ESANN'2005 proceedings - European Symposium on Artificial Neural Networks
Bruges (Belgium), 27-29 April 2005, d-side publi., ISBN 2-930307-05-6.

# Spike-timing-dependent plasticity in 'small world' networks

K. Kube[1], A. Herzog[1], B. Michaelis[1], A. Al-Hamadi[1], A.D. de Lima[2], T. Voigt[2] *

1- Otto-von-Guericke-University Magdeburg
Institute of Electronics, Signal Processing and Communications
P.O. Box 4120, D-39016 Magdeburg - Germany

2- Otto-von-Guericke-University Magdeburg
Institute of Physiology
Leipziger Str. 44, D-39120 Magdeburg - Germany

**Abstract**. Biologically plausible excitatory networks develop a stable synchronized pattern of activity due to synaptic refractoriness (short term depression). The introduction of spike-timing-dependent plasticity (STDP) modifies the weights of synaptic connections in such a way that synchronization of neuronal activity is considerably weakened. By changing network connections to include long-distance connections according a power law distribution ('small world' topology) we found that synchronization could be much better sustained, despite STDP influence.

## 1 Introduction

The occurrence of synchronous oscillatory activity seems to play an important role in the development of the mammalian CNS [1]. Imaging techniques and electrophysiological patch-clamp measurements in cell cultures showed that neocortical neuronal networks develop the same kind of synchronous oscillatory activity in a similar pace as in the intact brain [2]. When synapses are formed, neurons start to show spontaneous activity, first by bursting independently and later by showing simultaneous network spiking. The number of neurons that fire synchronously increases with time. Neurons which do not participate do not survive in culture conditions [3]. These results underline the interrelationship between synaptic development and network activity development. In [4] we have investigated intrinsic conditions and parameters for networks where oscillatory activity is emerging in single cells, and how to control the network externally. It has been shown that a mechanism of synaptic depression is a necessary requirement to render an episodic nature of activity-dependent network excitability by spontaneous activity [5] that is observed in vitro [6]. Here we combine these models with a biologically plausible hebbian learning algorithm, spike-timing-dependent plasticity (STDP), where the modification function depends on the time difference of pre- and postsynaptic action potentials [7]. The simplified models for STDP derived from neurophysiological experiments may improve synchronization in long distant areals [8], and is probably a key element

*Supported by the federal state of Saxony-Anhalt under FKZ 3431A/B/C/D-0302M and FKZ 0037M1/0002A.

when cultured networks are arbitrarily conditioned to oscillatory behavior [9]. Effects like STDP or short-term depression are recently being implemented in VLSI devices [10]. 'Small world' network topologies [11] are simple, flexible, and reminiscent of the connectivity patterns in the brain with an abundance of local intercellular connections and a few have long distance connections. The impact of long-distance connections in epileptic network synchronization of activity was demonstrated in various neuronal models [12].

