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**First author** Egert, Ulrich (speaker)

Workshop W06-1. Sat 03/07/2010, 12:30 - 15:30 - Room G103

Session 006 - Structure, dynamics and function in large scale neuronal ensembles

Abstract n° 006.1

Publication ref.: *FENS Abstr.*, vol.5, 006.1, 2010

**Authors** Egert U. (1, 2), Weihberger O. (1, 2, 3) & Okujeni S. (1, 2, 3)

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**Title** Targeted interaction with oscillatory network dynamics

**Text** The dynamics of activity in neuronal networks of any scale or level of observation is, if at all, barely understood. Most analyses of information processing in neuronal networks implicitly assume reproducible responses masked by unpredictable background activity that needs to be removed by data preprocessing. While this provided some understanding in sensory systems, in particular of anesthetized animals, the interaction of the ongoing network activity or network state with induced activity may be neither random nor additive, but could be non-linear, e. g. via state dependent sensitivity changes, network participation or propagation of activity. Some of these dynamics are observed in reduced preparations, e. g. cultures of dissociated neocortical tissue that develop spontaneous burst activity. This varies spontaneously in recurring patterns, extending across a wide range of temporal scales and spatial structures. To identify the rules by which ongoing activity influences stimulation outcomes, we analyzed the dependence of the efficacy and effect of weak electrical microstimulation (EMS) in neuronal networks on microelectrode arrays. In previous reports repetitive EMS led to frequency dependent adaptation of the responsiveness and suppressed spontaneous activity. In our networks, responses to EMS strongly depend on EMS timing to the phase of burst cycles and on the overall mode of this activity. EMS efficacy increased with its lag to the preceding spontaneous burst. Phase-locked EMS increased the reproducibility of the response. Furthermore, the magnitude of the response and the delays of its components were a function of the mode of spontaneous activity. The spatial pattern of activity propagation could be related to the propagation pathways of spontaneous bursts. EMS at sites activated early in spontaneous bursts appeared to recruit the same pathways from the stimulation site onward, suggesting the activation of local networks by the stimulus, rather than individual neurons. Our analyses outline the forces underlying intrinsic response variability and the options to implement reproducible interaction with neuronal networks.  
(Funded by the BMBF 01GQ0420 and 01GQ0830, and EC 12788.).

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**First author** Yim, Man Yi (poster)

Poster board D6 - Wed 07/07/2010, 11:15 - Hall 1

Session 200 - Basal ganglia 2

Abstract n° 200.6

Publication ref.: *FENS Abstr.*, vol.5, 200.6, 2010

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**Title** Weak input correlation enhances the signal-to-noise ratio in striatum network model

**Text** The striatum is the main input station of the basal ganglia and is associated with higher cognitive functions such as reward-based learning, action selection and motor control. Medium spiny neurons (MSNs) represent over 95% of the striatum neuron population. The recurrent connections among MSNs provide weak feedback (FB) inhibition. By contrast, GABAergic interneurons such as fast spiking interneurons (FSIs), which constitute only ~2% of striatum neurons, project extensively to the MSNs and provide strong feedforward (FF) inhibition. The striatum is innervated by massive excitatory afferents from various regions of the neocortex via the cortico-striatal projection neurons. Anatomical evidence suggests that the sharing of inputs between neighboring neurons is very low, however, afferents driving a single neuron may be correlated because they originate from functionally related brain regions [1].

To understand the representation of cortical inputs in the striatum, we simulated a spiking neuron network model of the striatum, based on existing experimental data. We characterized the input-output transfer function of the striatum network for correlated and uncorrelated inputs. Independent of the input correlation, activation of a small fraction of MSNs (~10%) resulted in a corresponding decrease in the spiking of unstimulated neurons. This was similar to the activity of MSNs recorded in behaving rats [2]. Moreover, we found that input correlation influenced the signal representation in a non-monotonic fashion. Specifically, we showed that weak pairwise correlations among cortical afferents to striatal neurons improved the representation by increasing the signal-to-noise ratio in the striatum.

