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KEYNOTE SPEAKER PRESENTATIONS

A1

Studying large-scale brain networks: electrical stimulation and neural-event-triggered fMRI

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BMC Neuroscience 2013, **14(Suppl 1):**A1

The brain is "the" example of an adaptive, complex system. It is characterized by ultra-high structural complexity and massive connectivity, both of which change and evolve in response to experience. Information related to sensors and effectors is processed in both a parallel and a hierarchical fashion. The connectivity between different hierarchical levels is bidirectional, and its effectiveness is continuously controlled by specific associational and neuromodulatory centers. In the study of such systems one major problem is the adequate definition for an elementary operational unit (often called an "agent"), because any such module can be a complex system in its own right and may be recursively decomposed into other sets of units. A second difficulty arises from the synergistic organization of complex systems and of the brain in particular. Synergy here refers to the fact that the behavior of an integral, aggregate, whole system cannot be trivially reduced to, or predicted from, the components themselves. Localizing and comprehending the neural mechanisms underlying our cognitive capacities demands the combination of multimodal methodologies, i.e. it demands concurrent study of components and networks; one way of doing this, is to combine invasive methods which afford us direct access to the brain's electrical activity at the microcircuit level with global imaging technologies such as magnetic resonance imaging (MRI). In my talk, I'll discuss two such methodologies: Direct Electrical Stimulation and fMRI (DES-fMRI) and Neural-Event-Triggered fMRI (NET-fMRI).
DES-fMRI can be used in hopes of gaining insight into the functional or effective connectivity underlying DES-induced behaviors. Yet, our first findings suggest that DES has an important limitation: It clearly demarcates all monosynaptic targets of a stimulated site, but it largely fails to reveal polysynaptic cortico-cortical connectivity.
NET-fMRI, on the other hand, appears to offer great potential for mapping whole-brain activity that is associated with individual local events. In the second part of my talk, I'll describe the characteristic states of widespread cortical and subcortical networks that are associated with the occurrence of hippocampal sharp waves and ripples; the brief aperiodic episodes associated with memory consolidation.

A2

The influence of metabolic energy on neural computation

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

A3

Rescuing the spike

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Sensory and motor variables are represented by large populations of neurons. We hypothesized that these representations are constrained such that they can be read-out linearly (synaptic integration) while limiting the metabolic cost. Such framework can predict many aspects of neural tuning,

the population fluctuations and the size of the basin of attraction that thus determines the onset and lifetime of persistent activity states. Moreover, individual neuronal activity turns out to be very irregular, switching between long periods of low firing rate to short burst-like states. We show that this is an effect of the strong coupling strength in the network combined with the finite memory time constant of the neurons. Thus, such irregular neuron dynamics can be a pure network phenomenon, and do not require intracellular bistability or additional high-variability noise as previously suggested.

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Electrodiffusive model for neuronal and astrocytic ion concentration dynamics

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Electrical signaling in neurons is typically modeled using the cable equation, where dendrites or axons are represented as one-dimensional electrical cables [1]. The cable model is based on the assumptions that the electrical currents along the cable are negligibly affected by (i) diffusion (due to ion concentration gradients), and (ii) variation in resistivities (due to varying ion concentrations). An electrodiffusive model, based on the Nernst-Planck equations, has been developed for situations when these assumptions do not hold [2]. Like the standard cable model, the electrodiffusive model assumes that transport phenomena are essentially one-dimensional. Unlike the standard cable model, the electrodiffusive model explicitly includes ion-concentration dynamics and its effect on diffusive currents and resistivities.

A limitation with the model [2] is that it only considered intracellular dynamics, whereas extracellular conditions were assumed to be constant. The extracellular space (ECS) comprises only about 20% of the total tissue volume, whereas the remaining 80% is the intracellular space (ICS) of various cells. When groups of cells perform similar functions simultaneously, the impact on ionic concentrations may therefore be of the same order in the ICS and ECS. For instance, during periods of intense neural signaling, the extracellular K^+-concentration may locally increase by several millimolars. Clearance of excess K^+ likely depends partly on diffusion in the ECS, partly on local uptake via astrocytic K+-uptake mechanisms, and partly by intracellular transport within astrocytes [3]. To model such processes, we need an electrodiffusive formalism that includes both the ICS and ECS explicitly.

Here, we derive a simple, general mathematical framework for modeling the dynamics of the membrane potential (v_M) and the ion concentrations (c_k) for a set (k) of ionic species in an intra- and extracellular domain. The formalism is based on the constraint of electroneutrality, except in the thin Debye-layers surrounding the capacitive membrane. Like the one-domain model [2], the formalism ensures (i) a consistent relationship between v_M and c_k, and (ii) accounts for diffusion and concentration dependent variations in resistivities. Unlike the one-domain model, the formalism ensures (iii) global particle/charge conservation, and (iv) that the charges on either side of a piece of membrane must be equal in magnitude and opposite in sign. The latter constraint is implicit when the membrane is assumed to be a parallel plate capacitor, an assumption made in most models of excitable cells (see e.g., (1-3, 16)).

The formalism was implemented in a model of ionic exchange between astrocytes and the extracellular space. By simulations, we estimated the contribution of astrocytes in K+ removal from high concentration regions, and revealed a (to our knowledge) novel mechanism that astrocytes may utilize to remove K^+ from extracellular high concentration regions.

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P123

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

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Pattern formation, i.e., the generation of an inhomogeneous spatial activity distribution in a dynamical system with translation invariant structure, is a well-studied phenomenon in neuronal network dynamics, specifically in neural field models. These are population models to describe the spatiotemporal dynamics of large groups of neurons in terms of macroscopic variables such as population firing rates. Though neural field models are often deduced from and equipped with biophysically meaningful properties, a direct mapping to simulations of individual spiking neuron populations is rarely considered. Here, we consider networks with regular topologies, such as rings and lattices, where neuron positions are distributed on regular grids.

Neurons have a distinct identity defined by their action on their postsynaptic targets, i.e., they act either excitatorily or inhibitorily. When the distribution of neuron identities is assumed to be periodic, pattern formation can be observed, given the coupling strength is supercritical, i.e., larger than a critical weight.

Intriguingly, this critical weight is strongly dependent on the characteristics of the neuronal input, i.e., it depends on whether neurons are mean-driven or fluctuation-driven, and very different linearizations of the full non-linear system are relevant in order to assess stability.

We present and analyze these two linearizations, one that is derived directly from the leaky integrate-and-fire dynamics [1], the other from linear response theory in the diffusive coupling limit [2,3]. In the subcritical weak-coupling regime both approaches describe the firing rates of individual neurons with equally good precision, and by analysis of the respective linear stability we can predict under what conditions the system becomes unstable to spatial perturbations, and which spatial firing pattern will be assumed.

We moreover analyze the effect of structural randomness by rewiring individual synapses or redistributing weights.

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Ideal-observer models of perceptual contrast enhancement

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In standard ideal-observer models of sensory cue integration [1], the perceptual estimate resulting from the combination of two cues lies in the interval bounded by the estimates of each cue separately. For example, this type of model accounts well for the psychophysical result - observers give an estimate in-between the haptic-alone and the visual-alone estimates, when asked to estimate ridges height with both vision and touch [2]. Nevertheless, a class of perceptual illusion is supposedly not accounted for by this type of model, namely contrast illusion, such as the size-weight illusion [3,4]. In the size-weight illusion, when asked to estimate the weight of two objects of the same mass but not the same size, observers estimate the larger as lighter. Using standard ideal-observer models, we showed that it is possible to account for this class of illusion provided that statistical correlation between each cue estimate is taken into account. Our argument is based on statistical inference models such as linear minimum-variance unbiased estimation, maximum a posteriori estimation, and least relative surprise estimation. This psychophysical model is general as long as the perceptual estimate deals with a physical quantity that is proportional to another physical quantity also available as a cue, such as mass and volume for a given material in the size-weight illusion.

