# International Conference on Mathematical Neuroscience

# **ICMNS 2024**





**JUNE 11-15, 2024** University College Dublin

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### About

### International Conference on Mathematical Neuroscience

The International Conference on Mathematical Neuroscience (ICMNS) is an inter-disciplinary conference series, bringing together theoretical/computational neuroscientists and mathematicians. The conferences are aimed at scientists interested in using or developing mathematical techniques for neuroscience problems.

#### **Conference co-chairs**

Daniele Avitabile (Vrije Universiteit Amsterdam, Inria MathNeuro Team) Áine Byrne (University College Dublin) Jonathan Rubin (University of Pittsburgh)

#### Scientific committee

Andrea Barreiro	Sue Ann Campbell	Stephen Coombes
Olivier Faugeras	Grégory Faye	Richard Gast
Chengcheng Huang	Zachary Kilpatrick	Sukbin Lim
Cheng Ly	Ernest Montbrió	Patricia Reynaud-Bouret
Pierre Roux	Susanne Solem	Massimiliano Tamborrino
Romain Veltz	Douglas Zhou	

### Timetable

PT: Plenary Talk, IT: Invited Talk, CT: Contributed Talk

### Tuesday, 11th of June

Time	Mason Hayes Theatre (room L024)	William Fry Theatre (room L143)
12:00 - 13:00	<b>Registration</b> (Gardiner Atrium)	
13:00 - 13:10	Opening	
13:10 - 13:50	IT1 André Longtin First return time calculation for a Rayleigh process model of brain rhythm bursts	
13:50 - 14:30	IT2 Louis Pezon Linking neural manifolds and field models of network dynamics	
14:30 - 14:50	Coffee break (G	Gardiner Atrium)
	<b>Contributed Talks 1A</b> <i>Chair: André Longtin</i>	<b>Contributed Talks 1B</b> Chair: Louis Pezon
14:50 - 15:20	<b>CT1A-1 Carlos Coronel-Oliveros</b> Whole-brain modeling in health and disease: from neurodegeneration to brain aging	<b>CT1B-1 John Parker</b> Modeling an output nucleus integrating multiple inhibitory pathways
15:20 - 15:50	CT1A-2 Stephen Coombes	CT1B-2 <del>Francesca Cavallini</del> Daniele Avitabile
	Understanding the effect of white matter delays on large scale brain dynamics	Numerical Methods for Uncertainty Quantification in Spatiotemporal Models for the Neural Activity
15:50 - 16:20	<b>CT1A-3 Stefano Spaziani</b> Coupled model of brain rhythms and neuronal activity: theoretical and functional connectivity estimation	
16:30 - 18:30	Poster Session 1	(Gardiner Atrium)

### Wednesday, 12th of June

Time	Mason Hayes Theatre (room L024)	William Fry Theatre (room L143)
09:00 - 10:00	<b>PT1 Benjamin Lindner</b> Spiking neurons: How are their spontaneous fluctuations and their response to time-dependent stimuli related?	
10:00 - 10:40	<b>IT3 Songting Li</b> Mathematical mechanism of hierarchical timescales in the large-scale brain network	
10:40 - 11:00	Coffee break (Gardiner Atrium)	
	<b>Contributed Talks 2A</b> <i>Chair: Benjamin Lindner</i>	<b>Contributed Talks 2B</b> Chair: Songting Li
11:00 - 11:30	<b>CT2A-1 Massimilano Tamborrino</b> Estimation of connectivity structures of brain regions before and during during epileptic seizure	<b>CT2B-1 Anca Radulescu</b> Using complex dynamics to compute brain networks
11:30 - 12:00	CT2A-2 Maliha Ahmed Modelling pre-treatment ictal connectivity differences and the role of sex steroid hormones in childhood absence epilepsy	<b>CT2B-2 Sage Shaw</b> Radial Basis Function Methods for Neural Field Models
12:00 - 12:30	CT2A-3 Yuxiu Shao Identifying the impact of local connectivity features on network dynamics	<b>CT2B-3 Thibaud Taillefumier</b> Exact analysis of the subthreshold variability for conductance-based neuronal models with synchronous synaptic inputs
12:30 - 14:00	Lunch t	preak

Time	Mason Hayes Theatre (room L024)	William Fry Theatre (room L143)
	<b>Contributed Talks 3A</b> <i>Chair: Jonathan Rubin</i>	<b>Contributed Talks 3B</b> <i>Chair: Daniele Avitabile</i>
14:00 - 14:30	<b>CT3A-1 Lousiane Lemaire</b> Integrate-and-fire neurons with potassium dynamics that capture switches in neuronal excitability class and firing regime	<b>CT3B-1 Heather Cihak</b> Robust representations of certainty in a metastable bump attractor model
14:30 - 15:00	CT3A-2 Alla Borisyuk Effect of Astrocytes in Neuronal	CT3B-2 Cheng Ly Coding odor modality in precortical
15:00 - 15:30	CT3A-3 Emre Baspinar A neural field model for ignition and propagation of cortical spreading depression	CT3B-3 Marcella Noorman Maintaining and updating accurate internal representations of continuous variables with a handful of neurons.
15:30 - 15:50	Coffee break (Ga	ardiner Atrium)
15:50 - 16:30	<b>IT4 Gemma Huguet</b> Exploring Oscillatory Dynamics in Neural Networks: Insights for Effective Communication	
16:30 - 18:30	Poster Session 2 (	Gardiner Atrium)

### Thursday, 13th of June

Time	Mason Hayes Theatre (room L024)	William Fry Theatre (room L143)
09:00 - 10:00 10:00 - 10:40	<b>PT2 Kathryn Hess Bellwald</b> Topological perspectives on the connectome <b>IT5 Rodica Curtu</b> Emergence of modulated-wave-patterns of activity	
	from the intrinsic dynamics of neuronal mean field models	
10:40 - 11:00	Coffee break (	Gardiner Atrium)
	<b>Contributed Talks 4A</b> <i>Chair: Kathryn Hess</i>	<b>Contributed Talks 4B</b> <i>Chair: Steve Coombes</i>
11:00 - 11:30	<b>CT4A-1 Kaitlyn Toth</b> The Influence of Synaptic Plasticity on Critical Coupling Estimates for Neural Populations	<b>CT4B-1 Rachel Nicks</b> Insights into neural oscillator network dynamics using a phase-isostable framework
11:30 - 12:00	CT4A-2 Iris Yoon Topological tracing of encoded circular coordinates between neural populations	CT4B-2 Maxwell Kreider Q-phase reduction of multi-dimensional stochastic oscillator networks
12:00 - 12:30	<b>CT4A-3 Nikolas Schonsheck</b> Relative Neural Population Size Modulates Learnability of Cyclic Features of Neural Code	<b>CT4B-3 Siddharth Paliwal</b> Metastability in networks of nonlinear stochastic spiking neurons
12:30 - 14:00	Lunch	break
14:00 - 14:40	<b>IT6 Tilo Schwalger</b> Stochastic neural-mass models for populations of spiking neurons	
14:40 - 15:20	<b>IT7 Paul Bressloff</b> The mean field limit of the Kuramoto model with stochastic resetting	
15:20 - 15:40	Coffee break (	Gardiner Atrium)

Time	Mason Hayes Theatre (room L024)	William Fry Theatre (room L143)
	<b>Contributed Talks 5A</b> Chair: Tilo Schwalger	<b>Contributed Talks 5B</b> Chair: Paul Bressloff
15:40 - 16:10	<b>CT5A-1 Rainer Engelken</b> Understanding and Optimizing Learning in Recurrent Networks using Dynamical Systems Theory	<b>CT5B-1 Shoshana Chipman</b> The Effect of Noise Structure on Spatio-Temporal Pattern Formation and Chaos
16:10 - 16:40	<b>CT5A-2 Katie Morrison</b> Emergent dynamic attractors from recurrent network connectivity	<b>CT5B-2 Madeline Edwards</b> State modulation in spatial networks with three interneuron subtypes
16:40 - 17:10	<b>CT5A-3 Pascal Helson</b> Mean Field Analysis of a Stochastic STDP model	<b>CT5B-3 Paola Malerba</b> The space-time dynamics of sleep oscillations and its implications for cognition and health.
19:00 - 22:00	Social at Brev	vdog Outpost

### Friday, 14th of June

Time	Mason Hayes Theatre (room L024)	William Fry Theatre (room L143)
09:00 – 10:00 10:00 – 10:40	<ul> <li>PT3 Martin Wechselberger</li> <li>Neural dynamics and geometric singular perturbation theory beyond the standard form</li> <li>IT8 Taro Toyoizumi</li> <li>Chaotic neural dynamics facilitate probabilistic computations through sampling</li> </ul>	
10:40 - 11:00	0 – 11:00 <b>Coffee break</b> (Gardiner Atrium)	
	<b>Contributed Talks 6A</b> Chair: Martin Wechselberger	<b>Contributed Talks 6B</b> Chair: Taro Toyoizumi
11:00 - 11:30 11:30 - 12:00	CT6A-1 Olivier Faugeras Pros and cons of mean field representations of large size networks of spiking neurons CT6A-2 Pau Clusella	CT6B-1 Zachary Kilpatrick Phase transitions in decision dynamics depend on information reliability CT6B-2 Jyotika Bahuguna Catalina Vich
	Exact low-dimensional description for fast neural oscillations with low-firing rates	Three-factor cortico-striatal plasticity shifts activity of cortico-basal ganglia-thalamic subnetworks to maximize reward rate in decision-making tasks
12:00 – 12:30	<b>CT6A-3 Liang Chen</b> Collective Dynamics in the Heterogeneous Izhikevich Network with Synaptic Delay	CT6B-3 Andrea Barreiro Sensory input to cortex encoded on low-dimensional periphery-correlated subspaces
12:30 - 14:00	Lunch	break
14:00 - 14:40	<b>IT9 Victoria Booth</b> Mapping developmental changes in	

sleep-wake behavior

Time Mason Hayes Theatre (room L024)	William Fry Theatre (room L143)
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Contributed Talks 7A

Chair: Victoria Booth

- 14:40 15:10 **CT7A-1 Andrew Flynn** Seizure onset mechanisms beyond bifurcations: New perspectives on critical transitions and early warning signals.
- 15:10 15:40 **CT7A-2 Fabrizio Lombardi** Explaining the coexistence of neural oscillations and avalanches in resting human brain
- 15:40 16:10 **CT7A-3 Pascal Chossat** Analyzing sequential activity in neural networks from the dynamical systems perspective

**Contributed Talks 7B** *Chair: Zachary Kilpatrick* 

#### **CT7B-1 Richard Gast**

Dynamics and Computations in Networks of Heterogeneous Spiking Neurons

#### **CT7B-2 James MacLaurin**

Population Density Equations for Balanced Neural Networks

#### **CT7B-3** Mathieu Desroches

Observing hidden neuronal states in experiments

16:10 - 16:15 **Closing** 

### **Useful Information**

### Location

The conference will be held in the UCD Sutherland School of Law. The **registration desk** will be just inside the building entrance.