Supported by the German Federal Ministry of Education and Research (BMBF 01GQ0420 to BCCN Freiburg, BMBF GW0542 Cognition and BMBF 01GW0730 Impulse Control).

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1. Zheng & Wilson (2002) *J Neurophysiol.* 87:1007-1017.
2. Barnes et al. (2005) *Nature* 437: 1158-1161.

**Theme** D - Sensory and motor systems  
Basal ganglia

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**First author** Vlachos, Ioannis (poster)

Poster board B58 - Wed 07/07/2010, 11:15 - Hall 1

Session 192 - Network interactions 2

Abstract n° 192.58

Publication ref.: *FENS Abstr.*, vol.5, 192.58, 2010

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**Title** How do distinct neuronal subpopulations in the central amygdala shape the fear response? A computational model

**Text** During a typical fear conditioning experiment a neutral stimulus is paired with a fearful one and after several trials the former acquires aversive properties. Such learning can be suppressed by repeated presentations of the initially neutral stimulus alone (fear extinction). The critical brain structures involved in these fear related processes are the lateral (LA), the basal (BA) and the central (CeA) nuclei of the amygdaloid complex. The CeA is a striatum-like structure containing almost exclusively GABAergic neurons [1]. It is known to be the major output nucleus of the amygdala and to control the fear response by its projections to the brainstem and hypothalamus.

To understand the interactions between the lateral (CeL) and medial (CeM) subdivisions of the CeA during fear conditioning and fear extinction, we built a spiking neuron network model of the CeA using the NEST simulator [2]. We modeled the CeA as a feedforward dis-inhibitory circuit, based on known anatomical and electrophysiological data. The input to the CeA was controlled by two distinct, fear and extinction specific neuron subpopulations within the BA [3, 4]. These inputs were crucial, as they altered the states of different subgroups within the CeA.

With our model we provide first insights about the computations performed by the CeA. In particular, we show how CeL and CeM neurons might process fear and extinction related activity of the BA in order to shape the fear response.

Supported in parts by BMBF grants 01GW0542 Cognition, 01GQ0420 to BCCN Freiburg and by Neurex Interreg-IV.

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2. Gewaltig & Diesmann (2007) *NEST. Scholarpedia*, 2(4):1430

3. Herry et al. (2008) *Nature* 454, 600-606

4. Vlachos et al (2009) *BMC Neuroscience* 2009, 10 (Suppl 1): P142.

**Theme** B - Excitability, synaptic transmission, network functions  
Network interactions - Signal propagation

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**First author** Rotter, Stefan (poster)

Poster board C135 - Sun 04/07/2010, 12:15 - Hall 1

Session 018 - Parkinson's 1

Abstract n° 018.6

Publication ref.: *FENS Abstr.*, vol.5, 018.6, 2010

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**Title** Task related phase locking of spikes in the STN of humans with Parkinson's disease

**Text** Motor dysfunction (e. g. tremor) is a common symptom of Parkinson's disease (PD), caused by a malfunction of dopaminergic projections. The depletion of dopamine in PD is also associated with multiple cognitive deficits and impaired decision making. While the neural correlates of motor dysfunctions can be observed in oscillatory local field potentials (LFP) and spiking activity in the sub-thalamic nucleus (STN), the neural mechanisms underlying impaired decision making remain poorly understood.

To study neural activity during decision making, we analyzed broadband extracellular field potentials recorded from the STN of 7 PD patients at different depths, while they performed a standard Go/NoGo task. The recordings were made during stereotactic surgery for deep-brain-stimulation with the consent of the patients.

We extracted multi-unit spiking activity (MUA) and LFP from the raw extracellular signals and compared the synchrony between the LFP and spiking activity in the Go and NoGo task. We found that the onset of a NoGo trial increased the pairwise correlations between the LFPs. There was no significant difference between Go and NoGo task triggered MUA. However, we found that MUA spikes were tightly locked to the phase of the LFP in the NoGo task. This finding suggests a critical role of the STN in NoGo behavior, imposing a strong constraint on computational models of the basal-ganglia network.