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

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Over the last couple of years, supercomputers such as the Blue Gene/Q system JUQUEEN in Jülich and the K computer in Kobe have become available for neuroscience research. These massively parallel systems open the field for a new class of scientific questions as they provide the resources to represent and simulate brain-scale networks, but they also confront the developers of simulation software with a new class of problems. Initial tests with our neuronal network simulator NEST [1] on JUGENE (the predecessor of JUQUEEN) revealed that in order to exploit the memory capacities of such machines, we needed to improve the parallelization of the fundamental data structures. To address this, we developed an analytical framework [2], which serves as a guideline for a systematic and iterative restructuring of the simulation kernel. In December 2012, the 3rd generation technology was released with NEST 2.2, which enables simulations of 10^8 neurons and 10,000 synapses per neuron on the K computer [3].

Even though the redesign of the fundamental data structures of NEST is driven by the demand for simulations of interacting brain areas, we do not aim at solutions tailored to a specific brain-scale model or computing architecture. Our goal is to maintain a single highly scalable code base that meets the requirements of such simulations whilst still performing well on modestly dimensioned lab clusters and even laptops.

Here, we introduce the 4th generation simulation kernel and describe the development workflow that yielded the following three major improvements: the self-collapsing connection infrastructure, which takes up significantly less memory in the case of few local targets, the compacted node infrastructure, which causes only negligible constant serial memory overhead, and the reduced memory usage of synapse objects, which does not affect the precision of synaptic state variables. The improved code does not compromise on the general usability of NEST and will be merged into the common code base to be released with NEST 2.4. We show that with the 4g technology it will be possible to simulate networks of 10^9 neurons and 10,000 synapses per neuron on the K computer.

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Noise decouples covariances from interaction strength

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Correlated neural activity is a known feature of the brain [2] and evidence increases that it is closely linked to information processing [1]. In our recent work we have shown how to map different network models, including binary networks, onto linear dynamics [4]. For binary neurons the mean-field approach takes random fluctuations into account to accurately predict the average activity in such networks [5]. Expressions for covariances follow from a master equation [3]. Binary neurons with a Heaviside gain function are inaccessible to the classical treatment [3]. Based on our earlier

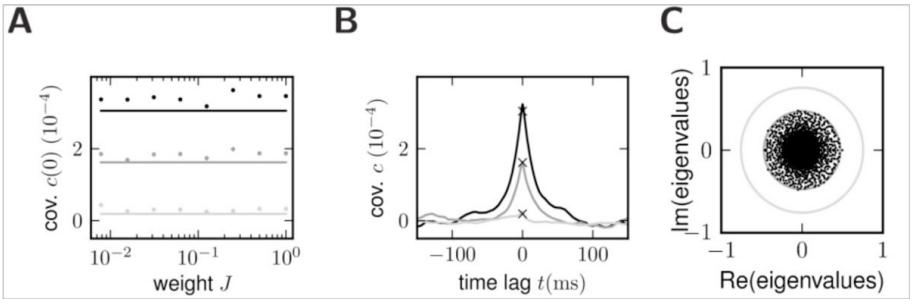

Figure 1(abstract P164) A Zero time-lag cross covariance averaged over pairs of excitatory (black) and inhibitory (light gray) cells and of one excitatory and one inhibitory neuron (gray) in simulation (dots) and theory (lines). **B** Cross covariance functions averaged as in A (same gray code) obtained from simulations at one coupling strength. Crosses show the analytical prediction. **C** Set of eigenvalues of a random connectivity matrix after linearization (black dots) with the corresponding spectral radius (gray circle) and the maximum radius for any synaptic strength (light gray circle).

preliminary results [6] here we show how random fluctuations generated by the network effectively linearize the system of binary neurons, including the case of the Heaviside gain function, and how they implement a self-regulating mechanism which renders population-averaged covariances independent of the synaptic coupling strength. Figure 1A, B illustrate this invariance.

The mechanism is based on the increase of fluctuations in the input signal in proportion to the synaptic weight. The fluctuations cause portions of the gain function with smaller slope to be visited more frequently, effectively reducing the transmission gain. This keeps the linearized system away from instability, with the eigenvalues of its effective connectivity matrix bounded by a constant less than unity (see Figure 1C). Although of local origin the mechanism controls global features of the network dynamics.

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Impact of inhibition in striatal decorrelation of cortical neuronal avalanches

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The Basal Ganglia represent subcortical structures that have a crucial role in determining when a given motor program should be selected and called into action [1]. The input region of the Basal Ganglia (the striatum) contains several distinct cell types. 90-95% of them are medium spiny projection neurons (MSNs) that have high threshold for activation and represent the sole source of the output. There is also a small population of fast-spiking interneurons (FSIs) that receive inputs from a wider range of distinct cortical regions compared to projection neurons [2]. Two sources of GABAergic inhibition onto MSNs are the feedforward inhibition via the FSIs and the feedback inhibition from the axon collaterals of the MSNs themselves. Feedforward inhibition is very powerful and may filter cortical information transmitted by striatal projection neurons [3]. In contrast, feedback inhibition between pairs of MSNs acts predominantly at the distal dendrites, but may still significantly control the overall level of activity of the spiny neurons [4].

We simultaneously recorded local field potentials (LFPs) in the cortex and striatum in order to determine how striatum processes cortical neuronal avalanches. Cortical neuronal avalanches represent activity clusters with a cluster size distribution that follows a power law with exponent -1.5 [5]. Analysis of experimental data revealed that activity clusters in striatum also follow power law distributions, but with an exponent significantly lower than what is observed in the cortex [6]. To understand what controls the LFP statistics observed in experiments, we developed an abstract model of the cortico-striatal network. We investigated to what extent the connectivity pattern between cortex and striatum as well as the inhibition within striatum can explain the experimental results [7]. Our model predicts that striatal inhibition plays a prominent role in shaping the observed striatal dynamics and decorrelating the striatal responses to cortical neuronal avalanches. To understand the contribution of feedforward vs feedback inhibition to the dynamics, we extended our abstract model to spiking networks. We used the model to quantify the role of feedback and feedforward inhibition for decorrelating MSNs, and preliminary results suggest that FSIs play a significant role.

