction change in MT underlies the overestimation of the perceived direction change. Our data demonstrate that both encoded and perceived direction changes are overestimated. The magnitude of encoded direction change

overestimations is almost twice as high for attended vs. unattended stimuli. Our results suggest that the role of attention is not only increasing the strength and accuracy of attended stimulus representation, but also enhancing the neuronal representation (the saliency) of change events.

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[T 3] Bayesian mapping reveals that attention boosts neural responses to predicted and unpredicted stimuli

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Predictive coding posits that the human brain continually monitors the environment for regularities and detects inconsistencies. It is unclear, however, what effect attention has on expectation processes, as there have been relatively few studies and the results of these have yielded contradictory findings. Here, we employed Bayesian model comparison to adjudicate between 2 alternative computational models. The “Opposition” model states that attention boosts neural responses equally to predicted and unpredicted stimuli, whereas the “Interaction” model assumes that attentional boosting of neural signals depends on the level of predictability. We designed a novel, audio-spatial attention task that orthogonally manipulated attention and prediction by playing oddball sequences in either the attended or unattended ear. We observed sensory prediction error responses, with electroencephalography, across all attentional manipulations. Crucially, posterior probability maps revealed that, overall, the Opposition model better explained scalp and source data, suggesting that attention boosts responses to predicted and unpredicted stimuli equally. Furthermore, Dynamic Causal Modelling showed that these Opposition effects were expressed in plastic changes within the mismatch negativity network. Our findings provide empirical evidence for a computational model of the opposing interplay of attention and expectation in the brain.

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Brain disease, network dysfunction and intervention

[T 4] Automated Validation and Comparison of Models of Neurophysiological Biomarkers of Psychiatric Disorders

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Research on psychiatric disorders has gradually shifted its focus from complex clinical phenotypes towards the identification of biomarkers and endophenotypic measures. Computational approaches have gained significantly more attention over the last years, and this has led to the emergence of 'Computational Psychiatry' as an independent discipline. Computational modeling of biomarkers promises to more readily shed light on the mechanisms underlying disorders and to facilitate the discovery of novel medications [1]. However, the development of computational models requires scientists to have an in-depth understanding of the current, relevant experimental data, the current state of computational modeling and the state-of-the-art of statistical testing [2]. In a field where both the number of experimental and computational studies grows rapidly, such as psychiatry, this becomes more and more impracticable. Omar et al. therefore proposed a framework for automated validation of scientific models, SciUnit [3]. Here, we propose to adopt this framework for the computational psychiatry community and to collaboratively build repositories of experimental observations, computational models, tests and tools. As a case in point, we built an experimental database for auditory steady-state response (ASSR) deficits in schizophrenic patients, based on observations from several experimental studies [4,5,6], and, building on SciUnit, we have implemented a set of tests that cover a range of ASSR deficits together with tools for advanced visualization of model data. Here, we demonstrate how existing computational models [6,7] can be validated against these observations and compared against each other. We included computational models that not only comprise biophysically detailed as well as abstract models, but that also differ in implementation (Python vs. Genesis vs NeuroML2), in order to demonstrate the flexibility of the approach. Furthermore,

our approach enables us to assess the variability of the produced model output. This is achieved by generating a distribution of model instances where certain parameters, such as the precise timing of noise inputs (however, not the strength/type of noise) or the precise connectivity (however, not the underlying distribution of connectivity), vary, which then is used to produce a distribution of model outputs. This can inform on the robustness of the findings and can also be compared against the variability of experimental observations.

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[T 5] Modeling ischemic stroke using NEURON's reaction-diffusion module

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In ischemic stroke, the accustomed network of synaptically-connected neurons is not the main source of relevant interneuronal effects. There are additionally a large number of toxins that are released, primarily from the central core area of ischemia. These agents spread out by diffusion at different rates, serving as spatial "signals" that produce different types and different severity of damage to cells, while triggering protective mechanisms as well. The ischemic core, this central area of irreparable damage, is surrounded by a penumbra where cells may be able to recover if adequate treatment is received. We postulate that multitarget, multitemporal (and multiscale) pharmacological solutions will be needed, where particular agents are directed at the correct time and correct locations to interfere with the dominant pathological processes occurring at that time and place.

The modeling of this complex phenomenology requires that we combine many different types of models to handle this multiscale, multiphysics problem. In brief, the sequence of damage begins with blood vessel occlusion which triggers cascades of interacting changes that include: 1. local glutamate release producing neuron activation which can activate other synaptically-connected cells; 2. increased extracellular potassium (spreading depression) 3. edema (cell swelling) which reduces extracellular volume; 4. production of reactive oxygen species (ROS), which diffuse quickly and directly damage proteins in other cells; 5. local inflammatory response with local invasion by a variety of cell types, some intrinsic to the brain and others coming in due to breakdown of the blood-brain barrier.

As an initial approach, we have assessed at the effects of spreading depression (spread of potassium) on cell firing, K^+ depolarizes cells, causing increased firing and increasing their vulnerability to cell death. In the context of cell swelling, extracellular space (ECS) volume is decreased which produces additional increase in ion concentration. We additionally consider the effects of astrocytes by modeling ECS as an active medium with K^+ uptake that will decrease with glial damage. At the cellular level, ischemia reduces ATP resulting in failure of the Na^+/K^+ pump and further rise in local K^+ .

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[T 6] Modelling the temporal and spatial dynamics of inflammatory lesion formation in multiple sclerosis

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We present a computational approach to lesion formation characterised by the interaction between pro-inflammatory T-cells, neurons and the microglial and astrocytic inflammatory reaction of the brain parenchyma around a venule using a tractable dynamical model. MS is a chronic autoinflammatory demyelinating disease of the central nervous system, ultimately leading to progressive neurological disability in affected patients. With our model we aim to understand the spatial and temporal sequence of immunopathological events observed in human histology and in vivo two-photon imaging experiments in mice. Specifically, we present a tractable low-dimensional computational model which incorporates the available experimental data to predict the dynamical and spatial structure of inflammatory reactions leading to neuronal cell death. We implement direct excitotoxic interactions between neurons and pro-inflammatory T-cells leading to elevated intracellular calcium levels [Siffrin, et al (2010)], as well as the neurotoxic effect by the inflamed tissue, e.g. by over-activated microglia, and effects due to suppressed astrocytic glutamate re-uptake. Our model gives an insight into the temporal and

spatial dynamics centred around a venule, and ultimately allows for a classification of lesion types into two groups: One in which neuronal damage is only caused and limited to interactions with infiltrated pro-inflammatory T-cells, and a second in which T-cells trigger an inflammation-induced reaction of the system, which causes widespread neuronal damage. We explore the limits between these two regimes with respect to structure, connectivity and susceptibility of the brain tissue, as well as duration of T-cell infiltration.

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[T 7] Neural modeling of developmental lexical disorders

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Standardized tests exist for the diagnostics of developmental lexical disorders, but it is still difficult to associate the resulting behavior of a child while speaking with functional deficits in the child's brain. The mental lexicon is part of the speech and language knowledge repository of individuals. It enables humans to produce as well as to understand speech. The computational frameworks we used for implementing a model of the mental lexicon and speech processing are the NEF (Neural Engineering Framework, Eliasmith et al. 2012, Eliasmith 2013) and the SPA (Semantic Pointer Architecture, Eliasmith et al 2012, Stewart & Eliasmith 2014). These frameworks allow modeling of large scale neural networks, comprising sensory, motor and cognitive components. The modeled task is the WWT 6-10 (Word range and Word Retrieval Test, see Glück 2011), which comprises 95 items and is a picture naming and word comprehension task. In case of incorrect answers semantic and phonological cues are also given in order to facilitate word production. A major goal of this study is to introduce a quantitative neurocomputational model for lexical storage as well as for lexical retrieval. A further goal of this study is to associate neural dysfunctions with deficits in speech behavior. Concretely, the deficits of interest are in lexical storage and lexical access. The dysfunctions introduced here are the lesioning of specific neural SPA-buffers and of specific neural connections between these buffers. Based on the behavioral data given by the WWT, we are now able to associate functional neural deficits with symptomatic behavioral data. This allows us to identify potential dysfunctions at neural level for word retrieval and word storage.

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Computational connectomics

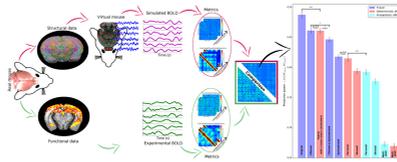
[T 10] Predictive power of connectome-based models

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Connectome-based models are an appealing framework for investigating brain dynamics since they allow a potential causal investigation of large-scale mechanisms. The connectome is the basis of this sophisticated apparatus and, consequently, the tools used to reconstruct it can affect the modeling outcomes. Here we quantify how model prediction reliability depends on the connectome reconstruction. We simulate resting state dynamics in mouse brain using both (i) tracer connectome built through The Virtual Brain (Sanz-Leon et al., 2013, Melozzi et al., 2017) exploiting the Allen Institute dataset (Oh et al., 2014) and (ii) diffusion Magnetic Resonance Imaging (dMRI) connectomes, from 19 animals, processed both with deterministic and probabilistic tractography. The goodness of the simulated data is tested against experimental functional data through head-fixed fMRI resting state experiments in the same 19 mice. To quantify the contribution of connectome's characteristics in shaping different simulated functional pathways, we built surrogate connectomes, where the hypothesized differences were enhanced. We demonstrate that preserving individual variability, i.e. not averaging dMRI data between subjects, is crucial for obtaining reliable predictions. In line with the finding we show that the predictions achieved by deterministic processed dMRI data are more reliable than the ones obtained by probabilistic processed data, since deterministic tracking preserves connectome specificity. The observation challenges the supposed superiority of probabilistic algorithms in the large-scale models framework; a similar finding in the graph theory context has been reported by Zalesky et al., 2016. We find that the tracer-based connectomes' ability in predicting resting state activity is greater than the one of the dMRI-based connectomes. From the analysis of the potential causes of this discrepancy we identify two, independently sufficient, factors: the dMRI inability of detecting fiber directionality and of resolving complex fiber pathways. We identify the areas whose connections, reconstructed with dMRI, mostly negatively affect brain predictions. On the other hand we find the areas whose connections, since highly characteristic for each brain, are better reconstructed by dMRI method than by the tracer one, highlighting once again the importance of individual variability in modeling predictions.



The left part of the figure summarizes the workflow used to model resting state dynamics in mouse brain. The right part of the figure shows the predictive power ability of differently derived connectomes.

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Data analysis, machine learning, neuroinformatics

[T 11] Instrumenting network simulations with the NESTConnectionApp

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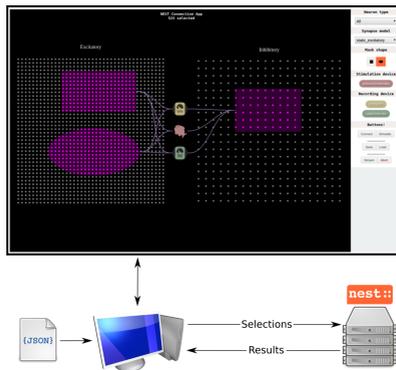
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Just as neurophysiologists attach recording and stimulation devices to brain areas in vivo, we should be able to instrument in silico network models. We present here the NESTConnectionApp, providing a graphical user interface to connect stimulation and recording devices to spatially structured neuronal network models. The app will allow computational neuroscientists to set up in silico experiments more easily than by scripting. We anticipate that such graphical user interfaces will be increasingly requested by users as network models grow in size and complexity. Beyond in silico neurophysiological experiments, the app will also be useful in connecting artificial brains to robotics systems, connecting robot sensors and actuators to brain models.

The NESTConnectionApp consists of two parts, the server side and the client side. On the client side, the application is independent of NEST and is implemented in JavaScript, using the libraries Three.js, D3.js, and React. Layer representations are created from a JSON file providing information on neuron locations and properties. The user selects neuron populations to be connected by marking rectangular or elliptic masks in a layer of neurons. Additional specifications for the selection can be made, such as what type of neurons to selected, and what synapse type to use for connections. A connection to a specific device is then specified by dragging a line from the selection mask to a device. Ultimately, all connection specifications are represented in JSON format and can be

sent to the NESTConnectionApp server or stored locally for later retrieval. Note that the JSON format only stores information about the selection masks and other selection criteria.

To instantiate connections in a NEST simulation, both the network layout and the connection specifications are sent to the NESTConnectionApp server in JSON format. The server, implemented in Python, runs a Flask microframework and PyNEST. On receiving network and connection specifications from the client, the server creates the layers, devices, and connections between the two. Optionally the server can run a simulation and send results back to the client. Work is currently under way to extend the app to support three-dimensional networks and to integrate the NESTConnectionApp in the Human Brain Project Collaboratory.



Instrumenting a two-population network: Two areas in the excitatory and one area in the inhibitory population are instrumented with a Poisson generator, a spike detector and a voltmeter.

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[T 12] Binding of visual features in the macaque prefrontal cortex

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Feature binding, the process through which different feature dimensions are combined into a unitary percept, is a critical concept in cognitive neuroscience. Although different previous studies have made efforts in understanding the underlying neural mechanism, it remains poorly understood. Here we show that the activity of lateral prefrontal cortex can be linked to binding of visual features in the macaque brain. For this purpose, we trained two monkeys in a visual delayed match to sample task, to match the sample and test stimulus in each trial based on their color and direction of motion. The sample stimulus consisted of one patch of moving, random dots, while the test stimulus consisted of two overlapping patches of moving dot patterns, one coherently moving towards a direction and the other moving randomly towards an arbitrary direction. In half of the trials, the color which the monkey had to match was assigned to the coherent patch (bound condition) and in the other half, the color was assigned to the randomly moving patch (unbound condition). Monkeys had to report by pressing a button, if the sample and test stimuli matched in terms of their color and direction of motion. Each animal was implanted with a 96 channel electrode array in its IPFC. LFPs were recorded from the electrode arrays while the monkeys performed the task. Using a Bayesian classifier, we show that this coding occurs exclusively in low frequency bands. By applying a 10-fold cross-validation based on wavelet coefficients as the input features, binding condition is decoded significantly in Theta (4-8 Hz) and Alpha (8-12 Hz) bands with up to 67% and 66% accuracy, respectively. We conclude that feature binding is coded in IPFC - a high-level cortical area - which receives input from both dorsal and ventral visual pathways. Additionally, this coding occurs in low frequency bands, theta and alpha, suggesting that the coding is widely distributed across neurons of IPFC.

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[T 13] Spiking Neural Networks. A new methodology for fMRI data analysis.

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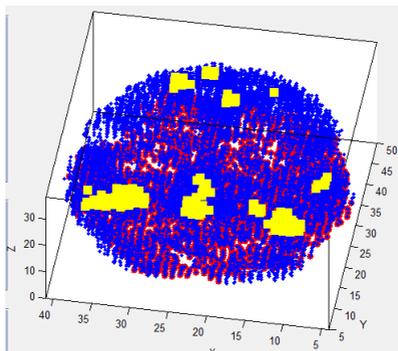
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Classical methods of analysis in functional connectivity in MRI, as Independent Component Analysis (ICA) or Sliding-Window Seed-Based Correlation Analysis (SWBCA), have showed some limitations to recognize the dynamic evolution of the brain networks [1]. The use of new computational approaches derived from Artificial Intelligence such as Deep Learning or Artificial Neural Networks are increasing due to their successful results. However further research of this technology is needed to validate the approach

This work shows a proof-of-concept of a novel methodology that uses the NeuCube framework [2] for data where a no priori hypothesis or huge reduction of dimensionality has been applied. However, its most promising feature is the capability to develop a multimodal platform where different type of data (fMRI, EEG, DTI or epigenetics) could be combined to identify new information and biomarkers or create predictive models in the future.



DMN captured in real brain fMRI data

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[T 14] Spatio-temporal Spike Pattern Detection in Massively Parallel Recordings

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Cell assemblies [1], i.e. interacting groups of neurons, were suggested as the building blocks of information processing in the cortex. Modern electrophysiological techniques allow to record hundred(s) of neurons simultaneously and thereby hopefully increase the chances to observe active cell assemblies. Their activity is assumed to be expressed by synchronous or spatio-temporal spike patterns (STPs) [2,3]. We developed SPADE [4], a statistical method that detects synchronous spike patterns in massively parallel spike data (MPST), i.e. in the order of 100 or more parallel spike trains. The method is able to deal with the huge number of patterns occurring by chance in such high-dimensional data by employing a combination of 1) frequent item set mining [5] to efficiently extract and count repeating spike patterns and 2) a Monte-Carlo approach to evaluate the statistical significance of the found pattern under the null hypothesis of independence. To avoid a massive multiple testing problem we reduce the dimensionality of the pattern candidates by pooling patterns of same number of neurons (size) and number of occurrences. In [6] we applied SPADE to MPST extracted from electrophysiological data recorded from motor and premotor cortex of non-human primates [7]. The monkeys performed a delayed reach to grasp task, where they had after a preparatory period to pull and hold an object using a side or precision grip and with high or low force. We hypothesized that different cell assemblies are activated at different points in time in relation to the behavior. Therefore recordings of the same set of neurons were analyzed for the occurrence of significant spike patterns in different behavioral epochs and in the four different behavioral conditions (combinations of object loads and grip types). We found a variety of significant patterns that show specificity to the behavior. We recently extended the SPADE method to also detect STPs [8, 9], i.e. patterns composed of spikes occurring with temporal delays. It turned out that the same statistical framework as in SPADE for synchronous patterns can be applied, although the number of chance patterns is much larger than in the synchronous case. Preliminary analysis of the same data as in [6] reveal that STPs do occur, also primarily during the movement, and are formed by a larger number of neurons. All together these findings provide evidence for the existence of higher-order patterns occurring in relation to behavior.

Acknowledgements

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[T 15] Neural system identification for large populations separating “what” and “where”

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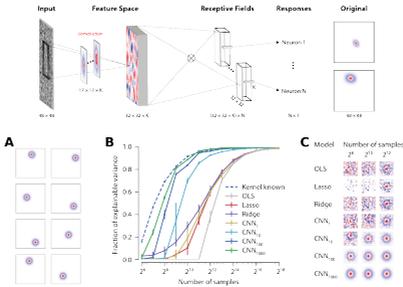
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Neuroscientists classify neurons into different types that perform similar computations at different locations in the visual field. Traditional neural system identification methods do not capitalize on this separation of “what” and “where”. Learning deep convolutional feature spaces shared among many neurons provides an exciting path forward, but the architectural design needs to account for data limitations: While new experimental techniques enable recordings from thousands of neurons, experimental time is limited so that one can sample only a small fraction of each neuron’s response space. Here, we show that a major bottleneck for fitting convolutional neural networks (CNNs) to neural data is the estimation of the individual receptive field locations – a problem that has been scratched only at the surface thus far. We propose a CNN architecture with a sparse pooling layer factorizing the spatial (where) and feature (what) dimensions. Our network scales well to thousands of neurons and short recordings and can be trained end-to-end. We explore this architecture on ground-truth data to explore the challenges and limitations of CNN-based system identification. Moreover, we show that our network model outperforms current state-of-the-art system identification models in the mouse visual system.



(Top) Our proposed CNN architecture in its simplest form. It consists of a feature space module and a readout layer. (Bottom) Feature sharing in homogeneous population: A) Simulated neurons, B) Model comparisons, C) Learned filters.

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[T 16] **A Statistical Field Model of Spatiotemporal Neural Point-Processes Applied to Large Scale Neuronal Recordings**

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High-density multi-electrode arrays now enable detailed recording of large-scale spatiotemporal spiking activity, and there is a growing interest in point-process state-space models for describing collective dynamics in population spiking activity. Conversely, statistical field models of spatiotemporal neural activity have been influential in explaining qualitative phenomena in collective neural dynamics. This work connects neural field theories with state-space point-process models, building on recent work that combines latent field models with point-process observations for simulation and inference in chemical reaction-diffusion systems. We develop a Bayesian spatiotemporal point-process filter for neural fields, based on a master equation formulation of the three-state “Quiescent-Activated-Refractory” model. We demonstrate a state inference algorithm on spontaneous waves in the developing mouse retina, which can approximate the posterior distribution for mean-field intensities and their spatial correlations. This work lays the groundwork for integrating statistical neural field theories with data-driven state-space models of spiking population dynamics.

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[T 17] Automated parameter fitting and testing of detailed neuronal models

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Although biophysically detailed multi-compartmental neuronal models can be useful tools in understanding and predicting the behavior and function of neurons, building such a model is still a complex task. Typically, many parameters of such a detailed model have not been experimentally determined. These parameters are often tuned using manual methods, with the aim of reproducing a few specific features of the real cells. As a result, there are often many different models available for the same cell type, which were developed and used for different purposes, and it is usually unknown how they would behave outside their original context.

In order to make principled and reproducible model building possible, we are developing software tools for the automated fitting of unknown parameters and for the automated testing of model behavior in different situations.