## 2 Methods

Networks of neurons were modelled using the NEURON environment [13]. Single neurons were represented as electrical models with two compartments for the purpose of decoupling synaptic integration and impulse generation dynamics in different compartments [14]. This model reflects some electrical effects caused by size parameters (for a study of the effects of geometrical structure of a neuron contributing to signal propagating properties, see [15].) The dynamics of the membrane potentials $V_m$ is defined by $C_m \frac{d}{dt} V_m = \frac{E_m - V_m}{R_m} + \sum_k I_{syn} + \frac{V_m - V_m(soma)}{R_a}$ and $C_m \frac{d}{dt} V_m = \frac{E_m - V_m}{R_m} + \frac{V_m - V_m(dend)}{R_a} + I_{HH} + I_\zeta$ for dendrite and soma, respectively, describing the dendritic synaptic input integration and the somatic action potential elicitation. $\frac{E_m - V_m}{R_m}$ is the leaky current, $I_{HH}$ is a current resulting from active Hodgkin-Huxley conductions (standard HH equations [16]) and $\sum_k I_{syn}$ is the sum of all $k$ synaptic currents. $I_\zeta$ is a discrete stochastic component, which causes spontaneous elicitation of action potentials, with highly variable intervals between spikes, erratically but Poisson-distributed [17]. $I_\zeta$ is implemented as a current pulse, activated at distinct time points driven by a Poisson-process. The time of an action potential is determined as the first point in the rising phase that exceeded 0 mV. *Synapses:* Synapses were simulated as an alpha-shaped postsynaptic conductance with refractoriness (differential STD effects): Empirical pharmacological intervention in cell cultures have shown a period of synaptic depression after each burst activity [6]. This is obviously caused by a consumption of some synaptic ressource (e.g. exhaustion of neurotransmitter). By extending the point-process described in [18], the postsynaptic membrane conductances now are also determined by the amount of available transmitter and the synaptic currents are defined by a differential system $\tau_g \frac{d}{dt} a = -a$, $\tau_g \frac{d}{dt} g = -g + a$, and $\tau_m \frac{d}{dt} m = 1 - m$. If an isolated presynaptic event arrives, a peak conductance of magnitude weight occurs at time $\tau_g$ after the event, $m \Leftarrow m - m_{spk}$ and $a \Leftarrow a + e^w$, provided that $m \geq m_{thr}$. The synaptic current then amounts to $I_{syn} = g(V_m - E)$ see Fig. 1(A). Synaptic latency was adjusted via parameter $\tau_m$ to endure about 85ms. In simulation, synaptic currents are calculated with initial values $a(0) = g(0) = 0$, $m(0) = 1$. An example with a test spiketrain is depicted in Fig 1 (A). If $m$ is below a critical threshold $m_{thr}$ spikes are not transmitted. *Synaptic plasticity:* The synaptic weights (peak conductance $g_{max}$) are changed during simulation, using the learning algorithm of synaptic weight changing dependent on time difference of pre- and postsynaptic cells (STDP), the relative weight change is

calculated as in [7], $F(\Delta t) = \{ \begin{smallmatrix} A_+ e^{\Delta t/\tau_+} if \Delta t<0 \\ A_- e^{-\Delta t/\tau_-} if \Delta t \geq 0 \end{smallmatrix}$, see Fig 1. The change of peak conductance is determined by the spike time difference of pre- and postsynaptic cells, exponentially decaying for longer times, with potentiation and depression rates, respectively. *Network:* A network section of $n = 400$ neurons was assembled, arranged on the planar area of 1 mm$^2$ (a simple representation allowing reproducing culture dish plating). Assuming the network structure to be homogenous (and prevent overlying of neuronal positions), the cells positions were de-clustered and scattered uniformly over the section's area using the neural-gas-algorithm according to [19], thus minimizing the spatial entropy of the cellular distribution. *Connectivity:* Each neuron was connected to its $k$ (distance $l$)-

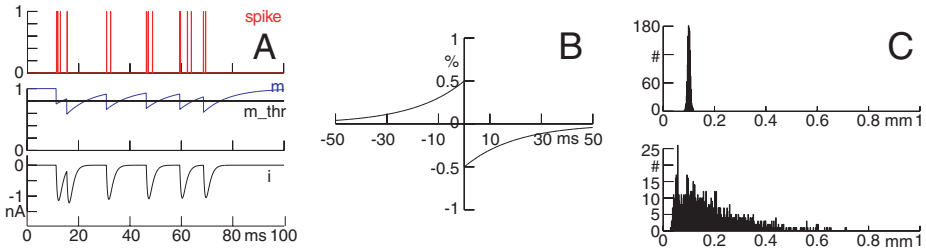


Fig. 1: **A** Simplified alpha-model of synapse with refractoriness: conductance of synapse $g$, transmitter $m$ and transmitter threshold $m_{thr}$ and transmitter consumption $m_{spk}$ per spike. Top panel: incoming spiketrain, middle panel: transmitter $m$ and threshold $m_{thr}$, bottom panel: resulting synaptic conductance. **B** Weight modification function of STDP [7]. **C** Connectivity probability function. Top panel: gaussian distributed locally coupled network, bottom panel: power distributed 'small world' network.

