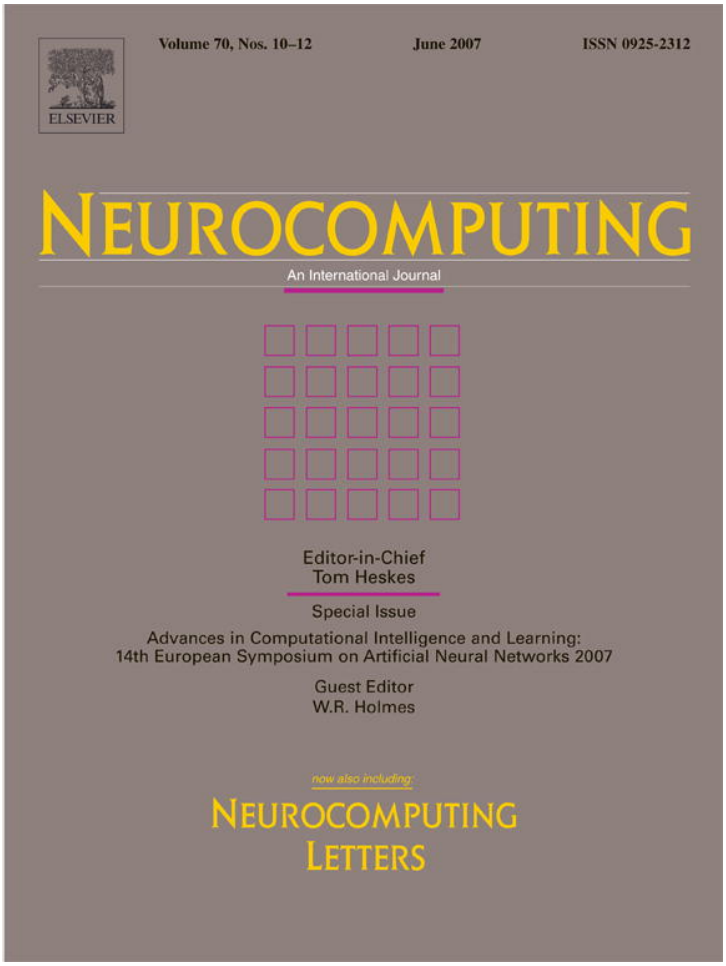

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Emergence of population synchrony in a layered network of the cat visual cortex

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Abstract

Recently, a quantitative wiring diagram for the local neuronal network of cat visual cortex was described [T. Binzegger, R.J. Douglas, K.A.C. Martin, A quantitative map of the circuit of the cat primary visual cortex, J. Neurosci. 39 (24) (2004) 8441–8453.] giving the first complete estimate of synaptic connectivity among various types of neurons in different cortical layers. Here we numerically studied the activity dynamics of the resulting heterogeneous layered network of spiking integrate-and-fire neurons, connected with conductance-based synapses. The layered network exhibited, among other states, an interesting asynchronous activity with intermittent population-wide synchronizations. These population bursts (PB) were initiated by a network hot spot, and then spread into the other parts of the network. The cause of this PB is the correlation amplifying nature of recurrent connections, which becomes significant in densely coupled networks. The hot spot was located in layer 2/3, the part of the network with the highest number of excitatory recurrent connections. We conclude that in structured networks, regions with a high degree of recurrence and many out-going fibres may be a source for population-wide synchronization.

Keywords: Recurrent network; Layered-cortical network; Synchrony; Cat area 17

1. Introduction

Random network models have emerged as a useful tool to understand the dynamical properties of local cortical networks. At its simplest, the cortical networks are modeled as homogeneous networks of spiking model neurons. These simple models have been successful in characterizing the dynamics of cortical networks [5]. However the cortex is not a homogeneous network. It can be clearly identified as a structure composed of up to six layers in sensory cortices, with each layer differing in neuron types, their density and connection probability [11,4,6]. Even though the heterogeneous nature of cortical networks was known for long [2,6], only few studies have attempted to model this heterogeneity [8,12,7].

This small number of studies on heterogeneous network dynamics was primarily due to a lack of detailed information on the neuron type specific inter- and intra-layer connectivity. Recent advances in techniques have greatly increased the knowledge of the cortical neuro-anatomy and a quantitative wiring diagram of the local neuronal network of cat visual cortex was described [3], which provided the first realistic estimate of synaptic connections among various neuron types in different cortical layers. Here we numerically studied the dynamics of the resulting heterogeneous layered network of spiking integrate-and-fire neurons, connected with conductance-based synapses.