For parameter fitting we are developing a general software tool called Optimizer (<https://github.com/KaliLab/optimizer>). Optimizer offers a GUI which helps non-expert users optimize models defined in Neuron using some common protocols. It also offers many advanced features for expert users, and its modular structure makes it possible to extend it by adding new ones. We describe how we improved Optimizer since its initial release, how it can be used to compare the performance of different algorithms, and how we used it to fit the parameters of a CA1 pyramidal cell model to reproduce somatic spiking features.

To automatically and quantitatively test and compare the behavior of different models, we are developing a python test suite called HippoUnit (<https://github.com/sasaray/neuronunit>), which is based on NeuronUnit and SciUnit. There are three tests implemented so far in HippoUnit that mimic experimental protocols to test the somatic spiking features and probe the integration properties of the oblique dendrites of hippocampal CA1 pyramidal cell models. The tests of HippoUnit allow the parallel run of the different stimulating protocols and save the model's response and the most important results for later use. For quantitative comparison between the model's response and experimental results it uses feature-based error functions. The tests of HippoUnit have been integrated into the Brain Simulation Platform of the Human Brain Project.

These tools should encourage collaborative research by making possible the systematic and reproducible building, testing and comparison of detailed neural models.

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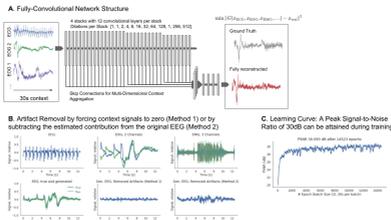
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[T 18] **Fully Convolutional Signal Transcription for Artifact Removal and Normalization**

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Electrophysiological recordings are usually prone to artifacts that are commonly removed by visual inspection and signal decomposition techniques such as ICA in clinical and research contexts. In this work, we propose a generative deep learning model for EEG signals inspired by the WaveNet architecture conditioned on biosignals from other modalities (EOG, EMG and ECG) that are expected to capture relevant context information on artifacts that are simultaneously recorded in the EEG signal. As our main contributions, we show that it is possible to capture non-trivial correlations between time-series from multiple modalities in a fully-convolutional neural network. By dampening the context signals during inference, it is possible to remove the caused artifacts in the EEG signal by (1) generating a possibly uncorrelated EEG sequence from a dampened version of the context signal (2) estimating the context signal's contribution to a particular kind of artifact and removing this effect from the original EEG sequence, preserving the signal information. In this poster, we present preliminary results on the openly available MASS cohort-3 sleep database, comprising recordings from various patients recorded during sleep.



A. Proposed WaveNet-inspired network architecture. Using recordings from ECG, EOG and EMG recordings as context signals, the network generates an EEG sequence matching the context signals. B. Demonstration of artifact removal C. Network learning curve

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[T 19] Computational Based Approach to Explore & Decode Human Consciousness: Using Artificial Consciousness & Artificial Neural Mathematics (ACANM)

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Human Brain is complex phenomenon especially when one can look at its functionality, behaviour and evolutionary organization. Certainly brain is a different planet in human body or embedded system which is associated with all integral functional actions executed by itself as per need or thought. We can call this our thought converted into auxiliary and supplementary behaviours into actions. Still even in 21st century where advancement and innovation in medical technology more precisely medical imaging, medical physics gives enough signature and insights to decode human brain fully but in reality we are far away to explore human brain completely. One of the key challenges to decode and explain consciousness, and also how our brain process mathematical problems. Understanding mathematics or its ability fully depends on consciousness. We cannot compute everything what we have in our conscious mind and it is also true that conscious thinking or mind cannot control or simulated by a computer. Decoding & exploring such complex problem really a herculean task. In our study and proposed model we are introducing some artificial parameters by known behaviours and assumptions which are governed by conscious thinking. These proposed parameters are called Baye's conscious thinking where we are already aware about probability of outcomes. We train such conscious thinking with artificial neural mathematical ability and classify such outcomes and compare them with controlled set of ideal parameters. We observe and draw a conclusion that trained and untrained brain has huge potential difference in conscious thinking or having conscious mind. Our proposed computational model will solve some fundamental issues and key questions related to human brain. Of course computational based model has certain limitation and could not meet natural parameters because brain is dynamical with chaotic system and need some causal constraints. One can also bring medical imaging for brain scan to check & establish new paradigm.

Keywords: Brain, Medical Imaging, Medical Physics, Encoding, Computational Model.

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[T 20] Tools, Formats and services for efficient data management, collaboration and reproducibility in neuroscience

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Management of scientific data, including consistent organization and storage of data, is a challenging task. Data needs to be annotated with metadata to provide information about the underlying experiment to ensure reproducibility. Accessing and managing data from multiple workplaces while keeping it in sync, backed up, and easily accessible from within or outside the lab, is even more demanding. To minimize the time and effort scientists have to spend on these tasks, we here present formats and tools designed for comprehensive and reproducible management of scientific data. To easily store, select, retrieve and share data using an open format we provide the NIX[1] format, which offers convenient organization of data and metadata, supporting various data types including electrophysiology and imaging, and enables to effectively link data and corresponding analysis results as well as the associated metadata. NIX builds on the odML[2] metadata format and is supported by the Neo[3] Python package for electrophysiology, enabling Neo users to store their data in a common open format. Keeping data organized in the lab is made easy via the GIN[4] services. GIN keeps track of changes to the contents and organization of the files and provides secure remote access, making it convenient to work from multiple workplaces while keeping all data available and in sync. Data can be managed from web and file browsers or through a command line interface, enabling even integration into data acquisition and analysis procedures. The system works with any kind of directory structure and file types, using established technologies to keep previous versions accessible when datasets are updated. The service furthermore makes it straightforward to share data within a lab or with off-site collaborators and to work on it together. Any data hosted with the service can easily be made persistently available for publication using digital identifiers. Combining GIN and NIX allows streamlining data workflows and eases the sharing of well-annotated datasets within the lab, among collaborators between labs, or with the public.

Acknowledgements

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[T 21] Detecting causality from time series

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Determining causal relations from time series observations is a non-trivial task and recently gained paramount interest in many scientific fields including neuroscience. The knowledge of causal relations is particularly important in the treatment of patients with drug-resistant epilepsy, where the only treatment option is the surgical removal of epileptic focus. In this case, clearly, there is no opportunity to execute experiments and the least invasive solution is to implant electrodes into brain tissue and try to localise the epileptic focus from extracellular field potential observations.

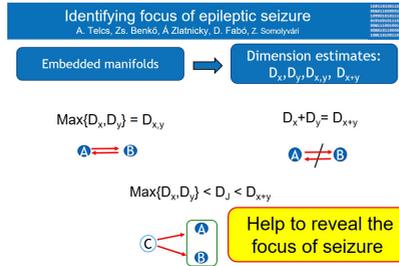
When we observe correlation between two time series it does not determine the exact causal relation between the variables. That it is possible that one is a cause of the other or vice versa or both and it is also possible that there is a hidden common cause triggering the correlation between the two variables.

In the '50-s Norbert Wiener proposed a predictive principle for determining directed causal relations from time series observations. In the next decade the first practical implementation was made in an autoregressive modelling framework by Clive Granger. In the early 21st century transfer entropy was introduced as nonparametric method for determining causal relations using the very same Wiener principle. A few years later George Sugihara et. al. published a causality detection method using the dynamical systems abstraction. It is based on Takens' theorem and the topological equivalence of manifolds in state-spaces reconstructed with time-delay embedding from time series.

However these methods can be used for retrieving directed causal information from observations there have been no single method existed yet that could identify all causal cases, for example none of the aforementioned methods can detect a hidden common cause.

Here we present a new causality detection method which uses manifold dimensions to determine causal relation from time series data. This method can identify independence, directional and circular causal effects as well as hidden common causes properly by assigning probabilities to each possible case.

We demonstrate our method on simulated examples and on neurophysiological measurements.



Relation between intrinsic dimensions reveals causal connections

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Learning, plasticity and memory

[T 23] Input-dependent synaptic consolidation in Hebbian cell assemblies

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Long-term synaptic plasticity plays a major role in the formation of memory representations in the brain. It exhibits an early phase with changes lasting up to several hours and a late phase with changes lasting up to several days [1, 2, 3, 4]. The transfer from the early to the late phase is referred to as synaptic consolidation. It requires a synaptic tag and protein synthesis in the postsynaptic neuron (synaptic tagging and capture hypothesis [2, 3]). Protein synthesis as well as the formation of tags depend on ongoing neuronal activity and external stimuli. Some research has been done on these dynamics using single-synapse models of synaptic consolidation [5, 6, 7], but the role of consolidation in network phenomena such as Hebbian cell assemblies remains elusive. Hebbian cell assemblies are groups of neurons with significantly strengthened interconnections. Their neurons tend to fire together, in this way making them serve as memory representations [8]. We assume that the impact of synaptic consolidation on the formation, stabilization and interaction of cell assemblies is of great importance.

Here, we study the stabilization of cell assemblies following different stimulus protocols, that is, the input-dependent transfer of assembly-related synapses from the early-phase to the late-phase state. For this, we use a spiking network model with early- and

late-phase synaptic plasticity based on a model widely used for single neuron dynamics [6, 7, 9]. Depending on the strength of the stimulus, our results exhibit three different scenarios: consolidation in the whole assembly, consolidation restricted to the core of the assembly and no consolidation at all. In the latter case, only the early phase of synaptic plasticity is induced. Our work provides a further step in understanding the consolidation of memory representations in biologically realistic neuronal networks. Further studies shall yield the impact of our findings on the interaction between cell assemblies.

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[T 24] How should interneuron activity be regulated to homeostatically control principal cell firing rates?

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Neuronal networks in the brain respond to chronic manipulation of activity with changes at a variety of different scales, both spatial and temporal [1]. For instance, changes can be observed in the firing rates of various interneurons, which respond over different time courses following experimental intervention [2-4]. The functional role of these responses are poorly understood, but one plausible interpretation is that the activity of certain interneurons is regulated such that the firing rates of excitatory cells are brought back to a homeostatic set-point. Because interneurons project to many excitatory cells, such a form of homeostasis would act on the network level, rather than cell-autonomously, as most classical homeostatic mechanisms [1].

Here, we address the question: Which rules should govern interneuron-based forms of homeostasis? That is, which neurons in the network should change their activity

in response to deviations in the firing rate of a single excitatory cell? Is a local rule sufficient—one that changes those interneurons to which the excitatory cell projects—or does homeostasis require a more elaborate, non-local scheme?

Using numerical simulations, we first show that for local rules operating on excitatory-to-inhibitory synapses (analogous to [5]), excitatory cells compete to control inhibition, driving most of the network into a quiescent state. This competition arises when input heterogeneities are combined with the fanout in recurrent connectivity, producing de-localized inhibition when local inhibition is asked for. Such local rules regulate neither individual cells nor population activity.

Having shown that local rules are not homeostatic, we derive a gradient-based rule—with the objective of minimizing the total squared-error of all individual excitatory firing rates with respect to the homeostatic rate. While the resulting rule is biologically implausible, we discovered that it can be simplified to yield two plausible, though non-local rules: one rule relying on retrograde signals, and the other relying on more generic diffusion-like signals. Both change the activity of each interneuron in response to their postsynaptic excitatory population, which makes them effective in controlling the population rate.

Our results suggest that if interneuron activity is to homeostatically control principal cell firing rates, then it must rely on a non-local measurement of population activity, rather than a cell-autonomous mechanism.

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[T 25] On the capacity of sequence learning neural networks

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There is a long-standing interest in the relationship between sequences of brain activity and behavior and cognition. Hebb theorized that sequential activation of cell assemblies (“phase-sequences”) would form the basis of “the thought process” [1]. Sequential neural activity that is time-locked to behavior has been observed in the neural circuits of the hippocampus [2], cortex [3] and in the “high vocal center” (HVC) in birds [4]. Revealing the underlying neural mechanisms of these sequential neural activity patterns and explaining how these correlates participate in information processing remains an open task in computational neuroscience. The question of how neural systems can reliably self-organize to learn and reproduce sequences has been long studied in the field [5]. Recently there has been a promising modelling attempt at investigating sequence learning in the cortex [6] and dynamical sequence warping in striatum [7]. The proposed models however have only been studied with a limited number of sequence patterns and it still remains unknown how they perform in conditions with a larger sequence memory load. This is an important question in the context of network scaling properties. In this work we propose a non-spiking attractor network model capable of recalling and learning sequences of attractor memories. This model extends the previous work [5], where a spiking attractor network model can learn sequences of attractor states, manifested as quasi-stable cell assembly activations, by means of the the Bayesian Confidence Propagator Neural Network (BCPNN) synaptic plasticity rule. Since the question about the storage capacity of the proposed network in various configurations has not yet been addressed systematically, we have conducted a comprehensive quantitative analysis in this work. In essence, our contribution is two-fold: i) we put forward a firing-rate system than can self-organize with the adequate stimulus to both learn and reproduce sequences with biologically relevant constraints and architecture, and ii) we advance a methodology to parameterise the sequence space which facilitates a systematic study of sequence storage capacity in models.

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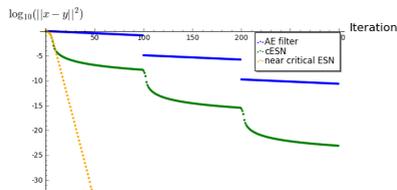
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[T 26] Arithmetic encoding, reservoir computing, criticality and biological implications

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Arithmetic encoding (AE) [1] and reservoir computing have so far been handled as two complete unrelated topics. We demonstrate that an arithmetic encoder can easily be modified into a linear online filter. This form of the encoder relates to the initially independent ideas about one type of recurrent neural networks, that are echo state networks, because this filter fulfills the echo state property (ESP). Thus, it is a reservoir in the sense of reservoir computing. At this point we see important implications and insights mostly on reservoirs. In the past many researches have indicated that the performance of reservoirs significantly depends on their dynamic features and recurrent connectivity. Near edge of chaos approaches [2] have been proposed as peak performers with regard to reproducing trained output capabilities. This performance may be caused by the fact that in the case of a strong recurrent connectivity a larger window of the input history is available for information processing. However, the onset of strong dependence of initial settings (one might call that "chaos") sets a limit to recurrent connectivity [3,4]. Since due to their physical or mathematical design all types of reservoirs have a limit of their information capacity, features of applicable memory compression may turn out to be very useful to get even a longer time span of the history into play in order to train an output. The present contribution details out the above mentioned version of the AE that fulfills the ESP, discusses implications on multi-neuron reservoirs and also goes into the field of critical echo state networks (ESNs), which is also connected with features of AE. Also in critical ESNs the memory is only limited by the entropy of the input time series in relation to the reservoir's capacity. The results show that cESNs are potentially a good concept for memory compression in reservoirs. We are further discussing how multi-neuron cESNs can be designed, biological implications of cESNs with regard to power law statistics found in brain slices (also cf. [5]). As an additional feature, it is possible to evaluate probabilities of future inputs (following ideas in [6,7]) out of the reservoir dynamically which might raise the possibility of incremental learning. So, the purpose of the poster is to bring together ideas about recurrent neural networks, memory compression, input prediction and incremental learning.



Semi log plot of the difference of two initially different internal states of otherwise identical neural networks is retained over time if both networks receive identical input. Input is synthetic, where 99 iterations the input is as expected with $p=0.99$ while at one iteration another input occurs.

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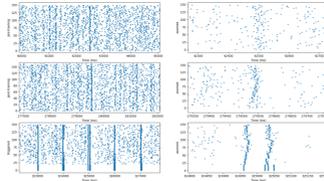
[T 27] Unsupervised Sequence Learning and Sharp Wave-Ripple-Like Spontaneous Replay in a Biologically Plausible Self-Organizing Neural Network

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The learning of spatiotemporal sequences by the cortex and hippocampus is of fundamental importance to navigation, pattern recognition, motor learning, and numerous other vital cognitive functions. This action is particularly remarkable in light of its complex and unsupervised nature, when most modern machine learning techniques require extensive supervision for such learning. Within the cortical-hippocampal loop, phenomena known as sharp wave-ripples (brief bursts of increased and semi-synchronous activity occurring primarily during rest or sleep in which previously observed activation patterns are frequently embedded) are theorized to be critical for the consolidation of memories and long-term pattern storage (Diekelmann and Born 2010). We present here a unified model combining a sparse inhibitory and recurrent excitatory population of simple conductance-based leaky integrate-and-fire neurons with two synaptic plasticity mechanisms (one causal Hebbian and the other homeostatic) and a homeostatic intrinsic neuronal plasticity mechanism (Lazar et al. 2009, Hartmann et al. 2015, Miner and Triesch 2016). All plasticity rules are local. The model's spontaneous activity is driven by a combination of the homeostatic intrinsic plasticity and intrinsic noise. We find that the model is capable of learning (via repeated unsupervised exposure), storing, and recalling (at an accelerated timescale) a sequential spatiotemporal pattern upon receiving a cue. We also find that reducing the intrinsic driving noise level in the model, in a clear analog to reduced sensory input and activity in the rest of the brain, as during sleep or rest, results in sporadic brief spontaneous bursts of synchronous activity, which, after pattern learning, tend to acquire embedded structure and order. Earlier work (Jahnke

et al. 2015) has also shown similar learning and sharp wave-ripple-like spontaneous replay functionality, but relied on the presence of complex dendritic nonlinearities, and, perhaps more notably, lacked the obvious rest-analog which could introduce or remove the presence of this sharp wave-ripple-like phenomenon. We believe this model, and the particular set of plasticity mechanisms that allow it to self-organize, demonstrates a simple and parsimonious explanation for diverse vital neural phenomenon (in this case, specifically, rapid memory replay within sharp wave-ripples) , many of which have never been simultaneously expressed before in such a relatively simple model.



Raster plots showing spikes during and surrounding spontaneous synchronous events before and after repeated exposure to sequentially structured stimuli ("training") and cue-triggered recall.

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[T 28] **Why working memory is not a reservoir: the role of transient dynamics and attractors when processing unreliably timed inputs**

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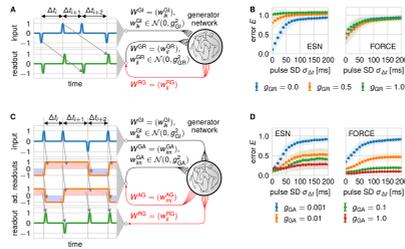
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Working memory (WM) refers to the ability of humans and animals to store and process the continuously incoming stream of stimuli and information on short time scales [1]. The neuronal dynamics implementing these two core functions of WM, to store and to process information, are still a matter of debate. It is unclear whether working memory relies on attractor dynamics [2] or on transient dynamics [3]. Experimental evidence and theoretical considerations provide support for both of these seemingly contradictory hypotheses.

We approach this debate by considering the fact that, when interacting with the environment, subjects cannot rely on precisely timed input stimuli. The consequence of unreliability of input stimuli on the operation of WM has been psychologically studied in the popular N-back task. In this task, introducing unpredictability of the occurrence timing of the stimuli does not significantly influence the subject's performance [4]. We investigate which kind of neuronal dynamics enables a network to perform the N-back task with a comparable level of robustness with respect to variances in the stimuli timing.

The most widely used network model of transient neuronal dynamics is the framework of reservoir networks [5, 6]. We test the performance of reservoir networks trained with different learning algorithms and with different feedback topologies on the N-back task. Interestingly, we find that introducing already small variations in the timing of the input stimuli reduces the performance of reservoir networks in the N-back task significantly. We show that the performance can be restored by explicitly training the network to represent past input stimuli via the activity states of feedback loops. As this, in turn, effectively introduces attractor states into the network, we conclude that only by exploiting the properties of both, attractor states as well as of transient dynamics, a neuronal network is able to achieve a performance comparable to the one found in WM experiments. Task-relevant information is stored in attractor states and processing of information is accomplished by transient dynamics. We predict that in the N-back task, an explicit recall stimulus should avoid a drop in performance resulting from introducing delays between stimulus perception and action execution. Thus, we provide an experimentally verifiable hypotheses about the underlying dynamics of WM ruling out purely transient reservoir networks as a plausible model [7].



A) Benchmark N-Back task with a purely transient network. Red synapses are adapted during learning. B) Performance of the reservoir. C) Same as in A but with additional, specially-trained readout neurons. D) Performance of the reservoir with additional readouts.

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[T 29] Presynaptic inhibition provides a rapid stabilization of recurrent excitation in the face of plasticity

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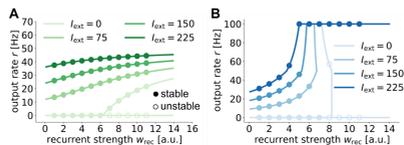
Synaptic plasticity in recurrent neural networks is believed to underlie learning and memory in the brain. One practical problem of this hypothesis is that recurrent excitation forms a positive feedback loop that can easily be destabilized by synaptic plasticity. Numerous homeostatic mechanisms have been suggested to stabilize plastic recurrent networks [1], but recent computational work indicates that all these mechanisms share a major caveat: An effective rate stabilization requires a homeostatic process that operates

on the order of seconds, while experimentally observed mechanisms such as synaptic scaling occur over much longer timescales [2].

