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# Comparison of dynamical states of random networks with human EEG

Ralph Meier[a,c,*], Arvind Kumar[a], Andreas Schulze-Bonhage[b,c], Ad Aertsen[a,c]

[a]Neurobiology and Biophysics, Institute of Biology III, Albert-Ludwigs-University, Freiburg, Germany
[b]Center for Epilepsy, Department of Neurosurgery, University Clinics, Freiburg, Germany
[c]Bernstein Center for Computational Neuroscience Freiburg, Freiburg, Germany

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**Abstract**

Existing models of EEG have mainly focused on relations to network dynamics characterized by firing rates [L. de Arcangelis, H.J. Herrmann, C. Perrone-Capano, Activity-dependent brain model explaining EEG spectra, arXiv:q-bio.NC/0411043 v1, 23 Nov 2004; D.T. Liley, D.M. Alexander, J.J. Wright, M.D. Aldous, Alpha rhythm emerges from large-scale networks of realistically coupled multicompartmental model cortical neurons, Network 10(1) (1999) 79–92; O. David, J.K. Friston, A neural mass model for MEG/EEG: coupling and neuronal dynamics, NeuroImage 20 (2003) 1743–1755]. Generally, these models assume that there exists a linear mapping between network firing rates and EEG states. However, firing rate is only one of several descriptors for network activity states. Other relevant descriptors are synchrony and irregularity of firing patterns [N. Brunel, Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons, J. Comput. Neurosci. 8(3) (2000) 183–208]. To develop a better understanding of the EEG we need to relate these state descriptors to EEG states. Here, we try to go beyond the firing rate based approaches described in [D.T. Liley, D.M. Alexander, J.J. Wright, M.D. Aldous, Alpha rhythm emerges from large-scale networks of realistically coupled multicompartmental model cortical neurons, Network 10(1) (1999) 79–92; O. David, J.K. Friston, A neural mass model for MEG/EEG: coupling and neuronal dynamics, NeuroImage 20 (2003) 1743–1755] and relate synchronicity and irregularity in the network to EEG states. We show that the transformation between network activity and EEG can be approximately mediated by linear kernel with the shape of an $\alpha$- or $\gamma$-function, allowing us a comparison between EEG states and network activity space. We find that the simulated EEG generated from asynchronous irregular type network activity is closely related to the human EEG recorded in the awake state, evaluated using power spectral density characteristics.


*Keywords:* Simulated EEG; EEG model; Human EEG; Cortical dynamics; Brain state; Asynchronous irregular activity; Parallel computing; Power spectral density

## 1. Introduction

Cortical activity can be recorded at various levels of details ranging from in vivo intracellular recording (microscopic activity) to global population activity such as LFP, ECoG, and EEG (macroscopic activity). While there is a good understanding of the origin of the microscopic activity, very little is known about the origin of the macroscopic activity. It has long been speculated that the macroscopic cortical activity is generated as a consequence of network activity [5,11,10]. In fact, several modeling studies have been able to relate network dynamics to EEG states by assuming a linear mapping between the network firing rates and oscillations in the EEG [1,7,4]. However, network dynamics is not only characterized by firing rates, but also by synchronization in neural populations and irregularity of single-neuron firing patterns [3]. To understand how cortical background activity states generate the EEG we need to relate these state descriptors to EEG states. We show that the mapping between the population activity in the network can be approximated by a linear kernel described by either an $\alpha$-function or a $\gamma$-function. The simulated EEG (see materials and methods) corresponding to asynchronous irregular (AI) and synchronous irregular states showed a

*Corresponding author. Neurobiology and Biophysics, Institute of Biology III, Albert-Ludwigs-University, Freiburg, Germany. Tel.: +49 761 2032864; fax: +49 761 2032860.
*E-mail address:* meier@biologie.uni-freiburg.de (R. Meier).

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good match with the human EEG – especially in theta and delta bands. Heterogeneous network simulations resembled the human background EEG even better – also in the alpha and beta bands.