The **taks** will take place in the **Mason Hayes Theatre** and **William Fry Theatre**. The Mason Hayes Theatre is located on the ground floor of the Sutherland Building and the William Fry Theatre is located on the first floor.

Coffee breaks will be held on the the ground floor of the Sutherland Building.

The **poster session** will be held on Tuesday and Wednesday evening on the **ground floor** of the Sutherland Building.

Wi-Fi is available through the *eduroam* network and the UCD Wireless network.

#### How to get to UCD?

University College Dublin is situated in the south of Dublin, approximately 20-30 mins (via bus) from the city centre. The 39A will drop you on campus, while the 46A, 145 and 155 will drop you at the main entrance to campus (see maps below). You must have cash (exact change) or a Leap card to board these buses. Leap cards can be bought at the airport and in most convenience stores. See https://www.transportforireland.ie/fares/leap-card/ for more information on paying for public transport in Dublin.

From the airport, take the Aircoach 700 and disembark at *UCD (University College Dublin)*. Tickets can be purchased in advance at www.aircoach.ie/ or on board with card or cash. This journey takes roughly 60 mins via public transport or 30-40 mins in a taxi. Taxis are by the meter and you can expect to pay around €40.



### Social

The conference social will be held on Thursday evening from 7pm at Brewdog Outpost. **Address:** Three Locks Square, 4, Grand Canal Dock, Dublin 2, D02 E5R7. **Getting there:** Take the 47 bus (runs every 30 mins) from the same location as the 46A, 145 and 155 (marked on above map) Google maps directions

#### **Restaurants and cafes**

There are a range of different cafés and restaurants on campus. See this interactive map.

The most convenient options for lunch include:

- The Village FoodHall
- Confucius Chinese Restaurant
- University Club (you will need to show your badge at reception)
- Pi Restaurant

On Thursday, there will be food trucks on campus at this location.

#### Accommodation

Check in for on campus accommodation is from 3pm on the date of arrival. Early check-ins may be facilitated subject to availability but the UCD accommodation office is unable to confirm this prior to arrival. The check out is 11am on the date of departure. There is a luggage storage room where bags can be left prior to check-in and after check-out.

See above map for check-in location (pink circle). This is a 10-15 minute walk from the 46A, 145, 155 and Aircoach bus stops. Click here for the Google maps location.

For enquiries about accommodation please use the contacts below, mentioning the booking associated with the International Conference on Mathematical Neuroscience (reference number 777):

Email: stay@ucd.ie Phone: +353 1 716 7000

### **List of Posters**

### Tuesday, 11th of June

	Presenter	Title
1	Robert Allen	Phase-Isostable Reduction of Oscillatory Neural Mass Networks with Delays in local dynamics and network connections
2	Diego Becerra	Information integration ( $\Phi$ -ID) and high order interactions in Caenorhabditis elegans sleep-wakefulness neural dynamics
3	Oliver Cattell	Travelling fronts in a generalised neural field model that couples to the extracellular space
4	Jordan Culp	A Markovian neural barcode representing mesoscale cortical spatiotemporal dynamics
5	Michel Davydov	Propagation of chaos and Poisson Hypothesis for replica-mean-field models of intensity-based neural networks
6	Rodrigo Echeveste	Calibrated variational inference for low-level visual perception
7	Luc Falorsi	Population density dynamics of spiking neurons with absolute refractory period
8	Niamh Fennelly	Mean-field approximations for networks with synaptic plasticity
9	Camille Godin	Deciphering the Effects of Transcranial Electrical Stimulation with an Izhikevich Spiking Network
10	Ruaraí Goodfellow	Stability Analysis of the Coombes-Byrne Model
11	Maria Guasch-Morgades	Multiparameter optimization for whole-brain model personalization

	Presenter	Title
12	Ryosuke Hosaka	Liquid state machines with inhibitory synaptic STDP learning
13	Akke Mats Houben	Excitation-to-inhibition ratio determines pattern forming instability type in networks of pulse coupled phase oscillators
14	Henry Kerr	Analysing travelling waves and wave packets in laterally-inhibited grids of integrate-and-fire neurons
15	Fernando Lehue	Emergence and occupation of dynamical brain states in different states of consciousness using a biophysical model
16	Élia Lleal-Custey	Modeling plasticity for Neural Mass Models under tES
17	Pedro Maia	Terminal patterns: a data-driven analysis of the brain's last signals
18	Ana Mayora-Cebollero	Bifurcation and Arnol'd tongue structure of a next generation neural mass model: the effect of gap junction coupling
19	Carmen Mayora-Cebollero	Adaptive Coupling in Mean-Field Models of Neural Populations
20	Khadija Meddouni	Particle filters for neural fields
21	Nima Mirkhani	Mathematical model of coupled oscillators to predict the effects of phase-locked stimulation
22	Morten Gram Pedersen	Revisiting mixed-mode oscillations due to mutual inhibition in neuronal systems
23	Anca Radulescu	Dynamics of coupled Wilson-Cowan systems with distributed delays
24	Safura Rashid Shomali	Higher-order interactions reveal the hidden shared motifs across mouse brain regions
25	Lena Salfenmoser	Optimal control of oscillations and synchrony in nonlinear models of neural population dynamics
26	Natalie Schieferstein	Linear response of escape-noise models depending on hazard function
27	Oleg Senkevich	Stochastic gene expression drives correlated synaptic noise

	Presenter	Title
28	Ronja Strömsdörfer	The Wilson-Cowan neural field model with non-linear adaptation for traveling waves of slow oscillations
29	Hélène Todd	The role of gap junctions and clustered connectivity in emergent synchronisation patterns of spiking inhibitory neuronal networks
30	Hannah van Susteren	A biophysical model of neuronal dynamics and synaptic transmission during energy deprivation
31	Xiaoqi Xu	Analysis on EEG spatiotemporal patterns via Laplacian eigenmodes
32	Viktoria Zemliak	Continual familiarity detection in spiking neural networks

### Wednesday, 12th of June

	Presenter	Title
1	Daniele Andrean	The Schwann cell hug: mere support or active player in the neuromuscular junction's efficiency?
2	Logan Becker	Full moment and co-variability analysis of conductance-based neuron models
3	Rebecca Brady	Recurrent Cortical Detection Models to Assess Multisensory Mechanisms in an Audio-Visual Reaction-Time Task
4	John Butler	Mathematical Modelling of Visual-Vestibular Neuronal Processing and Behavioural Responses
5	Jennifer Crodelle	A functional role for stage II retinal waves on the receptive field development of neurons in primary visual cortex
6	Farzaneh Darki	Touch stimulation to enhance separation of sound sources
7	Rosa Maria Delicado Moll	Excitatory and Inhibitory synaptic conductances. How can they be estimated?

	Presenter	Title
8	Jérôme Emonet	A Retino-cortical model of anticipation
9	Matthew Fellows	Reduced descriptions of oscillatory neural field models via higher order phase interaction functions
10	Richard Foster	Modeling human temporal EEG responses to VR visual stimuli
11	Rainer Engelke	A time-resolved theory of chaos suppression and information encoding in recurrent neural networks
12	Nils Erik Greven	Mesoscopic dynamics of spiking neural networks with stochastic rewiring
13	Devika Khurana	First-passage times of SDEs to curved boundaries
14	Pedro Lima	Existence results and estimates for neural fields with diffusion
15	Matteo Martin	Analysis of Mixed-Mode Oscillations in Cortical Neurons: the role of M and HCN channels
16	Victor Matveev	Effects of Ca2+ buffers with multiple binding sites on Ca2+ signals and synaptic facilitation
17	James McAllister	Heterosynaptic plasticity rules induce small-world topologies
18	Hil Meijer	On choosing activation functions for neural fields
19	Diego Pazó	Discontinuous transition to chaos in a canonical random neural network
20	Alina Podschun	Does the rich club control brain state transitions? A network control theoretical investigation
21	Georg Reich	Temporally Asymmetric Hebbian Learning is All You Need for Forward and Reverse Replay
22	Flavio R. Rusch	Topology Influence on the Critical Behavior of Modular Hierarchical Networks of Stochastic Neurons
23	Maria Luisa Saggio	Bifurcations and bursting in the Epileptor
24	Kazuya Sawada	Detecting causality between neural spike trains by mutual prediction of inter-spike intervals

	Presenter	Title
25	Helmut Schmidt	The effect of frequency filtering and noise on functional brain connectivity and its relation to brain structure
26	Andrew Shannon	Next Generation Neural Mass Models Reproduce Features of Neural Speech Processing.
27	Brian Skelly	A mean field model for beta bursts and non-averaged neural data
28	Anna Thomas	Computational model investigating the role of rodent substantia nigra pars reticulata projections to pedunculopontine nucleus in long-term motor recovery in Parkinsonian rodents
29	Ka Nap Tse	Understanding Neuronal Clustering through Evolution of Adaptation Distribution
30	Yota Tsukamoto	Outstanding pattern discrimination ability of spatiotemporal learning rule
31	Pablo Vizcaíno García	Validating the use of simplified models for studying gap junction coupling in the brain
32	Marius Yamakou	Diversity-Induced Decoherence in a Slow-Fast Neuron Model