nearest neighbors. The distance $l$ is distributed with probability $\rho$ according to a power-law. This kind of connectivity is consistent both with published anatomical data [21] as well as with theoretical models [11]. Connections are synaptic couplings (model see above) and the conduction time was calculated from the euclidean distance between pre- and postsynaptic cells. The conduction velocity was assumed to be $0.5\frac{m}{s}$ ([20]), which leads to synaptic delays for each pre-post-pair. *Synchronization measures:* Perfect or nearby perfect synchronization can easily be detected by the raster plot of neuron activity, when all neurons fire synchronously (e.g. within a few milliseconds). A mathematical measure to know whether all neurons are located in a network fire synchronously is needed. The 'near spike' measure for neuron $i$ with $j$ generated spikes. For each neuron $i$, each spike $j$, the distances to all other spikes are weightened by a gaussian: $z_{ij} = \frac{1}{m}\sum_{p=1}^{m}\frac{1}{n_i}\sum_{k=1}^{n_i}\frac{1}{\sigma\sqrt{\pi}}e^{\frac{\left(t_i{}^j - t_p{}^k\right)^2}{2\sigma^2}}$ for each neuron leads to a synchronization index of $z = \frac{1}{mn}\sum_{m}^{i=1}\sum_{n}^{j=1} z_{ij}$. Frequency and synchronization of the network activity were analyzed in 10ms-epochs. Average firing frequency was determined by binning the rasterplot in 10ms slices (Fig. 1 (C)).

## 3 Results and Discussion

Neurons were initialized to a resting membrane potential of -65 mV. To initiate spontaneous activity in the network, we triggered single, non-repetitive action potentials by applying driving currents ($I_\zeta$), at time points randomly chosen from a poisson distribution (mean firing rate timing parameter $\lambda$, 0.1nA, lasting 10 ms), were applied. Fig. 2 (D)(E) shows the weight development of synchronization from initial values. The unfolded intrinsic behavior depended on coupling parameters. Two out of three known activity modi described by Netoff et al [12] in circular networks could clearly be distinguished: 'normal' and 'bursting' activity. A 'seizing' mode was extremely unstable in our open network topology. In Fig. 2 the network activity is descending from 'bursting' mode (in which the oscillatory frequency is determined by synaptic refractoriness) into 'normal' mode.

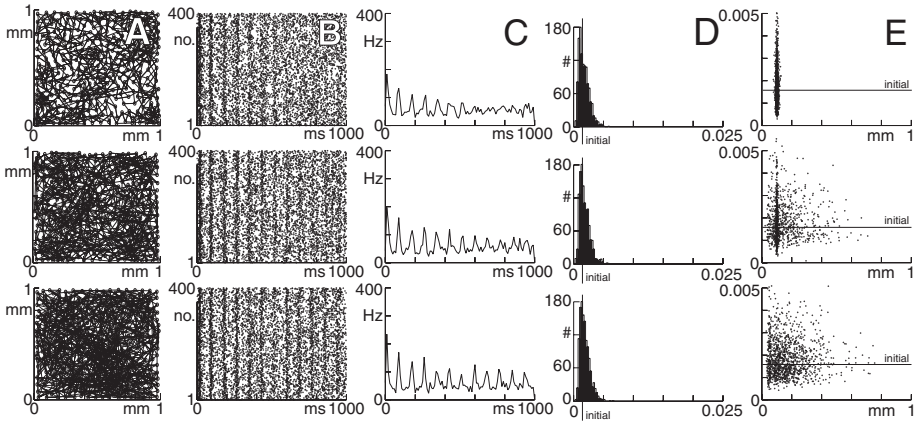


Fig. 2: Influence of Network structure on synaptic development. **A** Network topology, **B** Rasterplot of neuron activity, **C** mean firing frequency, **D** distribution of weights, **E** connection distance vs. connection weight strenght. The ratio of the power-law distributed long-distance connections (see Fig. 1 (C)) amounts to 0, 50%, and 100% for the respective simulation runs.