Here, we suggest presynaptic inhibition as a compensatory process, which does not suffer from this discrepancy in timescales. Experimental studies have revealed that excess network activity can trigger an inhibition of transmitter release at excitatory synapses through the activation of presynaptic GABA(B) receptors, which effectively weakens synaptic strength [3]. This attenuation of recurrent interactions has been observed to be fully reversible and act on timescales of 100s of milliseconds, thus constituting a candidate mechanism for the rapid compensation of elevated recurrent excitation induced by synaptic changes.

To highlight the beneficial properties of presynaptic inhibition in excitatory recurrent circuits, we analyzed a simple rate-based recurrent network model. Presynaptic inhibition is mimicked by multiplicatively scaling down recurrent excitatory weights in response to excess population activity. Using analytical and numerical methods, we show that presynaptic inhibition ensures a gradual increase of firing rates with growing recurrent excitation, even for very strong recurrence (Fig. 1A). In contrast, classical subtractive postsynaptic inhibition is unable to control recurrent excitation once it has surpassed a critical value (Fig. 1B). Moreover, we find that presynaptic inhibition stabilizes firing rates in a recurrent population subject to plasticity while allowing synaptic homeostasis to operate on biologically plausible timescales.

In summary, the multiplicative character of presynaptic inhibition provides a powerful compensatory mechanism to rapidly reduce effective recurrent interactions. Remarkably, presynaptic inhibition conserves the underlying network connectivity and might therefore set the stage for stable learning without interfering with plasticity at the level of single synapses.



Steady- state firing rates as a function of recurrent strength for different input intensities. A. Presynaptic inhibition B. Postsynaptic inhibition

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[T 30] **Gap junction plasticity as a mechanism to regulate network-wide oscillations and to support neuronal communication through synchronisation.**

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Abstract Cortical oscillations are thought to be essential for many biological functions and cognitive processes. Several mechanisms have been proposed to promote oscillations. One prominent but understudied mechanism is gap junction coupling. Gap junctions are ubiquitous in cortex between GABAergic inhibitory neurons. Moreover, recent experiments indicate that the strength of gap junctions can be modified in an activity-dependent manner, similar to chemical synapses. We hypothesised that activity-dependent plasticity of gap junctions acts as a potential mechanism to regulate oscillations in the cortex. To test this, we developed a computational model of gap junction plasticity in a recurrent cortical network. Gap junction coupling divides the network activity in two regimes, either asynchronous irregular for low coupling or synchronous regular for strong coupling. We showed that indeed gap junction plasticity can regulate oscillations. Moreover, we showed that gap junction plasticity allows effective communication between neuronal assemblies, through neuronal synchronisation. Cortical networks oscillating at gamma frequencies (40 Hz) can therefore successfully transmit signals up to 10 Hz using electrical synapses.

Results Gap junction (GJ) coupling divides the network activity in the asynchronous irregular (AI) regime where sparse firing dominates, and the synchronous regular (SR) regime where bursting activity prevails (Figs. A and B). Haas et al. shows that bursting activity leads to GJ long-term depression (gLTD). We modelled their bursting protocol to infer the gLTD learning rate. We assumed that sparse firing would lead to GJ potentiation (Fig. C). GJ plasticity regulates oscillations and finds a balance between AI and SR regimes. The network reacts to a step current by oscillating, which can make readout neurons fire. If they are plastic, GJs are depressed due to the bursting activity associated with the oscillations. The network then leaves the SR and downstream neurons stop firing. Thus, the regulation of oscillations mediated by GJ plasticity allows for sparse but salient information transfer (Fig. D). Moreover, gap junction plasticity allows robust cross-network synchronisation (Figs. E-F). Finally, gap junction plasticity increases robustness of information transfer through frequency modulation (Figs. G-H).

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[T 31] Variably Sized Place Fields: Learning and Replay Model

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Hippocampal place cells have been studied extensively in rodents, both in experiments and models. Activity of place cells during free exploration has been found to be replayed later, in sharp-wave-ripple (SWR) events during rest. The observed variability in place field size, however, has not been considered in most existing models of place cell activity and sequence replay. These different sizes create an ambiguity in the order of cells. For example, if one cell has an earlier activity onset, but a later peak and end of activity compared to a second cell. On the other hand, the different sizes offer an additional degree of freedom to encode path information. We explore three methods of building a recurrent neural network (RNN) from place cell activity, using either the start, middle or end of place cell activity to define an order relation. The resulting RNNs are discrete-time dynamical systems that use the thresholded function proposed in Medina and Leibold (2013). We then study the properties of the resulting RNNs with a special emphasis on properties related to place field size. All three order relations result in sequence replay and have distinct dynamical properties. These differences include the number of time steps that each cell is active, as well as the distribution of field sizes among active cells. The connection matrix of each RNN and their differences are visualized as a vector field and diffusion tensor. For a crossing path, each order relation requires different values of feedback inhibition to replay the correct sequence.

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[T 32] Simple mathematical model of delay eyeblink conditioning in the cerebellum

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Timing and motor control are two classic cerebellar learning tasks. Starting from the seminal work of Marr[1] and Albus[2], a number of theories have been developed describing the underlying mechanisms[3,4]. In general, timing is regarded as the more fundamental task, since sequencing and timing of motor primitives is a prerequisite to dexterous motor control. We challenge this basic notion by showing that a simple cerebellar model can utilize gain adaptation, the core element of motor learning, to reproduce time intervals in a Pavlovian eyeblink conditioning paradigm.

We mathematically model delay eyeblink conditioning as a gain adaptation task in which the cerebellum drives an external integrator as a timer. The external integrator can be neuronal (e.g., a line attractor network) or mechanical (e.g., a muscle). We discuss the results and consequences of our model, many of which are consistent with biophysical lesioning, recording, stimulation, and reversible inactivation studies (see, e.g., [5,6]). We augment the discussion with previous results from computer simulations and robotic experiments.

Acknowledgements

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[T 33] Quantifying memory in the spike generation of single neurons in a neural population

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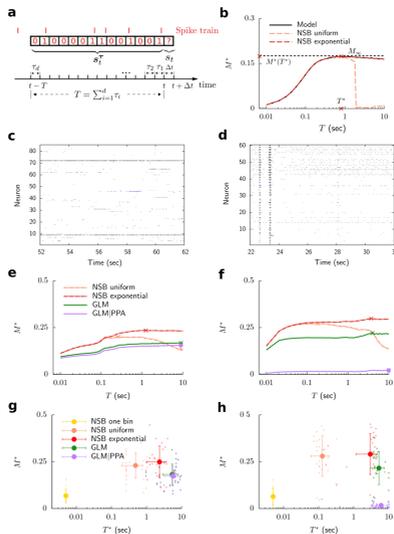
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For neural coding it is crucial to which extent a neuron's spike generation carries information about its own and the network's past activity. This can be quantified using information storage and transfer [1] that are powerful methods as they do not require knowledge about the exact biophysical mechanisms. However, they are difficult to estimate from data and crucially depend on the chosen representation or embedding of the spiking activity. In past studies, these embeddings were commonly chosen to simplify the estimation (e.g. only one past bin), thereby possibly ignoring relevant information about the spike generation. We overcome this by introducing a systematical approach to find the most informative embedding that still allows reliable estimation. We benchmarked our method for information storage on a realistic model neuron [2] with analytical solution, showing the strong effect of embedding on the inferred storage and the robustness of our method (b). The approach was then used to compare memory effects of spike generation in vivo (awake rat hippocampus [4]) (c) and in vitro (rat

cortex [5]) (f). Memory is quantified as the mutual information (MI) between the future state s_t (spike or no spike) and the past activity \mathbf{s}_t^T as a binary sequence of spike counts for uniform ($\tau_i = \tau, \forall i$) or exponential ($\tau_i = \tau \exp(\kappa i)$) embedding (a). To make results comparable we study the relative memory $M = MI/H \in [0, 1]$, with H being the Shannon information of the spiking process without history. We use a Bayesian estimator (NSB [3]) to estimate M from data and optimize the embedding parameters to maximize memory for fixed embedding depth T under the constraint of reliable estimation. By systematically increasing T we find the T^* for which the inferred memory is maximal (b), giving a lower bound for the minimal past time range containing all relevant information. We found that in vivo and in vitro past activity of at least 500 ms, or 100 ms, respectively, contributes information about the neuron's spiking (e,h), with one bin being insufficient. Furthermore, we fitted a GLM to the data and found interesting differences in the memory in vivo and in vitro when we also incorporated the recorded population past activity (PPA) in the model. In vitro, there is a substantial decrease of memory when PPA is given, meaning that the memory is highly redundant, while in vivo it is clearly non-redundant.



a) Representation of spiketrains as binary sequences. b) Estimated and true memory in the model for optimized exponential embedding in comparison to uniform embedding. c), e) show application to in vivo, d), f), to in vitro MEA recordings in rat. g) and h) show $M^*(T^*)$, averaged over all neurons.

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[T 34] Detecting hippocampal, prefrontal and parietal cell assemblies during online and offline cognition

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In 1949, Donald Hebb suggested that the repeated coactivation of a group of neurons leads to the reinforcement of their shared synaptic connectivity, forming what he called a cell assembly (1). The idea is that this altered connectivity matrix, once formed, would then induce assemblies to display coherent activity patterns either spontaneously or whenever the original stimulus conditions are reinstated. Leveraging this proposed excess in pattern occurrence rates, we recently developed a statistical method to detect assembly activations in non-stationary multiple single units recordings. The method performs a fast, non-stationarity-corrected parametric test over a large number of activity patterns and temporal scales, treating them as free parameters to be determined for each specific dataset (2).

Here we present preliminary results from analyses of CA1-prefrontal-parietal co-recordings from sleeping and behaving rats. During performance of a maze task, assembly activation across a range of timescales correlated with a range of salient features, including position, running trajectory and reward. As expected, we observe some reactivation of task-related assemblies during sleep (3-4). While such reactivation is found in about the same proportion in all the three regions, we found that the percentage of assemblies modulated by the hallmark oscillations of non-REM sleep – ripples, spindles and delta waves – is much higher in the hippocampus than in the neocortical regions. Moreover, while CA1 assemblies were typically activated during these oscillatory events, the majority of PFC and PC assemblies were inhibited.

Current analyses are quantifying to timescales of assembly activation, and the experience-dependent interrelationships between assemblies active on the maze and during subsequent sleep.

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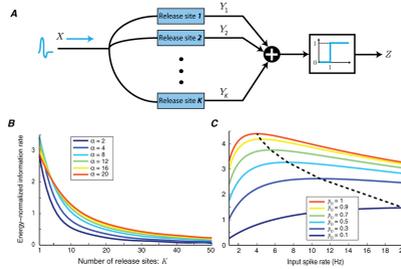
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[T 35] Energy-efficient information transmission in depressing synapses

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Synaptic short-term depression and facilitation regulate the information conveyed between neurons [1-2]. The reliability of a synapse depends on the number of synaptic release sites, which varies across different synapses from a single release site to hundreds of release sites. More release sites enhance the reliability of the synapse by increasing spike-evoked release probability, but at the cost of an increased probability of spontaneous release. To study the effect of the number of release sites on synaptic information efficacy, we model a synapse with K independent release sites. Each release site is treated as a binary asymmetric communication channel whose state is determined by the release history of the site [3]. We assume that one vesicle from any site releases enough neurotransmitter to activate all the receptors on the post-synaptic site (Fig. 1A). After each release, the release site is inactivated and recovers slowly back to the normal state. Energy efficiency is a critical aspect of information transmission. Without taking energetic cost into consideration, we had previously shown that the optimal number of release sites ranges from 6 to 16 for realistic parameter values of synaptic depression and input spike rates between 2 and 20 Hz [4]. Here we compute the energy-normalized information rate, by assuming that one unit of energy is consumed for each vesicle release. The energy-normalized information rate is highest when the synapse has only a single release site (Fig. 1B). This finding is consistent with the low number of release sites found in most central nervous system synapses. We also calculate the optimal input spike rate for synapses with one release site ($K = 1$) and show that the optimal range of input spike rates is between 4-8 Hz, provided that the spike-evoked release probability of the synapse (p_0) varies between 0.5 and 1 (Fig. 1C). The calculated range of spike rates is also compatible with the average spike rates in cortical white matter.



A) Information transmission through multiple release sites of a synapse. B) Energy-normalized information rate vs. the number of release sites, for different input spike rates. C) For $K=1$, energy-normalized information rate vs. input spike rate for various spike-evoked release probabilities.

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[T 36] Time-series Learning through Hidden Population Shaping by Somatic Nudging

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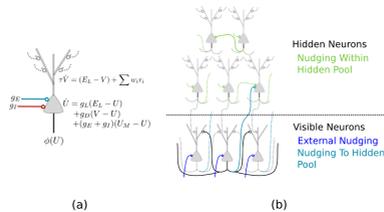
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Neuronal activation sequences are the basis for many animal behaviours. An active research question is how the brain learns these neural time-series. This question is directly related to the research topics of learning in recurrent neural networks and reservoir computing. In contrast with many biologically unrealistic algorithms like FORCE learning [1] or Back-propagation through time [2], we propose a biologically plausible way for reservoir shaping and time-series learning.

Our network consists of two-compartment neurons as introduced by Urbanczik and Senn [3] (see Figure 1a). In these neurons the membrane potential U is a combination of the dendritic voltage V and the somatic voltage $U_M = \frac{g_I E_I + g_E E_E}{g_E + g_I}$, which functions as a teacher, by weakly nudging the neuron's membrane potential towards the desired value U_M . During learning the dendritic synapses will adapt in order to minimise the error between the dendritic prediction V and the true membrane potential U : $\Delta w_{ij} = \eta(\phi(U_i) - \phi(V_i^*))\phi(U_j)$. We utilize this property to shape the hidden population. We start with a dendritically fully connected network. From that network, as shown in

Figure 1b, a subset of neurons is chosen as visible neurons. These neurons will receive an external teaching signal, representing the desired time-series, in the form of somatic nudging (blue connections). Teaching input in the hidden layer is provided by somatic inputs from other hidden neurons (green connections) and crucially by somatic inputs from the visible subpopulation (cyan connections). Note that there is no somatic teaching input from the hidden towards the visible population. During the learning the external teaching signal will be provided to the visible population. After learning the external teacher is removed and in the case of successful learning the visible neurons can reproduce the teacher.

This setup is capable of learning non-Markovian patterns as well as time-series with activity gaps that are longer than the neuronal decay. The memory extension of the visible population, necessary for these tasks, is due to the formation of delay lines and loops in the hidden population. Especially neuronal loops can keep information alive over long periods without any visible activation. Hence we conclude that our biological plausible approach is successful in shaping the hidden reservoir for time-series prediction.



(a) Two-compartment neuron, with dendritic and membrane voltage V and U , teacher U_M defined by excitatory and inhibitory conductances g_E and g_I and firing rate function $\phi()$. (b) Network showing visible and hidden pool with external (blue) and intra-network (cyan, green) nudging.

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[T 37] Exploring the role of interneurons in sensory representation during reward learning

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Adult brains are plastic, although specific behavioral contexts may be required to trigger plasticity as opposed to continuous remodelling in juvenile brains. Rewards are among the behavioral cues that trigger plasticity. When mice receive a reward after seeing one particular stimulus, responses of layer 2/3 pyramidal neurons in visual cortex change such that task-relevant stimuli, and especially the rewarded stimulus, are more distinctly represented after learning (Poort et al. 2015, Goltstein et al. 2013). The underlying changes in excitatory and inhibitory circuitry are however unclear. Recently, interneuron networks turned out to be crucial regulators of learning and synaptic plasticity (Letzkus et al. 2011, Fu et al. 2015). Moreover, interneurons might be the main target of top-down reward signals, as they receive cholinergic input (Froemke et al 2007, Letzkus et al 2011); cholinergic fibres from reward-processing regions such as the forebrain project to visual cortex (Chubykin et al. 2015). Therefore, we hypothesize that interneuron networks are involved in adjusting stimulus representations. Here, we explore different network motifs that could underlie the changes in stimulus representation. In particular, we use computational models of layer 2/3 mouse visual cortex consisting of excitatory pyramidal neurons, and different interneuron populations, corresponding to somatostatin (SST)-positive, parvalbumin (PV)-positive and vasoactive intestinal peptide (VIP)-expressing interneuron types.

Acknowledgements

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[T 39] **Compartmental model of calcium coupling for synaptic cross-talk**

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This study aims to address the fundamental principle of inter synaptic interactions in synaptic cross-talk through homosynaptic and heterosynaptic plasticity. We propose that calcium signal coupling between neuronal spines, dendrites and the smooth endoplasmic reticulum as well as back-propagated action potentials, provides an intriguing mechanism for inter synaptic cross-talk. We have built a compartmental model based on calcium dynamics, and present a detailed scheme of calcium interactions between stimulated and unstimulated spines through the calcium diffusion process in dendrites and calcium induced calcium release (CICR) in the smooth endoplasmic reticulum (SER). By extracting rate parameters from two-photon calcium imaging observations of calcium decay kinetics in spines, our results can recapitulate the calcium dynamics in spines and dendrites. In addition, our work predicted a 'Mexican hat' profile in response to high frequency stimulation and a clustered long term depression (LTD) by low frequency stimulation. This compartmental model for heterosynaptic plasticity provides a promising mechanism for the interpretation of cooperative and competitive interactions between synapses, and reveals the complexity of synaptic plasticity and its potential functions in neural circuits.

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Motor control, movement, navigation

[T 40] A Cortico – Basal Ganglia Model for Bimanual Reaching in Hemiparetic Stroke

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We propose a cortico-basal ganglia model capable of performing bimanual reaching tasks under normal and hemiparetic stroke conditions. The areas modeled in each hemisphere include the prefrontal cortex, motor cortex, proprioceptive cortex, motor neurons of the spinal cord and the basal ganglia. The proposed architecture has two semi-independent systems, coupled at the level of the motor cortex, controlling their respective arms. The architecture on each hemisphere consists of an outer cortical loop and an inner basal ganglia loop. The two systems control the reaching movements of two simple kinematic arms (Fig. 1A). Experimental studies on bimanual reaching in hemi-paretic stroke patients' shows alteration in velocity profiles of the paretic and non-paretic arm in different conditions (Rose and Winstein 2004). In the model, stroke-like conditions were simulated by lesioning a part of the motor cortex. The two motor cortices were then coupled (through a coupling factor, ϵ), to simulate bimanual reaching. Both arms had their respective targets and two types of reaching tasks were performed using this setup. First is the unimanual reaching task where both the arms were allowed to reach the target independently ($\epsilon = 0$) and second is the bimanual reaching task, where they had to reach the targets simultaneously. This was done using both inhibitory coupling ($\epsilon < 0$) and excitatory coupling ($\epsilon > 0$). It was found that both inhibitory and excitatory coupling influenced the arms, i.e. when the nature of coupling from the paretic arm to the non-paretic arm was excitatory and the coupling from non-paretic arm to the paretic arm was inhibitory, the peak resultant velocity (PRV) of the paretic arm in bimanual reaching was higher than that of its unimanual counterpart; the converse was observed in the non-paretic arm (Fig. 1B and 1C). This model behavior is in accordance with the experimental data (Rose and Winstein 2004). Thus the model suggests that the interhemispheric communication between the two motor cortices in bimanual reaching is both excitatory and inhibitory in nature. We anticipate future development of this model as an elaborate test bench for bimanual reaching tasks in order to develop improved rehabilitation strategies for stroke patients.

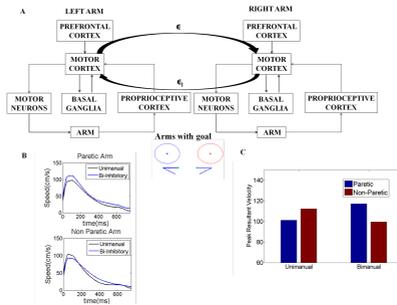


Fig 1: (A) The cortico-basal ganglia model architecture to simulate the bimanual task and the arms with their respective targets. The velocity profiles (B) and the peak resultant velocity (PRV) (C) of the parietic and the non-parietic arm in the unimanual and bimanual reaching tasks.

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[T 41] Distinct representations of planned reach trajectories in human premotor and posterior parietal cortex

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Goal-directed movements of the hand are often directed straight at the target, e.g. when swatting a fly; but when drawing or avoiding obstacles, hand trajectories can also become quite complex. Studies on movement planning have largely neglected the latter case and the question of whether the same neural machinery is planning straight, saccade-like vs. complex hand trajectories. Using time-resolved fMRI during delayed response tasks we examined planning activity in human superior parietal lobule (SPL) and dorsal premotor cortex (PMd). We show that the recruitment of both areas in trajectory planning differs significantly: PMd represented both straight and complex hand trajectories while SPL only those that led straight to the target. This implies that complex and computationally demanding reach planning is governed by a frontal pathway while a parietal route could warrant an alternative and faster way to put simple plans into action.