Supported by the BMBF grant 01GQ0420 to BCCN Freiburg, BMBF GW0542 Cognition & BMBF 01GW0730 Impulse Control.

**Theme** C - Disorders of the nervous system  
Parkinson's disease - Human studies and therapies

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**First author** Reinartz, Sebastian (poster)

Poster board B67 - Tue 06/07/2010, 12:15 - Hall 1

Session 130 - Network interactions 1

Abstract n° 130.67

Publication ref.: *FENS Abstr.*, vol.5, 130.67, 2010

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**Title** The concept of learning transferred to low-level modifications of stimulus/response relationships

**Text** Although changes in synaptic efficiencies are considered as the functional basis for learning (Hebb, 1949), it remains unclear how selective synaptic changes are realized and directed in neural networks. Our goal was to investigate selective changes within neuronal networks that might underlie the process of learning and its required activation processes. We reproduced a learning paradigm introduced by Shahaf and Marom (2001) for neuronal networks cultured on microelectrode arrays. Low-frequency stimulation (0.1-0.5Hz) was applied to induce plasticity. If the response probability of a previously selected electrode within a defined time window was doubled, the stimulation was paused for 5min to consolidate the new connection weights. Learning was defined as successful when the number of stimuli needed to fulfill the stop criterion (NS) decreased significantly during the 5h experiment. This was achieved only in a fraction of experiments (6/23), whereas NS increased in 7/23 or fluctuated in 9/23. Our results motivated a search for hidden causes for these variable outcomes. In sham learning experiments with similar pseudo-stimulus/response pairs, only 1 of 17 experiments would be classified as successful learning. Additionally, the length of a stimulation cycle was negatively correlated with the subsequent spontaneous activity but not with the preceding. Response latencies, increased with the ratio of the inter-event intervals of spontaneous network bursting to stimulation period. NS correlated positively with the response latencies. Thus, stimulation-induced adaptation could either be one of the hidden causes for variable outcomes, or be itself an important mechanism for learning as defined in the paradigm. Supported by the German BMBF (FKZ 01GQ0420) and by the EC (NEURO, No. 12788).

**Theme** B - Excitability, synaptic transmission, network functions  
Network interactions - Oscillations and synchrony

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**First author** Okujeni, Samora (poster)

Poster board B42 - Wed 07/07/2010, 11:15 - Hall 1

Session 192 - Network interactions 2

Abstract n° 192.42

Publication ref.: *FENS Abstr.*, vol.5, 192.42, 2010

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**Title** Neuronal cluster formation supports self-excitation of isolated cortical networks

**Text** Brain architectures evolve considerably on the basis of activity-dependent neuronal differentiation processes. The context-dependent integration of neurons is thereby partly reflected in the cellular morphology evolving under specific tissue-dependent constraints and input scenarios. Likewise, neurons developing in isolated cultures *ex vivo* integrate themselves into networks based on comparable fundamental principles. The biochemical machinery translating a specific developmental context into the adequate neuronal morphology thereby strongly depends on proteins that regulate dynamical properties of the cytoskeleton. We show that cortical neurons developing under chronic inhibition of Protein Kinase C form increased dendrites and reduce their clustering behavior resulting in overall more homogeneous networks. Emerging spontaneous activity dynamics exhibit stronger and more compact network-wide bursting (NB) in line with proposed higher connectivity. Lower activity levels due to lower NB frequencies, however, suggest a reduced ability of network self-excitation. We assess origins of NB by simultaneous extracellular recordings from 1000 electrodes sampling from all areas of developing networks of 200000 neurons. By electrical stimulation via microelectrode arrays at different sites we probe how activity propagates within different network architectures. We show that homogeneous networks formed under PKC inhibition are potentially able to operate at much higher activity levels driven by electrical stimulation. In contrast, networks that developed under normal conditions cannot be driven far beyond spontaneous activity levels when electrically stimulated. We conclude that the self-organized formation of neuronal clusters in isolated networks is important for the generation of intrinsic activity by local recurrent amplification of spontaneous excitation supporting the idea that specific brain circuitries partly emerge within function-follow-form scenarios. Patrick Pauli and Ute Riede are gratefully acknowledged for technical assistance. Supported by the German BMBF (FKZ 01GQ0420) and by the EC (NEURO, No. 12788).