2. Network

Binzegger et al. [3] specified the total number of neurons in cat area 17 to be approx. 31×10^6. However, it is still not possible to simulate such large networks, so we downscaled the network to a size of 10,000 or 50,000 neurons. While downscaling the complete network of area 17, we conserved the proportion of excitatory (NE) and inhibitory

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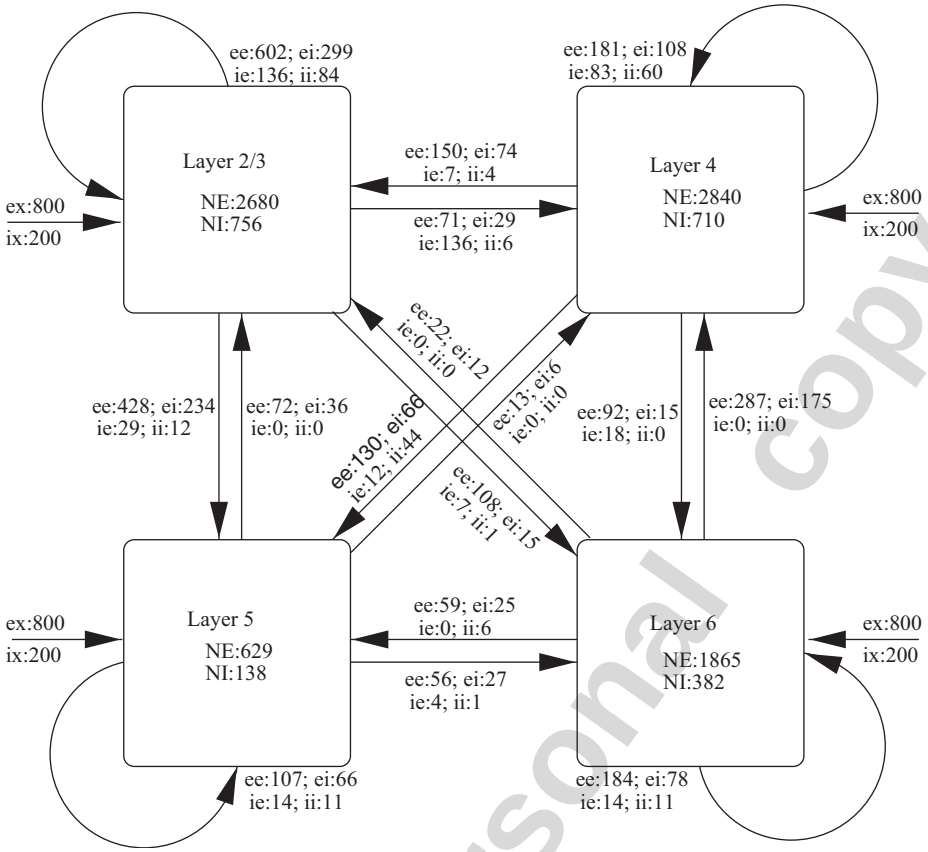

Fig. 1. *Schematic diagram of the network:* NE and NI are the numbers of excitatory and inhibitory neurons, respectively. The labels $xy\{\varepsilon\,\mathrm{ee},\mathrm{ei},\mathrm{ie},\mathrm{ii}\}$ for each arrow indicate the number of synapses of type x projecting onto a neuron of type y, where e stands for 'excitatory' and i for 'inhibitory'.

(NI) neurons across the layers. The number of synapses within a layer was restricted to have a maximum network connectivity (fraction of possible couplings that are realized) of $\varepsilon = \frac{K}{N} = 0.1$. As neurons in different layers received different numbers of synapses due to layer-specific wiring, the resulting connectivity ε was also different in all layers. The neurons were modeled as point neurons with leaky-integrate-fire dynamics. All neurons had identical parameters (membrane capacitance 250 pF, leak conductance 16.7 nS, spike threshold 15 mV above rest). Besides the inter and intra layer connectivity, neurons also received a balanced external input ($\nu_{\mathrm{extGround}}$), mimicking the cortico-cortical inputs the area 17. Synaptic inputs were modeled as conductance transients using the same α-functions (time constant 0.3 ms) for excitation and inhibition. Fig. 1 shows the resulting circuit of a network with 10^3 neurons. The simulations were performed using a parallel kernel of NEST [10].