While the network behavior remained stable, if plasticity was switched off [4], the presence of STDP re-distributed synaptic weights, from homogeneously initial values into Poisson-law distributed weights (Fig. 2). This effect was independent of small-world topology. Interestingly, STDP weakened the synchronization of neuronal activity, probably by mostly non-causal coincidences [7]. Networks having 'small world'-topologies can better sustain synchronous behavior under same conditions. This can be seen in relation to [12]. However, it does not seem to exist a direct correlation between a connections length and weight Fig. 2 (E). Varying the other model parameters in realistic ranges (e.g. number of neurons) had no qualitative effect on the results. The values of the

model parameters used in the simulations, except those mentioned otherwise in the text, are given in Table 1.

## 4 Conclusion

Here we investigated the impact of STDP and 'small world' topologies on the activity dynamics of excitatory networks. In our simulations STDP tended to desynchronize neuronal activity, redistributing weights of connections to fit a Poisson-law. The transformation of the locally coupled network to a network with 'small world' topology by adding a few long connections prevented desynchronization without affecting weight distribution. These results suggest that a small world network topology contribute to maintain synchronized activity in neuronal networks, in spite of destabilizing mechanisms like synaptic plasticity.

| Symbol | Description | Value | Unit |
|---|---|---|---|
| $n$ | number of neurons | 400 | – |
| $I_\zeta$ | current,duration | 0.1,10 | $nA$,$ms$ |
| $\lambda$ | mean of poisson dist | $0.001dt^{-1}$ | $ms^{-1}$ |
| $diam_{soma}$ | somatic diameter | 10 | $\mu m$ |
| $L_{soma}$ | somatic length | 10 | $\mu m$ |
| $diam_{dend}$ | dendritic diameter | 5 | $\mu m$ |
| $L_{dend}$ | dendritic length | 50 | $\mu m$ |
| $\rho$ | ratio of long distance connections | *varied* | – |
| $l$ | connection length | 0.1 | $mm$ |
| $k$ | number of postsyn. cells | 3 | – |
| $A_+$ | STDP potentiation factor | 0.15 | – |
| $A_-$ | STDP depression factor | 0.15 | – |
| $\tau_+$ | STDP pot. time constant | 20.0 | $ms$ |
| $\tau_-$ | STDP depr. time constant | 20.0 | $ms$ |
| $c_{vel}$ | conductance velocity | 0.5 | $\frac{m}{s} = \frac{mm}{ms}$ |
| $\tau_g$ | monoexp. rise/decay param | 3.0 | $ms$ |
| $\tau_m$ | decay param for refractoriness | 50.0 | $ms$ |
| $m_{thr}$ | synaptic ressource threshold | 0.8 | – |
| $m_{spk}$ | consumption per spike | 0.75 | – |
| $E$ | reversal potential | 50.0 | $mV$ |
| $w_{init}$ | initial synaptic weight | 0.00165 | - |
| $t_{stop}$ | simulation time | 1000 | $ms$ |
| $dt$ | integration width | 0.05 | $ms$ |

Table 1: Used parameters.

## References

[1] Ben-Ari Y, Developing networks play a similar melody. *TRENDS in Neurosciences* Vol. 24 No.6 June 2001.

[2] Voigt T, Opitz T, de Lima AD, Synchronous Oscillatory Activity in Immature Cortical Network is Driven by GABAergic Preplate Neurons. *J Neurosci*, Nov 15, 21(22):8895-8905, 2001.

[3] Voigt T, Baier H, and de Lima AD, Synchronization of neuronal activity promotes survival of individual rat neocortical neurons in early development. *Eur. J. Neurosci.*, 9: 990-999, 1997.