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Modeling brain functional connectivity at rest

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Well organized spatio-temporal low-frequency fluctuations (< 0.1 Hz), observed in blood-oxygen-level-dependent (BOLD) signal during rest, have been used to map several consistent resting state networks (RSNs) in the brain [1-3]. It has been hypothesized that these correlated fluctuations reflect synchronized variations in neural activity of particular brain areas, which are dynamically coupled to one another forming functional connections within networks of brain. Furthermore, it has been suggested that resting state functional connectivity (FC) is strongly shaped by underlying anatomical connectivity (AC). However, although RSNs reflect anatomical connections between brain areas comprising the networks in focus, FC cannot be understood in those terms alone [4]. Here, we combine experimental and modeling approach to investigate dynamics underlying correlated behavior of distant cortical regions and formation of the so called functional networks. We aim to address complicated interplay between network structure, dynamics of its components and emerging global behavior, as key ingredients of the networks complexity [5]. We study how functional connectivity arise from anatomical connections and compare obtained data with the networks simulated on the empirically derived FC networks from resting state fMRI data. We compare two distinctive networks: one with 90 brain regions defined using the Automated Anatomical Labeling (AAL) template [6], and another with 100 regions organized into seven distinctive resting state functional networks [7]. We choose to model local network dynamics by excitable FitzHugh-Nagumo oscillators subject to uncorrelated white Gaussian noise and time-delayed interactions to account for the finite speed of the signal propagation along the axons. We discuss FC between brain regions without apparent anatomical connections, exploring dynamics that underlie these correlations.

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

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How could phasic variations in dopamine level affect the learning outcome of a spiking neural network? How may neuromodulation affect the network's instantaneous response to simultaneously arriving glutamatergic inputs? How may this depend on the brain regions involved?
In our spiking phenomenological model for signal transmission across the synapse and along the dendritic tree, we propose a new approach for the influence of dopamine-like neuromodulators on the ascribed aspects, which unifies diverging views on its role in (reinforcement) learning and (attentional) contrast.
We call into question the common practice of simulating dopaminergic influence on an STDP rule as a third factor, and instead show how an instantaneous effect of a dopamine-like neuromodulator on postsynaptic activity can also lead to reinforced learning outcomes.
As the phasic change of neuromodulator needs to be present during glutamatergic transmission in our model, we do not account for delayed reward as stated in the distal reward problem. Instead, we assume an involvement of hippocampus and cortical working memory for long delays of reward. However, as our transmission-based model does not interfere with the standard two-factor STDP rule, it may be freely combined with existing extensions to STDP if needed.

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Single cell neuro-sensory dynamics: Ca^{2+} chemoreceptor-guided sea urchin sperm motility

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Thomson and Kristan investigated in their study [2] the encoding of stimulus location based on two P cells with overlapping receptive fields and analyzed the differences of their spike counts and latencies. They found that latency differences allow reliable discrimination of touch locations with a distance of 4°. In contrast, discrimination based on spike count differences requires a touch location distance of 13°.
In order to investigate how the three types of leech mechanosensory cells respond to tactile stimulation, we recorded intracellularly from pairs of these neurons while stimulating the skin mechanically. Tactile stimuli varied in intensity and location. Responses of cell pairs were analyzed by calculating the differences of latencies and of spike counts. Discrimination performance was evaluated for location distances and intensity differences based on a pair-wise classification.
We found:
1. All three types of mechanosensory cells respond to strongly overlapping intensity ranges (≥ 50 mN). Spike count and response latency of all cell types depend on touch intensity as well as location. These results suggest that N cells could be involved in the encoding of touch stimuli.
2. For the estimation of touch location, we found in agreement with Thomson and Kristan [2] that latency difference of both P cells leads to reliable classification of small touch location distances, when touch stimuli of higher intensities (e.g. 50 mN) are used. Locations of touch stimuli of lower intensities (e.g. 10 mN) can better be discriminated based on latency difference of two T cells. Combinations of T and P cell responses do not improve discrimination.
3. Stimulus intensities are optimally discriminated by spike counts of single P cells. Relative response features do not improve the estimation of intensity, neither for pairs of the same type nor for different cell types.
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SPIKY: a graphical user interface for monitoring spike train synchrony

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With the growing availability of multi-unit recordings there is increasing demand for methods which provide the possibility to study similarity patterns of activity across many neurons. Accordingly, a wide variety of approaches to quantify the similarity (or dissimilarity) between two or more spike trains has been suggested. Recently, the ISI- and the SPIKE-distance [1,2] have been proposed as parameter-free and time-scale independent measures of spike train synchrony. The key property of both measures is that they are time-resolved since they rely on instantaneous estimates of spike train dissimilarity. This makes it possible to track changes in instantaneous clustering, i.e., time-localized patterns of (dis)similarity among multiple spike trains. The SPIKE-distance also comes in a causal variant [2] which is defined such that the instantaneous values of dissimilarity are defined from past information only so that time-resolved spike train synchrony can be estimated in real-time.
For both the regular and the real-time SPIKE-distance, there are several levels of information reduction [3]. The starting point is the most detailed representation in which one instantaneous value is obtained for each pair of spike trains. This results in a matrix of size 'number of sampled time instants' × 'squared number of spike trains' (i.e. $\#(t_n)N^2$). By selecting a pair of spike trains one obtains a bivariate dissimilarity profile whereas the selection of a time instant yields an instantaneous matrix of pairwise spike train dissimilarities which can be used to divide the spike trains into instantaneous clusters, i.e., groups of spike trains with low intra-group and high inter-group dissimilarity. Another way to reduce the information is averaging. The spatial average over spike train pairs yields a dissimilarity profile for the selected (sub)population, whereas temporal averaging leads to a bivariate distance matrix for the selected interval or the selected trigger points. Finally, application of the remaining average results in one distance value which describes the overall level of synchrony for a group of spike trains over a given time interval.
The Matlab source codes for calculating and visualizing both the ISI- and the SPIKE-distance have been made publicly available and have already been widely used in various contexts. However, the use of these codes is not very intuitive and their application requires some basic knowledge of Matlab. Thus it became desirable to provide a more user-friendly and accessible interface. Here we address this need and present the graphical user interface SPIKY [4,5]. This interactive program facilitates the application of the ISI- and the SPIKE-distance to both simulated and real data. SPIKY includes a spike train generator for testing purposes, as well as masks for selecting the analysis window and the neuronal subpopulation of interest. Once given a set of spike train data, it calculates the desired measure and allows visualization of all the different representations mentioned above (such as measure profiles and pairwise dissimilarity matrices). It even includes the possibility to generate movies which are very useful in order to track the varying patterns of (dis)similarity. Finally, we also have increased the high computation speed even further by transferring the most time-consuming parts of the original Matlab code to Matlab executables (MEX) with the new subroutines written in C.
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Synfire chains and gamma oscillations: two complementary modes of information transmission in cortical networks

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Background: The cortex is thought to process sensory stimuli from the environment by flexible routing of neuronal activity across a hierarchy of functionally specialized neuronal networks. This routing necessitates mechanisms that allow for high fidelity communication of neuronal activity between these networks [1]. It was suggested that synchronization of spiking activity plays a pivotal role in this communication process, based on which two seemingly different mechanisms were proposed. The synfire chain hypothesis postulates the existence of highly organized divergent/convergent connections, which allow the generation and faithful transmission of synchronous spike volleys generated by common drive from presynaptic neurons [2]. By contrast, another model proposes that communication between different brain areas is achieved by creating consistent phase relations between population level oscillations entrained by distinct neuronal networks. These oscillations emerge as a consequence of local interactions between excitatory and inhibitory neurons. So far, synchronization driven by oscillations and synchronization due to a common drive have been considered as dynamical processes of a different

nature. Here, we outline a new theoretical framework, which views the appearance of coherent oscillations as a manifestation of common input synchrony spreading along diluted feed-forward networks (FFNs), which, initially, fail to create stable propagation of excitatory spike volleys due to insufficient weight and number of connections. We have tested this working hypothesis by implementing numerical simulations of diluted FFNs. In our network model, each FFN group consisted of recurrently connected leaky integrate-and fire neurons with an excitation-inhibition ratio of 4:1.