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[T 42] **Grid-cell activity – from running back and forth on a linear track: one-dimensional slices through two-dimensional hexagonal grid fields?**

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Grid cells in rodent medial entorhinal cortex are thought to play a critical role for the neural representation of space and, in particular, for spatial navigation. When the animal is freely moving in an open arena the firing fields of each grid cell form an imaginary hexagonal lattice spanning the environment [1]. For movements along linear tracks the cells seem to respond differently. They show multiple response fields that are not periodically arranged and differ strongly depending on the running direction. In addition, peak firing rates vary widely from field to field [2]. Further analysis showed that the firing fields from runs in one direction are compatible with slices through two-dimensional hexagonal firing fields [3]. This study did, however, not address the relation between left-wards and right-wards fields. Here, we show that a joint hexagonal firing pattern explains the linear-track data for both running directions if additional translational shifts are allowed for each direction. Importantly, a rotation or scaling of the grid is not required. The agreement is further improved if the firing rates of the underlying 2D grid field can vary from field to field, as suggested by recent studies [4]. We also analyzed firing fields from linear tracks that extended across different rooms and found that the underlying 2D pattern is the same apart from a translational shift patterns.

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[T 43] **Sensor-motor maps for hopping – influence of changes in muscle properties**

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In bipedal locomotion (e.g. walking or hopping) cortical and spinal networks (with CPGs and sensory feedback) work in concert to generate appropriate motor commands (for a given task and environment). It is suggested that the human physiology (e.g. muscle properties) facilitate the neuromuscular system to adapt to sudden changes of the environment more rapidly [1-3]. In this simulation study, the influence of different Hill-type muscle properties (e.g. force-length- and force-velocity-relationship) on hopping patterns is investigated.

Human hopping was simulated using a simplified biomechanical model [4] which additionally represents different sensory feedback pathways from proprioceptive signals (Golgi tendon organ: tendon force, muscle spindles: fiber length and velocity). These pathways

were blended to generate motor commands of one leg extensor muscle-tendon complex [5]. In this framework, force-length (F-l) and force-velocity-relationship (F-v) were varied in different types of approximation (constant, linear, non-linear) [2] to evaluate the individual contribution of the muscle properties on the predicted combinations of sensor-pathways enabling stable hopping. We call these pathway combinations sensor-motor maps. In our previous work, the topology of the sensor-motor map was found to be invariant to changes in the morphological design (e.g. tendon compliance, body mass, segment lengths) [5]. This indicates the stabilizing function of intrinsic muscle properties [3].

In this study, we aim at identifying the required level of the representation of muscle properties to enable functional and robust sensor-motor maps for stable hopping.

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[T 44] Grid Cells as Transition Encoders

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One of the primary cells that are believed to participate in spatial navigation in the rodent brain are grid cells, found in the Entorhinal Cortex (Hafting et al. 2005). However, the origin and purpose of the hexagonal arrangement of their response fields with respect to the spatial location of an animal, called grid fields, remains enigmatic. Several computational models have been proposed to shed light on the puzzle, for instance oscillatory interference (Burgess et al. 2007), self-organizing (Kropff & Treves 2008), or continuous attractor models (Fuhs & Touretzky 2006), all of which produce phenomenologically convincing results. In these models, the purpose of grid cells is commonly believed to be path integration (Burak & Fiete, 2009). On the other hand, theoretical investigations showed that grid cells optimally encode spatial locations (Mathis et al. 2012, Wei et al. 2015).

Here, we propose a novel purpose of grid cells which is related both to path integration as well as localization, namely transition encoding. It is shown that encoding of transitions of sequences in two dimensional Euclidean space is optimal for hexagonally arranged

encoders. Transition encoding was previously suggested to be part of the entorhinal-hippocampal loop (Cuperlier et al. 2004), however without considering optimality.

The results of the theoretical analysis were used to derive an error function and model for grid cells. The cells perform transition encoding in form of dendritic computation. The model is evaluated for varying sizes of receptive fields and using realistic trajectories. Except for numerical considerations, the model yields cells with high gridness scores.

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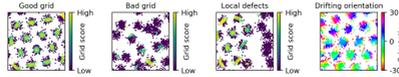
[T 45] A local grid score for individual spikes of grid cells

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The location-specific firing of cells in the entorhinal cortex is subject to extensive experimental and theoretical research. When classifying the tuning properties of entorhinal cells, researchers distinguish between grid cells, which fire on a hexagonal grid of locations, cells that fire periodically but without hexagonal symmetry and cells without periodic firing patterns [1, 2, 3]. This classification requires a measure for the symmetry of spatially modulated firing patterns — a grid score. The most established grid score is computed in multiple stages [4, 5]. First, spike locations are transformed into a rate map. Subsequently, an autocorrelogram of the rate map is cropped, rotated and correlated with its unrotated copy. The final grid score is obtained from the resulting correlation-vs-angle function at selected angles. This procedure results in a global grid score for the firing pattern. Here we suggest a new approach that computes a local grid score — and the local grid orientation — for each individual spike, directly from spike locations. Averaging over spikes, we obtain a global grid score and show that it is at least as reliable as existing grid scores in quantifying the global hexagonal symmetry of a firing pattern. The new score enables the plotting of spike locations, color coded with the local grid score or the local orientation of the grid and could thus simplify the visualization of experimental data. More specifically, it could be used to quantify and

highlight recent experimental findings on local properties of grid patterns, like boundary effects in asymmetric enclosures [6] and drifts in grid orientation along the arena [7]. The grid score is applicable to any n-fold symmetry. We provide a public Python package (using SciPy and NumPy) that efficiently determines the grid score directly from spike locations.



From left to right: Spikes of good grid cell, bad grid cell and grid cell with defects on the right half of the arena. Each spike is color coded with its individual grid score. Right: Grid with drifting orientation. Each spike is color coded with the local orientation of the grid.

Acknowledgements

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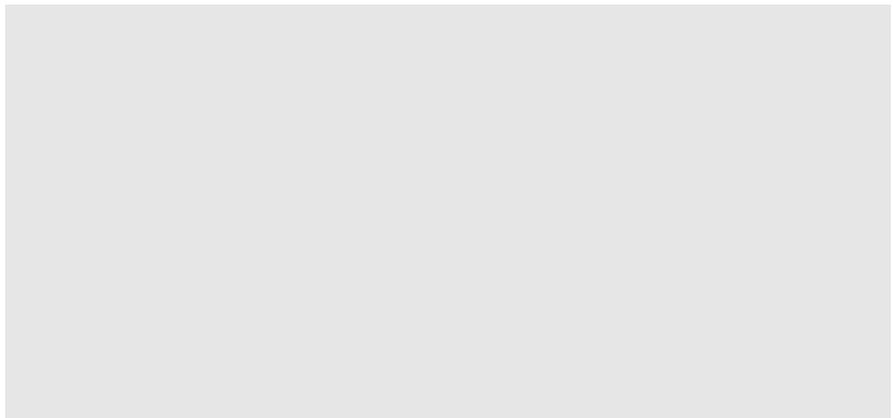
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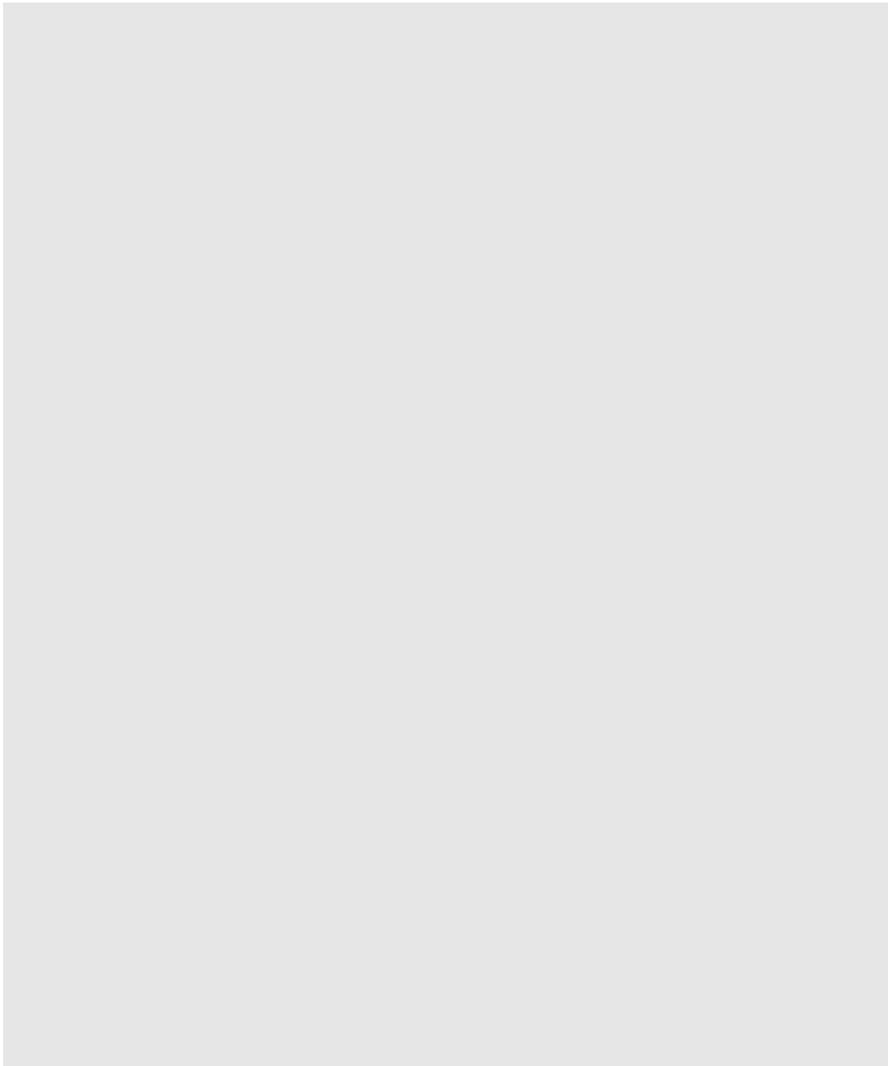
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[T 46] **Withdrawn**





Neurons, networks, dynamical systems

[T 47] Amplification of gamma- and theta-band inputs by distinct cortical interneuronal populations

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Despite the fact that cortical neurons receive hundreds or thousands of synaptic inputs per second, their outputs are sparse, on the range of only a few hertz. This low output rate suggests that information is encoded not by a single neuron's spikes but rather by the population's collective activity. By characterizing the neuronal transfer function, or dynamic gain function, one can understand the population's ability to encode rapidly varying inputs as well as identify their preference for certain input frequencies. A pronounced maximum at a certain frequency means that the neuron selectively amplifies input components at this frequency while others are suppressed. By means of patch-clamp recordings in acute brain slices of mice from different genetic lines in which interneurons could be targeted, we characterized the neuronal transfer function of distinct interneuron classes under different noise regimes. We show that, at the populational level, fast-spiking interneurons exhibit remarkable preference for stimulus in the gamma-frequency band, while adapting interneurons respond preferentially to theta- or gamma-frequencies, depending on the input's correlation time. These results extend known evidence of the contribution of these interneuronal classes to oscillations in these frequency bands as well as indicate that, at the population level, adapting interneurons may switch from one oscillation rhythm to another depending on the input statistics.

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[T 48] How rapid spike initiation reduces network chaos and localizes Lyapunov vectors.

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Spike initiation is a bottleneck for neural information transmission. Recent studies showed that the bandwidth of information encoding is limited by spike onset rapidness. Experiments revealed that neocortical neurons have a surprisingly broad encoding bandwidth. How this impacts the collective network dynamics is not well understood. Here we show that increasing the spike onset rapidness leads to decreasing attractor dimension, chaos and dynamical entropy production, which vanishes at a critical value. We numerically calculated all Lyapunov exponents and derived exact upper and lower bounds for attractor dimension and dynamical entropy production rate of random spiking networks. Analysis of large networks with more realistic structure indicate the generality of these findings. This demonstrates that spike initiation drastically shapes the entropy production rate by which information about the initial state is erased by the chaotic

recurrent network dynamics. The effect of spike onset on chaotic entropy production surpasses the effect on the bandwidth of information encoding by orders of magnitude.

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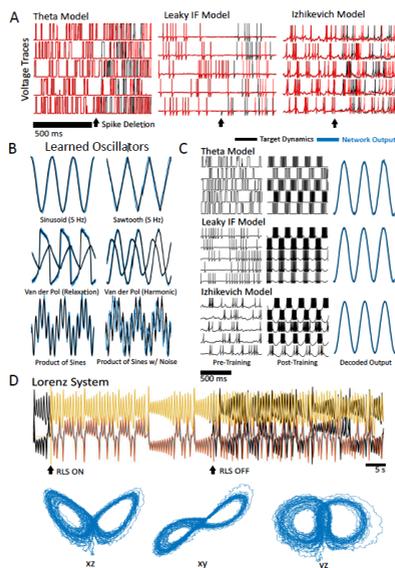
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[T 49] Supervised Learning in Spiking Neural Networks with FORCE Training

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Populations of neurons display an extraordinary diversity in the types of problems they solve and behaviors they display. Techniques have recently emerged that allow us to create networks of model neurons that solve tasks of similar complexity. Examples include the FORCE method, a novel technique that harnesses chaos to perform computations. We demonstrate the direct applicability of FORCE training to spiking neurons by training networks to mimic various dynamical systems in addition to more elaborate tasks such as input classification, storing sequences, reproducing the singing behavior of songbirds, and spontaneous replay of a scene from a movie. The networks can be analyzed post-training to yield illuminating information not easily obtainable in rate networks such as spike-timing statistics and peri-stimulus time histograms. With suitable biological constraints, future FORCE trained spiking networks can serve as models for neural circuits such as the HVC-RA circuit responsible for songbird replay, or the hippocampal circuit involved in the encoding and replay of episodic memories.



FORCE training with spiking neurons

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[T 50] Robustness of neural circuits with disparate components to intrinsic and synaptic perturbations

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Functionally equivalent neuronal circuits can generate similar activity patterns despite disparate intrinsic and synaptic properties [1]. Given these intrinsic and synaptic differences, we asked how the circuits respond to neuromodulators or perturbations caused by environmental changes: do all circuits respond in the same way because of the same output, or do they respond differently as reflected in their different components? One example is given by the stomatogastric nervous system of crustaceans: The pyloric rhythm is essential for the feeding behavior of these animals and needs to maintain its functionality over a wide temperature range. The operating ranges differ significantly across different animals of the same species, such that some circuits seem to be more robust than others [2]. We aimed to uncover the interplay between circuit properties (intrinsic and synaptic) and their contribution to circuit stability, by classifying changes in circuit output due to various kinds of perturbations. High dimensional Hodgkin-Huxley conductance-based models were used to examine the output of half-center circuits composed of two such non-identical neurons coupled with mutual inhibition. We propose a new measure of stability to classify the robustness of different circuits to changes in the maximum conductance value of specific channel types in the individual neurons. These changes are applied either separately for each channel type, or in combination, to examine whether the effects of different perturbations add up. Generally, we find that circuits which are robust to changes in one conductance, remain robust to changes in other conductances, despite their different components. We discuss robustness of circuit output to these changes regarding the individual intrinsic and synaptic properties, which has implications for neuromodulation, plasticity and environmental changes that can dynamically alter these characteristics. Furthermore, we consider stability by looking at extrinsic perturbations, including noise with different correlation structure and periodic drive with different frequencies.

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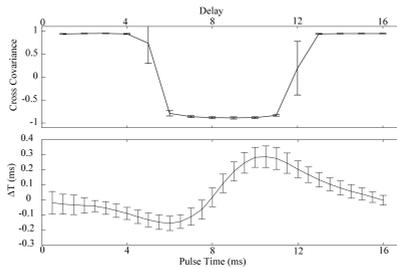
[T 51] Manipulating phase relation between neuronal population by time delay

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Cortical neurons in awake animals show irregular activity due to receiving excitation and inhibition from other connected neurons in a way that the mean of input of single neuron is below threshold and neurons almost fire stochastically [1, 2]. Regardless of this irregular activity, when an area of cortex involved in signal processing the synchronous state will emerge in different frequency ranges [3]. It is hypothesized that the coherence among the oscillations of brain regions affects neuronal communications and the changes in phase relation between the rhythms of these regions will change effective communication routes [4]. We have studied that how the phase lag of two coupled balanced networks (BN) is affected by the time delay in the transmission of signals between the two networks. In particular, we questioned if the theory of weakly coupled limit cycle oscillators can be applied in the case of two coupled balanced networks [5-8]. We have generated the balanced neural network using the conductance based model neurons. The networks are set to in Synchronous-Irregular state at which the network has a degree of coherence in a gamma range while the dynamics of single neurons is irregular. We then numerically calculated phase resetting curve (PRC) of the networks by applying a pulse to 50% of the neurons in the networks and recording the phase shift due to the applied pulse. The theory predicts that with the delay in the positive and the negative range of the PRC slope, anti-phase locking and in-phase states are respectively stable. The main idea is that by knowing the PRC of network it is possible to predict the behavior of interacting networks.



Coherency change of coupled balanced network. Changing the delay between two balanced neuronal network, can change their synchronization from out of phase to in-phase synchronization.

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[T 52] Achieving a qualitative reproduction of: 'Polychronization: Computation with spikes'

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Any modeler who has attempted to reproduce a spiking neural network model from its description in a paper has discovered what a painful and time-consuming endeavor this is. To study the potential pitfalls in reproducing such a model, we reproduce a seminal study by Izhikevic on Polychronization for which, unusually, source code was provided at time of publication. For all simulations NEST is used and the analysis is written in Python 2.6 and snakemake is used as workflow manager. In an initial attempt, we create a spiking model based on an analysis of the text description in the original paper and the source code, that nonetheless yields results that bear little resemblance to the described network dynamics. To uncover the causes for the network dynamics disparity, we import all random elements of the model (connectivity, initialisation of membrane potentials, stimulus) from the original source code into the NEST implementation, and deconstruct the original code to develop modular tests. This process allows us to make the NEST implementation exact down to numeric precision. We then achieve a qualitatively similar implementation, which is what is usually understood as reproducing a study, by rewriting the neuron model and STDP algorithm to use standard expressions of parameters and standard numerics for the equations as well as generating the sources of randomness within NEST. We conclude that the original model is sensitive to the specific random realisation of the network, and derive a number of recommendations that can help increase the reproducibility of future spiking neuronal network models.

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[T 53] Ion channel cooperativity enables cellular short-term memory via graded persistent firing

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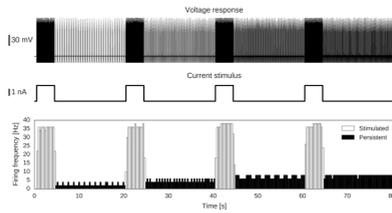
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Ion channels shape the electrical signalling of neurons, like the generation of spikes. A widely accepted assumption is that individual channels gate independently: their opening and closing is determined by other variables, like membrane voltage or calcium concentration, but channels do not directly influence each other's state transitions. Experimental observations, however, show that some channels gate cooperatively. For example, CaV1.3 channels in hippocampal neurons have recently been shown to assemble in small clusters and, when opening, to enhance the open probability of neighbouring channels [1] - a hallmark of channel cooperativity. Despite multifaceted implications of such inter-channel interactions for neural computation, the functional consequences of ion channel cooperativity have received relatively little attention, see for example [2,3].

In this study, we focus on small clusters of voltage-gated, cooperative ion channels. Based on conductance-based neuron models, we demonstrate that such clusters can impose a multistability of the firing response, thus enabling a neuron to retain a cumulative short-term memory of recent inputs. Specifically, we show how a small fraction of cooperative, clustered calcium channels (that are not per se required for the generation of action potentials) can mediate several levels of persistent firing that encode the number of recently present transient inputs, see Figure 1. Similar graded persistent activity has been observed in in-vitro experiments of several brain regions [4,5]. Here, we used in-vitro dynamic clamp experiments [6] to "equip" mouse perirhinal cortex neurons with clusters of cooperative channels. In the "presence" of these cooperative channels, cells switched from a normal firing response to graded persistent activity. This effect was very stable and recorded neurons did not have to fulfill specific requirements, indicating that the hypothesized cooperativity mechanism is very robust and generalizable across cell types.

In summary, our combined mathematical-experimental analysis leads us to conclude that clusters of cooperative ion channels constitute an interesting and potentially overlooked mechanism that widens the computational repertoire of single neurons. In particular, ion channel cooperativity may implement a cell-intrinsic mechanism for neuronal persistent activity - a representation of memory that usually has been thought to require recurrent network connectivity.



A single-neuron model with cooperative clusters of Ca²⁺ channels exhibits a similar type of graded persistent activity as cells in the entorhinal cortex (compare Egorov et al [4]). The rate of activity persisting beyond the actual presence of an input increases with each stimulation pulse.