[4] Kube K, Herzog A, Spravedlyvyy V, Michaelis B, Opitz T, de Lima A, Voigt T.: Modelling of Biologically Plausible Excitatory Networks: Emergence and Modulation of Neural Synchrony, *ESANN'2004 Proc.*, d-side pub., ps 211-216, April 28-30, Bruges (Belgium), 2004.

[5] Tabak J, Senn W, O'Donovan MJ, Rinzel J, Modelling of Spontaneus Activity in Developing Spinal Coord Using Activity-Dependent Depression in an Excitatory Network, *J Neurosci*, April 15, 2000, 20(8):3041-3056, 2000.

[6] Opitz T, de Lima AD, Voigt T, Spontaneous Development of Synchronous Oscillatory Activity During Maturation of Cortical Networks In Vitro. *J Neurophysiol*, 10.1152/jn.00316.2002, 2002.

[7] Song S, Miller KD, Abbott LF, Competitive Hebbian learning through spike-timing-dependent synaptic plasticity, *Nature Neurosci* vol 3 no 9 September, 2000.

[8] Knoblauch A, Sommer FT, Spike-timing-dependent synaptic plasticity can form "zero lag links" for cortical oscillations, *Neurocomputing* 58-60 (2004) 185-190, 2004.

[9] Eytan D, Breenner N, Marom S, Selective Adaptation in Networks of Cortical Neurons, *J Neurosci*, 23(28):9349-9356, 2003.

[10] Indiveri G, Chicca E, Douglas R, A VLSI reconfigurable network of integrate-and-fire neurons with spike-based learning synapses, *ESANN'2004 Proc.*, d-side pub., pps. 405-410, April 28-30, Bruges (Belgium), 2004.

[11] Watts DJ, Strogatz SH, Collective dynamics of 'small-world' networks. *Nature* 393:440-442, 1998.

[12] Netoff TI, Clewley R, Arno S, Keck T, White JA, Epilepsy in Small-World Networks *J. Neurosci.* Sep 2004, 24, 8075-8083, 10.1523/JNEUROSCI.1509-04.2004.

[13] Hines ML, Carnevale NT, The NEURON simulation environment. In: *The Handbook of Brain Theory and Neural Networks*, 2nd ed, edited by M.A. Arbib. Cambridge, MA: MIT Press, pp. 769-773, 2003.

[14] Bower JM, Beeman D, *The book of genesis*, 2nd edition, Springer Verlag New York, 1999.

[15] Spravedlyvyy V, Herzog A, Kube K, Michaelis B, Braun K, Schnabel R, Influence of dendritic spines shape changes by learning. *IJCNN 2004 proc.*, 25-29 Juli, Budapest pp. 3107 - 3112, 2004.

[16] Hodgkin AL, Huxley AF, A quantitative description of membrane current and its application on conduction and excitation in nerve *J Physiol*, 117:pp500-544, London, 1952.

[17] Harsch A, Robinson HPC, Postsynaptic Variability of Firing in Rat Cortical Neurons: The Roles of Input Synchronization and Synaptic NMDA Receptor Conductance *J. Neurosci.*, 20: 6181 - 6192, 2000.

[18] Hines ML, Carnevale NT Expanding NEURON's Repertoire of Mechanisms with NMODL. *Neural Computation* 12:995-1007, 2000

[19] Fritzke B, *Vektorbasierte Neuronale Netze*, Habilitation Thesis, Universitaet Erlangen-Nuernberg, Shaker Verlag Aachen, 1998.

[20] Bartos M, Vida I, Frotscher M, Meyer A, Monyer H, Geiger JR, Jonas P, Fast synaptic inhibition promotes synchronized gamma oscillations in hippocampal interneuron networks. *Proc Natl Acad Sci U S A* 99:13222-7, 2002.

[21] Buzsaki G, Geisler C, Henze DA, Wang XJ, Interneuron Diversity series: Circuit complexity and axon wiring economy of cortical interneurons, *TRENDS Neurosci* Vol.27 No.4, 2004.