## 2. Material and methods

### 2.1. Networks

We performed simulations of homogeneous and heterogeneous networks consisting of 50,000 leaky integrate and fire type neurons (80% excitatory and 20% inhibitory neurons), representing $\approx 0.5\,\mathrm{mm}^2$ slice of cortex [2]. The neurons were connected randomly with a connection probability of 0.1. In a homogeneous network all neurons had identical passive properties. To introduce heterogeneity into the network, the passive properties (membrane capacity $C$ and conductivity at resting condition $G_{\mathrm{rest}}$) and the spiking threshold ($V_{\mathrm{thresh}}$) of the neurons were chosen from a normal distribution (mean ± SD); $C = 250 \pm 25\,\mathrm{pF}$, $G_{\mathrm{rest}} = 16,7 \pm 1.5\,\mathrm{nS}$ and $V_{\mathrm{thresh}} = -55 \pm 5.5\,\mathrm{mV}$. The mean values of the passive parameters in the heterogeneous network were identical to the values of the passive properties in the homogeneous network. The simulations of the neuronal networks were performed using the NEST [9,12] simulation environment. The output of the spiking network simulations (spike patterns of sustained activity in dynamical networks recorded over several seconds) were then used for further analysis.

We obtained a network population signal ($N_{\mathrm{pop}}$) by binning the spikes (binwidth = 2 ms) of all neurons in the network. To characterize the dynamical states of simulated network activity, both at the level of single neurons and neuron populations, we employed the following descriptors, see [6] for details:

*Mean firing rate* of the activity was estimated as the mean spike count per second of the neurons in the network.

*Synchrony* in the network was measured by the pair wise correlations ($\rho_{\mathrm{net}}$) in the network. A population of identical independent Poisson processes yield a $\rho_{\mathrm{net}} = 0$, any mutual dependence results in an increase in $\rho_{\mathrm{net}}$.

*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, and a clock-like pattern yields $CV^2 = 0$. By contrast, $CV^2 = 1$ indicates Poisson-type behavior (cf. Fig. 1a and b).

### 2.2. Generating simulated EEG

The spectral bandwidth of $N_{\mathrm{pop}}$ is much wider than the EEG signal bandwidth. To draw a comparison between $N_{\mathrm{pop}}$ and EEG, it is required to limit the bandwidth of $N_{\mathrm{pop}}$. To achieve that we transformed $N_{\mathrm{pop}}$ to Sim-EEG by convolving $N_{\mathrm{pop}}$ with either an $\alpha$-function or a $\gamma$-function shaped kernel (cf. Fig. 1c). The parameters for the two kernels were determined in an optimization process by maximizing the similarity of power spectral densities (PSDs) obtained from the Sim-EEG generated using one AI network state (see Section 3) and EEG data from two healthy, awake subjects who were asked to focus on a fixation point. After the optimization process, the kernel parameters were fixed. In an independent test set (both networks and EEG data) we then compared different network states (both homo- and heterogeneous) as the basis for Sim-EEG using the aforementioned kernels and comparing the resulting Sim-EEG to the human background EEG data from more than 90 subjects (cf. [8]). We used the cross correlation ($\rho$) between the powerspectra of the SIM-EEG and the recorded EEG (data was kindly provided by the Center for Epilepsy, University Clinics Freiburg) to quantify the similarity between the two signals.

## 3. Results

### 3.1. Network activity dynamics

A large random network of integrate and fire neurons exhibits a continuum of activity states, depending on the intensity of external excitatory inputs ($\nu_{\mathrm{ext}}$), and on the recurrent inhibition/excitation balance ($g$). The firing pattern of individual neurons varies between regular ($R$) ($CV_{\mathrm{ISI}} \approx 0$) and irregular ($I$) ($CV_{\mathrm{ISI}} \approx 1$), population activity varies between synchronous ($S$) ($\rho_{\mathrm{net}} \approx 1$) and asynchronous ($A$) ($\rho_{\mathrm{net}} \approx 0$). Still, the network activity state can be attributed to one of four characteristic states, viz. AI, SI, AR, or SR as a function of $\nu_{\mathrm{ext}}$ and ($g$) [cf. 3,6]. Of these, it is the AI regime where network activity is considered to most closely resemble cortical spiking activity in vivo (Fig. 1a and b). Note that in the AI regime $\rho_{\mathrm{net}} \approx 0.002$, this results in transient synchrony in the network [6]. The $\rho_{\mathrm{net}}$ can be further reduced by introducing heterogeneities in the network (data not shown). The homogeneous and heterogeneous networks, however, do not differ systematically in the repertoire of states they exhibit.