**Theme** B - Excitability, synaptic transmission, network functions  
Network interactions - Signal propagation

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**First author**      Kremkow, Jens (poster)

Poster board B29 - Wed 07/07/2010, 12:15 - Hall 1

Session 192 - Network interactions 2

Abstract n° 192.29

Publication ref.: *FENS Abstr.*, vol.5, 192.29, 2010

**Authors**            Kremkow J. (1, 2), Aertsen A. (1, 2) & Kumar A. (1, 2)

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**Title**                Temporal gating of signal propagation in spiking neural networks by delayed correlation between excitation and inhibition

**Text**                Both spontaneous and natural stimulus driven neuronal activity are dominated by transients. Selective gating of these transients is mandatory for proper brain function and may, in fact, form the basis of millisecond fast decision making and action selection.

Recently, Vogels & Abbott [1] proposed a gating mechanism based on the detailed balance of excitation and inhibition to control the propagation of neuronal activity in feedforward networks.

This gating strategy, however, is only effective in controlling the propagation of tonic components of neuronal activity modulation - transients 'escape' from such gating and propagate to the next stage in any case. As a consequence, gating based only on detailed balance of excitation and inhibition is truly limited in its applicability.

Here, we propose a simple, biologically feasible mechanism (temporal gating) that exploits timing differences between excitation and inhibition to control the propagation of spiking activity transients.

To systematically study the concept of temporal gating, we embedded a signal path in a large-scale recurrent cortical network model. The signal path was a feedforward network, consisting of three successive groups of neurons.

We show that small differences between the timing of excitation and inhibition can act as a modulator of neuronal integration time and, thereby, serve as a highly selective gate for both transients and tonic components of neuronal activity. This gating mechanism presents an example of exploiting a single neuron level property to modulate network behavior, such as to control the propagation of spiking activity within the network. Interestingly, the relative latencies between excitation and inhibition used here are well within the range recently measured experimentally in vivo [2].

Taken together, the proposed mechanism of temporal gating allows for both, gating of onset transients and correlated rate inputs in millisecond time scales. Thereby, it presents a definite improvement over earlier proposed mechanisms.

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Okun&LampI (2008) *NatNeurosci* 11:535–537.

**Theme**             B - Excitability, synaptic transmission, network functions  
Network interactions - Signal propagation

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**First author** Kandler, Steffen (poster)

Poster board B36 - Tue 06/07/2010, 11:15 - Hall 1

Session 130 - Network interactions 1

Abstract n° 130.36

Publication ref.: *FENS Abstr.*, vol.5, 130.36, 2010

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**Title** Synchronization of activity and initial connectivity in dissociated culture follows native cortical development

**Text** The structural and functional embedding of single neurons into complex networks as found in the mammalian cortex is not fully understood. Recent findings suggest that spontaneous network synchronization during early development supports the formation of basic functional circuits. It implies that this activity state has a fundamental influence on the emerging computational properties of the developing network.

To investigate the embedding of individual neurons into developing networks, we performed simultaneous intracellular and extracellular recordings of dissociated cultures obtained from frontal cortex of newborn rats. Monitoring these networks with dual patch-clamp electrodes and 60-site microelectrode arrays (MEA), we gained insight into the network connectivity and dynamics on a single-neuron and population level.