### Abstracts – Talks

#### Tuesday, 11th of June

### First return time calculation for a Rayleigh process model of brain rhythm bursts

M. Roman<sup>1</sup>, I. L'Heureux<sup>1</sup>, A. Powanwe<sup>1</sup>, A. Longtin<sup>1</sup>

<sup>1</sup> University of Ottawa

Local field potential (LFP) data display strong oscillatory "bursts". These epochs have random onset times and durations, likely due to synaptic noise in excitatory and inhibitory populations. Linearization of the relevant stochastic Wilson-Cowan system yields a 2D Ornstein-Uhlenbeck process. The problem then reduces to understanding epochs when the amplitude (radius) of this process crosses and returns to a threshold. It simplifies further by transforming the 2D OU process into a 1D amplitude Rayleigh process. Fokker-Planck analysis of the first return time (FRT) for any stochastic process to come back to its starting point generates a non-normalizable probability distribution. We apply the trick of Artime et al. 2018 to find an analytical expression for the FRT distribution of the Rayleigh process. Analytical expressions matched simulations very well for FRTs, for motion either below and above the threshold. The work is also relevant to criticality studies, assuming white noise drives avalanches.

#### Linking neural manifolds and field models of network dynamics

#### <u>L. Pezon<sup>1</sup></u>, V. Schmutz<sup>2</sup>, W. Gerstner<sup>1</sup>

<sup>1</sup> Ecole Polytechnique Federale de Lausanne
 <sup>2</sup> University College London

While the analysis of large-scale neural recordings indicates that the activity of heterogeneous neuronal populations lies on low-dimensional "neural manifolds", this picture is hard to reconcile with the classical view of precisely tuned neurons interacting with each other in some ordered circuit structure, eventually captured by field models. We provide a conceptual link between these two contrasting views, by introducing a field model interpretation of low-rank recurrent neural networks. We first show that there is no unique relationship between the circuit structure and the emergent low-dimensional dynamics that characterise the population activity. We then propose a method for retrieving the circuit structure from recordings of the population activity. Our approach provides not only a unifying framework for circuit and field models on one side, and low-rank networks on the other side, but also opens the perspective to identify principles of circuit structure from large-scale recordings.

### Whole-brain modeling in health and disease: from neurodegeneration to brain aging

<u>C. Coronel-Oliveros</u><sup>1,2,3</sup>, R. Gonzalez-Gomez<sup>1,4</sup>, K. Ranasinghe<sup>2</sup>, A. Sainz-Ballesteros<sup>1</sup>, A. Legaz<sup>7</sup>, S. Fittipaldi<sup>1,2,7</sup>, J. Cruzat<sup>1</sup>, R. Herzog<sup>1</sup>, G. Yener<sup>5,6</sup>, M. Parra<sup>8</sup>, D. Aguillon<sup>9</sup>, F. Lopera<sup>9</sup>, H. Santamaria-Garcia<sup>10,11</sup>, S. Moguilner<sup>1,7</sup>, V Medel<sup>1,12,13</sup>, P. Orio<sup>3,12</sup>, R. Whelan<sup>2</sup>, E. Tagliazucchi<sup>1,14</sup>, P. Prado<sup>1,15</sup>, A. Ibanez<sup>1,2,8,16</sup>

- <sup>1</sup> Universidad Adolfo Ibáñez
- <sup>2</sup> University of California San Francisco
- <sup>3</sup> Universidad de Valparaíso
- <sup>4</sup> Universidad Adolfo Ibáñe
- <sup>5</sup> Izmir University of Economics
- <sup>6</sup> Dokuz Eylül University
- <sup>7</sup> Universidad de San Andrés
- <sup>7</sup> University of Strathclyde
- <sup>9</sup> University of Antioquia
- <sup>10</sup> Pontificia Universidad Javeriana
- <sup>11</sup> Hospital Universitario San Ignacio
- <sup>12</sup> University of Sydney
- <sup>13</sup> Universidad de Chile
- <sup>14</sup> University of Buenos Aires
- <sup>15</sup> Universidad San Sebastián
- <sup>16</sup> Trinity College Dublin

Alzheimer's disease (AD) and behavioral variant frontotemporal dementia (bvFTD) lack well-understood characterization in non-stereotypical and underrepresented populations. Electroencephalography (EEG) is a high-resolution, cost-effective technique for studying dementia globally, but lacks mechanistic models and produces non-replicable results. A promising solution in the field is the use of whole-brain models. These computational models can be used to test mechanistic hypotheses ascribed to neurodegeneration. We developed a novel approach using whole-brain modeling with high-order functional connectivity, anatomical priors, and a perturbational approach to investigate brain dynamics related to dementia in the Global South. We tested two possible mechanisms ascribed to neurodegeneration dementia: structural connectivity disintegration and E/I balance alterations. As a step forward, we used the same model and mechanisms to generate normative models of healthy brain aging, utilizing EEG source data from participants of Global North and South. The results provide a novel agenda for developing diagnostic methods and model-inspired therapies.

# Understanding the effect of white matter delays on large scale brain dynamics

#### <u>S. Coombes<sup>1</sup></u>, H.G.E. Meijer<sup>2</sup>

<sup>1</sup> University of Nottingham,
 <sup>2</sup> University of Twent

We present a new set of mathematical tools to help unravel the contributions of spacedependent axonal delays to large-scale spatiotemporal patterning of brain activity. We first analyse a single neuronal population Wilson-Cowan neural mass model with self-feedback and a fixed delay and show how to construct periodic orbits for a Heaviside firing rate. We perform linear stability analysis by augmenting Floquet theory with saltation operations. We then show how to treat the synchronous oscillatory state in networks of nonsmooth neural masses with multiple and heterogeneous delays. A complementary numerical approach utilising the harmonic balance method is also developed for smooth sigmoidal firing rates. To augment this advance in understanding of how network states synchronise and destabilise to more novel functional connectivity patterns, we also present direct numerical simulations. Finally, we discuss state-dependent delays and present preliminary results for a new form of biologically motivated white matter plasticity rule.

# Coupled model of brain rhythms and neuronal activity: Theoretical results and functional connectivity estimation

#### S. Spaziani<sup>1</sup>, T. Leblanc<sup>2</sup>

<sup>1</sup> Université Côte d'Azur

<sup>2</sup> École Normale Supérieure

In neuroscience, functional connectivity can be seen as an ensemble of interactions among local field potential (LFP) rhythms and individual neuronal activity. The set of active interactions in a particular time interval can be associated with the cognitive state and helps understand high-cognitive processes such as learning.

In this talk, we introduce a method to assess at once directed interactions from spike-LFP multi-frequency brain data.

The framework is built from a new type of stochastic process that describes the coupled interactions between the oscillatory activity of the LFP and the spiking activity of the neurons. These equations allow to model a wide range of known biological phenomena.

In order to create a robust statistical functional connectivity estimator we prove some theoretical properties of the process, such as stationarity conditions and concentration inequalities.

We validate and employ this procedure to recover heterogeneous spike-LFP functional connectivity graphs from simulated and real data.

#### Linking neural manifolds and field models of network dynamics

<u>*T. Taillefumier*<sup>1</sup></u>, *L. A. Becker*<sup>1</sup>, *B. Li*<sup>1</sup>, *N. J. Priebe*<sup>1</sup>, *E. Seidmann*<sup>1</sup>

<sup>1</sup> The University of Texas at Austin

The spiking activity of cortical neurons exhibits a striking level of variability, even in sensory pathways responding to identical stimuli. This has led to the proposal that neural networks operate in an asynchronous state, where neurons receive independent synaptic inputs. However, recent experimental measurements indicate that the membrane potential of neurons is also highly variable. It is unclear whether asynchronous networks can satisfactorily account for this form of subthreshold variability. We propose a new analytical framework to rigorously quantify the subthreshold variability of a single conductance-based neuron in response to synaptic inputs. Our formulation allows us to examine both asynchronous networks as well as those with prescribed degrees of synchrony. We find that achieving realistic levels of subthreshold membrane potential variability in an analytically tractable, biophysically relevant, neuronal model necessitates synaptic input synchrony in amounts compatible with the weak but nonzero observed spiking correlations.

#### Modeling an output nucleus integrating multiple inhibitory pathways

#### <u>J. Parker<sup>1</sup></u>, A. Aristieta<sup>2</sup>, A. H. Gittis<sup>2</sup>, J. E. Rubin<sup>1</sup>

<sup>1</sup> University of Pittsburgh

<sup>2</sup> Carneige Mellon University

Study of the basal ganglia (BG) commonly focuses on the interactions of competing pathways thought to gate actions and to become imbalanced in pathologies such as Parkinson's disease (PD). This investigation focuses specifically on the substantia nigra pars reticulata (SNr), a key BG output nucleus, and its responses to optogenetic stimulation of the globus pallidus (GPe) and striatum (STR) in murine models with reduced dopamine levels, akin to PD. Traditionally, GPe and STR inputs to SNr are considered as inhibitory, but in recent studies they induced a surprisingly diverse spectrum of responses in the SNr. We emulate in-vivo firing patterns and responses through incorporation of stochastic processes and cellular diversity into a biophysical conductance-based model of a network of SNr neurons. Through this approach, we refine our understanding of how SNr integrates GPe and STR inputs and determine key factors governing its activity across physiological and pathological contexts.