### 3.2. Sim-EEG

As the AI state resembles the ongoing activity in vivo most closely, we assumed the AI state to be the network activity underlying the EEG obtained from healthy awake human subjects and used the corresponding $N_{\mathrm{pop}}$ to optimize the time constant of the convolution kernels ($\alpha$-function and $\gamma$-function). The resulting optimal width of the $\alpha$-function was estimated to be $\approx 40$ ms (cf. Fig. 1c). Examples for the resulting power spectra for the generated SIM-EEG based on different network states in comparison to the recorded EEG are shown in Fig. 1d. To quantify the similarity between the power spectra we chose to calculate the mean correlation coefficient for relevant frequency bands. Fig. 1e shows the correlation coefficients in the independent test set ($\rho$) between PSD of Sim-EEG

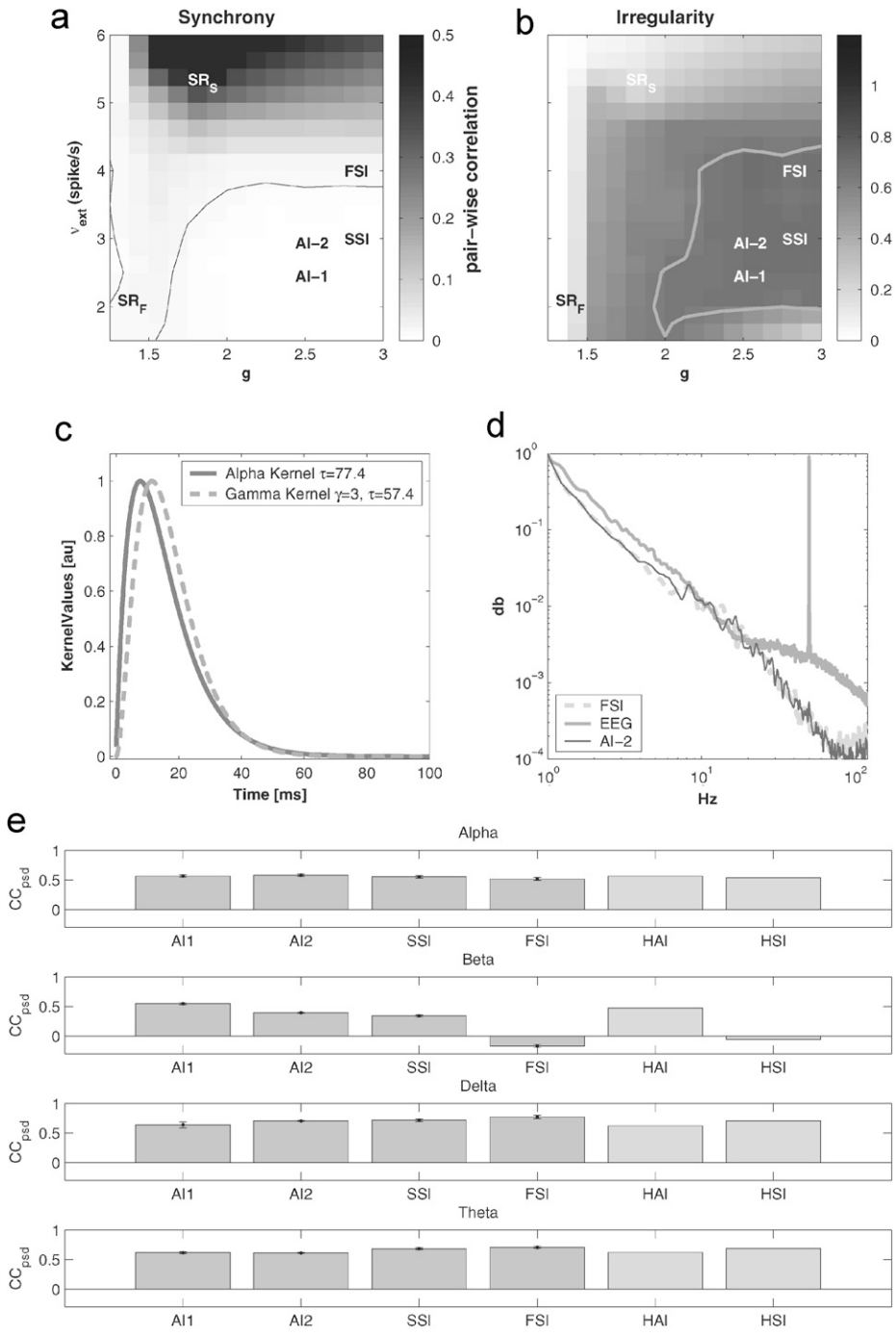


Fig. 1. Network activity states, kernels, and power spectra; correlation of SIM-EEG to EEG: (a) synchrony and (b) irregularity in the homogeneous network as a function of $\nu_{\text{ext}}$ and $g$. The contour ($\rho_{\text{net}} = 0.02$) in (a) separates synchronous states from asynchronous states, while the contour ($CV^2 = 0.8$) in (b) separates regular states from irregular states. Characteristic network states (e.g. SR, AI, and SI) are indicated. (c) Visualization of estimated $\alpha$- and $\gamma$-kernel with respective parameters. (d) Exemplary power spectra for two different simulated network states (nAI and AI–SI) and human EEG. (e) Correlation coefficients ($\rho$) between different characteristic bands ($\alpha, \beta, \delta$, and $\theta$) for the PSDs of SIM-EEG obtained from homogeneous networks mapping different states (asynchronous irregular (AI-1), nearly AI (AI-2), slow synchronous irregular (SSI), and fast synchronous irregular (FSI)) and heterogeneous networks with AI and SI states (HAI and HSI) to human EEG representing normal and awake behaving activity.

(homogeneous and heterogeneous networks) and PSDs of EEG recorded in human subjects, in four different frequency bands (viz. $\alpha[8-13\,\text{Hz}]$, $\beta[14-35\,\text{Hz}]$, $\theta[4-7\,\text{Hz}]$, and $\delta[\leqslant 3\,\text{Hz}]$). The Sim-EEG corresponding to the AI-2 and SI states showed a good match with the human EEG ($\rho \geqslant 0.6$) in the $\theta$ and $\delta$ bands, less in the $\alpha$ and $\beta$ bands. The Sim-EEG generated from the heterogeneous network simulations resembled the human EEG even better ($\rho \geqslant 0.7$), now also in the $\alpha$ and $\beta$ frequency bands.

We observed a small mismatch between the Sim-EEG and human EEG, which could be due to an inappropriate choice of the convolution kernel. Therefore we changed the