Acknowledgements

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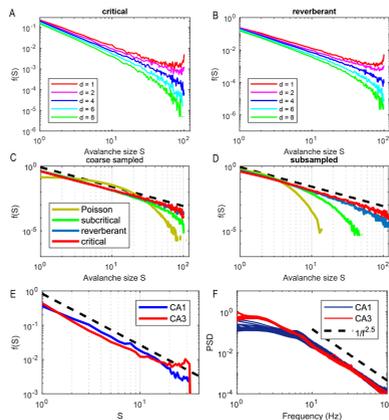
[T 54] Effects of sampling techniques on the assessment of neural dynamics

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It is hypothesized that the brain can operate in different dynamical states. One of these states is thought to be directly at a critical phase transition, maximizing properties such as information processing capacity and dynamic range [1,2,3]. In many systems, criticality is associated with avalanches of activity, with the avalanche size s following a probability distribution $f(s) \sim s^{-\alpha}$. Such a $f(s)$ has been observed in experiments in vitro and in vitro based on coarsely sampled neural activity (e.g. LFP, EEG) [4,5]. In contrast, results based on spiking activity, which are inevitably subsampled, suggests that the brain operates not at criticality but on a reverberant regime on the subcritical side [6,7]. To understand the origin of this contradiction, we model how coarse sampling

affects the observed $f(s)$ and other measures. We find a number of effects that can mask the true nature of the underlying system. For instance, a low inter-electrode distance (IED) d can make a critical system appear as supercritical, showing an initial power-law with an excess near system size (Fig.1A). A large IED, however, can make it appear as subcritical, described by a power-law $f(s)$ with an exponential cutoff. The same is true for a system in the reverberant state (Fig. 1B). Moreover, even with identical conditions the analysis of coarse sampled data is unable to distinguish critical and reverberant states (Fig. 1C). A better distinction can be achieved based on subsampled data, i.e. spikes (Fig. 1D). We conclude that more observables besides $f(s)$ are needed in order to assess criticality. For example, one can measure the autocorrelation time of spiking activity [7] or the power-spectral density (PSD) of the coarse signal [8], which are independent of IED. We analyze a dataset of rat hippocampus LFP recorded simultaneously from CA1 and CA3 during sleep. Assuming that the dynamics is bounded by criticality (because supercriticality results in runaway activity), Fig. 1E suggests that CA3 is closer to criticality than CA1 by directly comparing them under identical conditions. This is confirmed by a PSD analysis of each electrode channel, where we find the PSD of the CA3 channels decay closer to a pure $1/f^\beta$ decay with frequency f , which means a smaller distance to criticality. In conclusion, coarse sampling can bias the inference of the dynamical state, and to overcome ambiguities combinations of various measures is necessary.



A,B. Avalanche size probability distribution $f(s)$ for critical and reverberant systems with IED d . C,D. Comparison of $f(s)$ for coarse sampled (C) and subsampled (D) data with $d = 4$. E. $f(s)$ from rat LFP data for CA1 and CA3. F. PSD for all electrode channels from (E).

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[T 55] Sodium channel activation kinetics determines cut-off frequency of the dynamic gain

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Action potentials are initiated by activation of sodium channels. How precisely action potentials are locked onto temporal fluctuations in the neuron's input, is determined by the dynamics of action potential initiation. The precision of action potential timing is often expressed in the frequency domain as "dynamic gain". It expresses the neurons susceptibility as Hertz per Ampere, i.e. as a modulation of the firing rate per modulation of the input current. Importantly, the limited timing precision of action potentials is reflected in a steep cut-off of the dynamic gain in the high frequency limit. Although the limited bandwidth of the dynamic gain and the related limitation of action potential timing precision are a fundamental limit for the information processing in the cortex, there is currently no understanding of the biophysical factors that limit the bandwidth. Earlier, we presented experimental data indicating that the amount of axonal sodium channels plays an important role in the bandwidth of the dynamic gain, because reduction of the sodium channel density or sodium current density decreased the cut-off frequency. However, it is not known, whether this density is also the factor limiting the bandwidth of the unperturbed neuron. Here we combine precision measurements of sodium channel kinetics, measurements of dynamic gain curves and simulations of multi-compartment neurons. Our results indicate that the kinetics of sodium channel activation is ultimately limiting the bandwidth of the dynamic gain. The high-frequency behaviour of the gain curve is therefore set by a molecular parameter.

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[T 56] Enhancing synchronous spike propagation between layers of networks by feedback matching network resonance properties

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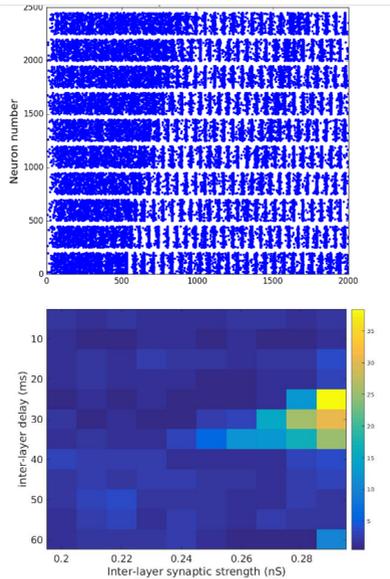
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A reliable propagation of spiking activity across weakly and sparsely connected neuronal networks is crucial for the brain function. Usually feed-forward networks (FFN), with or without recurrent connectivity within each layer, are used as a model to understand the propagation of spiking activity [1]. Such models have revealed conditions under which synchronous spike volleys (pulse-packets: PP) can be propagated [2]. Two similar mechanisms have been proposed to allow for the propagation of weak and asynchronous PPs. The 'communication through coherence' (CTC) mechanism requires that the sender and receiver networks oscillate at the same frequency and phase [3], while 'communication through resonance' (CTR) requires that the sender and receiver networks exhibit resonance frequency at the same frequency [4]. Recently, Moldakarimov et al. [5] showed that feedback connections between all layers of a FFN can allow for the propagation of weak and asynchronous PPs provided the feedback connection delay matches with the temporal precision of the PP. This mechanism while precludes the need for network resonance and coherent oscillations, increases the connectivity and thereby wiring cost.

Here we show that it is sufficient to have excitatory feedback connections between only one pair of layers in an otherwise feed-forward network to reliably transmit weak and asynchronous PPs. We found that the stable transmission of PPs depends on the feedback delay. Two ranges of feedback delays support the stable propagation of the PPs. When the feedback delay is smaller than the temporal precision of the PP, feedback excitation re-ignites the PP before it diminishes to boost the propagation [5]. (2) When the feedback delay is matched with the period of network resonance frequency, the pair of layers connected by feedback form a 'resonance pair' and locally amplify the weak PP [4] to enable a stable propagation. Thus, we demonstrate that a small modification in the FFN network (i.e. few feedback excitatory connections between a pair of layers) can enable propagation of weak signals through a weakly connected FFN without any fine tuning to obtain coherent oscillations or identical resonance frequency in each layer of the FFN.



A pulse packet can propagate successfully if a feedback loop between first and second layer exists, and also inter-layer delay matches the frequency period of the network . Signal-to-noise ratio for 10th layer of the network shows propagation happens only for a suitable amount of inter-layer delay.

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[T 57] Effects of monocular-deprivation induced plasticity on the dynamics of recurrent networks

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Monocular deprivation (MD) as an experimental paradigm has long been used to study mechanisms guiding the development and plasticity of visual cortical circuits. While most studies examine plasticity in the binocular region of the primary visual cortex (V1b), where competition between inputs from the two eyes takes place, the effects of MD in the monocular cortex (V1m) are less well studied. However, experimental results suggest that different plasticity mechanisms operate in these regions during MD [1]. We aim at understanding the dynamical aspects of network plasticity in V1m following MD using network models of cortical circuits. We incorporate recent experimental findings on the firing rate dynamics from long term in vivo recordings in rodents [2], as well as known changes in thalamocortical and intracortical synapses following MD [1, 3] to determine how they affect cortical network dynamics. Underlying our studies is a sparse random network of inhibitory and excitatory units [4]. We use dynamical systems theory and simulations of large-scale spiking networks to study the effects of plasticity induced by MD at thalamocortical and intracortical synapses. We find that the effects of cortical plastic reorganisation change qualitatively depending on the operating regime of the recurrent network, both in terms of the average firing rate and the auto- and cross-correlations of the neurons' output spike trains. The regime in the basic model is controlled by the recurrent coupling scale and the structure of inputs driving the network. Incorporating strong feedforward inhibition and decoupling the background input (from other cortical areas) from the thalamic drive strongly modulates the nature of these dynamical changes induced by cortical plasticity. Extending this, we also incorporate a second type of inhibitory interneurons which are driven by background input, delivering a type of feedback inhibition. These cells selectively modulate the responses of excitation and feedforward inhibition and we study how these two types of inhibition interact to shape network dynamics, as well as other computational properties of cortical circuits in the mature visual cortex. Our aim is to understand how structure in cortical networks that suits the computational demands emerges during development, and how it is shaped by experience.

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[T 58] Limiting the effect of temperature on firing rates in small neural networks

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Temperature fluctuations challenge the physiology of living organisms [1], as they affect almost any physico-chemical process, with consequences for metabolism as well as signalling [2,3]. Because of a temperature change's immediate effect on ion channels and cellular electrophysiology, the functionality of neural processing is put at risk. Specifically, temperature effects on action potential shape and frequency as well as synaptic transmission [4] limit the temperature range across which neural networks can be expected to work reliably. These restrictions apply not only to (cold blooded) ectotherms but also to (warm blooded) endotherms, whose activity-based fluctuations in brain temperature are known to exceed 2°C [5] – a value sufficiently large to impair nervous system function, like during febrile seizures or hot-water epilepsy [6,7]. Therefore, a temperature robust design of the nervous system function should have been favoured by evolution in both ectotherms and endotherms.

Here, we focus on a potential role for specific network motifs in establishing temperature robustness and test the hypothesis that circuits of parallel excitation and inhibition, which are often encountered in vertebrate and invertebrate systems [8], help to constrain temperature effects, because of the opposing (and hence countermanding) nature of the involved processes [9]. To this end, we use computation models of small networks and explore the effect of temperature on firing frequency in motifs with and without parallel excitation and inhibition. Implemented effects include temperature-dependent ion channel dynamics in conductance-based neuron models (for example, the Connor-Stevens and Traub-Miles model) as well as a phenomenological temperature-dependence of synaptic transmission.

Our results predict an interesting trend: robustness of the output firing rate is supported by parallel excitation and inhibition if the temperature dependence of inhibitory transmission exceeds that of the excitatory one. This finding can be reproduced with different synapse and conductance based neuron models and agrees with experimental observations reported in the literature [10]. Our study demonstrates the advantages of

specific neural network designs for achieving robustness to a global perturbation – a principle that may well extend beyond the effect of temperature as it is discussed here.

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[T 59] Detailed spiking network model of the human mirror neuron system

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The human mirror neuron system is considered the neural basis of our interpersonal understanding [1]. The neurons found in the motor cortex of the primate fire when the monkey is making a movement and also when it is observing a similar movement [2]. For humans, the assumption was derived that we recognize the emotions and also the intentions of other persons by simulating their motor state in our own motor system [3]. One way to learn more about the human mirror neuron system without directly measuring the activity of individual neurons consists in the theoretical modeling of the involved cell assemblies. The mathematical description of the activity of neuron networks makes it possible to calculate the indicators of the non-invasive measurement methods and to compare them with the actually measured values. Using statistical optimization methods, the free parameters of the model can be fitted to the experimental data. Thus, statements about the physiology of the cell assemblies are possible since the parameters of the model are directly related to biophysical properties, e.g. cellular and synaptic conductivities and resting potentials. Here, we use a highly detailed network model of the prefrontal cortex which all neurons and synapses parameters are determined by anatomical and in vitro electrophysiological data, and which has previously been shown

to statistically reproduce a wide range of measures from in vivo prefrontal data [4]. This spiking network model has been adapted to the motor cortex where the mirror neurons are located. Then it was adjusted to the fMRI data, in the first step the effective connectivity between the activated regions has been identified by Dynamic Causal Modeling (DCM) with comparing 424 models and then some modifications were done on DCM approach to using the (nonlinear) Wilson-Cowan-type model instead of the standard DCM schemes [5]. The global connectivity was inferred from Wilson-Cowan model and the connections between regions were made according to the best model which was found from DCM. The input-output functions of the neurons in the firing-rate model are matched with the predicted data from DCM analysis by comparing the resulting outputs, thus realizing the transfer from the macro- to the micro-level. This model will be used to predict the task performance and also to make predictions about a completely different set of data in order to make statements about the physiology properties of human mirror neuron system.

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[T 60] Neuronal Connectivity Options along the Edge of Bounded Neural Networks – Analysis of Network Structure and Dynamics

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Neurons which cannot make enough afferent or efferent connections will not survive in neural networks [1, 2]. In biological neural networks in-vivo this is found under pathological situations such as stroke areas or epileptic foci. In-vitro, neurons located at the boundary of cultured networks encounter a special situation where putative partners are available in one direction only. Therefore, axons of neurons located near the edge respond in different ways to increase their connectivity within the boundary. In biologically realistic scenarios, axons reaching the margin may: 1-stop growing and make local dense tree near the established axon; 2- bifurcate and expand the local area by smaller branches; 3- follow the network's edge and make new contacts in more distant locations (Fig. 1). We compared these three edge schemes along with

the unrealistic scenario that the axons can extend out of the network, in a bounded balanced neural network with anisotropic axons [3, 4]. Using this model, we simulated 50,000 neurons in a circular network (radius 3 mm, simulated in NEST [5]), where neuronal somata self-organize into clusters and anisotropic axons of excitatory neurons bundle to create fascicules. Changing the connectivity of the neurons on the edge has pronounced effects on structural features of the network such as degree distributions, local excitation/inhibition balance and motif distributions especially near the boundary. The edge options, depending on the extent of their impacts on the boundary, can alter the distribution of recurrent and cycling motifs near the boundary, which will impact the activity dynamics properties such as firing rates and bursting behavior of the networks. The heterogeneity produced by clustering and bundling forms higher variability in network structure and as a result richer patterns of network dynamics with shorter and more intense bursts emerge.

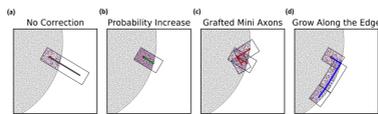


Fig. 1 – (a) Initial random axon direction. (b) “Probability Increase” method mimics local dense tree near the axon. (c) “Grafted Mini Axons” models bifurcation of mini-branches in the local area. (d) “Grow Along the Edge” represents the growth of the axon near the edge of the network.

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[T 61] Structural Properties of Burst Origination and Termination Regions in Simulated Neural Networks

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Synchronous neural activity is a typical activity in biological neural networks in-vivo and in-vitro. While it is known that the bursts of neural activities originate and terminate in distinct regions [1-3], it is, however, unclear what properties define the time and region of onset of such activity. To investigate these properties, we have developed a computational neural network model that mimics essential properties of cortical networks in vitro, in which such bursting is ubiquitous, and it includes anisotropic connectivity, clustering of somata, and fasciculation of axons. These networks showed activity dynamics that in many respects were similar to cultured neural networks. We analyzed the spatial distribution and mesoscale structure of connectivity in relation to the participation of local subnetworks in different stages of extra- and intra-burst activity. Our findings show that bursts usually start from areas with balanced excitation and inhibition or excess inhibition, while they end in areas receiving excess excitation. The spatial distribution of initiation areas is non-random and associated with recurrent connectivity at network boundaries. These results can help to understand how stimulation of distinct brain regions intercept synchronous events in some brain disorders, like epilepsy and Parkinson's Disease [4, 5].

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[T 62] Phase precession via synaptic facilitation in the hippocampal formation

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During the traversal of a firing field, both hippocampal place cells and entorhinal grid cells elicit action potentials at increasingly earlier phases of the theta oscillation in the local field potential – an effect called phase precession (O’Keefe and Recce, 1993). There is an ongoing debate about the brain structures and mechanisms involved in the generation of phase precession and its role in episodic memory (Jaramillo and Kempster, 2017). Thurley et al. (2008) proposed a model for phase precession in CA3 pyramidal cells based on short-term synaptic facilitation in response to strong, theta phase-locked input via a single mossy fiber synapse from the dentate gyrus. However, phase precession can already be detected further upstream in the medial entorhinal cortex (MEC) and might just be passed on from there to downstream regions like CA1 or CA3 (Jaramillo et al., 2014). Here we investigate an extension of the facilitation model for multiple inputs. In this case, the generation of both spatial tuning and monotonous phase precession in the target cell is not trivial. By exploring the parameter constraints in our model, we arrive at experimentally testable predictions about electrophysiological and anatomical features of a potential region of origin for phase precession.

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[T 63] Reconstructing neural dynamics from experimental data using radial basis function recurrent neural networks

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Neural recordings often constitute complex, noisy and high-dimensional time series. To assess underlying network dynamics, analysis methods should target the recorded population as a whole. A popular class of methods for simultaneously reducing dimensionality while reconstructing smooth trajectories and capturing different noise sources is the statistical framework of State Space Models (SSMs) [1]. The idea behind SSMs is that there is an underlying low-dimensional latent dynamical system generating the observations, with latent dynamics and observations having separate noise terms. While

linear SSMs are widely used to recover hidden neural trajectories [2], they are only able to reproduce the linear aspects of the underlying neural dynamics and cannot capture the complete system [3].

We examine a nonlinear SSM that includes radial basis function (RBF) basis expansions for the latent state dynamics, originally developed in [4]. With such an RBF expansion, in theory arbitrary dynamical systems can be approximated [5]. To estimate model parameters, an Expectation Maximization (EM) algorithm combined with an Extended Kalman Filter-Smoother is used. A major advantage of Gaussian RBFs is that all integrals for the expectation values required in the maximization step can be solved analytically, making this method computationally efficient.

To examine this approach in experimentally realistic scenarios, its effectiveness in cases where observations result from only an incomplete subset of latent variables, or where some of the latent states are translated into highly noisy observations, is tested. For this purpose, benchmark dynamical systems are simulated in different regimes, and states are projected into higher-dimensional observation spaces in which some of the latent states may be missing or highly obscured. Inference of dynamics through SSMs is compared to a direct time series modeling approach without latent states as developed in [6]. While both methods work well in the (empirically unlikely) case of complete latent state information, only the SSM is able to recover dynamics from incomplete observations.

The SSM-RBF model is also applied to an fMRI dataset obtained during a working memory task. With this approach, the neural dynamics can be visualized as trajectories in state space, showing that different task phases can be separated well in this low-dimensional space, or as flow fields, exposing attractor dynamics of the system.

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[T 64] Transition to chaos and short-term memory in driven random neural networks

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Recurrent networks of randomly coupled rate neurons display a transition to chaos at a critical coupling strength [1]. Their rich internal dynamics emerging near the transition has been associated with optimal information processing capabilities [2]. In particular, the dynamics becomes arbitrary slow at the onset of chaos similar to 'critical slowing down'. However, the interplay between time-dependent input signals, network dynamics, and the resulting consequences for information processing are poorly understood. Here, we investigate the effect of time-varying inputs on the transition to chaos. Using dynamic mean-field theory we determine the largest Lyapunov exponent, which quantifies the rate of exponential divergence or convergence of close-by trajectories. We analytically obtain the phase diagram for the transition when varying coupling strength or input amplitude (Figure 1, a). The transition is shifted to significantly larger coupling strengths than predicted by linear stability analysis of the local Jacobian matrix. This difference corresponds to the emergence of a novel dynamical regime, which combines locally expansive dynamics with asymptotic stability. To study information processing capabilities we evaluate the capacity to reconstruct a past input signal based on a linear readout of the present state, the so-called memory curve [3]. We find that for a given input amplitude the memory capacity peaks within the novel dynamical regime (Figure 1, b). This result indicates that locally expansive while asymptotically stable dynamics is beneficial to store information about the input in the network dynamics.

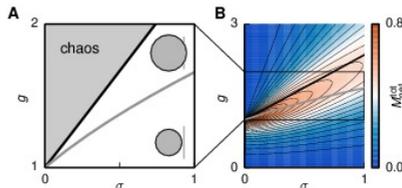


Figure 1: (a) Phase diagram for signal amplitude σ and coupling strength g . Black curve: Phase transition to chaotic regime. Gray curve: condition for loss of local stability. (b) Network memory capacity encoded in color. Global and local transition curves (black and gray) as in (a).

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[T 65] A theory of mesoscopic neural activity

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Having models of large-scale brain activity that are linked to the properties of single neurons and connectivity is vital for a mechanistic interpretation of mesoscopic and macroscopic neural data. Phenomenological models such as Wilson-Cowan equations, neural mass or field models are widely used but lack a clear link to the underlying microscopic parameters and often fail to correctly reproduce fast macroscopic dynamics. Using a refractory density approach [1,2], we systematically derive mesoscopic activity equations for several interacting populations starting from a microscopic network of generalized integrate-and-fire (GIF) neurons or generalized linear models (GLM) [3]. Each population consists of 50 – 2000 neurons of the same type but different populations account for different neuron types. Our mesoscopic theory captures important properties of population activity such as finite-size fluctuations due to the limited number of neurons per population and pronounced spike-history effects caused by refractoriness and adaptation on the cellular level. The mesoscopic model accurately reproduces the dynamics of the original microscopic spiking neural network model including stochastic transitions between multistable states and synchronization in balanced networks of excitatory and inhibitory neurons. We also demonstrate that the mesoscopic model correctly predicts non-stationary neural activity in a multi-laminar model of a cortical microcircuit consisting of eight neuron types under thalamic inputs [4].