We show that distance-dependent pairwise correlations in the firing within spontaneous network bursts (NB) increased during network maturation, which is similar to the native cortex. The onset of NBs was further restricted to a distinct set of network sites, suggesting that the spatial arrangement and connectivity of neurons in these populations support NB initiation and propagation. Pairwise correlations between intracellular and extracellular NB firing were highly dependent on the NB onset latencies, and to a minor degree on the spatial distance to NB onset site. In 95 pairwise intracellular recordings we found that neurons less than 400microns apart had connections with a probability of 49%. Most connections thereby were unidirectional (23%) or bidirectional (20%) excitatory; only about 5% of all pairs had inhibitory connections.

We conclude that a high degree of excitation and interconnectivity supports spontaneous network activation and synchronization in networks of cortical neurons in vitro and in vivo.

Supported by the German BMBF (FKZ 01GQ0420) and by the EC (NEURO, No. 12788).

**Theme** B - Excitability, synaptic transmission, network functions  
Network interactions - Oscillations and synchrony

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**First author** Häussler, Ute (poster)

Poster board C18 - Sun 04/07/2010, 11:15 - Hall 1

Session 015 - Epilepsy 1

Abstract n° 015.18

Publication ref.: *FENS Abstr.*, vol.5, 015.18, 2010

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**Title** Unraveling the spatial relation between neurogenesis and the spread of epileptiform activity in a mouse model of mesial temporal lobe epilepsy

**Text** Temporal lobe epilepsy (TLE) is associated with severe hippocampal restructuring including granule cell dispersion (GCD). We have previously shown that GCD is accompanied by the loss of neurogenesis and caused by displacement of mature neurons in the intrahippocampal kainate (KA) model for TLE in mice (Heinrich et al., 06, JN; Müller et al., 09, Exp. Neurol.). In contrast, epileptic activity (EA) triggered an acute increase in hippocampal neurogenesis in other TLE models. To address this controversy, we used the intrahippocampal KA model in mice to clarify the relation between GCD, neurogenesis and EA propagation.

We recorded status epilepticus (SE) and recurrent EA with electrodes implanted along the hippocampal septo-temporal axis. In parallel, cell proliferation was displayed by systemic BrdU injection. Cell fate was determined by immunolabeling for doublecortin (DCX, newborn neurons) and glial markers. Physiological properties and network integration of newborn granule cells were characterized with patch clamp recordings in acute slices from KA-injected DCX-DsRed transgenic mice.

KA-triggered SE was measurable at all positions in the ipsi- and contralateral hippocampus, but was strongest in the temporal regions. During the following three weeks recurrent EA developed in the whole hippocampus, but amplitudes were largest where GCD was not maximal, but still pronounced. At the cellular level, we found a spatial correlation between GCD and increased gliogenesis around the injection site. In contrast, more distally, a decline in GCD coincided with recovery of neurogenesis, which was strongly increased even in the temporal ipsi- and entire contralateral hippocampus. Newborn granule cells showed high input resistances, action potential firing and network integration. These data suggest that propagation of SE triggers neurogenesis which in turn may increase the overall network excitability, but is not sufficient for the generation of EA in TLE.

Funding: DFG SFB TR3, SFB 780.

**Theme** C - Disorders of the nervous system  
Epilepsy - Circuits and systems

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**First author** Froriep, Ulrich (poster)

Poster board C14 - Sun 04/07/2010, 11:15 - Hall 1

Session 015 - Epilepsy 1

Abstract n° 015.14

Publication ref.: *FENS Abstr.*, vol.5, 015.14, 2010

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**Title** The relation of entorhinal cortex and dentate gyrus in a model of temporal lobe epilepsy

**Text** Mesial temporal lobe epilepsy, the most common classifiable form of focal epilepsies in humans, is often accompanied by histological changes within the hippocampal formation, denoted as hippocampal sclerosis (HS).

We use the intrahippocampal kainate mouse model, in which epileptiform activity (EA) and HS is observed after focal injection of kainic acid (KA) into the dentate gyrus (DG). Although the hippocampal network of the injected side is likely responsible for EA generation [1], hippocampal slices taken close to the injection site are unable to generate or sustain EA [2]. This suggests that a more complex network may contribute to initiation of EA.