#### Observing hidden neuronal states in experiments

#### D. Amakhin<sup>1</sup>, A. Chizhov<sup>2</sup>, G. Girier<sup>3</sup>, <u>M. Desroches<sup>2</sup></u>, J. Sieber<sup>4</sup>, S. Rodrigues<sup>3,5</sup>

<sup>1</sup> Sechenov Institute of Evolutionary Physiology and Biochemistry of RAS

<sup>2</sup> Inria Centre Université Côte d'Azur

<sup>3</sup> Basque Center for Applied Mathematics

<sup>4</sup> University of Exeter

<sup>5</sup> Ikerbasque, the Basque Foundation for Science

In this talk, I will present recent work in which we have constructed systematically experimental steady-state bifurcation diagrams for type-I neurones. A slowly ramped voltage-clamp electrophysiology protocol serves as closed-loop feedback controlled experiment for the subsequent current-clamp open-loop protocol on the same cell. In this way, the voltage-clamped experiment determines dynamically stable and unstable (hidden) steady states of the current-clamp experiment. The transitions between observable steady states and observable spiking states in the current-clamp experiment reveal stability and bifurcations of the steady states, completing an  $\epsilon$ -approximation of the steady-state bifurcation diagram ( $\epsilon$  being the speed of the ramp). Furthermore, combining the output of the current-clamp protocol together with that of the voltage-clamp protocol gives an experimental verification of the slow-fast dissection method by J. Rinzel. I will explain how to understand why this is possible, using elements of slow-fast dynamical systems theory.

### Wednesday, 12th of June

# Spiking neurons: How are their spontaneous fluctuations and their response to time-dependent stimuli related?

#### **B. Lindner**<sup>1</sup>

<sup>1</sup> Bernstein Center for Computational Neuroscience, Berlin

Neurons often exhibit considerable fluctuations in their spontaneous (stimulus-free) activity, characterized by correlation functions. Neurons also react to time-dependent stimuli described by response functions. Because the neural raison d'etre is information processing and transmission (shaped by both their fluctuation and response properties), one might wonder whether the two aspects (correlation and response functions) are related and if so how? In statistical physics such relations have been studied under the label of fluctuation-dissipation theorems. In neuroscience fluctuation-response relations (FRR) for spiking neurons have been discovered only recently. I review my own results on FRRs for general integrate-and-fire models that include spike-frequency adaptation, a refractory period, a colored Gaussian or a Poissonian shot noise. Several applications of FRRs are considered: i) calculation of so-far unknown correlation statistics, ii) the extraction of otherwise inaccessible noise statistics, and ii) identification of constraints for the response function of neurons in a recurrent network.

## Mathematical mechanism of hierarchical timescales in the large-scale brain network

#### <u>S. Li</u>¹

#### <sup>1</sup> Shanghai Jiao Tong University

In the brain, while early sensory areas encode and process external inputs rapidly, higherassociation areas are endowed with slow dynamics to benefit information accumulation over time. Such a hierarchy of temporal response windows along the cortical hierarchy naturally emerges in an anatomically based model of primate cortex. The emergent property raises the question of why diverse temporal modes are well segregated rather than being mixed up across the cortex, despite high connection density and an abundance of feedback loops. In this talk, we will address this question by mathematically analyzing a large-scale brain network model and identifying crucial conditions of synaptic excitation and inhibition that give rise to timescale segregation in a hierarchy. In addition, we will discuss the mathematical relation between timescales segregation and signal propagation in the brain.

### Estimation of connectivity structures of brain regions before and during during epileptic seizure

S. Ditlevsen<sup>1</sup>, <u>M. Tamborrino<sup>2</sup></u>, I. Tubikanec<sup>3</sup>

<sup>1</sup> University of Copenhagen

- <sup>2</sup> University of Warwick
- <sup>3</sup> University of Klagenfurt

We aim to infer the connectivity structures of brain regions before and during epileptic seizure. First, we propose a 6N-dimensional stochastic differential equation for modelling the activity of N coupled populations of neurons in the brain, further developing the (single population) stochastic Jansen and Rit neural mass model. Second, we construct a reliable and efficient numerical splitting scheme for its simulation. Third, we propose an adapted Sequential Monte Carlo Approximate Bayesian Computation algorithm for inferring all parameters, in particular those 0,1-valued describing the coupling directions among the N modelled neural populations. Fourth, after illustrating and validating the statistical approach on simulated data, we apply it to multi-channel EEG data recorded before and during an epileptic seizure. The real data experiments suggest, for example, a larger activation in each neural population and a stronger connectivity on the left brain hemisphere during seizure. (Joint work with Susanne Ditlevsen and Irene Tubikanec)

#### Modelling pre-treatment ictal connectivity differences in the thalamocortical circuit associated with childhood absence epilepsy

#### <u>M. Ahmed<sup>1</sup></u>, S. A. Campbell<sup>1</sup>

#### <sup>1</sup> University of Waterloo

Childhood absence epilepsy (CAE) is a paediatric idiopathic generalized epilepsy disorder with a confounding ability to spontaneously resolve in adolescence in a majority of cases. There have been several factors hypothesized to be mechanisms behind remission, however, there remains an inadequate understanding of some of these factors. According to some functional connectivity studies, there exist pre-treatment connectivity differences between patients who ultimately experience remission and those who do not. In this study, we have built a conductance-based model of the thalamocortical circuit consisting of multiple types of single-compartment thalamic and deep layer cortical neurons corresponding to different parts of the cortex. In particular, our model improves on previous well-established models in the literature by incorporating a more comprehensive cortical component. Through our results we aim to gain an understanding of the role of different neuron types, and differences in synaptic connectivity towards generating distinct networks modelling remitting and non-remitting behaviour.

#### Identifying the impact of local connectivity features on network dynamics

#### <u>Y. Shao<sup>1,2</sup></u>, S. Recanatesi<sup>3</sup>, D. Dahmen<sup>4</sup>, E. Shea-Brown<sup>5</sup>, S. Ostojic<sup>2</sup>

- <sup>1</sup> Beijing Normal University
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- <sup>3</sup> Technion Israel Institute of Technology
- <sup>4</sup> Research Centre Julich
- <sup>5</sup> University of Washington

Understanding how connectivity structure shapes network dynamics is paramount in the field of neuroscience. A newly released synaptic physiology dataset highlighted the strong presence of motifs – specific connectivity patterns between pairs and triplets of neurons–beyond the scope of mean connectivity. However, it is a priori not clear which of the experimentally identified connectivity motifs exert a strong influence on neural dynamics. Here we show that chain motifs have a strong impact on the dynamics of neural activity. Our results show that an overrepresentation of chain motifs induces an additional eigenmode with an eigenvalue of sign opposite to the dominant one, thus modifying the network's effective rank. This additional eigenmode substantially influences network dynamics, offering a new perspective on how local El motifs shape the network's excitability.

#### Using complex dynamics to compute brain networks

#### <u>A. Radulescu<sup>1</sup></u>, S. Muldoon<sup>2</sup>, J. Nakuci<sup>3</sup>, S. Evans<sup>4</sup>, A. Augustin<sup>5</sup>, A. Cooper<sup>1</sup>

- <sup>1</sup> State University of New York at New Paltz
- $^2$  University of Buffalo
- <sup>3</sup> Army Research Labs
- $^{\rm 4}$  Dartmouth College
- <sup>5</sup> Fordham University

Global behavior in dynamic networks emerges from the interplay between the network's connectivity profile and the node-wise dynamics. We study this interplay with a novel modeling approach: we equip each of the network nodes with discrete quadratic dynamics in the complex plane, and we study the behavior of the resulting complex quadratic network (CQN). We combine methods from complex dynamics and graph theory to tie patterns in network architecture to the topology of the network equi-M set (an extension of the traditional Mandelbrot set). We then visit outstanding questions in computational neuroscience and address them in our canonical CQN framework. We finally illustrate how the topology of asymptotic sets can provide valuable means of classification of both brain connectivity and functional dynamics, when applied to human subject connectomes (as provided by the Human Connectome Project).

#### **Radial Basis Function Methods for Neural Field Models**

<u>S. Shaw<sup>1</sup></u>, D. Avitable<sup>2,3</sup>, Z. P. Kilpatrick<sup>1</sup>

<sup>1</sup> University of Colorado Boulder
 <sup>2</sup> Vrije Universiteit Amsterdam
 <sup>3</sup> Inria Centre at Universite C<sup>o</sup>ote d'Azur

Neural field models are non-linear systems of integro-differential equations intended to model large-scale neural activity. There is growing interest in identifying efficient and accurate schemes for simulating neural field models as they can capture activity dynamics that spread across wide swathes of tissues and that reflect highly complex neural architecture. Recently, a framework has been put forth for analyzing neural field solvers (Avitable 2023) that separates the error due to the numerical representation of the solution (projection) and the error due to approximating the integral operator (quadrature). In this talk, we will discuss using Radial Basis Function (RBF) interpolation and quadrature methods to combine and simplify this error analysis and to create efficient, robust, and high-order-accurate neural field solvers. We will demonstrate their utility in solving neural fields over 2D manifolds and discuss their application to modeling cortical spreading depression.

#### Quantifying the Uncertainty in Spatiotemporal Models of Neural Activity: Forward and Inverse problems, and their numerical solutions

F. Cavallini<sup>1</sup>, <u>D. Avitabile<sup>1</sup></u>, S. Dubinkina<sup>1</sup>, G. Lord<sup>2</sup>

<sup>1</sup> Vrije Universiteit Amsterdam
 <sup>2</sup> Radboud University

While uncertainty is a crucial component in neurodynamics, its quantification in computational and mathematical models of neural activity is still in its infancy.

The talk lays the foundations of uncertainty quantification in nonlocal, nonlinear neurobiological models, by developing a framework similar to the one for PDEs with random input data.

After discussing a functional and probabilistic setup for the Wilson-Cowan model, we address the Forward Problem, and study how the model solution propagates uncertainty in random inputs (synaptic kernel, firing rate, initial condition, and external input).