We also show how our theory can be extended to other biological features such as synaptic background noise and synaptic short-term plasticity. In conclusion, our theory offers a general quantitative framework for modeling cortical information processing on a mesoscopic level that can be constrained by microscopic parameters.

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[T 66] Behavior-dependent functional connectivity of beta and low-frequency hub units in the macaque fronto-parietal grasp network

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Dynamic communication within cortical single-unit network is only coarsely known. To better understand how selective communication is organized within and between brain areas relevant for motor control, we examined the fronto-parietal grasp network that is involved in a broad variety of perceptual and cognitive processes related to hand motor control including vision, decision making, and movement generation. In order to analyze the dynamics in the neuronal network communication, we implanted 64 electrodes in each the ventral premotor cortex (area F5) and the anterior intraparietal area (AIP) in two monkeys. Spiking activity of single units and local field potential (LFP) activity were recorded simultaneously from all electrodes while monkeys performed a delayed grasping task, in which monkeys were either instructed to grasp a target with one of two possible grip types, or could freely choose one of the grips. Dynamic network interactions between single units and LFPs were assessed using pairwise phase consistency (PPC) as a measure of functional connectivity (FC). PPC is largely independent of spike-rate variations and therefore provides an accurate estimate of the phase coherence in different task epochs and conditions. Beta (18-35Hz) and low frequency (2-8Hz) functional connections dominated the network, with beta being stronger during fixation and memory epochs, whereas low frequency was stronger during movement. Beta functional connections were found almost exclusively between single units of AIP and LFPs in both areas, whereas single units in F5 synchronized predominantly with LFPs of both areas in low frequencies. Network connectivity was heterogeneously distributed for all task conditions, with a small group of single units (hubs) synchronizing consistently with a large part of the network. On average, pairwise FC was constant across task conditions. However, conditional differences became apparent when the dynamic FC network was projected into a state space where each dimension represented the FC of one spike-field pair. Beta FC network trajectories showed larger cross-conditional differences during the memory epoch, while low frequency FC network trajectories varied mainly during the movement epoch. These findings suggest that behavior-relevant changes in the cortical motor network are coordinated by distinct groups of hub units synchronizing selectively in the beta or low frequency range.

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[T 67] **Balanced networks with overlapping assemblies and dense inhibition produce sequences with Markov dynamics**

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Following experimental measurements of clustered connectivity in the cortex, modelling studies have found that clustering connections in simulated spiking networks causes transitions between high and low firing-rate states in subgroups of neurons [1]. An open question is to what extent the pattern of transitions in such networks can be related to computational functions, such as the generation of sequences. We present several studies of the relationship between connection structure and network dynamics in balanced spiking networks with dense nonspecific inhibition. We demonstrate that balanced networks which contain overlapping assemblies with equal levels of recurrent excitatory connectivity can produce “winnerless competition” sequences of high activity states, in which a single assembly is in a high-activity state at any one time. This activity is reflected in the power spectrum of spiking activity as a peak in the low-frequency delta range. Sequences can be described with a Markov chain framework, which we use to verify and quantify the non-uniformity of probabilities of transitions between specific states. We furthermore investigate which qualities of the network connection matrix support the generation of state sequences and what determines the specific structure of transitions between states. At moderate levels of overlap, transition dynamics can be compared mechanistically to “latching” models of sequence generation, in which activity is passed between overlapping attractors with overlapping basins of attraction [2]. The switching transitions can be related to theoretical computational concepts such as chaotic itinerancy, and could potentially have relevance to biological instances of Markov sequence generation such as the song production of some species of birds. The results clarify the computational capabilities of clustered spiking networks and their relationship to experimental findings. We conclude that clustered networks could provide a supporting intermediate link between abstract models and biological instances of sequence generation.

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[T 68] Input-output relations of inhibition-dominated networks with a 3-dimensional input ensemble to describe color selectivity

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In previous work it was shown that contrast-invariant neuronal responses to oriented visual stimuli occur in random network models of primary visual cortex, provided recurrent inhibition dominates excitation [1]. Through further mathematical analysis it was found that selective attenuation of the stimulus-independent input mode, common to all neurons, can explain this property. The input space is 2-dimensional in this well studied experimental setup, where elongated gratings of light and dark stripes in the visual field evoke a neuron-individual response specific to the angle of the grating. In our present study, the same network architecture is used to investigate the input-output relations for higher-dimensional input ensembles. Color vision in trichromats is an important example of a 3-dimensional setting, where the selective response of neurons in several areas of the visual cortex is well documented [2]. In our model, the rate of external non-homogeneous inputs to the network is weakly tuned according to the individual neuron's "preferred feature" of the tuning curve. Our simulation results show that the firing rate response of neurons, as it was the case for a 2-dimensional input space, again exhibits strong tuning as well as attenuation of the common mode.

Acknowledgements

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[T 69] **EI balance: necessary or inevitable?**

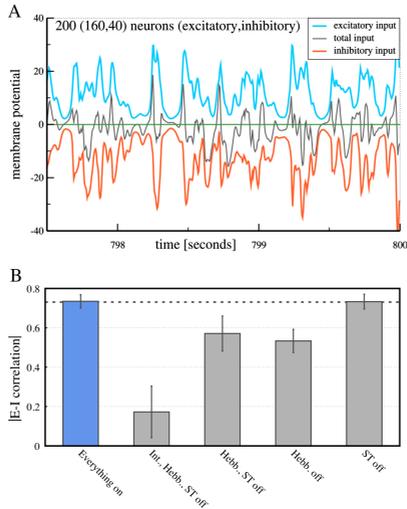
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Spontaneous brain activity is characterized by an asynchronous chaotic state for which otherwise large excitatory (E) and inhibitory (I) inputs balance each other [V96]. Such states may be obtained when the synaptic weights scale, in the absence of strong correlations in the neural activity, as $1/\sqrt{K}$, where K is the number of afferent synapses. The variance remains in this case constant [B16], with the mean excitatory and inhibitory input contributions individually diverging as \sqrt{K} . The question is then if and how (a) the chaotic state and (b) EI balance are obtained in fully autonomous networks, i.e. with ongoing dynamics coupled to both intrinsic plasticity (IP), and synaptic plasticity, considering here both Hebbian (Hebb.) and short term plasticity (ST) .

We examine here networks of continuous-time rate encoding neurons which adapt their afferent synaptic weight according to a self-limiting Hebbian learning rule deduced from the stationarity principle for statistical learning [E15]. The pruning of synaptic weights crossing zero allows then to study networks obeying Dale's law. The synaptic learning rules are considered in our study to be identical for excitatory and for inhibitory neurons [S17], for which have considered both a 1 : 1 or a 4 : 1 ratio. For the stabilization of the average activity level we use an intrinsic adaption rule for the threshold $b=b(t)$ entering the sigmoidal [M12].

Under these conditions, we find that EI balance robustly emerges in a self-organized fashion, with inhibition closely tracking excitation (see Fig. A), without the need for an explicit $1/\sqrt{K}$ synaptic scaling. This effect is mainly produced by the intrinsic regulation (IP) of the mean neural activity, and is present even in the absence of synaptic plasticity, but is further increased by the effect of synaptic renormalization induced by the ongoing Hebbian plasticity (cf. different bars in Fig. B). Further introduction of short term plasticity, which can produce fast changes in the effective connectivity between neurons, is not able to disrupt this state, making our results remarkably robust, and reinforcing the idea that EI balance is not just desirable in these networks, but almost inevitable under these conditions. Our results show furthermore that networks of rate-encoding neurons evolve, under the influence of self-limiting Hebbian plasticity, to a chaotic state fluctuating strongly on timescales of (30 – 50) ms.



(A) For a representative neuron: total input (in black) and separate inputs from excitatory (in blue) and inhibitory (in red) neurons. Network config.: 160 exc./40 inh. neurons, connectivity 20%. (B) Avg. correlation coefficient of E-I inputs, with different forms of plasticity switched on/off.

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[T 70] Which abstraction level to choose for the leech local bend network model?

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In the tactile system of the medicinal leech (*Hirudo medicinalis/verbanus*), sensory information is processed locally by a network of interneurons in each of the 21 midbody ganglia [1]. Applying voltage-sensitive dye (VSD) imaging, the activity of up to 100 neurons in each ganglion can be simultaneously recorded [2]. By intracellular current injection to mechanoreceptors or by applying tactile stimulation in semi-intact preparations, we identified neurons involved in the processing of tactile stimulation and mediating the local bend behavior. These neurons were further investigated via intracellular recordings. In this study, we aimed at constructing a neuronal model of the local bend network. On the single cell level, our goal was to find out if and how characteristic response features of different leech neurons can be fitted with the standard Hodgkin–Huxley model [3]. Our approach comprised two steps: 1) Passive membrane parameters (time constant, membrane resistance and capacitance) calculated from experimental data were implemented in the model. 2) Active model parameters (reversal potentials and conductances of Na⁺ and K⁺) were systematically varied to fit the empirical number of spikes, spontaneous spike activity and spike amplitude, but the model failed to replicate the intracellularly recorded dependency of spike counts on intracellular current stimulation amplitude. On the network level, the data from VSD experiments and intracellular recordings were incorporated into a feed-forward network model inspired by [4]. Neurons were modeled as simple leaky-integrate-and-fire models. The model reproduced the amplitude and time course of most of the motor neuron responses to sensory cell stimulations as measured in [5]. We conclude that both, the basic single cell – and network – model are not sufficient to simulate the fundamental neuronal response properties even of this simple nervous system. Future work needs to combine optimization of channel kinetics on both levels to obtain a more accurate model of the leech local bend network.

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[T 71] Temporal Fluctuations in Functional Networks of the Human Brain: Modeling brain transition between cognitive states

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New technologies of imaging allow the mapping of activity on whole-brain and to infer interdependence between cortical areas with high spatio-temporal resolution. An increasing number of studies show that higher cognitive functions can be better understood by considering the brain an interactive dynamical complex network. In this context, we are developing a data driven computational model to study functional cortical interactions of human brain. Our broad aim is to create numerical tools to characterize properties of functional patterns, and their evolution over time, at rest and during a task execution. We derived connectivity maps from fMRI and DTI experiments to build structural networks. The dynamic processes are simulated by embedding into this complex network a combination of oscillations and Balloon-Windkessel hemodynamic response models. We evaluate how dynamical properties may influence functional interactions. Our initial model is already able to recover remote synchronization in the network, reaching a reasonable agreement with experimental functional network (see references). We show that the best agreement between model and experimental data is reached for dynamical states that simultaneously maximize synchronicity and metastability. The main contribution of this work is to create a model of networks reconfigurations that will pave the way for the understanding of transient interactions in the cognitive processes.

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[T 72] Optimal wiring fixes cortical hypercolumn sizes across species

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The cerebral cortex is arranged in discrete columns wherein neurons respond to a single modality across cortical layers [1]. A hypercolumn is a broader definition of a cortical column; it is made up of the neurons representing a full set of values of a receptive field parameter [2]. Orientation selective neurons in the visual cortex form hypercolumns representing all possible orientations between 0 and 180 degrees at a finite amount of precision [3]. Within these hypercolumns, neurons are cyclically arranged regarding their orientation preference and form so-called pinwheels [4]. Although it has been shown that pinwheel density normalized by the hypercolumn area is constant [4], the absolute pinwheel density is not constant [5]. Using a novel method of modelling neural placement that predicts pinwheel structures from optimal wiring considerations [6], we find that cortical map structure appears at a fixed number of neurons per hypercolumn independently of the overall network size. These results are consistent with existing biological data that show constant numbers of neurons per pinwheel across a variety of mammalian species. Observed differences in the absolute pinwheel density can accordingly be explained just by variations in the neuronal density of the visual cortex and probably have no functional implication. Thus our findings further support the presence of a relatively uniform architecture in mammalian visual cortex.

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[T 73] Fading reverberations in collective spiking dynamics in vivo

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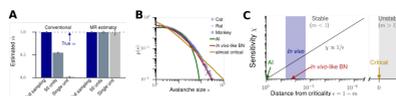
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Collective spiking dynamics are likely linked to the function of neuronal networks. However, to date two contradictory hypotheses prevail: The first proposed an asynchronous irregular (AI) state [1], which minimizes redundancy [2] and promotes fast network responses. The second proposed a critical state [3], which is characterized by long range correlations in space and time and in models maximizes certain information processing properties. Distinguishing between these two states is straight forward when the activity of all neurons is known. However under subsampling [4,5], classic approaches can mistake a network close to critical as AI: We showed that single neurons exhibit exponential inter-spike-interval distributions, and the Fano factor of single neurons is always close to unity.

We derived a novel estimator, which can infer the dynamical state even under strong subsampling, in principle from the activity of a single neuron (Fig A). In this framework the dynamical state is characterized by the average number m of postsynaptic spikes triggered by one presynaptic spike.

We applied this estimator to spiking activity in monkey prefrontal cortex, cat visual cortex, and rat hippocampus. Consistently, in vivo dynamics is in a narrow regime of fading reverberations (median $m = 0.984$), situated between AI ($m = 0$) and critical ($m = 1$). A model with the same m as in vivo could predict the bin size dependent spike count cross correlations between neurons, Fano factors, inter-spike-interval distributions, rate distributions, and the avalanche size distributions of the in vivo recordings (Fig B). The latter clearly differed from power-laws, which have been used as marker of criticality. Despite the small difference in m , the fading reverberations state is clearly distinct from criticality: e.g. it limits the sensitivity and intrinsic timescale of the network, which both diverge at criticality (Fig C).

Our findings may account for the contradictory experimental results on cortical population dynamics: spiking activity appeared AI-like, as under subsampling correlations are underestimated. In contrast, coarse measures like LFP potentially overestimated correlations, making networks appear critical. Instead, spiking dynamics in vivo is situated between the two regimes and may combine their computational benefits by allowing integration of information over limited timescales, while avoiding the risk of instability or slowing down associated with criticality.



- A. The novel MR estimator can infer the correct dynamical state even under strong subsampling. B. The in vivo-like network could predict the in vivo avalanche size distributions. C. The fading reverberations state clearly differs from criticality in terms of dynamics.

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[T 74] IDTxI – The Information Dynamics Toolkit xl: a Python package for the efficient analysis of multivariate information dynamics in networks

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We present IDTxI, a new open source toolbox for effective network inference from multivariate time series using information theory, available from github. IDTxI utilises a greedy or iterative approach with multivariate transfer entropy for building sets of parent sources for each target node in the network. This iterative conditioning is designed to both remove redundancies and capture synergistic interactions in building each parent set. Rigorous statistical controls (based on comparison to null distributions from time-series surrogates) are used to gate parent selection and to provide automatic stopping conditions for the inference. The toolkit is a next generation combination of the existing TRENTOOL and JIDT toolkits, extending TRENTOOL's pairwise transfer entropy analysis to a multivariate one, and adding a wider variety of estimator types. Further, IDTxI is Python3 based, requiring no proprietary libraries, with parallel computing engines for both GPU and CPU platforms. The toolkit is highly flexible, providing various information-theoretic estimators for the user to select from; these handle both discrete and continuous time-series data, and allow choices, e.g. using linear Gaussian estimators (i.e. Granger causality) for speed versus nonlinear estimators (e.g. Kraskov-Stoegbauer-Grassberger) for accuracy. IDTxI also automates parameter selection for the

user, including selecting source-target delays and constructing non-uniform embeddings of the sources via conditional mutual information. Tools are included for group-level analysis of the inferred networks, e.g. comparing between subjects or conditions. Finally, IDTxI includes additional tools for studying the dynamics of various information flows on the inferred networks. The primary application area for IDTxI lies in analysing brain imaging data (import tools for common neuroscience formats, e.g. FieldTrip, are included). However, the toolkit is generic to analysing multivariate time-series data from any discipline. We will demonstrate the efficacy of IDTxI in inferring networks from various synthetic data sets.

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[T 75] Changing correlation structure during activity deprivation in visual cortex of freely behaving rodents

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Neuronal networks are faced with the difficult challenge of maintaining stability for the execution of precise behavioral actions and flexibility to be able to change following learning and experience-dependent plasticity. Various forms of homeostatic mechanisms exist to maintain stable function by globally adjusting overall synaptic weights and intrinsic excitability. A good model to study these processes is the visual cortex (V1) of rodents during the classical critical period (4-6 weeks after birth). This period is characterized by a high degree of plasticity that can be induced by manipulating visual experience, for example, depriving animals of vision in one eye, known as monocular deprivation (MD). It has been previously shown that MD induces an initial drop in firing rates followed by a homeostatic recovery of firing rates despite continued deprivation [1,2]. We asked what other properties of cortical network dynamics change in addition to firing rates during prolonged MD. We obtained extensive datasets of extracellular

chronic recordings over 9 days of the joint activity of many cells from the cortices in freely behaving rodents during MD using tungsten multielectrode arrays [1,2]. We focused on cortical dynamics in the monocular region of V1 and analyzed 5 hemispheres ipsilateral to the deprived eye (unaffected, control) and 6 contralateral to the deprived eye (affected, deprived). To characterize changes in the network, we computed the pairwise correlation matrix for all recorded cells. We found that the correlations considerably weakened during early MD, and then recovered during late MD. Moreover, the changes were consistent among different cell types (excitatory and inhibitory) and different behavioral states (wake, REM and non-REM sleep), and were not due to changes in firing rates because shuffling spike times eliminated the effects. The observed drop and gradual recovery of correlations in deprived hemispheres was very different compared to control hemispheres. In control hemispheres we observed a slight increase in the correlations as a function of age, but only during light conditions and in the presence of visual input. These results suggest that powerful homeostatic mechanisms regulate network interactions following activity deprivation independent of single neuron firing rate homeostasis.

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[T 76] **A biologically mechanistic model for orientation map development**

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The architecture of iso-orientation domains in the primary visual cortex (V1) of primates and carnivores apparently follows species invariant quantitative laws [1,2]. The emergence of this common design has been recreated by an abstract class of mathematical models for neural circuit self-organization [1,3]. So far no biologically detailed model has been shown to conform with all features of this common design. Recently, Stevens et al. [4] made available a biologically mechanistic model which mimics visual pathways and can be trained by natural stimuli. We examine and characterize this biologically mechanistic model in order to understand whether detailed models of Hebbian learning for the formation of nerve cell networks quantitatively match the common design in the visual cortex.

Our results show that, when covering a substantial fraction of the period of juvenile plasticity [5], the statistics of pinwheel layouts including average pinwheel densities generated by the model are time-dependent and typically drop below experimentally observed values. We also find that the orientation tuning properties of single neurons continue changing at high rates long after the emergence of orientation selectivity.

Orientation maps in the simulations typically become more regular over time. The banded geometry of the orientation domains at the boundaries of V1 increasingly dominate the center of the simulation area. This process is driven by pinwheel generation events at the boundaries of V1 followed by pinwheel annihilation events at the center. The time scale of pinwheel survival is roughly maintained over the course of the simulation, with fluctuations depending on the learning rate of the system. The switching behavior between the prominence of orientation stripes from the lateral and from the superior/inferior boundaries prevents the pinwheel density to decrease to crystal-like values, concealing a potential energy ground state of the system. This indicates that boundary effects strongly influence the layouts in this model.

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[T 77] Weak-noise-induced transitions with inhibition and modulation of neural oscillations

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We analyze the effect of weak-noise-induced transitions on the dynamics of a computational neuron model in a bistable state consisting of a stable fixed point and a stable unforced limit cycle. Bifurcation and slow-fast analysis give conditions on the parameter space for the establishment of this bi-stability. In the parametric zone of bi-stability, weak-noise amplitudes may strongly inhibit the neuron's spiking activity. Surprisingly, increasing the noise strength leads to a minimum in the spiking activity, after which the activity starts to increase monotonically with increase in noise strength. We investigate this inhibition and modulation of neural oscillations by weak-noise amplitudes by looking at the variation of the mean number of spikes per unit time with the noise intensity. We show that this phenomenon always occurs when the initial conditions lie in the basin of attraction of the stable limit cycle. For initial conditions in the basin of attraction of the stable fixed point, the phenomenon however disappears, unless the time-scale separation parameter of the model is bounded within some interval. We provide a theoretical explanation of this phenomenon in terms of the stochastic sensitivity functions of the attractors and their minimum Mahalanobis distances from the separatrix isolating the basins of attraction.