3. Network dynamics

3.1. Descriptors of network activity dynamics

To characterize the activity states of the network both at population level and single neuron level we used the following state descriptors:

Synchrony in the network was measured by pair wise correlations (PwC)

$$\mathrm{PwC}[C_i, C_j] = \mathrm{Cov}[C_i, C_j]/\sqrt{\mathrm{Var}[C_i]\mathrm{Var}[C_j]}, \quad (1)$$

where C_i and C_j are the joint spike counts.

Mean firing rate was estimated from the spike counts collected over 1 s simulation time, averaged over all neurons in the network.

Irregularity of individual spike trains was measured by the squared coefficient of variation of the corresponding inter-spike interval (ISI) distribution. Low values reflect more regular spiking, a clock-like pattern yields $\mathrm{CV} = 0$. On the other hand, $\mathrm{CV} = 1$ indicates Poisson-type behaviour.

3.2. Dynamics of network activity

In vivo the cortical activity is characterized by irregular spike trains of individual neurons and with a low pairwise correlation among neurons in the network [1]. The membrane potential of individual neurons is close to threshold, and the spikes are elicited by synaptically induced membrane potential fluctuations. In our simulations we excited the network with a balanced input ($\nu_{\mathrm{extGround}}$) to a uniform asynchronous-irregular (AI) activity state [5] with similar average firing rates in each

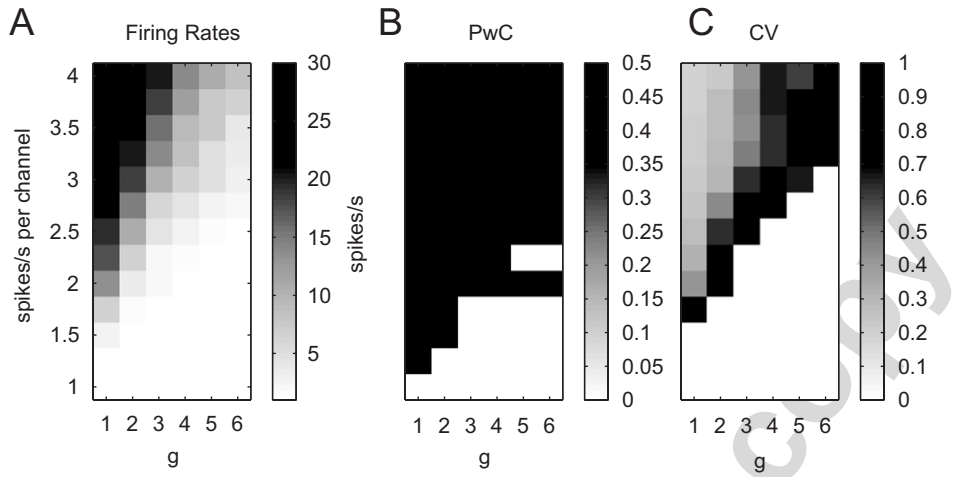

Fig. 2. *Network dynamics:* The network was excited with $\nu_{\text{extGround}}$ to a uniform AI activity state and the stability was studied by systematically varying the ratio of recurrent inhibition and excitation (g) and the external excitatory input (ν_{ext}) which was added to $\nu_{\text{extGround}}$. The firing rates and irregularity (CV) showed the expected behaviour, they increased and became more regular with increasing ν_{ext} while stronger inhibition resulted in lower rates and more irregular spike-trains (A, C). Interestingly, the synchrony (PwC) was already high at low rates ($\rho \approx 6$ Spike/s at mark in A) and irregular spike trains (CV ≈ 0.9).

layer. Here we assumed a uniform AI state (firing rate ≈ 2 spikes/s) across layers. The across-layers distribution of firing rates in real brains is not known. To study the stability of the AI state of the layered networks we systematically varied the ratio of recurrent inhibition and excitation (g) and the external excitatory input (ν_{ext}) which was added to $\nu_{\text{extGround}}$.