Specifically, we present proofs and numerics illustrating how the proposed Stochastic Collocation scheme 1) converges exponentially with the number of interpolation points, under regularity assumptions on the parametric inputs; and 2) is sensitive to the dimension of the parameter vector, and to the support of its probability density function. Time permitting, we discuss preliminary findings on the numerical solution to the Inverse Bayesian Problem.

# Integrate-and-fire neurons with potassium dynamics that capture switches in neuronal excitability class and firing regime

#### <u>L. Lemaire<sup>1</sup></u>, M. Behbood<sup>1</sup>, J. Schleimer<sup>1</sup>, S. Schreiber<sup>1</sup>

<sup>1</sup> Humboldt-Universität zu Berlin

In conductance-based models, spiking-induced ion concentrations fluctuations can modify single neurons' excitability. What are the consequences in networks? To study this, simple models capturing ion concentration dynamics realistically are needed. We propose a method to derive a phenomenological model capturing the coupled extracellular potassium and voltage dynamics from a given class 1 conductance-based model.

Rather than fitting voltage traces, we fit the bifurcation structure of the target model, thereby capturing parameter heterogeneity and rich dynamics. The resulting model extends the quadratic integrate-and-fire model, with extracellular potassium accumulation altering voltage dynamics by increasing the reset voltage.

We apply our systematic reduction procedure to the Wang-Buzsáki model. Its phenomenological version exhibits quantitatively comparable dynamics and replicates the reshaping of the phase response curve associated with the transition from SNIC to HOM spikes at elevated potassium. To illustrate the derived model's applicability, we explore how changes in potassium concentration influence synchronization in networks.

#### Effect of Astrocytes in Neuronal Networks

#### A. Borisyuk<sup>1</sup>, G. Handy<sup>2</sup>, A. Liu<sup>1</sup>, C. Johnson<sup>1</sup>

<sup>1</sup> University of Utah

<sup>2</sup> University of Minnesota

Astrocytes are glial cells playing multiple important roles in the brain, e.g. control of synaptic transmission. We are developing tools to include "effective" astrocytes in neuronal network models in an easy-to-implement computationally-efficient way. In our approach we first consider neuron-astrocyte interaction at fine spatial scale, and then extract essential ways in which the network is influenced by the presence of the astrocytes. For example, by using a DiRT (Diffusion with Recharging Traps) model we find that a synapse tightly ensheathed by an astrocyte makes neuronal connection faster, weaker, and less reliable, and subsequently astrocytes can push the network to synchrony and to exhibiting strong spatial patterns, possibly contributing to epileptic disorder. Further, the calcium signals in the astrocyte initiate a loop of calcium-sodium-potassium trans-membrane activity. This activity modulates the extracellular concentrations of these ions and, consequently, the excitability of the nearby postsynaptic cell, further modifying the neuronal network activity.

## A neural field model for ignition and propagation of cortical spreading depression

## <u>E. Baspinar</u><sup>1</sup>, M. Simonti<sup>2,3</sup>, H. Srour<sup>1</sup>, D. Avitabile<sup>4,5</sup>, M. Desroches<sup>3</sup>, M. Mantegazza<sup>2,3,6</sup>

<sup>1</sup> Paris-Saclay University

- <sup>2</sup> Université Cote d'Azur, Institute of Molecular and Cellular Pharmacology (IPMC)
- <sup>3</sup> CNRS, Institute of Molecular and Cellular Pharmacology (IPMC)
- <sup>5</sup> Vrije Universiteit Amsterdam
- <sup>5</sup> Inria Center at Université Cote d'Azur
- <sup>6</sup> INSERM, France.

Cortical spreading depression (CSD) is a wave of neuronal depolarization that slowly spreads across the cortex. It is followed by a neuronal silence that may last for several minutes. Recent electrophysiological recordings demonstrated that CSD was associated with migraine aura. So far, modeling CSD dynamics has concentrated only on the ignition phase. To complement this, we propose a neuronal population framework which models the CSD dynamics of both ignition and propagation phases. The model is based on an excitatory-inhibitory neuronal population pair which is coupled to a potassium concentration variable. It is spatially extended to a cortical layer patch, generalizing the ignition dynamics to the propagation dynamics. The novelty is that the model takes into account the ionic modulation of the neuronal transfer functions. The model simulations has been confirmed by biological experiments.

### Robust representations of certainty in a metastable bump attractor model

#### <u>H. L. Cihak<sup>1</sup></u>, Z. P. Kilpatrick<sup>1</sup>

#### <sup>1</sup> University of Colorado Boulder

Persistent neural activity in the cortex can represent estimates of parametric variables during delays of working memory tasks and data show that stimulus presented for longer periods results in higher amplitude activity and lower response errors. Organizing activity patterns according to cell feature preference reveals activity "bumps" that stochastically wander in ways that predict errors in delayed estimates. Continuum neural field models support such solutions and we extend them to consider a staircase-shaped firing rate function to achieve a discrete gradation of possible stable activity levels in response to stimulus features. Using nonlinear asymptotics, we derive equations of the bump position and amplitude as perturbed by inputs and noise, as well as predict position variance (response error) and mean transition time between states. Indeed, higher amplitude bumps are more robust to perturbations, mimicking behavioral data. Currently, we work to include short term plasticity to examine its role in distractor resilience.

#### Coding odor modality in precortical and cortical regions

C. Ly<sup>1</sup>, M. Craft<sup>2</sup>, A. K. Barreiro<sup>3</sup>, S. H. Gautam<sup>4</sup>, W. L. Shew<sup>4</sup>

<sup>1</sup> Virginia Commonwealth University

<sup>2</sup> The Federal Reserve Bank of Richmond

<sup>3</sup> Southern Methodist University

<sup>4</sup> University of Arkansas

How are sensory signals decoded from noisy cortical activity? We study this question in olfaction where there are two modes of sensing: from the front (sniffing) or rear (eating/exhaling), i.e., ortho and retro, respectively. We recently showed that ortho versus retro information (modality) is encoded in the olfactory bulb (OB, a precortical region). But can modality information be extracted in olfactory piriform cortex (aPC)? With simultaneous recordings of in OB and aPC in anesthetized rats, we show that a recently developed unsupervised and biologically plausible algorithm based on CCA can extract odor modality from aPC spiking. Consistent with our theory, when OB encodes sensory information with low noise correlation between OB+aPC, modality in aPC is encoded at optimal limits in low-dimensional subspaces. Also, this algorithm can indeed improve encoding of ortho/retro in aPC in pairs of cells compared to standard methods (PCA with LDA).

#### Maintaining and updating accurate internal representations of continuous variables with a handful of neurons

#### <u>M. Noorman<sup>1</sup></u>, B. K. Hulse<sup>1</sup>, V. Jayaraman<sup>1</sup>, S. Romani<sup>1</sup>, A. M. Hermundstad<sup>1</sup>

<sup>1</sup> Janelia Research Campus

Many animals rely on persistent internal representations of continuous angular variables for working memory, motor control, and navigation. Theories have proposed that such representations are maintained by a class of recurrently connected networks called ring attractor (RA) networks. These networks rely on large numbers of neurons to maintain continuous and stable representations and to accurately integrate incoming signals. The head direction system of the fruit fly, however, seems to achieve these properties with a remarkably small network. These findings challenge our understanding of RAs and their putative implementation in neural circuits. By constructing and analyzing an energy function for this small system, we analytically determine parameterizations that enable small networks to generate RAs. Further, we show how RAs emerge in small threshold linear networks through the coordination of a discrete set of line attractors. More broadly, this work informs our understanding of the functional capabilities of small, discrete systems.

# **Exploring Oscillatory Dynamics in Neural Networks: Insights for Effective Communication**

#### G. Huguet<sup>1</sup>

#### <sup>1</sup> Universitat Politècnica de Catalunya

Oscillations are widespread in the brain and are associated with various cognitive functions, including perception and attention. The Communication Through Coherence theory (Fries, 2005, 2015) posits that synchronized oscillations at specific phases are essential for effective neuronal communication. In this presentation, we will delve into the study of oscillations in neuronal networks using a new generation of exact mean-field models. Through detailed analysis, we will explore phase-locking patterns and optimal conditions for communication. Our approach integrates tools from the field of dynamical systems, such as the parameterization method for invariant manifolds, along with recent numerical advancements enabling efficient analysis of dynamics through phase-amplitude description and strategies for regulating oscillatory frequency and phase alignment for communication. This methodology enhances our understanding of oscillatory dynamics in neural networks, providing insights particularly relevant for neural communication.

### Thursday, 13th of June

#### Topological perspectives on the connectome

#### K. Hess<sup>1</sup>

#### <sup>1</sup> Ecole Polytechnique Federale de Lausanne

Over the past decade or so, tools from algebraic topology have been shown to be very useful for the analysis and characterization of networks, in particular for exploring the relation of structure to function. I will describe some of these tools and illustrate their utility in neuroscience, primarily in the framework of a collaboration with the Blue Brain Project.

# Emergence of modulated-wave-patterns of activity from the intrinsic dynamics of neuronal mean field models

#### <u>*R. Curtu*<sup>1</sup></u>

#### <sup>1</sup> The University of Iowa

Integrodifferential equations have been successfully used in the past to study patterns of neural activity in the brain. Implemented under the name of ""neural field models"", these equations describe the spatiotemporal dynamics of the membrane potential of neurons, say u(X; t), from a certain domain D by coupling it with other (one or more) local variable(s), v(X; t).

In this talk I will discuss a two-dimensional mean-field model for neuronal activity and show that, depending on the choice of parameters, it could give rise to several interesting spatiotemporal patterns. We implemented methods from bifurcation theory to prove the existence of traveling waves, standing waves and modulated waves, and identify transitions between them. In particular, we found that the modulated waves – spatiotemporal patterns characterized by two distinct oscillatory frequencies – can occur through two distinct mechanisms, depending on the parameter selection. Our theoretical results are supported by numerical simulations.