Results: In our simulations, external stimulation with rhythmic pulse packets was followed by network activity oscillations, which were a consequence of mutual interactions between the excitatory and inhibitory pools. These oscillations progressively amplified in strength with each new input presentation. They synchronized excitatory activity in each FFN pool and facilitated the propagation of excitatory spike volleys along weak and sparse divergent/convergent connections. Several oscillation cycles were needed to transmit spike volleys across the entire FFN in contrast to synfire activity, in which excitation is propagated in one sweep. We also hypothesized that the precise timing inherent to coherent oscillations may induce synaptic potentiation, which would reduce the number of oscillation cycles necessary to propagate synchrony and drive the network towards synfire chain dynamics. Indeed, our simulations confirmed that an increase of synaptic weights between groups of the FFN transformed oscillation chains into classical 'synfire chains', in which synchrony was transmitted in a single wave. In summary, we propose a conceptual link between the concepts of synfire chains, coherent oscillations and synaptic plasticity. We suggest that coherent oscillatory dynamics presents an immature case of spike volley transmission across multiple neuronal networks, which may lead to secured transmission, without the need for oscillations, via the results of synaptic plasticity.

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Effects of single neuron firing patterns on network dynamics

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The interaction of activities at multiple levels, ranging from molecular to cellular to network level, is a fundamental aspect of information processing in the brain. In vitro studies show that the firing patterns of neurons in response to an input current are highly diverse. Recent experimental data suggest that parvalbumin and somatostatin expressing interneurons, differing in their connectivity and firing patterns, influence the orientation selectivity of pyramidal neurons differently. However, under which conditions low-level neuron properties like spiking patterns affect the network activity dynamics remains to be understood. Therefore, we studied the dynamics of spiking neuronal networks (SNN) by systematically varying the firing pattern of inhibitory interneurons from fast spiking to bursting, keeping the excitatory population as regular spiking.

In an SNN with sparse and homogeneous connectivity, global network properties such as population synchrony and mean firing rates did not show significant differences with variations in the type of inhibitory neurons. Interestingly, local properties such as burstiness of the individual neurons were determined by the global network state for instance in the asynchronous activity state both fast spiking neurons and bursting neurons exhibited similar spiking patterns. Thus, the global network state, instead of the neurons' intrinsic properties, determined the spiking pattern of the neurons.

Because the effect of neuronal spike patterns could be obscured by the random and homogeneous connectivity, we considered two specific deviations: First we introduced hubs into the network by allowing the fast spiking and bursting neurons to form up to eight times more connections. The overall out degree in such a network with hubs was kept the same as in the random homogeneous networks. Such topology did not affect the network dynamics qualitatively.

Next, we separated the inhibitory population into two sub-populations, connected in feedforward (FF) and feedback (FB) manner. With this connectivity scheme, we initially found that neuronal spike patterns can affect the oscillation frequency of the population activity if the fast spiking inhibitory neurons were present as the FB population and FF population was altered between the various ratios of fast spiking and bursting neurons. However, the differences in the oscillations frequency could be attributed to the different gain of the two types of neurons. That is, it was an effect of differences in the degree of recurrent inhibition and not that of spiking activity patterns.

In summary, our results suggest that for random homogeneous and the two types of inhomogeneous recurrent SNNs, the individual neuronal patterns do not affect the global dynamics. Any differences in global dynamics with change in single neuron firing patterns is due to the ensuing difference in the firing rate of the neuron types. On the other hand, the global activity state influenced the local parameters like burstiness.

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Controlling the Go / No-Go decision threshold in the striatum

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A typical Go/No-Go decision is thought to be implemented via the activation of direct and indirect pathways in the basal ganglia. Indeed, optogenetic activation of the direct pathway increased ambulation, whereas that of the indirect pathway induced freezing [1]. Striatal neurons participating in these two pathways express D1 and D2 type dopamine receptors [2]. Furthermore, D1 and D2 expressing MSNs also differ in their passive properties [2] and recurrent connectivity [3]. To understand striatal function it is, therefore, important to identify factors that regulate the balance of activity in D1 and D2 MSNs. Here we used both, a reduced firing rate model and numerical simulations of the striatal networks to study the dynamic balance of spiking activity in D1 and D2 MSNs.

Specifically, we show that: (i) Because D1 MSNs receive higher recurrent inhibition from FSIs [4] and D2 MSNs [3], they require a stronger cortical drive to overcome this inhibition. (ii) D1 and D2 firing rates change non-monotonically as a function of cortical input rates. For small cortical input rates, D1 MSNs have higher firing rates than D2 MSNs, due to the stronger synaptic input from cortex. For higher cortical input rates, D2 MSNs activity surpasses D1 MSN activity because cortical input rate is no longer sufficient to balance the strong inhibition coming from FSIs. The cortical rate at which D2 MSNs activity exceeds that of D1 MSNs is termed the decision threshold. (iii) The decision threshold depends on the strength of cortico-striatal synapses and the firing rate of FSIs. (iv) The STN could control the decision threshold via the massive pallidostriatal back-projections [5], via inhibition of the FSIs. (v) Finally, the difference between D1 and D2 firing rates is also modulated by the input correlations [6].

These observations help us to explain several experimental and behavioral findings involving the basal ganglia. The model suggest that under dopamine depletion conditions, even for weak cortical inputs, D2 MSNs activity is higher than D1 MSNs, which is consistent with the fact that Parkinson's disease (PD) patients have difficulty in initiating voluntary

actions. We also observed that dopamine depletion reduced the parameter regime supporting D1 MSNs activation, by shifting the decision threshold towards lower cortical inputs. This suggests that under dopamine-depleted conditions, the striatum would require arbitration by the STN-GPe network, even for a low conflict task, providing a plausible explanation of increased reaction times in PD patients. Finally, increased activity in GPe under the influence of deep-brain stimulation (DBS) could also reduce the activity of FSIs on an average, thus shifting the decision threshold towards higher cortical input rates. Taken together, the model provides a mechanistic explanation of impulsive behavior in PD patients with DBS.

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

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The neuronal network of the nematode *Caenorhabditis elegans* (*C. elegans*) is comprised of 302 sensory, motor and inter-neurons. Near-complete connectivity data for the gap junctions and chemical synapses connecting these neurons (its connectome) have been resolved [1]. In addition, current experiments measure the response of various neurons to input stimuli. A description of these responses cannot be drawn from the static connectivity data alone. These studies suggest that computational modeling can assist in describing neural dynamics and their relation to the connectome. However, simulations of C. elegans neural dynamics are challenging since the single neuron dynamics do not appear to be characterized by standard spiking neuron models. Indeed, genomic sequencing and electro - physiological studies have consistently failed to observe classical Na^+ action potentials in C. elegans neurons [2].