The network activity states were characterized using the descriptors introduced above. The firing rates showed an expected trend: they increased with ν_{ext}, while increasing g reduced the firing rate (Fig. 2A). The irregularity of the individual spike trains, increased with the firing rates in the network (Fig. 2C). In the parameter space we explored here, the AI state was observed only in a small region (Fig. 2B). Intriguingly, the pairwise correlation (PwC) showed high values even at low firing rates (Fig. 2A,B).

4. Population burst

The high degree of synchrony at low firing rates was caused by a population wide synchronization in the network. Fig. $3A_1$ shows the raster diagram of the state marked (*) in Fig. 2A. Neurons are arranged in layers, with layer 2/3 on top. The black and gray lines define the excitatory and inhibitory population, respectively, within a layer. The population bursts (PB) occurred in a stochastic fashion (Fig. $3A_1, C_1$), however the frequency and regularity of the PB increased with ν_{ext} (Fig. $3B_1$). The PB followed a stereotypic temporal evolution (Fig. $3A_2$). It started in layer 2/3, invaded layer 5, and then spreaded across the remaining network. To demonstrate that this indeed was a general feature, we performed PB-triggered averaging, (Fig. $3B_2$), which revealed a clear temporal structure, with layers 2/3 and 5 leading the activity (Fig. $3B_2$).

However, why does the PB start in layer 2/3? Layer 2/3 differs from other layers in three main aspects which explain the origin of the PB: it is characterized by highest connectivity (ε), highest recurrent excitation, and maximum out-degree to other layers. Due to the high recurrence any transient synchrony gets amplified, and the high out-degree spreads the synchronous activity from layer 2/3 to other layers, where it eventually causes all layers to fully synchronize and thereby create the PB.

The strong excitatory recurrence of layer 2/3 seem to be important to determine the initiating layer, however, would it also be possible to change the probability of the PB by reducing the effect of recurrence e.g. by reducing synaptic strength? To further support that the excitatory recurrent connections in layer 2/3 (L2toL2EE) are indeed critical for the occurrence of PBs, we reduced their strength by about 50% from 0.25 to 0.13 mV peak amplitude at resting potential. This was reasonable since synaptic strengths are reported to be as low as 0.1 mV and can reach up to several millivolts [13,9]. This weakening reduced the frequency of the PBs (compare Fig. $3C_1$ and C_2), emphasizing the sensitivity of the network dynamics for this particular parameter.

5. Discussion

Using a more realistic network model, based on the circuitry of a hypercolumn of the cat visual cortex [3], we studied the consequences of a layer-specific connectivity on the network dynamics, in particular the stability of the AI state. The layered network exhibited, among other states, an interesting asynchronous activity with intermittent population-wide synchronizations, leading to high pairwise correlation even at low firing rates. The cause of this PB was the correlation amplifying nature of the recurrent network, which becomes significant when the network is densely coupled. As soon as any one layer entered a transient state of high correlations, these correlations were