### The influence of synaptic plasticity on critical coupling estimates for neural populations

#### <u>K. Toth<sup>1</sup></u>, D. Wilson<sup>1</sup>

#### <sup>1</sup> University of Tennessee

The presence or absence of synaptic plasticity can dramatically influence the collective behavior of populations of coupled neurons. Here, we consider spike-timing dependent plasticity (STDP) and its resulting influence on phase cohesion in computational models of heterogeneous populations of conductance-based neurons. STDP allows for the influence of individual synapses to change over time, strengthening or weakening depending on the relative timing of the relevant action potentials. Using phase reduction techniques, we derive an upper bound on the critical coupling strength required to retain phase cohesion for a network of synaptically coupled, heterogeneous neurons with STDP. We find that including STDP can significantly alter phase cohesion as compared to a network with static synaptic connections. Our analysis highlights the importance of the relative ordering of action potentials emitted in a population of tonically firing neurons and demonstrates that order switching can degrade the synchronizing influence of coupling when STDP is considered.

#### Hebbian learning of cyclic features of neural code

#### <u>N. Schonsheck<sup>1</sup></u>, C. Giusti<sup>2</sup>

<sup>1</sup> University of Delaware
 <sup>2</sup> Oregon State University

Cyclic structures are a class of mesoscale features ubiquitous in both experimental stimuli and the activity of neural populations encoding them. Important examples include encoding of head direction, orientation tuning, and grid cells. The central question of our present work is: how does the brain faithfully transmit cyclic structures between regions? Is this a generic feature of neural circuits, or must this be learned? If so, how? Our primary results are 1) feedforward networks with connections drawn from inhibitory-biased random distributions do not reliably propagate cyclic features, 2) updating network connections with a biologically realistic Hebbian learning rule robustly constructs networks that transmit cyclic features, and 3) the inhibition and propagation of such features can be modulated by the size of the output neuron population. These results suggest that "unpacking" geometry through high-dimensional encodings is necessary when applying biologically inspired learning rules in the context of naive neural connectivity.

# Topological tracing of encoded circular coordinates between neural populations

# <u>I. Yoon<sup>1</sup></u>, G. Henselman-Petrusek<sup>2</sup>, L. Ziegelmeier<sup>3</sup>, R. Ghrist<sup>4</sup>, S.L. Smith<sup>5</sup>, Y. Yu<sup>5</sup>, C. Giusti<sup>6</sup>

- <sup>1</sup> Wesleyan University
- <sup>2</sup> Princeton University
- <sup>3</sup> Macalester College
- <sup>4</sup> University of Pennsylvania
- <sup>5</sup> University of California Santa Barbara
- <sup>6</sup> Oregon State University

Topological methods are effective at detecting periodic, quasi-periodic, or circular features in neural systems. Once we detect the presence of circular structures, we face the problem of assigning semantics: what do the circular structures in a neural population encode? To address this problem, we introduced the method of analogous bars. Given two related systems, say a stimulus system and a neural population, or two related neural populations, we utilize the dissimilarity between the two systems and Dowker complexes to find shared features between the two systems. We then leverage this information to identify related features between the two systems. In this talk, I will briefly explain the mathematics underlying the analogous bars method. I will then present applications of the method in studying neural population coding and propagation on simulated and experimental datasets.

# Insights into neural oscillator network dynamics using a phase-isostable framework

#### <u>*R. Nicks*<sup>1</sup>, *R. Allen*<sup>1</sup>, *S. Coombes*<sup>1</sup></u>

#### <sup>1</sup> University of Nottingham

Here we discuss a framework for studying coupled oscillator networks where each oscillator is described by its phase on limit cycle and its slowest decaying isostable coordinate, allowing for representation of trajectories away from (but near) the limit cycle. The resulting phase-isostable network equations can be used compute existence and stability conditions for phase-locked states in networks of identical nodes, extending known results for phase-reduced equations beyond the weak coupling limit. The descriptive ability of the approach is compared with higher-order phase reductions for the mean-field complex Ginzburg-Landau equation, and we demonstrate the power of the general framework by considering small and large networks of Morris-Lecar neurons. We observe phenomena including the emergence of quasiperiodic behaviour that cannot be captured using first-order phase reduction and results are shown to be in good qualitative agreement with the dynamics of the original network through numerical simulations and bifurcation analysis.

#### Q-phase reduction of multi-dimensional stochastic oscillator networks

#### <u>*M. Kreider*<sup>1</sup>, *P. J. Thomas*<sup>1</sup></u>

#### <sup>1</sup> Case Western Reserve University

The governing dynamics of large-scale brain oscillations is an active area of research. One approach is to represent neuron populations as systems of oscillators: ODEs with stable limit-cycle solutions. Phase reduction is a tool that simplifies analysis by representing the synchronized dynamics of deterministic systems as a one-dimensional phase (timing) variable for each oscillator. However, the activity of neuron populations is noisy. Phase concepts can be extended to stochastic oscillators with the Q function, the slowest decaying mode of the backward Kolmogorov (stochastic Koopman) operator. Here, for the first time, we compute the Q function of coupled stochastic oscillators. We provide analytic solutions for linear systems of arbitrary dimension and coupling structure, and compare with numerical solutions of non-linear systems. We also describe an order parameter to quantitatively measure the synchrony of coupled stochastic oscillators. We argue that our approach can contribute to the analysis of large-scale electrophysiological recordings.

#### Metastability in networks of nonlinear stochastic spiking neurons

#### <u>S. Paliwal<sup>1</sup></u>, G. K. Ocker<sup>2</sup>, B. A. W. Brinkman<sup>1</sup>

<sup>1</sup> Stony Brook University
 <sup>2</sup> Boston University

Neurons in the brain continuously process the barrage of sensory inputs they receive from the environment. A wide array of experimental work has shown that the collective activity of neural populations encodes and processes this constant bombardment of information. How these collective patterns of activity depend on single neuron properties is often unclear. Single neuron recordings have shown that individual neural responses to inputs are nonlinear, which prevents a straightforward extrapolation from single neuron features to the emergent collective states. In this work, we use a field theoretic formulation of a stochastic leaky integrate-and-fire model to study the impact of nonlinear intensity functions on macroscopic network activity. We show that the interplay between single-neuron nonlinearities and membrane potential resets can i) give rise to bistability between active firing rate states, and ii) can enhance or suppress mean firing rates and membrane potentials in opposite directions. The membrane potential reset plays a crucial role in stabilizing the dynamics of these networks despite the superlinear firing intensity for individual neurons.

#### Stochastic neural-mass models for populations of spiking neurons

#### T. Schwalger<sup>1</sup>

#### <sup>1</sup> Technical University Berlin

Neural-mass or firing-rate models are widely used in computational neuroscience to describe neural population activities. However, these models are heuristic models that do not capture fast non-stationary responses and fluctuations of realistic finite-size populations of spiking neurons. I present a systematic reduction of a network of integrate-and-fire neurons with a stochastic firing intensity to an efficient, stochastic neural-mass model. I begin with stochastic population density equations for large but finite network size N, which are stable and generate correct fluctuations. Even though these equations are infinite-dimensional, they offer a unique starting point for a further reduction to low-dimensional stochastic differential equations via an eigenfunction expansion. Keeping the first dominant eigenmode already reproduces well the non-stationary response properties and fluctuation statistics of the underlying spiking neural network. Our ""bottom-up"" model provides a highly efficient way to simulate large brain areas at the mesoscopic scale, where fluctuations are crucial.

#### The mean field limit of the Kuramoto model with stochastic resetting

#### P. Bressloff<sup>1</sup>

#### <sup>1</sup> Imperial College London

In this talk we consider the mean field limit of the classical Kuramoto model of coupled phase oscillators in the presence of local or global stochastic resetting, where the phase of each particle independently or simultaneously resets to its original value at a random sequence of times generated by a Poisson process. In each case we derive the Dean-Kawasaki (DK) equation describing hydrodynamic fluctuations of the global density, and then use a mean field ansatz to obtain the corresponding nonlinear McKean-Vlasov (MV) equation in the thermodynamic limit. In particular, we show how the MV equation for global resetting is driven by a Poisson noise process, reflecting the fact that resetting is common to all of the oscillators, and thus induces correlations that cannot be eliminated by taking a mean field limit. We conclude by analyzing the dynamics with resetting on the Ott-Antonsen manifold.

# Understanding and Optimizing Learning in Recurrent Networks using Dynamical Systems Theory

#### *R.* Engelken<sup>1</sup>, *L.* Abbott<sup>1</sup>

#### <sup>1</sup> Columbia University

Addressing the temporal credit assignment problem is vital for modeling how organisms link stimuli and outcomes over time. Gradient-based training of recurrent neural circuit models for temporal tasks with long time horizons presents challenges, potentially leading to vanishing or exploding gradients. We connected this issue to the Lyapunov exponents of the forward dynamics, describing how perturbations grow or shrink in forward passes.

We propose "gradient flossing", a method to address gradient instability in recurrent spiking and firing rate networks by controlling the Lyapunov exponents of the forward dynamics throughout learning. We regularize Lyapunov exponents towards zero, ensuring that the corresponding directions in tangent space grow or shrink only slowly, for more robust propagation of learning signals over long time horizons.

This approach improves RNN stability and training success on temporal tasks by regulating the norm and dimensionality of the gradient signal in backpropagation through the dynamic adjustment of Lyapunov exponents.

#### Emergent dynamic attractors from recurrent network connectivity

#### <u>K. Morrison<sup>1</sup></u>, C. Parmelee<sup>2</sup>, J. L. Alvarez<sup>3</sup>, C. Curto<sup>3</sup>

<sup>1</sup> University of Northern Colorado

- <sup>2</sup> Keene State College
- <sup>3</sup> Pennsylvania State University

While the experimental technology for identifying connectomes has taken off, the mathematical technology for analyzing them is still somewhat limited. It is mathematically challenging to infer properties of a network's dynamics from its underlying architecture, even in idealized settings of simple deterministic dynamics where the precise connectivity graph is known. Given this complexity, how do we determine which features of a network's connectivity are important to its computational function? We focus on a simplified model known as Combinatorial Threshold-Linear Networks (CTLNs) to understand how neural connectivity, as encoded by a directed graph, shapes the emergent nonlinear dynamics of the network. We find that important aspects of these dynamics are controlled by the fixed points of the network, which can often be determined via graph-based rules. These rules provide a direct link between the structure and function of these networks and give insight into network motifs that produce dynamic attractors.