Our study combines the known connectome data [1] with a physiologically appropriate neuron model [3] to simulate the dynamics of the full neural network in response to stimuli over time. We model single neuron dynamics by graded electrical potentials using the findings of electrophysiological studies and biophysical considerations [3]. Since the parameters of the model are not well known, we first investigate their effect on the behavior and stability of the system. We use a genetic algorithm to explore this high dimensional parameter space. Once the parameters are set, we investigate the input-to-output response of the network. Specifically, we stimulate input sensory neurons, as is often done in experiments, and characterize the response elicited in the network. This is the first study of its kind *computationally* relating the sensory input with the resultant dynamical behavior of the inter- and motor-neurons. Figure 1 shows a prototypical example of the neural response when a chemosensory neuron AQR positioned in the head receives periodic input. A dominant pathway (A→B→C) shows how the signal propagates through inter-neurons to the tail chemosensory neurons PHAL/R. This example demonstrates that AQR and PHAL/R are highly correlated even with no direct static connection in the connectome. We call such a connection a "dynamical connection" between neurons, and our computational study discovers such dynamical connections.

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

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Prior to receiving visual stimuli, spontaneous, correlated activity called retinal waves drives activity-dependent developmental programs. Early-stage waves mediated by acetylcholine (ACh) manifest as slow, spreading bursts of action potentials. They are believed to be initiated by the spontaneous firing of Starburst Amacrine Cells (SACs), whose dense, recurrent connectivity then propagates this activity laterally. Their extended inter-wave intervals and shifting wave boundaries are the result of the slow after-hyperpolarization of the SACs creating an evolving mosaic of recruitable and refractory cells, which can and cannot participate in waves. Recent evidence suggests that cholinergic waves may be modulated by the extracellular concentration of ACh [1].Here, we have constructed a simplified, yet biophysically realistic, reaction-diffusion model of cholinergic retinal waves capable of recapitulating wave dynamics observed in mice retina recordings (Figure 1A). The dense, recurrent connectivity of SACs is modeled through local, excitatory coupling occurring via the volume release and diffusion of ACh. This novel approach is used to determine how extracellular ACh may modulate wave activity. In contrast with previous, simulation-based models (*e.g.* the model of Hennig [2]), we are able to use non-linear wave theory (traveling fronts, pulses, singular perturbation analysis, *etc.*) to connect wave features to underlying physiological parameters, making our model useful in determining appropriate pharmacological manipulations to experimentally produce waves of a prescribed spatiotemporal character (Figure 1B).

This is the first mathematical analysis of its type on retinal waves. However, a number of theoretical issues remain unresolved. The distribution of wave sizes has been reported to obey a power-law distribution, suggesting the developing retina may exist in a critical state [2]. Are these findings compatible with our theoretical model? We present preliminary results suggesting that our model possesses a configuration in which wave sizes are distributed according to a power-law (Figure 1C). We adapt analyses typically used in neural field equations to understand the effects of stochasticity and heterogeneity on wave size statistics [3], and therefore provide theoretical arguments characterizing the potential for criticality in retinal development.

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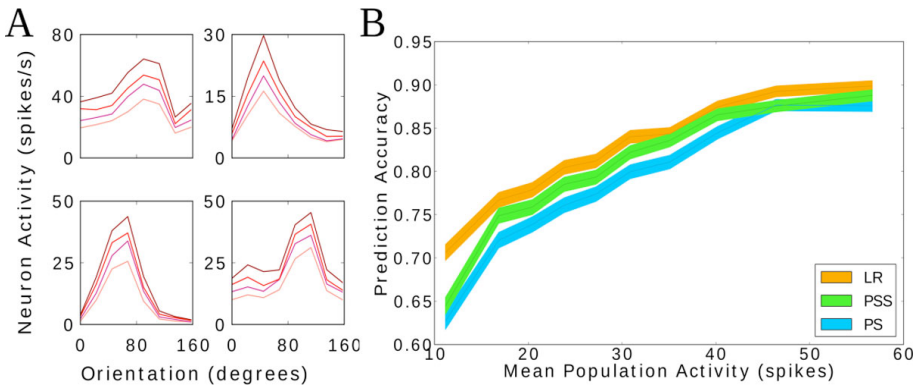

Figure 1(abstract P281) A: TCs conditioned to four different mean population activity bins, ranging from high mean population activity (upper line) to low mean population activity (lower line). B: Decoding performance of stimulus orientation as a function of mean population activity for four different decoding models.

identical. Therefore, spontaneous population activity fluctuations display highly non-random features, boosting TCs by shifts and multiplicative factors that gate information processing.

Results: We found that TCs are shifted and scaled by a multiplicative factor proportional to mean population activity (Figure 1A). The amount of each contribution was neuron dependent. We studied whether the scaling in the TCs induced by population activity fluctuations had an impact on information processing. To this end, we decoded orientation on a trial-by-trial basis from neuronal activity as a function of population activity (Figure 1B). We found that higher mean population activity resulted in better decoding accuracy. This result is surprising because the amount of information conveyed by V1 neurons, even for the same stimulus, depends on the mean population activity.

Methods: The observed boosting properties of TCs motivated us to build a statistical model with Poisson-like neurons that includes a multiplicative scaling factor (PS). We observed that the effect of population activity on TCs is not purely multiplicative, and we introduced therefore a shift (PSS). Our models approached the performance of state-of-art decoding techniques (SVM, and logistic regression -LR-) and provided higher accuracy than other tested decoders based on population vector and independent Poisson neurons. Experimental methods are as in [1].

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

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Recurrent circuits of simple model neurons can provide the substrate for cognitive functions such as perception, memory, association, classification or prediction of dynamical systems [1-3]. In Alzheimer's disease (AD), the impairment of such functions is clearly correlated to synapse loss [4]. So far, the mechanisms underlying this correlation are only poorly understood. Here, we investigate how the loss of excitatory synapses in sparsely connected random networks of spiking excitatory and inhibitory neurons [5] alters their dynamical and computational characteristics. By means of simulations, we study the network response to noisy variations of multidimensional spike-train patterns.

We find that the loss of excitatory synapses on excitatory neurons (decrease in excitatory-excitatory indegree; vertical arrow in Figure 1) lowers the network's sensitivity to small perturbations of time-varying inputs, reduces its ability to discriminate and improves its generalization capability [6].

A full recovery of the network performance can be achieved by firing-rate homeostasis, implemented by scaling up the remaining excitatory-excitatory synapses (horizontal arrow in Figure 1). Homeostasis may therefore explain the absence of clinical symptoms in early AD, despite cortical damage. The onset of clinical symptoms may result from an exhaustion of homeostatic resources.

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Syntax processing properties of generic cortical circuits

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Higher cognitive functioning is assumed to be largely symbolic/representational and compositional in nature. At various processing stages, from perceptual to motor, discrete structural elements with intricate temporal dependencies are combined into increasingly complex constructs [1]. Mapping such complex computational processes to the underlying neuronal infrastructure and assessing the properties of the neuronal system responsible for their implementation is not straightforward, but it is likely to yield important insights into the nature of neural computation.