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[T 78] V1 Populations Exhibit Stimulus-Specific Transient Dynamics

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Visual responses in V1 are dynamic in time even when the stimulus is static. These dynamics arise on the backbone of an intricate web of internal connections. Evidence suggests that these connections have been carefully shaped during development by Hebbian plasticity[1] to encode the statistical structures of the natural environment[2,3]. Here, we recorded multi-unit activity (32 electrodes, GrayMatter) from awake monkey V1 during a passive viewing task and we investigated the response dynamics evoked by different categories of visual stimuli. The stimuli included structured (natural scenes (NS) and geometric shapes (GS)) and unstructured (phase-scrambled (PS) images and random noise (RN)) images. Stimulus representations by the neuronal population in V1 can be described as trajectories in neuronal state space and visualized by lower dimensional projections via Principal Component Analysis (PCA). Over time, the PCA projections of population responses (spike counts over 50 ms bins) to different stimuli traced distinct trajectories: the responses started with a short onset transient (100 ms) and then settled into stimulus-specific sub-regions in the PC space (500 to 2000ms). For all stimulus categories, the velocity of trajectories was high during the transient and low once the trajectories were stabilized. The distance between the trajectories to different stimuli in GS, NS and PS conditions peaked during the onset transient and remained above the baseline during stability, while the distance between the RN trajectories stayed at baseline for the entire duration of the trial. Interestingly, the trajectories for structured stimuli (NS and GS) were well separated throughout the trial, whereas those for unstructured stimuli (PS and RN) were less separable, despite having nearly identical firing rate profiles. In conclusion, our preliminary analysis suggests that V1 neural responses to static images exhibit distinct dynamics over time for different stimuli, with a fast initial transient that contains a large amount of information about stimulus identity. When the visual stimuli are structured (NS and GS), the response trajectories settle into stimulus-specific sub-regions in the state space.

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[T 79] Dynamic Population Encoding in a Type II Neuron

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Cortical neurons can realize fast population encoding [4, 5, 8, 1, 7]. The underlying biophysical mechanisms however are not well understood. Action potential onset rapidness is proposed to be closely associated with the encoding ability [6]. Reducing the onset rapidness of pyramidal cells can impair the bandwidth of their linear response functions [4]. In a recent multi-compartment model [3], using a standard sodium activation function, cutoff frequencies above 100Hz were found. This high frequency encoding ability is maintained in the case without a dendritic tree. The observation above seems to indicate that fast population encoding can be realized without action potential onset rapidness.

In this work, we analyzed the underlying mechanism in a simplified multi-compartment model for realizing fast population encoding. We first reproduced the linear response curve for the model without a dendritic tree. The linear response curve is insensitive to the temporal correlation of the input, which differs from experimental observations in cortical neurons [8]. Through calculating the F-I curve of the model, we found that this is a type II neuron. The F-I curve has a pronounced discontinuity at the threshold current, which implies that the neuron is capable of high frequency repetitive firing. Examining the histogram of the inter-spike intervals generated by a stochastic stimulus, we found that the peak of the histogram fits with the resonance frequency in the linear response curve. This indicates that the type II model is more likely to generate pieces of high frequency repetitive firing when responding to the stochastic input. In this way, it enhances the bandwidth in the high frequency regime which results in a high cutoff frequency of the linear response curve.

Comparing with another multi-compartment model proposed by Brette [2], we found that an active soma and a complete spike generation mechanism with both sodium and potassium channels are important for the pronounced discontinuity in the F-I curve and high bandwidth in the linear response curve. Removing the ion channels in the soma will reduce the size of the discontinuity in the F-I curve. If we further replace the complete spike generation mechanism with resetting the voltage by hand at threshold, the high frequency repetitive firing breaks down. The neuron model then becomes type I and exhibit a low encoding ability.

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[T 80] Homeostatic plasticity explains different dynamic states of neural populations in vivo and in vitro

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In vivo and in vitro neural spiking activity clearly differ. While in vivo networks often show continuous activity[1–3], networks in vitro develop strong bursts separated by periods of silence[4–6]. This is puzzling considering that both networks presumably share the same single-neuron dynamics and plasticity rules. We propose homeostatic plasticity as a mechanism to account for the strong differences of in vivo and in vitro network dynamics. Analytically treating a mean-field neural network in terms of a branching process allows us to disentangle the recurrent network dynamics from stochastic external input. For a given input strength, homeostatic plasticity tunes the recurrent connections and thus alters the dynamic state of the network, generating bursts under in vitro conditions, and reverberant, fluctuating dynamics under in vivo conditions. We verify our conjecture by numeric simulations and compare to experimental results under both in vivo and in vitro conditions. Our results suggest that homeostatic plasticity may be exploited by adding stochastic input to in vitro cultures, thus tuning their dynamic state to be comparable to in vivo dynamics.

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Sensory processing and perception

[T 81] Mapping complex receptive fields in primate cortical area MSTd with reverse correlation

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Area MSTd in primate extrastriate visual cortex is assumed to play a central role in the encoding and perception of optic flow stimuli, i.e. the large-scale motion patterns on the retina caused by the movement of the visual environment relative to the organism. Correspondingly, MSTd neurons show tuned responses to the direction of linear motion stimuli, as well as of 'spiral motion stimuli', which include expansion, contraction, rotation and their mixtures, arranged in a continuous circular dimension. MSTd cells have been reported to be position-invariant in their responses to spiral motion stimuli. Here we report a study aimed to determine the exact motion patterns MSTd neurons are most responsive to. We used reverse correlation, a linear method which has been successfully used to characterize receptive fields in V1 and MT. Our reverse correlation stimuli were large complex random dot patterns, formed by the smooth variation of local dot direction and speed between a virtual grid of positions in the stimulus where local direction and speed parameters were chosen randomly every 100ms. We investigated whether these patterns are a more appropriate description of the specific motion preferences of individual MSTd neurons. We also determined the position dependency of MSTd responses to spiral motion patterns. We recorded from more than 140 MSTd cells in three rhesus monkeys. For around a quarter of the 86 cells where sufficient data with the reverse correlation stimulus was recorded, our analysis recovered significantly structured spatial motion preference maps. The recovered maps show a preference for similar linear motion directions across the receptive field. This raises the question whether the method was only successful on cells that have homogeneous linear motion preferences. Interestingly, almost half of the cells whose receptive field could be mapped with our reverse correlation stimulus showed position invariant responses to spiral motion patterns. Additionally, the responses of the mapped cells to spiral and linear motion patterns significantly correlated with the patterns' similarity to the spatial motion maps obtained by the reverse correlation analysis. Our findings are consistent with the hypothesis that the linear component of MST motion encoding is suited for

simple linear motion direction preferences, while the responsivity to and selectivity for complex motion patterns is a non-linear encoding aspect of the population of MST neurons.

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[T 82] The relation between eye and head movements and cortical activity in freely moving mice

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The control of gaze is an essential component of vision. As the mouse is becoming an increasingly prominent model in vision research it is important to understand the phenomenology and neural consequences of gaze- and eye-movements in mice. While experiments in head-fixed mice provide important insights into processes that stabilize or shift the gaze with respect to the external world, it is not well understood how freely moving mice move their eyes, nor how eye- and head-movements affect sensory neural responses in mice.

To address such questions, we developed a miniature lightweight head-mounted video camera system (1.3 grams) for mice, which we combined with movement sensors and chronic multielectrode implants for *in vivo* electrophysiology in behaving animals. The camera system allows for simultaneous monitoring of multiple behavioural variables including eye and whisker pad movements and pupil dilation in freely exploring mice. We show that the head-mounted video camera system does not affect neural recording quality and generates stable video recordings with an average jitter per frame of 5 micrometers, less than 0.5% of pupil diameter. Using a semi-automatic behavioural segmentation algorithm, we find that mouse behaviour is similar with and without the camera system.

We used the method to quantify covariations of behavioural variables and neural activity in sensory cortex in different behavioural states. We characterized the relation between head and eye movements by training different models to predict horizontal and vertical eye position based on measured movements of the head. A simple linear model was able to predict a large fraction of variation in eye positions (51% horizontal, 78% vertical). This suggests that, as reported for freely moving rats (Wallace et al., 2013), many eye movements are directly coupled to active changes in head orientation. We also found that activity of neurons in primary visual cortex was either suppressed or enhanced by head movements, even in the absence of visual input. Using the head-mounted camera system, we were able to dissociate the effects of different behavioural variables on neural activity, demonstrating for example that responses were more directly related to head movement-related signals than to eye or whisker movements.

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[T 83] Receptive field organization in motion detecting circuits in *Drosophila*

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Visual motion cues are fundamental for the navigation of many organisms. Classical models of motion computation consist of temporal correlations of incoming light signals from two spatially restricted sensors. The fruit fly *Drosophila melanogaster* is a powerful genetic model organism with a comparably small brain that responds behaviorally to motion cues. For these reasons, combining connectomics, physiology, genetics, and behavior allows significant progress in the mapping of motion detection circuits in the fly. In the fly visual system, there are two specialized pathways that detect moving light increments (ON) or decrements (OFF). Pathway splitting occurs in the lamina, one synapse downstream of photoreceptors, with neurons L1 and L2 & L3 giving rise to the ON and OFF pathways, respectively. Interneurons in the medulla then connect the L1, L2 and L3 inputs to the dendrites of the first direction-selective neurons. These circuits are believed to represent the neural substrate of the local correlation-type models, since most receptive fields on the circuit are restricted to about one column of the fly eye. Nonetheless, the functional architecture of the circuits remains incompletely understood. In particular, one of the medulla interneurons (Tm9) that is behaviorally required in the OFF pathway shows wide-field responses under some stimulus conditions. This finding challenges the purely local correlation assumption. Furthermore, recent studies support a model that implements three-point correlations. We aim to understand the circuit at the level of single cell visual response properties to be able to map the algorithmic steps leading to motion detection. Here we investigate the receptive field properties of the behaviorally critical Tm9 neuron for motion detection using *in vivo* two-photon calcium imaging with the genetically encoded GCaMP6f sensor, using a variety of visual stimuli. In order to understand how complex receptive fields are shaped by presynaptic inputs, we are combining physiological measurements of the input elements with computational simulations of the receptive field of postsynaptic, direction selective neurons. We will ultimately explain how subcomponents of a receptive field can shape neural computations and animal behavior using measurements of downstream direction-selective neurons or optomotor responses.

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[T 84] Mechanisms behind cortical processing of reverberated natural sounds

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Reverberation is a major source of noise in acoustic signals, masking both temporal and spectral features of the stimuli. For processing and recognition of acoustic signals, a neural representation invariant to reverberation is desirable. It has been shown that such invariant representations arise along the mammalian auditory pathway, especially in auditory cortex [1]. However, mechanisms promoting this processing remain largely elusive. One candidate mechanism is delayed feed-forward inhibition, potentially cancelling out the echo signal. Such feed-forward inhibition is provided by an important subtype of inhibitory interneurons (parvalbumin-positive – PV+).

Here, we aim to test processing of reverberated sounds in mouse auditory cortex, using a set of animal vocalizations. In a first step, we want to compare auditory cortical activity based on responses of populations of simultaneously recorded single neuron between non-reverberated and reverberated stimuli and reconstruct the stimuli based on population responses. Furthermore, we aim to investigate the role of inhibitory PV+ cells on processing of reverberation. For this purpose, the activity of PV+ neurons was increased by prolonged, low-level optogenetic depolarization, using a stable step-function opsin (SSFO) variant of ChR2. This optogenetic manipulation was performed during extracellular recordings from A1 of awake and chronically implanted animals.

In a first step, we observed that individual neurons responded specifically to different vocalizations and within the vocalization to different motives. Comparing the neuronal activity of reverberated to non-reverberated stimuli we noticed that reverberation leads mostly to a deterioration of the temporal structure of neural responses. To our surprise, we also observed some neurons that responded selectively to the echo in the reverberated stimuli. Enhancement of feed-forward inhibition mostly resulted in overall reduction of activity in both clean and reverberated sounds. Detailed results on stimulus reconstruction and the effect of enhancement of inhibition and consequences for cortical mechanisms will be discussed.

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[T 85] Different noise sources determine the optimal tuning curve heterogeneity in population coding

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The efficient coding theory states that neural populations in sensory systems have evolved to encode a maximum of sensory information given biophysical constraints. A common framework for quantifying the amount of information about a stimulus encoded by neurons is Shannon's mutual information. Early work studied how single neurons maximize information transmission, however, since information processing in sensory systems is performed by many neurons in parallel, interest has switched to population coding. Recent work has thus tried to answer what shapes of tuning curves are optimal (i.e. maximize information) and how tuning curves optimally diversify in populations of neurons [1,2,3,4]. The common result of all these studies is that in general the number of distinct tuning curves monotonously increases with decreasing noise. Some studies assumed a single (information limiting) channel [1,2], others studied small populations of up to two neurons [3,4] and only recently considered the interaction of different noise sources [4]. Here, we investigated how, in a finite population of neurons independently coding a one dimensional stimulus variable, optimal tuning curve diversity depends on two different sources of noise. One noise source is additive input noise arising from presynaptic signal corruption, the other is output noise present in the spike generating mechanism - here we consider a common noise model where neurons generate spikes following Poisson statistics. We assumed binary neurons since they maximize information in the presence of (biologically realistic) high output noise, they are a good model of neuronal responses in some sensory systems [5,6] and because they facilitate calculations. Mutual information was numerically maximized using several local and global optimization algorithms. For up to four neurons and finite noise levels, the number of diverse tuning curves in general increases stepwise with both decreasing input or output noise, a result which is in accordance with previous results. However, for five and more neurons over a range of input and output noise values the optimal number of diverse tuning curves changes non-monotonically with noise strength, in contrast to previous results. This unexpected result yields interesting predictions for the structure of tuning curves in different sensory systems in the presence of two sources of noise.

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[T 86] Functional diversity of mouse retinal ganglion cells in 4096-electrode CMOS array recordings

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The retina is a complex neural network, responsible for breaking down the visual scene into its main features such as color, motion and local contrasts. Retinal ganglion cells, which form the output layer of the retina, provide the only input of visual information to the brain. The population of ganglion cells displays considerable diversity, with more than 30 types currently expected based on anatomical and physiological considerations, and individual types are known to relay distinct visual features to specific areas of the brain. To understand what information is provided to downstream areas of the brain, as well as to tease out how visual information is processed in the retinal circuitry, it is important to cope with this functional diversity.

Previously, much of this diversity has been catalogued by pooling ganglion cell responses over many individual retinas. Here, we stimulate a mouse retina using a standard battery of light stimuli and simultaneously record the electrical activity from a large population of ganglion cells – in whole-mount preparations – using 4096-electrode arrays. This allows us to recover spikes from 500 to 1000 ganglion cells in a typical experiment. For each recorded ganglion cell, we measure the receptive field size and temporal dynamics, spike-train autocorrelation function, direction-selectivity, and orientation-selectivity. Ganglion cells with either significant direction- or orientation-selectivity are correspondingly labeled. For the remaining cells, we use an unsupervised learning method (spectral clustering) to cluster ganglion cells into groups of functionally similar cells. Ganglion cells in each group are finally tested for homogeneity in their responses to light stimuli, axonal conductance and whether a minimum pairwise distance (tiling) is respected between their receptive fields.

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[T 87] Challenges for a colorful brain: the integration of shape and color in the primary visual cortex

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Color vision was lost in mammals during the nocturnal bottleneck when our ancestors were small and dark-dwelling between 205 to 65 Million years ago (Ma). Among modern mammals old world monkeys and great apes (re-)invented trichromacy 30-40 Ma. The newly developed color vision inserted new pathways into visual cortical architecture, potentially perturbing the layout of orientation domains in the primary visual cortex (V1) through non-orientation selective cytochrome oxidase blobs (Livingstone and Hubel, J Neurosci. 1984). How much impact color vision had on the overall functional architecture of V1 in primates remains unclear and little is known about theoretically expected effects. Here we predict and test the experimental signatures of three distinct and paradigmatic model types for this evolutionary transformation of visual cortical architecture in primate evolution. The models are (i) a color vision dominated optimization model, in which we expand the coupling between layouts of orientation and color to lowest order (Reichl et al. PLoS CB 2015, Bressloff et al. PRL 2002). We find that models of this type can stabilize pinwheel-rich layouts without long-range connections but predict deviations from the common design, a set of layout rules of orientation domains that has evolved independently at least twice (Kaschube et al. Science 2010, Schotttdorf et al. PLoS CB 2015). (ii) a geometric distortion model that frees space in a layout of orientation domains to include color selective cells by displacing orientation domains. We show that models of this type leave the measures of the common design invariant, but are particularly sensitive to phase perturbation. (iii) a form vision dominated optimization model, in which orientation domains constrain the layout of color-selective cells. We find models of this type to be consistent with experimental data.

In summary, we uncovered a set of quantitative, specific and measurable predictions from the three model types that can be falsified given the precision of available data. Our study supports the view that orientation domains provide a scaffold for other functional layouts. The evolutionary expansion in primates induced only a minor perturbation to the design of form vision circuitry in V1.

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[T 88] From artificial to natural stimuli: encoding of visual signals in retinal bipolar cells

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Current mathematical models in the retina fail to reliably predict responses of retinal ganglion cells to natural stimuli. Often, in these mathematical models, it is assumed that the input neurons of the retinal ganglion cells behave linearly. In this work, we focus on the excitatory input neuron: the bipolar cell and test how well standard models – like the linear-nonlinear (LN) model – can predict bipolar cell responses to artificial and natural stimuli. To understand and predict bipolar cell responses to light, we started with simple full-field contrast changes while recording the membrane potential of the cells intracellularly. Here, we found some cells with linear and others with nonlinear responses to contrast changes, and for both types the LN model successfully predicted responses to new contrast sequences (R2 of 82%-98%). We then continued with spatially structured artificial stimuli. For these stimuli, the performance of the LN model varied, for some cells the prediction was accurate (e.g. R2 of 82%) while for other cells the prediction failed (R2 of 34%). When investigating the reasons for the failure, we found a novel bipolar cell response property: nonlinear integration in space, similar to Y-type ganglion cells. Furthermore, this novel Y-type-like property observed in some bipolar cells caused the LN model to fail. To finalize, we showed natural movies to study how well we can apply the knowledge learned from artificial stimuli to predict responses to natural stimuli. Here, again, the model can predict the responses to natural movies well for linear cells, but failed for nonlinear Y-type-like bipolar cells. The findings suggest that nonlinear signal integration can start already at the level of bipolar cells and that nonlinear computations are crucial properties that mathematical models in the retina have to take into consideration for predicting responses to artificial and natural stimuli.

Acknowledgements

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[T 89] The Role of Spikes for Information Transmission in Bipolar Cell Models

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Traditionally, most inner retinal neurons are thought to respond with graded voltage changes to visual stimulation. These analog signals would be converted into spikes only at the level of the retinal ganglion cells, the output neurons of the retina. However, a growing body of evidence instead argues that this signal conversion from 'analog' to 'digital' can already take place at the level of the bipolar cell synaptic terminals (Protti et al., 2000; Baden et al., 2013; Puthussery et al., 2013). This conversion impacts information transmission and coding, for example by increasing the precision of the signal, but at the expense of decreased reliability and higher energy consumption (Baden et al., 2011; Sengupta et al., 2014). To understand the biophysical regimes in which spiking can occur and to study the effects of spiking on information coding, we built a simple two-compartment model of bipolar cells. The first compartment is a Hodgkin-Huxley-like channel system, which incorporates four types of ion channels (Na^+ , K^+ , L-type calcium and T-type calcium channels). It is driven by a light stimulus with different statistics, which is pre-processed by a simple linear filter aimed to model linear photoreceptor input. The second compartment models a ribbon synapse (Sikora et al., 2005). This ribbon synapse transforms the voltage signal into calcium concentration, which in turn drives the dynamics of three different vesicle pools (Burrone et al., 1997). The release rate of these neurotransmitters act as the output signal of our model and a "fair currency" for comparing information rates in different regimes. First, we investigated the impact of channel densities, channel dynamics and different sources of noise on the responses to light stimuli. In this regard we identified parameter regimes in which the first compartment switches from a graded signaling to a spiking mode. As expected, response waveforms were dominated by sodium and potassium channels, but were in addition strongly influenced by the density of calcium channels. Next, we evaluated the information that can be read-out linearly from the release rate, with high information rates indicating regimes that may be favorable for coding. In summary, our analyses provide insights into the operating regimes of bipolar cells and their possible consequences on information transmission in vision.

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[T 90] Critical subnetworks for feature integration

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For understanding complex visual scenes, our brain has to integrate local, distributed information into global, coherent percepts. This computational process is fundamental for important functions such as contour integration, figure-ground segregation and, ultimately, object recognition (e.g. [1-2]). In our contribution, we propose that feature integration can be performed most efficiently when the neural dynamics of "figure"-encoding networks is close to a critical state [3]. In such a regime, avalanches of spiking events are observed on all length scales, thus rendering figures in a scene "visible" for coincidence detectors in higher visual areas.

We consider the analytically tractable network model of Eurich-Herrmann-Ernst (EHE) units [4] and we simulate a large network embedded with N_e subnetworks of recurrently, excitatorily coupled units, each representing a "figure". Each subnetwork of N_s units is poised at the critical state and competes with other subnetworks through inhibitory connections. We then present the network with semi-realistic stimulus pairs, mimicking a 2-AFC task: one containing a complete figure and K random background elements, and one without a figure, consisting purely of random elements. When a figure is present, the units belonging to the corresponding subnetwork are externally driven and the avalanche statistics of this subpopulation is critical while the rest of the network remains subcritical. When no figure is present, critical dynamics are not observed. We take advantage of this knowledge to conceive a read-out mechanism based on detecting avalanches above a threshold size s_0 and quantify the 2-AFC task performance.