#### Mean Field Analysis of a Stochastic STDP model

#### <u>*P. Helson*<sup>1</sup>, E. Tanré<sup>2</sup>, R. Veltz<sup>2</sup></u>

<sup>1</sup> KTH Royal Institute of Technology
 <sup>2</sup> Inria Centre Université Côte d'Azur

Analysing biological neural network models incorporating synaptic plasticity has proven challenging both on theoretical and numerical aspects. The intricate coupling between neuron and synapse dynamics, along with the heterogeneity introduced by plasticity, complicates the use of traditional theoretical tools. Additionally, the computational expense of synapses scaling in  $N^2$  hinders the numerical analysis. In this study, we study a stochastic STDP model integrated into a probabilistic Wilson-Cowan neural network with binary neuronal activity. The  $N^2$  weights impede the definition of a "typical" neuron. To address this, we introduce a new variable: the empirical distribution of the triplet (pre-synaptic neuron state, time elapsed since its last spike, synaptic weight from pre to post-synaptic neuron) over pre-synaptic neurons. Employing mean-field analysis on this typical neuron yields a substantially reduced model. This research could facilitate the derivation of mean-field limits for other models involving jumping particles with plastic interaction.

# The Effect of Noise Structure on Spatio-Temporal Pattern Formation and Chaos

#### S. Chipman<sup>1</sup>, B. Doiron<sup>1</sup>

#### <sup>1</sup> University of Chicago

Spatially-extended networks of recurrently coupled excitatory and inhibitory neural firing rates exhibit a rich suite of dynamics in two dimensions, such as spatio-temporal pattern formation, bulk oscillations, and deterministic chaos. Previous work shows that the addition of noise which is correlated across populations of neurons expands the chaotic regime in phase space. We found that spatially-correlated noise has the opposite impact. Both chaos and pattern formation were suppressed by noise with even relatively small spatial correlations, irrespective of the width of the recurrent connections of the network, as both positive and negative Lyapunov exponents trended to zero. Spatial correlations in noise had the effect of homogenizing and marginalizing system behavior, resulting in a network with much weaker correlations, but overall more uniformly predictable behavior across phase space.

#### State modulation in a spatial network with three interneuron subtypes

#### <u>*M. Edwards*<sup>1</sup>, J. Rubin<sup>1</sup>, C. Huang<sup>1</sup></u>

<sup>1</sup> University of Pittsburgh

Three inhibitory interneuron subtypes, parvalbumin (PV), somatostatin (SOM), and vasoactive intestinal peptide (VIP) expressing cells, are identified as key players in regulating sensory responses. However, the distinct contribution of each interneuron subtype for regulating network dynamics remains unclear. In this work, we systematically study the impacts of cell-type specific external inputs and connection strengths in a spatially organized spiking neuron network. We find that network dynamics transition across the same three activity states, characterized by population rates and coherence, as modulatory input varies to any one population. Strikingly, SOM rates fluctuate closely with network synchrony in all modulation cases. Changes to pre- and post-SOM synapses reveal the critical roles of SOM cells in regulating network activity states. This work explores the unique functionality of individual interneuron subtypes in shaping dynamics and broadens our understanding of top-down regulation of circuit responses to sensory inputs.

### The space-time dynamics of sleep oscillations and its implications for cognition and health

#### <u>*P. Malerba*<sup>1</sup>, S. C. Mednick<sup>2</sup></u>

<sup>1</sup> Ohio State University
 <sup>2</sup> University of California

Sleep is a state of highly rhythmic brain activity that is crucial for health and cognition. Studies show that these sleep oscillations serve to organize information processing and communication in relation to a specific behavior. Yet, the fundamental properties of brain oscillations that enable these behaviors are not understood. Present approaches to the study of human sleep, generally distinguish oscillations based on frequency alone while overlooking space-time profiles. Our ongoing research reflects the principle that sleep oscillations with the same frequency might not serve the same function. In this talk, I will show that that space-time patters of brain oscillations can be identified in a data-driven way, and that they can reveal biophysical differentiation relevant to information processing and brain function. This refined picture of space-time dynamics of NREM sleep oscillations, articulated across light and deep sleep stages, results in differentially organized space-time profiles that in turn can recruit different functional networks.

### Friday, 14th of June

# Neural dynamics and geometric singular perturbation theory beyond the standard form

#### M. Wechselberger<sup>1</sup>

#### <sup>1</sup> University of Sydney

An important feature of neural dynamics is that they evolve on multiple timescales. The Hodgkin-Huxley and the FitzHugh-Nagumo models are prototypical examples which can create 'four-stroke' relaxation oscillations, i.e., tonic spiking consisting of two slow and two fast phases per cycle. These neural models have been successfully analysed using the 'standard' geometric singular perturbation theory (GSPT) toolbox.

In this talk, I will introduce 'two-stroke' relaxation oscillators where spikes consist of only two distinct phases per cycle – one slow and one fast – which distinguishes them from the 'supposedly' paradigmatic four-stroke relaxation oscillators. These type of oscillators can be found in singular perturbation problems in 'non-standard' form where the multiple timescale splitting is not necessarily reflected in a multiple timescale variable splitting. I will introduce a mathematical framework for 'GSPT beyond the standard form' and its application to cell signalling problems of this kind.

# Chaotic neural dynamics facilitate probabilistic computations through sampling

#### T. Toyoizumi<sup>1</sup>

#### <sup>1</sup> RIKEN Center for Brain Science

Cortical neurons exhibit highly variable responses over trials and time. Theoretical works posit that this variability arises potentially from chaotic network dynamics of recurrently connected neurons. Here we demonstrate that chaotic neural dynamics, formed through synaptic learning, allow networks to perform sensory cue integration in a sampling-based implementation. We show that the emergent chaotic dynamics provide neural substrates for generating samples not only of a static variable but also of a dynamical trajectory, where generic recurrent networks acquire these abilities with a biologically-plausible learning rule through trial and error. Furthermore, the networks generalize their experience in the stimulus-evoked samples to the inference without partial or all sensory information, which suggests a computational role of spontaneous activity as a representation of the priors as well as a tractable biological computation for marginal distributions. These findings suggest that chaotic neural dynamics may serve for the brain function as a Bayesian generative model.

### Pros and cons of mean field representations of large size networks of spiking neurons

#### **O.** Faugeras<sup>1</sup>, R. Veltz<sup>1</sup>

<sup>1</sup> Inria Centre Université Côte d'Azur

We predict the changes of activity of N neurones when varying the parameters entering the models. The neurones are described by stochastic differential equations and N is large. Bifurcation theory is not available for such models. We finesse the problem by considering the case N infinite. The limit is described by a low dimensional nonlinear partial differential equation which is amenable to bifurcation analysis. The results can be used to predict changes of behaviours in the finite size population. We characterise the quality of the approximation of the finite size network by its thermodynamics limit as a function of N. Theoretical and numerical results are presented showing in the case of Fitzhugh-Nagumo neurones connected by electrical and chemical synapses that our strategy is effective.

### Exact low-dimensional description for fast neural oscillations with low firing rates

#### <u>*P. Clusella*<sup>1</sup>, E. Montbrió<sup>2</sup></u>

<sup>1</sup> Universitat Politecnica de Catalunya

<sup>2</sup> Universitat Pompeu Fabra

Recently, low-dimensional models of neuronal activity have been exactly derived for large networks of deterministic, Quadratic Integrate-and-Fire (QIF) neurons. Such firing rate models (FRM) describe the emergence of fast collective oscillations (>30 Hz) via the frequency-locking of a subset of neurons to the global frequency. However, the suitability of such models to describe realistic neuronal states is challenged by the fact that during fast collective oscillations, neuronal discharges are often irregular and have low firing rates compared to the global oscillation frequency. Here we extend the theory to derive exact FRM for QIF neurons to include noise, and show that networks of stochastic neurons displaying irregular discharges at low firing rates, are governed by exactly the same evolution equations as deterministic networks. Our results reconcile two traditionally confronted views on neuronal synchronization, and upgrade the applicability of exact FRM to describe a broad range of biologically realistic neuronal states.

# Collective Dynamics in the Heterogeneous Izhikevich Network with Synaptic Delay

#### L. Chen<sup>1</sup>, S. A. Campbell<sup>1</sup>

#### <sup>1</sup> University of Waterloo

We investigate collective dynamics of heterogeneous Izhikevich networks with global constantdelay coupling by means of an exact mean-field model, valid in the thermodynamic limit. Our study emphasizes the impact of the heterogeneity of the quenched input current, the adaptation intensity, and the synaptic delay on the emergence of collective oscillations. Furthermore, we address the question of whether the Ott-Antonsen-based mean-field system in the limit where the heterogeneity approaches zero can represent a system with extremely weak heterogeneity. Our perturbation and bifurcation analysis reveal that such a mean-field model remains consistent in dynamics, although the Ott-Antonsen theory is not conventionally considered the appropriate framework for deriving the mean-field model if the neurons are strictly identical. Synaptic delays mostly work like an excitatory drive to favor, even induce new collective dynamics. In particular, Torus bifurcations may occur in a single population of neurons without an external drive, and these are a crucial mechanism for the emergence of population bursting with two nested frequencies.