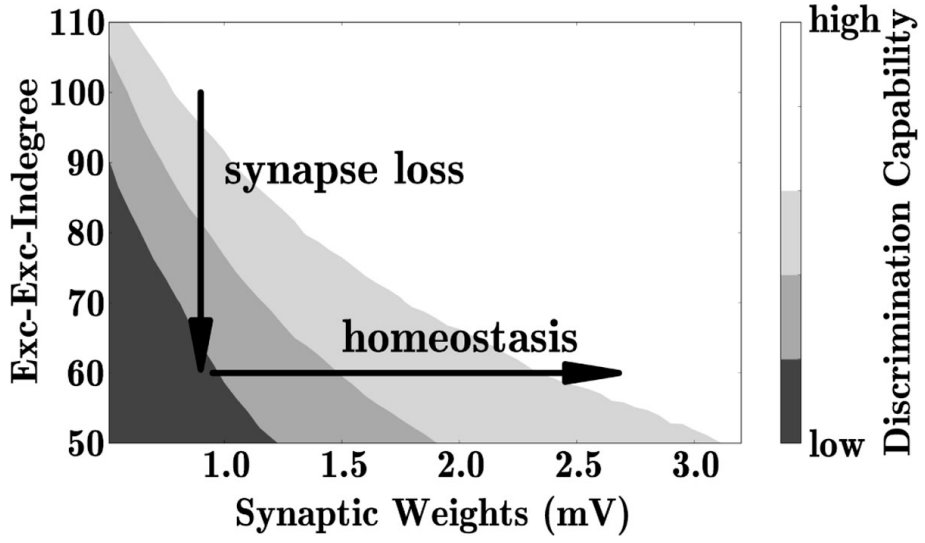

Figure 1(abstract P282) Loss of excitatory-excitatory synapses (vertical arrow) impairs discrimination capability (gray coded). Recovery of discrimination capability by firing-rate homeostasis (scaling up remaining excitatory-excitatory synapses; horizontal arrow).

In order to address these issues, we adopt ideas and formalisms developed by theoretical linguistics to study the nature of rule-like or compositional behavior in the language domain, namely the acquisition of formal (artificial) grammars. The Artificial Grammar Learning (AGL) paradigm has a long tradition in psycholinguistic research (see, e.g. [2] for an overview), as a means to study the nature of syntactic processing and implicit sequence learning.

With mere exposure and without performance feedback, human beings implicitly acquire knowledge about the structural regularities implemented by complex rule systems.

In this work, we investigate to which extent generic cortical microcircuits can support formally explicit symbolic computations, instantiated by the same grammars used in the human AGL literature and implementing various types of local and non-adjacent dependencies between the sequence elements, thus requiring varying degrees of computational complexity and online processing memory to be adequately learned. We use concrete implementations of input-driven recurrent networks composed of noisy, spiking neurons, built according to the reservoir computing framework and dynamically shaped by a variety of synaptic and intrinsic plasticity mechanisms operating concomitantly [3]. Additionally, we compare supervised and unsupervised learning rules for the decoding algorithms, with varying degrees of biological plausibility. We show that, when shaped by plasticity, these models are capable of acquiring the structure of simple (regular) grammars. When asked to judge string legality (in a manner similar to human subjects), the networks perform at a qualitatively comparable level. We uncover which plasticity mechanisms are crucial for the task, with the aim of specifying a minimal model. Furthermore, the capability of the networks to process (bounded) recursive constructions including multiple patterns of non-adjacent dependencies accurately reflects recent results of human performance, highlighting inherent limitations imposed by the nature of neuronal processing.

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P284

Aspects of randomness in biological neural graph structures

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In the past two decades, significant advances have been made in understanding the structural and functional properties of biological networks using graph-theoretic analysis. In general, most graph-theoretic studies are conducted in the presence of serious uncertainties, such as major undersampling of the experimental data. In the specific case of neural systems, however, a few moderately robust experimental reconstructions do exist, and these have long served as fundamental prototypes for studying connectivity patterns in the nervous system. Here, we provide a comparative analysis of several "historical" graphs, including areal connectivity graphs of the cat and macaque monkey cortex [1-3], as well as the neural connectivity graph of the nematode C. elegans [4,5].

While it is a common practice in applying graph-theoretic measures to empirical data to symmetrize the connectivity matrix prior to analysis, here we work with the graphs both in their unmodified directed, and symmetrized undirected forms, focusing on simple structural characterizations of their connectivity. This characterization includes the node degree distributions, the structural equivalence of graph nodes, as well as a nearest neighbor degree and assortativity analysis. All utilized measures are defined for directed graphs, but yield their forms known from the literature when applied to undirected graphs [6,7].

We find that the investigated networks share a strong component of randomness in their structural makeup, suggesting a mechanism of their formation which is much less constrained than that required for scale-free graphs. Specifically, fits of the node degree distributions are in accordance with a gamma model, supporting the idea of a simple local mechanism responsible for generating neural graphs. Secondly, the Euclidean distance of node adjacencies and node degree correlations are consistent with a independent random distribution of node connections for different nodes, but with strong correlations between in-coming and out-going connections for the same node. Finally, we find a weak disassortative tendency in the considered graphs, suggesting that in biological neural

mechanisms have been reported at several sites of the cerebellar model circuitry, thus including plasticity mechanisms not just at parallel fibers (a well-accepted plasticity site) but also at synaptic inputs of deep cerebellar nuclei (DCN) (from mossy fibers (MFs) [1,2]and Purkinje cells (PCs) [3,4]). The Marr and Albus model already hypothesized that parallel fiber (PFs) →Purkinje cell synapses presented both long-term potentiation (LTP) [5,6] and long-term depression (LTD) [5-7] plasticity so as to correlate the activity at parallel fibers with the incoming error signal through climbing fibers. Nevertheless, in subsequent studies, it has been demonstrated that many sites in the cerebellum show traces of plasticity [8-10]. But the way in which those distributed plasticity mechanisms may improve the operational capabilities of the cerebellum is still an open issue.

In this work we propose that the synaptic plasticity of mossy-fiber-to-deep-cerebellar-nucleus-cell and Purkinje-cell-to-deep-cerebellar-nucleus-cell may regulate the effect of Purkinje-cell activity on the cerebellar output, behaving as a distributed homeostatic mechanism [11]. The plasticity at the DCN afferents helps to keep the Purkinje-cell activity in an adequate range independently of the magnitude required for the cerebellar output, thus improving the precision of this output signal.

Since these plasticity mechanisms are capable of adapting the cerebellar behavior in the long-term, it is of necessity the presence of fast feedback for motor activities in order to perform precise movements. Thus, the presented work also explores the control implication that the inferior-olive-to-deep-cerebellar-nucleus-cell connection may possess in conjunction with the previously suggested plasticity mechanisms. As it is widely assumed, the climbing fiber activity that our cerebellar model implements is considered to be a teaching signal (targeting Purkinje cells). But we also explore its potential role as a control signal over the cerebellar output (targeting the deep cerebellar nuclei).

To investigate all these proposals, we have embedded the cerebellar model in a feed-forward control loop which is connected to a simulated 3-degree-of-freedom robot model.

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P330

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

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The relation between neural mass signals, like local field potentials (LFP) or electro-encephalograms (EEG), and the spiking activity of neurons in a network is still poorly understood. Recently, linear temporal filters have been used to map multi-unit activity (MUA) to LFP signals recorded at the same electrode [1]. Similar kernels have been previously identified relating simulated network activity to the human EEG [2]. However, currently there are no theoretical/computational models to explain the form of these filters that map MUA to LFP or EEG.

Here we studied the relation between MUA and LFP in a minimal network model of the neocortex. Using simplified statistical models of neurons [3,4], the firing rate response of neuronal populations to time-dependent inputs can be characterized as that of a high pass filter. At the same time, the LFP recorded in the neocortex can be interpreted as a measure of the summated synaptic input to the population of nearby neurons [5], filtered by the neuronal membranes and the recurrent network [6]. Combining these various filter operations, we arrive at the forward model (LFP to MUA) of a band-pass filter, which can be inverted to predict the LFP from the MUA. Our results explain the form of the experimentally obtained kernels [1] and provide insight into the encoding of a stimulus by local neuronal populations. Furthermore, our theory explains characteristic properties of the neocortical LFP, solely based on effective neuronal refractoriness, membrane filtering and recurrent connectivity.