In our simulations, we observe robustness of feature integration against increasing numbers K of activated background units. For a fixed observation time interval T , we find an optimal threshold s_0 for maximizing detection performance. Furthermore, we compute phase diagrams delineating regions in parameter space where detecting coincidences performs better than using rate detection schemes, and vice versa. Moreover, the use of the EHE model allows to analyze the observed dynamics in a simplified setting with purely excitatory couplings. As can be expected, feature integration without inhibition is less stable, but the analytical treatment reveals closed-form expressions for avalanche distributions, thus enabling a comprehensive understanding of our numerical observations.

Acknowledgements

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[T 91] Signatures of optimal population coding of sensory stimuli through latent variables

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Several studies observe power-law statistics consistent with critical scaling exponents in neural data, but it is unclear whether such statistics necessarily imply criticality. In this work, we examine whether the $1/f$ statistics of retinal populations are inherited from visual stimuli, or whether they might emerge from collective neural dynamics independently of stimulus statistics. We examine, in silico, a latent-variable encoding model of visual scenes, and empirically explore the conditions under which such a model exhibits $1/f$ statistics thought to reflect criticality. Specifically, we examine the Restricted Boltzmann Machines (RBMs) as a factorized binary latent-variable model for stimulus encoding. We find two surprising results. First, latent variable models need not exhibit $1/f$ statistics, but that the optimal model size, reflecting the smallest model that can faithfully encode stimuli, does. We illustrate that the optimal model size can be predicted from sloppy dimensions of the Fisher information matrix (FIM), which align with a subspace spanning the superfluous latent variables. Second, the optimal-sized model can exhibit $1/f$ statistics even when stimuli do not, indicating that this property is not inherited from environmental statistics. Furthermore, such models exhibit properties of statistical criticality, including diverging susceptibilities. This empirical evidence suggests that $1/f$ statistics are neither inherited from the environment, nor a necessary feature of accurate encoding. Rather, it suggests that parsimonious latent-variable models are naturally poised close to criticality, generating the observed $1/f$ statistics. Overall, these results are consistent with conjectures in other fields that a cost-benefit trade-off between expressivity and parsimony underlies the emergence of criticality and $1/f$ power-law statistics. Furthermore, this work suggests that in latent-variable encoding models, the emergence of $1/f$ statistics reflects true criticality and is not inherited from the environmental distribution of stimuli.

Acknowledgements

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[T 92] **How does the retina anticipate the motion of complex shapes ?**

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The visual system uses motion anticipation to compensate the delays in retino-cortical transmission. Neurobiologists first believed that anticipation only happened in the visual cortex, but recent studies have shown that it starts earlier, in the retina. Berry & al. (1999) [1] and later Chen & al. (2013) [2] emphasized the role of gain control mechanisms in retinal anticipation. Berry's model is able to reproduce the anticipated response of retinal ganglion cells (RGCs) to a translating bar stimulus with RGC contrast gain control. Chen's model accounts for two supplementary motion features: alert response to (i) the appearance and (ii) to the motion onset of a bar. This is done with an additional contrast gain control at the level of bipolar cells and a pooling function. These models simulate independant RGCs whereas these cells are indirectly connected in the retina via amacrine cells. Furthermore, the presented stimuli studied by the authors are simple (moving bars). We want to understand how these mechanisms act on the anticipation of more complex shapes motion in a population of connected RGCs. For this we have developed a retina simulator, PRANAS (<https://pranas.inria.fr/>) emulating the retina spike response to a visual scene, with two layers mimicking the Outer Plexiform Layer (photoreceptors, horizontal and bipolar cells) and the Inner Plexiform Layer (connected ganglion cells). PRANAS also provides tools to analyze the statistics of the population spike response to a general visual scene. We first implemented in PRANAS the gain control mechanisms of Berry & al. [1] and Chen & al. [2]. This allowed us to reproduce responses to moving bars. Then we developed a threshold adaptation mechanism tuned by the bipolar current and enhanced by a pooling function at the level of RGCs, to make the neuron more sensitive to changes in the input. Our aim is to compare the results of our two implementations in reproducing the three motion features introduced earlier. Anticipation has been studied on a shape more complex than a bar, in order to assess the effect of adaptation on the form of the translating object. We developed an algorithm to reconstruct the stimulus from the spike trains produced by PRANAS (Fig 1). Chen & al model produces anticipation but slightly blurred images. Blur is reduced by threshold adaptation and pooling. We conclude with a method to evaluate the similarity between the input and the output showing that the second model performs better.

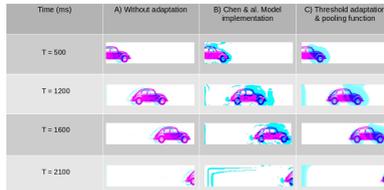


Figure 2: Reconstruction of a translating car stimulus from spike trains emitted by PRANAS. A) The simulation is done using linear spatio-temporal filtering at the level of bipolar cells and a discrete leaky integrate and fire model to produce spikes. B) The results of the implementation of Chen & al. Model (2) in PRANAS. There's a gain control implemented in the bipolar layer whereas the gain control at the level of the ganglion layer has been replaced by threshold adaptation as a function of the expected firing rate. C) The results using threshold adaptation as a function of θ_{bip} and the pooling of bipolar cells by ganglion cells.

Reconstruction of a translating car stimulus from spike trains emitted by PRANAS. The pink car represents the original stimulus and the blue signal the response of neurons.

Acknowledgements

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[T 93] Cortical state and natural movie responses in cat & mouse visual cortex

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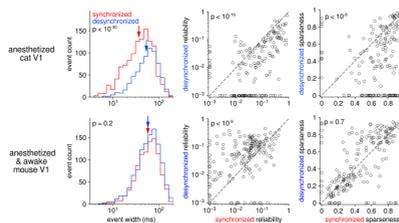
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Global brain activity changes spontaneously over time, ranging from slow synchronized activity to fast desynchronized activity [1]. This range of activity is similar in awake (quiescent vs. alert), asleep (slow-wave vs. REM) and anesthetized (deep vs. light) behavioural states, but is its effect on single unit firing patterns consistent across behavioural states and species? We recorded spikes and local field potential (LFP) from all layers of isoflurane-anesthetized cat primary visual cortex (V1), as well as isoflurane-anesthetized and awake mouse V1, while presenting 5 s long wide-field natural scene movie clips up to 400 times each. Spiking responses of single units to natural scene movie clips were remarkably precise, reliable and sparse, with lognormally distributed mean firing rates. Responses across trials exhibited distinct barcode-like events, some

as little as 10 ms wide in anesthetized cat and 20 ms wide in anesthetized and awake mouse, with most response events consisting of no more than one spike per trial.

Cortical state was quantified by the ratio of low (0.5-7 Hz) and high (15-100 Hz) frequency power of the LFP [2], and switched spontaneously between two extremes: the synchronized state (1/f distribution) and the desynchronized state (broadband distribution). Response precision was quantified as the temporal width of detected response events across trials. Single-unit response reliability was measured by binning single-trial responses into 20 ms wide bins at 1 ms resolution, and taking the mean pairwise correlation of binned trial responses across all trial pairs in a given state [3].

Surprisingly, responses in anesthetized cat V1 were more precise, reliable and sparse during the synchronized than desynchronized state. In contrast, in both anesthetized and awake mouse V1, responses were more reliable in the desynchronized state, but precision and sparseness did not differ between the two states (Figure 1). Greater reliability during the desynchronized state in mouse is consistent with several previous reports in rodents [3-7]. Our results therefore suggest a species-specific relationship between cortical state and the precision, reliability and sparseness of single unit responses. The presence of orientation maps in cat V1 may explain why the result in anesthetized cat differs from both anesthetized and awake rodents. This hypothesis predicts a similar result in V1 of other higher mammals, such as ferrets and primates.



Left: Response event widths in the synchronized and desynchronized state (Mann-Whitney U test). Arrows: geometric means. Middle: Scatter plots of response reliability in both states. Each point is a single unit (χ^2 test). Right: Scatter plots of response sparseness in both states (χ^2 test).

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[T 94] Phenomenological modeling of the electrically stimulated binaural auditory system

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Cochlear implants (CIs) restore hearing for profoundly deaf people by bypassing the degenerated inner hair cells and stimulating the auditory nerve fibers (ANFs) directly with electrical pulses. The responsiveness of the ANF to a particular electrical stimulus depends on several characteristics of the stimulus and of the ANF. Such information is vital for optimizing CI processing to improve CI users' ability to cope in complex auditory environments where interfering sounds and reverberation hinder listening to the target sound. However, the nerve's response can only be measured neurophysiologically which is not possible in humans.

Here, we present a functional model for the ANF response to pulse-train sequences and show how it can be applied, in conjunction with functional models for binaural-cue decoding (Takanen et al., 2014), to predict bilateral CI users' sensitivity to localization cues. The ANF model builds on the phenomenological biphasic leaky integrate-and-fire (BLIF) model (Horne et al., 2016), in which the ANF is thought to integrate incoming electrical current and to release an action potential if the membrane voltage exceeds a stochastic threshold and if the neuron is not hyperpolarized before it is ready to spike. Latency and jitter of the modeled ANF neuron depend on how greatly the threshold is exceeded. We have developed that model further by adding elements that simulate refractoriness and facilitation by affecting the threshold value of the model momentarily after supra- and subthreshold stimulation, respectively.

We show that the revised model can reproduce neurophysiological data considering refractoriness, facilitation, accommodation and spike-rate adaptation phenomena related to inter-pulse interactions that affect the responsiveness of the ANF to ongoing pulsatile stimulation. Outputs from the binaural CI model are shown to demonstrate pulse-rate dependency of just-noticeable differences for localization cues in electrical hearing. Consequently, the model offers a versatile instrumental tool for developers of CI coding strategies, providing accurate estimates of the responses that different stimulations evoke.

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[T 95] **Mixed latent variable model of attention in V1**

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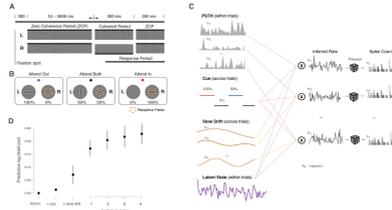
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Neurons show a high degree of variability of spike trains, even in responses to identical stimuli. This variability is often correlated between neurons of one population, however, the sources of the correlation remain unknown. According to one hypothesis, inter-trial fluctuation of an attentional signal can induce noise correlation [Cohen & Newsome 2008, Ecker et al. 2016]. To test this hypothesis in the primary visual cortex, we designed a novel cued change detection task in which attentional fluctuations are modulated across trials. We trained two monkeys to maintain fixation and to make a saccade toward coherent gratings among a series of two Gabor patches with randomly changing orientations presented simultaneously in the left and right visual field. The monkeys learned to attend either to the stimulus on one side or to both stimuli (Fig. 1 A, B).

To track the attentional state on a single-trial basis, we developed a model that multiplicatively accounts for the stimulus-driven variability of spikes and shared latent fluctuations of an attentional signal. The model describes the neuronal responses as a product of a stimulus response, attentional cue, slow drift, and shared latent variables (Fig. 1 C). The first two components are assumed to capture attentional modulation of the mean neuronal gain («classical» model of attention [Maunsell & Treue, 2006]). The slow modulator accounts for potential drift of individual neurons' firing rates throughout the recording session and is modeled by a Gaussian process across trials [Rabinowitz et al., 2015]. The shared attentional modulators are also assumed to be smooth, but with a faster timescale, and their within-trial dynamics are modeled by Gaussian Process Factor Analysis [Yu et al., 2009].

We trained the model on responses of V1 neurons in the change detection task. As expected, the gain of V1 neurons is increased by attention. We found that including shared latent variables improved predictive performance (Fig. 1 D) on held-out data compared with a model based on firing rates and attentional cue only. However, the improvement was small when including more than two latent variables. We are currently exploring properties of the learned latent components and how they relate to the animal's behavior. Overall, our model provides an interpretable account for the effects of spatial attention in V1 by learning the structure and timescales of fluctuations that affect shared neuronal variability.



A) Orientation change-detection task. B) Attention conditions in the task. C) Mixed latent variable model of a neuronal population (schematic). D) Average predictive log-likelihood of models with different components relative to a stimulus-driven model (\pm SEM).

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[T 96] Individual fitting of neural population latency distribution with a phenomenological auditory nerve fiber model for cochlear implant users

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The development of models fitted to individual cochlear implant (CI) patients can be used to individually optimize stimulation strategies with the aim to improve speech perception. In order to fit an individualized electrically evoked auditory nerve model, CIs can be used for measuring the nerve's compound activity in the vicinity of every channel-related neural cluster, resulting in a recorded response known as electrically evoked compound action potential (ECAP). Under the assumption that the ECAP is the compound discharge latency distribution of the whole population convolved with the unit response (single neuron in the auditory nerve) [1], latency distribution parameters can be obtained for an individual model using the measured data. In the present work, an individual model of temporal discharge behavior distribution was implemented using a novel phenomenological approach that models spike latency and jitter of the neural

response for a single pulse stimulation as a function of the stimulus amplitude [2]. The auditory nerve was represented by two populations of neurons extended along the cochlear length with different latency parameters related to peripheral and central areas. The stimulation weight was considered as a function of longitudinal distance between source electrode and nerve fiber, leading to a variation of input current amplitude along the neural populations. The compound discharge latency distribution was modeled as the combination of post stimulus time histograms obtained with the model output for both populations. The fitting procedure was based on the ECAP wave recording of selected electrode positions along the cochlear length at different stimulation levels.

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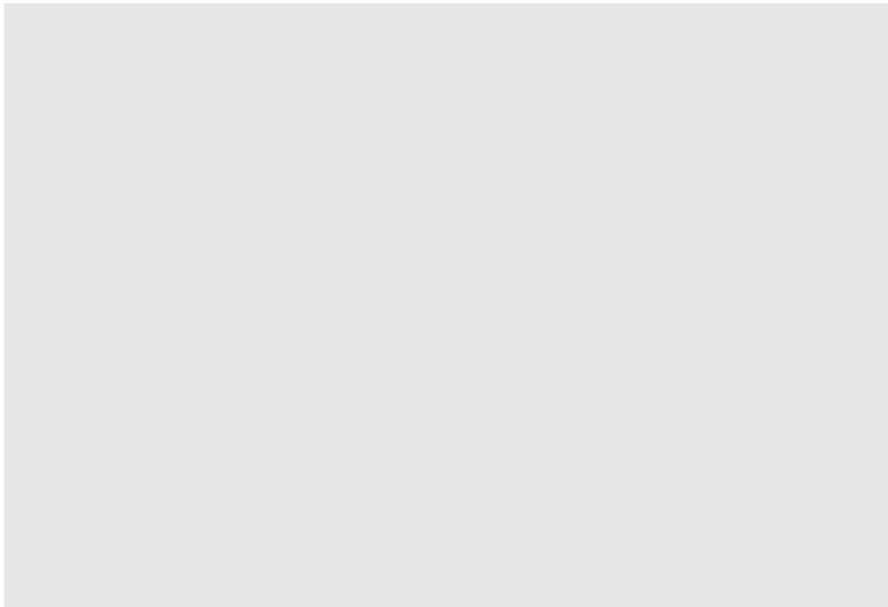
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[T 98] **Withdrawn**



[T 99] Neurons in macaque visual cortex encode information rhythmically

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The synchronization of activity across neurons has been a focus in many recent studies of information processing in networks of cortical cells and areas. One aspect of such inter-neuronal synchrony is the locking of spiking activity to local field potentials (LFPs). Such an interaction has been observed between the timing of individual action potentials ('spikes') of single neurons and the phase of low frequency (<15 Hz) oscillations of LFPs. However, the potential role of this phase-locking in neural encoding is unknown. To address this question, two behaving male macaque monkeys were trained to maintain their gaze on a central fixation point on a computer screen while two coherently moving random dot patterns (RDP) were simultaneously presented at eccentric locations, moving linearly in the same direction. One of the two RDPs was presented inside the receptive field of the recorded neuron and moved either in the neuron's preferred or anti-preferred direction LFPs and spikes were recorded from visual cortical area MT. To investigate if

phase-locking depends on the sensory properties of the visual stimulus, we measured the interconnection (locking) between spikes and the phase of low frequency LFP oscillations as a function of the stimulus' motion direction. We found that the phase-locking follows a tuning curve based on the presented stimulus' direction. This function is inverted compared to the tuning of the spike rate, i.e., the least spike-LFP coupling occurs for the preferred direction (based on the spike rate), while the strongest spike-LFP coupling is induced by the anti-preferred direction. This implies that those spikes added to a neuron's spike train in response to the preferred (rather than the anti-preferred) stimulus are inserted during LFP phases with a low spike rate, reducing the overall phase-locking. We tested this by comparing the neural discrimination calculated based on the spike-rate at the preferred vs. anti-preferred LFP phase. We found that the neural discrimination in the anti-preferred LFP phase is significantly larger than the preferred LFP phase. This suggests that neural information encoded in the spike rate varies with the LFP phase. Our data suggest that 1) the neural system harnesses spike-LFP coupling in the primate visual cortex to encode visual information and 2) the information coded by single neurons fluctuates relative to the surrounding LFP phase.

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Other

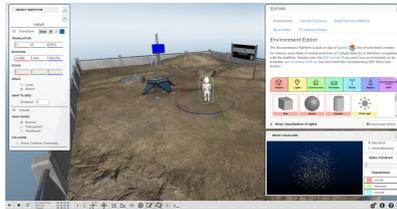
[T 100] **The Neurorobotics Platform: A simulation environment for brain-inspired robotics**

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The Neurorobotics Subproject of the Human Brain Project primarily develops the Neurorobotics Platform (NRP), which offers scientists all over the world the opportunity to connect brain models to robot bodies in various sensory-rich environments. To this end, the NRP consists of four essential components. The first component is a Robot Designer that allows users to design robot models that can be used in experiments. Robot models can be equipped with sensors and actuators or can also be biologically realistic musculoskeletal models. The Second component is an Environment Designer, where users can design their 3D environment models they want to run experiments in. Apart from the tools enabling users to design their own models, some template models of robots and environments are also provided with the NRP, which can allow rapid experiment prototyping. The third component is an Experiment Designer that offers users the capability of defining the experiment protocol by triggering certain events within an experiment. Events can be triggered when reaching a certain simulation time or when the robot is in a certain state for example. The fourth component is the Virtual Coach, a simple API that facilitates running batch experiments and modifying parameters between different runs. The Virtual Coach is especially useful when running

learning experiments. Additionally, the NRP offers an interface to easily couple neural networks to robots by connecting sensor readings to input neurons and neural outputs to robot actuators. These neurorobotics experiments are then executed in real-time on high performance supercomputing clusters through an easy to use web interface. Such infrastructure will facilitate running various types of experiments in multiple environments with different agents and will help us gain new insights into the causal relationships linking basic neural constituents to perception, cognition and behavior.



An experiment in the Neurorobotics Platform, where a Roboy robot model is loaded in the Space Bot Cup Arena. In the upper right corner, the Environment Designer can be seen. In the lower right corner, the Brain Visualizer can be seen, where users can visualize neurons spiking in real time.

Acknowledgements

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[T 101] Single Trial Visual Evoked Potential Estimation using Partial Least Squares Approach

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The nature of visual evoked potential (VEP) signals which is very low in power makes it difficult to be obtained except by gathering a series of time-locked electroencephalogram (EEG) epochs and performing ensemble averaging on these samples to improve the power. Averaging the EEG samples cannot reveal in details the variability across the trials. The variability across the trial helps in providing spatiotemporal resolution in the EEG-fMRI integration for example. In the BCI application, less trials helps in speeding its ITR. Thus, a plan based on single trial only VEP estimation that minimizes data loss and reduces recording time is highly desirable. In this work, a Partial Least Squares (PLS) based approach has been proposed to estimate VEP latencies from colored EEG noise. The proposed method uses the covariance matrices of the noisy signal, raw estimated signal and also the pre-stimulation EEG signal. The PLS is

used to describe the common structures of the covariance matrices by finding a pair of latent components which produce the maximum covariance. The proposed method then decomposes the corrupted VEP space into signal and noise subspace; VEP enhancement is achieved by removing the noise subspace and estimating the clean VEPs only from the signal subspace. Two subspace-based approaches for single-trial VEP latency estimation proposed by an author have also been evaluated and compared with PLS. Based on the comprehensively simulated data involving SNR from 0 to -10 dB indicate that the PLS schemes outperforms the TDE subspace approach but comparable in P100 estimation to GEVD subspace approach which prewhitens the input signal; although PLS is better in the estimation of P200 and N75 average error rate. PLS also has the narrowest standard deviation as compared to the other two subspace approaches. The results of forty seven real patient data further confirm the result in the simulated data. With the favourable performance demonstrated by the outcome of the simulated and real patient data, PLS estimators have the potentials to be used not only in the visual evoked potential signal estimator but also for other signal estimations from the brain where SNR values are relatively low.

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