# Phase transitions in decision dynamics depend on information reliability

#### N. W. Barendregt<sup>1</sup>, J. I. Gold<sup>2</sup>, K. Josic<sup>3</sup>, Z. Kilpatrick<sup>1</sup>

- <sup>1</sup> University of Colorado Boulder
- <sup>2</sup> University of Pennsylvania
- <sup>3</sup> University of Houston

Normative theories of evidence accumulation and decision making help quantify how different strategies balance objectives like efficient reward acquisition and information gathering. Recent developments focusing on adaptive strategies indicate how individuals cope with uncertainty in single, independent decisions, but in reality people and other animals make sequences of decisions in volatile environments under many constraints. Here we present a new theoretical framework describing how cost-constrained Bayes optimal agents accumulate information to make decisions across trials when correct choices are correlated as a discrete-state Markov process. Agents' behaviors depend strongly on their strategy and the task conditions, revealing a sharp transition from exploration to exploitation when choice feedback is more informative than environmental evidence.

# Three-factor cortico-striatal plasticity shifts activity of cortico-basal ganglia-thalamic subnetworks to maximize reward rate in decision-making tasks

#### <u>C. Vich<sup>1</sup></u>, J. Bahuguna<sup>2,3</sup>, J. E. Rubin<sup>4</sup>, T. Verstynen<sup>2,4</sup>

- <sup>1</sup> Universitat de les Illes Balears
- <sup>2</sup> Carneige Mellon University
- <sup>3</sup> University of Strasbourg
- <sup>4</sup> University of Pittsburgh

Understanding how cortico-basal ganglia-thalamic (CBGT) circuits influence decision making remains a challenge, especially considering the different decision policies a biological agent can adopt in response to environmental changes and feedback from past outcomes. We deconstruct the process of feedback-driven learning in a CBGT network into three aspects: (a) defining "what" is a decision policy, (b) identifying "where" in the CBGT network these decision policies are effectively generated (c) analyzing "how" CBGT pathways encode and modulate different aspects of decision policies. Specifically, we use evidence accumulation models to represent decision policies driven by the behavior of a spiking network model of the CBGT circuit learning a two choice task and canonical correlation analysis to map between policy parameters and network activity. Through this approach, we show that CBGT networks maximize reward rate via feedback-driven cortico-striatal synaptic plasticity by modulating three low dimensional CBGT subnetworks (control ensembles).

#### Sensory input to cortex encoded on low-dimensional peripherycorrelated subspaces

### <u>A. K. Barreiro<sup>1</sup></u>, A. J. Fontenelee<sup>2</sup>, C. Ly<sup>3</sup>, P. C. Raju<sup>2</sup>, S. H. Gautam<sup>2</sup>, W. L. Shew<sup>2</sup></u>

- <sup>1</sup> Southern Methodist University
- <sup>2</sup> University of Arkansas
- <sup>2</sup> Virginia Commonwealth University

As information about the world is conveyed from the sensory periphery to central neural circuits, it mixes with complex ongoing cortical activity. How do neural populations keep track of sensory signals, separating them from noisy ongoing activity? We demonstrate that sensory signals are encoded more reliably in low-dimensional subspaces defined by correlations between neural activity in primary sensory cortex and upstream sensory brain regions. We analytically show that these correlation-based coding subspaces can reach optimal limits as noise correlations between cortex and upstream regions are reduced, and that this principle generalizes across diverse sensory stimuli in the olfactory system and the visual system of awake mice.

#### Mapping developmental changes in sleep-wake behavior

#### V. Booth<sup>1</sup>

#### <sup>1</sup> University of Michigan

Mathematical models of physiological sleep-wake networks describe sleep-wake regulation by simulating the activity of wake- and sleep-promoting neuronal populations and their modulation by homeostatic and circadian drives. The resulting systems of ODEs are piecewisesmooth. Motivated by changes in sleep behavior during early childhood, we vary homeostatic and circadian modulation to analyze effects on developmental transitions of sleep-wake patterns, including napping and non-napping behaviors. To identify the types and sequences of bifurcations for sleep changes, we numerically compute circle maps representing model dynamics, which are non-monotonic and discontinuous. We find that the average daily number of sleeps exhibits period adding sequences, via saddle-node and border collision bifurcations, as homeostatic time constants are reduced. The inclusion of rapid eye movement (REM) sleep states disrupts these sequences with period-doubling bifurcations and bistability. Variations in the circadian rhythm, as can occur with seasonal changes, further modulates bifurcation sequences.

#### Seizure generation mechanisms beyond bifurcations: New perspectives on critical transitions and early warning signals

#### A. Flynn<sup>1</sup>, S. Wieczorek<sup>1</sup>, C. McCafferty<sup>2</sup>

<sup>1</sup> University College Cork
 <sup>2</sup> University College London

Three inhibitory interneuron subtypes, parvalbumin (PV), somatostatin (SOM), and vasoactive intestinal peptide (VIP) expressing cells, are identified as key players in regulating sensory responses. However, the distinct contribution of each interneuron subtype for regulating network dynamics remains unclear. In this work, we systematically study the impacts of cell-type specific external inputs and connection strengths in a spatially organized spiking neuron network. We find that network dynamics transition across the same three activity states, characterized by population rates and coherence, as modulatory input varies to any one population. Strikingly, SOM rates fluctuate closely with network synchrony in all modulation cases. Changes to pre- and post-SOM synapses reveal the critical roles of SOM cells in regulating network activity states. This work explores the unique functionality of individual interneuron subtypes in shaping dynamics and broadens our understanding of top-down regulation of circuit responses to sensory inputs.

### Oscillations and avalanches in an Ising-like class of adaptive neural networks

#### <u>F. Lombardi<sup>1,2</sup></u>, S. Pepic<sup>2</sup>, O. Shriki<sup>3</sup>, G. Tkacik<sup>2</sup>, D. De Martino<sup>4</sup>

- <sup>1</sup> University of Padova
- <sup>2</sup> Institute of Science and Technology Austria
- <sup>3</sup> Ben-Gurion University of the Negev
- <sup>4</sup> Biofisika Institute (CSIC,UPV-EHU) and Ikerbasque Foundation

Brain networks exhibit collective dynamics as diverse as scale-specific oscillations and scalefree neuronal avalanches. Although existing models account for oscillations and avalanches separately, they typically do not explain both phenomena, are too complex to analyze analytically or intractable to infer from data rigorously. Here we propose a feedback-driven lsing-like class of neural networks that captures avalanches and oscillations simultaneously and quantitatively. In the simplest yet fully microscopic model version, we can analytically compute the phase diagram and make direct contact with human brain resting-state activity recordings via tractable inference of the model's two essential parameters. The inferred model quantitatively captures the dynamics over a broad range of scales, from single sensor oscillations to collective behaviors of extreme events and neuronal avalanches. Importantly, the inferred parameters indicate that the co- existence of scale-specific (oscillations) and scale-free (avalanches) dynamics occurs close to a non-equilibrium critical point at the onset of self-sustained oscillations.

### Analyzing sequential activity in neural networks from the dynamical systems perspective

#### <u>P. Chossat<sup>1</sup></u>, E. K. Ersoz<sup>2</sup>, F. Lavigne<sup>3</sup>

<sup>1</sup> Inria Centre Université Côte d'Azur
 <sup>2</sup> INSERM, France
 <sup>3</sup> University of Nice

I present a simple, bio-inspired model for neural masses, which allows to apply fast-slow analysis in order to derive conditions for the occurrence of excitable chains of learned states (patterns) in a neural network. The key ingredients for this 'latching' dynamics are synaptic short term depression (STD) and overlaps between the learned states. Given any network satisfying these conditions, it can be decomposed into elementary 'building blocks' made of chains intersecting at a node. The case when a chain splits in two outgoing chains at some node has been investigated. Results applied to decision-making problems suggest that neural gain modulation by error signal could help fast decision making by changing the probability of transition to the outgoing chain in a changing environment (while synaptic adaptation alone would require relearning and oversight of previous memory before the change of strategy).

# Dynamics and Computations in Networks of Heterogeneous Spiking Neurons

#### <u>*R. Gast*</u><sup>1</sup>, *S. A. Solla*<sup>1</sup>, *A. Kennedy*<sup>1</sup>

#### <sup>1</sup> Northwestern University

Neurons express substantial heterogeneity within genetically defined cell types. How does this heterogeneity affect the dynamics and function of a neural network? Here, we address this question by studying networks of coupled spiking neurons. Applying the Ott-Antonsen ansatz, we derive mean-field equations for networks of Izhikevich neurons with heterogeneous spike thresholds and provide detailed comparisons between the mean-field equations and the spiking neural network dynamics. Leveraging the mean-field equations, we study how the level of spike threshold heterogeneity affects the dynamic regimes of neural networks via bifurcation theory. Moreover, we examine the memory and function generation capacities of spiking neural networks in dynamic regimes that we identified via the mean-field equations, to study the relationship between network dynamics and function. Our results suggest that neural heterogeneity should be considered as a crucial ""knob"" that can be adjusted in neural circuits to tune their dynamics towards a particular computational function.

#### **Balanced Neural Fields**

#### <u>J. MacLaurin<sup>1</sup></u>, M. Silverstein<sup>1</sup>, P. Vilanova<sup>2</sup>

<sup>1</sup> New Jersey Institute of Technology

<sup>2</sup> Stevens Institute of Technology

We study high dimensional neural networks with random connectivity, such that the excitatory and inhibitory inputs to any neuron are approximately balanced and of order one (with high probability). The neurons are embedded in a space (such as the ring  $S^1$  of the visual cortex). It is assumed that there are many cliques of neurons distributed throughout the space, such that the connectivities within neurons of the same clique can be highly correlated. The connectivities between neurons of different cliques are probabilistically independent. Upon taking the large size limit, we obtain an autonomous neural field equation. One of the most important novelties of this approach is that the large size limiting covariance equations are autonomous and do not possess a delay structure. This greatly facilitates the analysis of pattern formation and phase transitions, using standard dynamical systems techniques that have been successfully applied to other neural field models. To obtain the autonomous limiting equations it is necessary that one assumes that the gain function is linear. Joint work with Pedro Vilanova (Stevens Institute of Technology).

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