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P331

Nonlinear dynamics of large-scale activity in "networks of networks"

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As a first step toward understanding the macro-dynamics of brain-like systems, we study the large-scale dynamics of balanced random networks of excitatory and inhibitory integrate-and-fire neurons. Based on the dynamical equations of the model, a mean field approach was previously employed to reduce the dimensionality of the network dynamics [1,2]. Here, we analyze the joint activity dynamics of excitatory and inhibitory populations employing a pair of mutually interacting nonlinear differential equations. In absence of a voltage leak for individual neurons, and for negligible synaptic transmission delay, these equations take the form of Lotka-Volterra equations. These have been used to describe predator-prey systems, corresponding to excitatory and inhibitory populations of neurons in our case. For non-zero identical synaptic transmission delay, we obtain Lotka-Volterra equations with delay. We try to infer the parameters for the non-autonomous differential equations given a dataset from numerical simulations of such a network. Moreover, we attempt to analytically constrain the parameters and compare them with their statistical estimators. Using simulation results, the significance of the nonlinear dynamics becomes obvious in the vector field of excitatory-inhibitory activity, which corresponds nicely with the vector field of the analytical equations.

We have analyzed the stability of the network considering two bifurcation parameters: the relative strength of recurrent inhibition, "g", which

controls the balance between excitation and inhibition in the network, and the intensity of external input to the network, "η". We have found out that for a value of "g" that keeps the exact balance between excitation and inhibition, a bifurcation from unstable to stable network dynamics takes place. This bifurcation separates Synchronous Regular (SR) from Asynchronous Irregular (AI) activity of the network, similar to what was found in a previous study on the same network using a Fokker-Planck approach [3]. The influence of synaptic delays on the reduced dynamics of the network is currently under study.

It has been shown that Lotka-Volterra equations are capable of representing switching dynamics between different states of neural networks [2,4]. Our analysis represents a first step toward analyzing the dynamics of more complex "networks of networks" that are implicated in various cognitive abilities of the brain.

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P332

Going beyond Poisson processes: a new statistical framework in neuronal modeling and data analysis

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Cortical spike trains *in vivo* often show a high irregularity, reflected by a coefficient of variation (CV) close to one [1]. Such irregular single neuron spiking has consequently been associated with Poisson processes, and many models involving neuronal spike trains inherited this ideology. This viewpoint is further supported by the Palm-Khintchine theorem, which states that a superposition of a large number of renewal processes with very small intensity behaves like a Poisson process. It was demonstrated, however, that this theorem doesn't always apply to the superposition of neuronal spike trains [2-4]. Moreover, Poisson processes lack the temporal properties observed in population responses to input modulation either via a stimulus *in vivo* [5] or via electrical stimulation *in vitro* [6].

In this study, we report on new techniques to deal with non-Possonian aspects of stationary neuronal spike trains, as well as non-equilibrium population responses [7], based on Markov point processes (MPP), commonly known as continuous time Markov chains (CTMC). We compute the interspike interval (ISI) distribution by algebraically solving the first passage time problem for MPP neuron models, and compute the transient population responses with a similar technique. The same technique is used to compute exact cross-correlation functions for a shared input paradigm [8]. We advertise MPPs as a new powerful framework in neural network modeling and neural data analysis with many possible applications.

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P333

A computational view of area 3b of primary somatosensory cortex

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We investigated the development of topologically organized representations of a restricted region of skin in the primary somatosensory cortex (SI), more precisely, area 3b of SI. We devised a computational model based on the dynamic neural field theory and on an Oja-like learning rule at the level of feed-forward thalamocortical connections [1]. These connections reach area 3b through subthalamic and thalamic relays that convey information from the Merkel Ending Complexes (MECs), which are mechanoreceptors of the skin responsible for information related to touch and pressure. They have been modeled as a quasi-uniform grid while the rest of the relays have been neglected. Both the critical and the post-critical periods of the SI development [2] have been taken into consideration and the latter has been modeled as a long-term alteration of lateral connections. During the critical period, SI remains highly plastic and is able to cope with a vast number of alterations of the environment or of the body itself. This condition goes on during the post-critical period but in a less effective way [3]. In both periods SI is capable of reorganization in the presence of a cortical lesion [4] (e.g. stroke) or a sensory deprivation condition [5] (e.g. limb amputation). In order to examine if the model is capable of recovery from lesions, both cortical and sensory, we studied three different types of lesions on SI and on skin. As expected, the model is able to cope with such degenerative conditions and is able to recover a lot of the lost functionalities. More precisely, in the case of cortical lesions, neurons that are not affected can recover some of the lost representations while in the case of sensory deprivation, neurons that have lost their preferred input, tend to contribute to neighboring representations. Hence, the model confirms both cases and the mechanism of balance between excitation and inhibition seems to be the key for recovery. Attention is another aspect that has been investigated because of its prominent role in reshaping receptive fields during execution of demanding touch perception tasks [6]. In this context we simulated some attentional mechanisms in order to investigate how attention affects the receptive fields of the model. In the presence of an attentional signal, the model is able to gently adapt its receptive fields according to the position of the stimuli on the skin. On the one hand attention promotes the migration of the distant receptive fields towards the attended area and on the other hand proximal to attended signal receptive fields undergo shrinkage.

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whether further coordination is required to account for the observed multi-cell behaviour.

We developed a descriptive model of phase coding in individual place cells and used this model to investigate the cell assembly dynamics on a linear track. Under the assumption of independent phase coding, key experimental quantities were derived analytically and their relationship to behavioural variables was analysed and compared to experimental data (e.g., [1]). We showed that experimentally established relationships between behavioural variables such as running speed and cell assembly metrics such as the compression factor and lookahead can be reproduced and understood analytically in terms of the collective behaviour of independent phase coding units.

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P403

Probabilistic inference of synaptic dynamics in neocortical microcircuits

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Short-term synaptic plasticity (STP) is highly varied across brain area, cortical layer, cell type, and developmental stage (Reyes & Sakmann 1999). This variability is probably not coincidental and since synaptic dynamics shape neural computations, it suggests an important role of STP in neural information processing (Abbott & Regehr 2004). Therefore, an accurate description of STP is a key step towards a comprehensive understanding of neural systems. Many phenomenological STP models have been developed (Markram et al. 1998), but they have typically been fitted to experimental data using least-mean-square methods. With the Tsodyks-Markram model, we find that for typical synaptic dynamics such fitting procedures may give erratic outcomes. A Bayesian formulation based on a Markov Chain Monte Carlo method was introduced as a solution. This formulation provides the posterior distribution over the model parameters given the data statistics. We discovered that standard STP electrophysiology protocols yielded wide distributions over some model parameters. Based on this result we propose experimental protocols to more accurately determine model parameters. Next, the model parameters were inferred using experimental data from three different neocortical excitatory connection types: Pyramidal Cell-Pyramidal Cell (PC-PC), Pyramidal Cell-Basket Cell (PC-BC) and Pyramidal Cell-Martinotti Cell (PC-MC), (see Figure 1). This disclosed connection-specific distributions, which we used to classify synapses. This approach to determining connection-specific synaptic dynamics provides a more comprehensive representation of STP and unveils novel features from existing data.