As a major source of direct input to the DG, the superficial layers of the entorhinal cortex (EC) could be a key candidate. The EC appears to be preserved in this focal model as no neuronal degeneration could be detected within three days and up to three weeks after injection of KA. So far, most studies on the role of EC in animal models of epilepsy were performed after systemic application of pharmacological agents.

Hence, we performed simultaneous in vivo LFP recordings at the injection site and the EC to identify its contribution to EA. We characterized the relation of LFP signals in different frequency bands and detected a phase shift during EA when compared to interictal episodes as well as control animals. This phase shift between EC and DG network may be critical for input integration into the hippocampus.

Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420).

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**Theme** C - Disorders of the nervous system  
Epilepsy - Circuits and systems

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**First author** Dini, Patrick (poster)

Poster board B14 - Wed 07/07/2010, 11:15 - Hall 1

Session 192 - Network interactions 2

Abstract n° 192.14

Publication ref.: *FENS Abstr.*, vol.5, 192.14, 2010

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**Title** High-resolution mapping of single neurons provides insight into neuron structure and LFP generation

**Text** In the context of network state modulation, variability of targeted stimulation and LFPs, we need to better understand the factors influencing the variability of the electrical properties of individual neurons. These are difficult to assess in situ because of the 3D structure and the invasiveness of multielectrode probes. A model for such studies are generic 2D neuronal networks, which are recorded here with HD MEAs featuring 11k electrodes and a high temporal resolution. Such devices allow us to monitor the spatial structure of external electric field potentials along the soma and the dendritic region of neocortical neurons. Spike triggered averaging was used to compute the footprint of these neurons, which represents the flux of ions through the membrane near the electrodes locations. A study of such footprints allows us to obtain insight into the spatial structure of single neurons, the propagation of the electrical influx and the identification of different types of cells. We furthermore examined potential sources of variability of the electrical field of single neurons, such as spontaneous modulation of the individual neuron activity and the influence of the network activity. It is known that spontaneous and induced activity propagates along several reliable pathways. We therefore studied the influence of such dynamics on the electrical field of individual neurons. Our data could contribute to a better understanding of the origin of LFPs and their interpretation as a reflection of network dynamics. Supported by the German BMBF (FKZ 01GQ0420) and by the EC (NEURO, No. 12788).

**Theme** B - Excitability, synaptic transmission, network functions  
Network interactions - Signal propagation

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**First author** Cardanobile, Stefano (speaker)

Workshop W06-3. Sat 03/07/2010, 12:30 - 15:30 - Room G103

Session 006 - Structure, dynamics and function in large scale neuronal ensembles

Abstract n° 006.3

Publication ref.: *FENS Abstr.*, vol.5, 006.3, 2010

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**Title** Non-linear interactions in populations of spiking neurons

**Text** Dynamic properties of large-scale spiking networks are difficult to study analytically, due to the highly non-linear nature of interactions within and between neuronal populations. A mathematical theory of spiking network dynamics would indeed be instrumental to understand the computational properties of biological or simulated networks, and to develop procedures for the analysis of real data. We explain how such a theory can be obtained by resorting to a probabilistic description of local populations of spiking neurons [1]. Specifically, we introduce a Wiener cascade model, roughly equivalent to an integrate-and-fire neuron with exponential escape noise. Interacting populations of such units can be dealt with analytically in terms of continuous rate equations that very precisely capture their expected non-linear dynamics [2].

In a recent study, we could further exploit these results to predict and mathematically characterize the computational properties of various spiking neuronal systems, on different levels of the central nervous system [3]. First, we discuss an implementation of a winner-takes-all dynamics and use it to construct a spiking network that is able to discriminate the direction of moving stimuli. Second, we construct a simple model for the interaction between striatum and basal ganglia, which leads to a Lotka-Volterra oscillator and can account for some properties of the oscillations observed in Parkinson's disease.

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