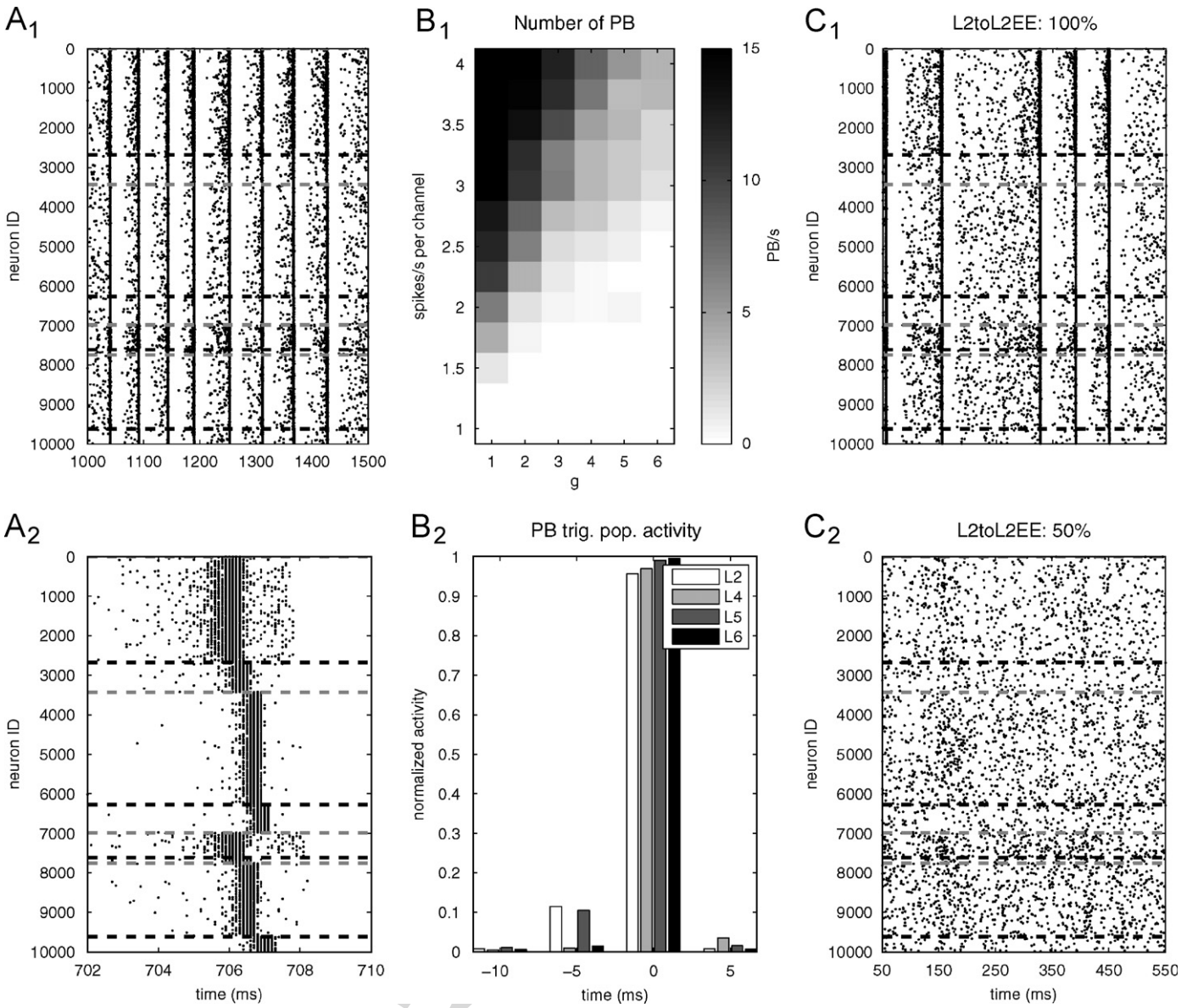

Fig. 3. *Population burst:*Synchronous events (population burst or PB), affecting almost all neurons in a numerical simulation of the network (A_1). Here the black and gray lines define the beginning of the exc. pop. and inh. pop., respectively, of a layer, starting with layer 2/3 at neuron ID = 0. The frequency of the PBs showed the same trend as the firing rate of the network (compare B_1 and Fig. 2A). Zooming into a PB revealed a temporal-laminar structure with layer 2/3 initiating the burst (A_2), that was underpinned by averaging the population activity, triggered on the PBs (B_2) (here and in the following L2 refers to L2/3). The PB was caused by the high excitatory recurrence in layer 2/3. The probability of PBs could be altered by reducing the synaptic strength of the excitatory recurrent connections in layer 2/3 (L2toL2EE). In C_2 the strength of the L2toL2EE connections was reduced by about 50% and a clear reduction in PB frequency could be observed (compare C_1 and C_2).

amplified and transferred to the other layers, resulting in a PB, recruiting all the neurons in the network. The layer of origin was dependent on the level of excitatory recurrent connections, which was highest in layer 2/3. PBs occurred for all the network sizes studied (up to 50,000). However, the characteristics of the PBs (e.g. the probability of their occurrence) were susceptible to changes in the network architecture. So we conclude that in a heterogeneously structured network, the regions with a high excitatory recurrence and large number of out-going connections may become a *hot spot* to induce population-wide synchronization.

In this work we ignored any specific thalamo-cortical and cortico-cortical inputs, and focused on the intrinsic dynamics of the laminar network, elicited by non-specific Poissonian inputs. A natural extension of the work would be to study how the stable stationary state of the network (without the PB) would interact with transient and/or structured thalamo-cortical and cortico-cortical inputs.

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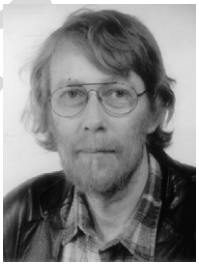

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