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P404

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

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The tilt illusion is a well-studied visual phenomenon, whereby the perceived angle of a center stimulus is misjudged in the presence of a differently aligned surround stimulus (e.g. [1]). The dependence of V1 neuron activity

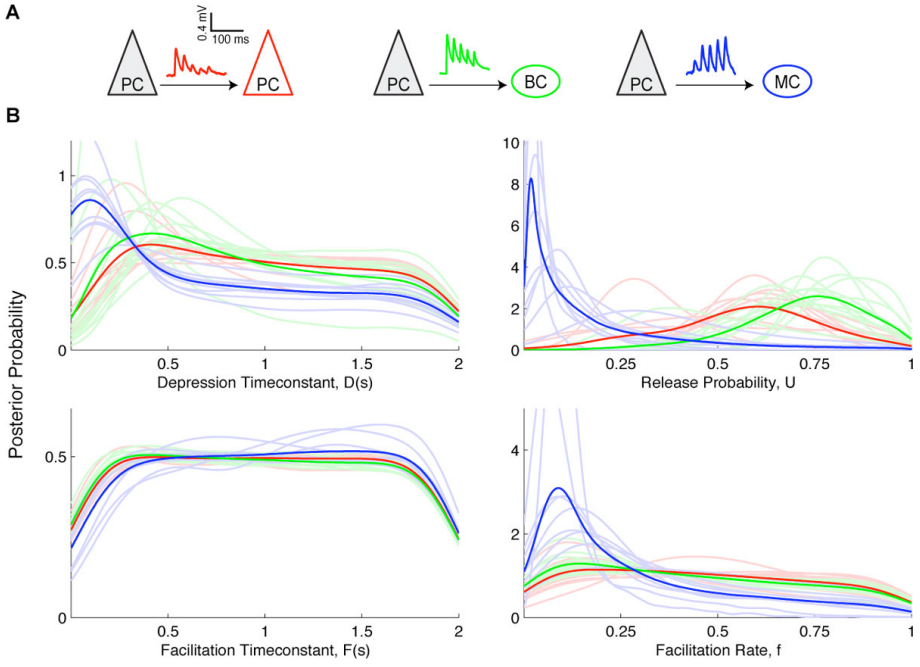

Figure 1(abstract P403) Posterior distributions of STP parameters from experimental data from visual cortex layer-5. (A) Sample experimental STP traces are shown for PC-PC (red), PC-BC (green), and for PC-MC (blue) connections. (B) Marginalized posterior distributions obtained using slice sampling from the three different excitatory connections show connection-specific distributions. Light colored lines show individual distributions, while dark colored lines correspond to their average.

on center-surround interactions has been studied extensively (e.g. [2]). These center-surround interactions can be used to explain the tilt illusion, as they result in tuning curve modulations. When population activity is decoded using these modulated tuning curves, the tilt illusion arises [2]. In this work, we examine two factors affecting the tilt illusion:

First, we examine is the effect of the tuning curve width on the tilt illusion. Tuning curves widths vary widely in vivo [3]. Although changes in tuning curve width due to center-surround interactions have been shown to potentially contribute to the tilt illusion [2,4], how the tuning curve width itself affects the illusion is less well understood. Using a firing rate model, we show here that for narrower tuning curves the tilt illusion lessens, and that it disappears almost completely for narrow, but still realistic, tuning curves.

Secondly, we consider the consequences of recent experimental findings on the tuning of surround modulation. Most models assume that V1 neurons experience most suppression when the surround stimulus is aligned with the neuron's preferred orientation. However, a recent study showed that for the majority of V1 neurons, the suppression effect depends much more on the relation between center and surround orientation, being strongest when they are co-aligned, regardless of the preferred orientation [5]. We use a firing rate model based on [5] to take these new finding into account, and show that, counter-intuitively, the tilt illusion is not impacted, once we control for changes in the tuning curve widths.

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

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On line monitoring and event-driven stimulation are promising techniques for neuroscience studies, especially in behavioral experiments [1]. Weakly electric fish have an electric organ and electroreceptors to generate and detect electric fields [2]. They use their electric pulses to 'see' their environment and also to communicate, changing their inter pulse intervals depending on the behavioral context. We conducted 2 behavioral monitoring experiments with *Gnathonemus petersii* where stimuli were triggered by (1) the fish position in the tank and (2) the fish's own electrical activity.

(1) We built a virtual fence isolating the fish in a given area in the tank, using a camera and video-event driven stimulation. Fast on line tracking was achieved by subtracting consecutive frames. When the fish crossed the virtual fence, electric stimuli were delivered. We observed that artificial stimuli as high frequency signals were more efficient to create a virtual fence than pre-recorded *Gnathonemus petersii* waveforms from another fish.

(2) We monitored in real time the electrical activity of the fish and delivered electric pulses. Fish's electrical activity was acquired in real time from 5 dipoles by a DAQ board and the pulses from the fish were detected by a computer. Once a fish's pulse was detected, a 3 V pulse stimulus was delivered to the fish with a delay τ. Fish responded by shortening their inter pulse intervals (IPIs) for short τ values (Figure 1A) and discharging longer IPIS (Figure 1B) or not altering their IPIs for longer τ. We tested τ = 10 ms, 20 ms, 50 ms, 40 ms, 70 ms and 100 ms and we obtained similar results as shown in Figure 1A and τ = 160 ms, 200 ms, 280 ms, 400 ms with similar results as shown in Figure 1B.

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Assisted closed-loops for brain-computer interfaces

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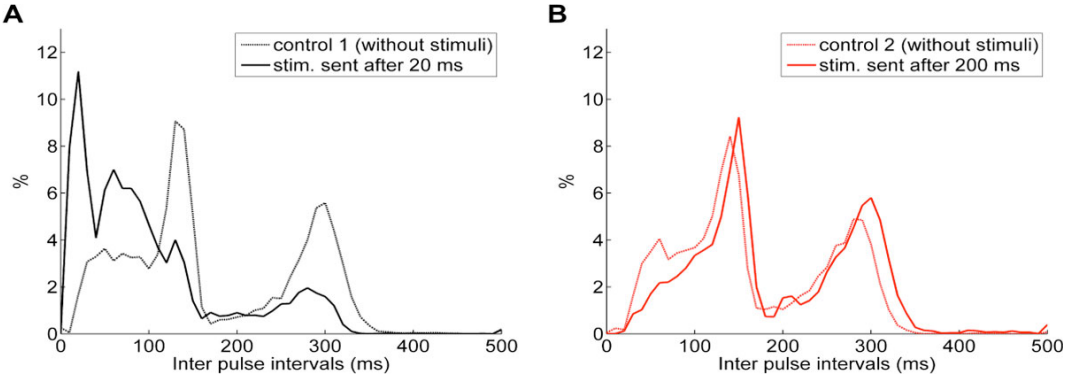

Figure 1(abstract P405) IPI histograms when the fish was without stimulus for 30 min (dashed lines) and stimulated by a electric pulse sent 20 ms (A) and 200 ms (B) after detecting a pulse from the fish (black and red lines respectively). **A**. The fish increased its frequency (shorter IPIs) compared to the control with a new high peak in 11 ms and discharging less 130 ms and 300 ms IPIs. **B**. There was a decrease in the frequency of the fish (longer IPIs) under stimulation, the peaks changed from 140 ms to 150 ms and 290 ms to 300 ms.