kernel function to be a $\gamma$-function, and repeated the function fitting and re-evaluated the correlations of the PSDs. We found that convolution of $N_{pop}$ with a $\gamma$-function kernel gave a slightly better fit, measured by correlation of PSD bands with the human EEG (data not shown).

## 4. Discussion

Here we presented a first attempt to relate the spiking activity of cortical network, to the macroscopic activity of the brain, as captured by the scalp EEG. Generally, the models AI state very closely resembles cortical activity in vivo in awake, behaving animals. Therefore, we assumed that healthy human EEG recordings correspond to an AI state in the cortical network model.

Our comparison of Sim-EEG based on AI state networks and recorded EEG from awake humans supports this assumption. For this comparison we started with an $\alpha$-function shaped kernel. This choice was motivated by the fact that the network activity is low-pass filtered by the cortical tissue and the skull. The $\alpha$-function shaped kernel resulted in a reasonably high correlation between Sim-EEG and real EEG. However, there were also notable differences between the SIM-EEG and EEG. Therefore we used another kernel ($\gamma$-function), which additionally allowed us to control the rising behavior of the kernel. The $\gamma$-function shaped kernel indeed resulted in a higher correlation and the spectra of SIM-EEG resembled recorded human EEG slightly better. This might be due to the additional degree of freedom allowed in the kernel-estimation process.

For this study we ignored the orientation of cortical cells with respect to the recording surface electrode. We assumed that all neurons in our simulations contribute equally to the surface background EEG. From previous modeling studies it is known, that the state space of networks studied here does not change considerably when the total number of neurons is increased [6]. This might allow us, in a first approach, to consider only neurons contributing to (SIM-) EEG recordings. Moreover, since a comparison of spectral properties from both, SIM-EEG and EEG, eliminates temporal causality, potential intracortical firing patterns in neurons of different orientations cannot currently be studied by this approach. However, this basic approach, with the aim of building up a simple phenomenological connection between the background EEG and microscopic network activity, can be extended to connect the activity of layered/oriented networks to EEG – where it naturally would be important to characterize the role of parallel currents.

Currently we are investigating the potential mapping of various network activity states to clinically and behaviorally relevant EEG states. Though there is good hope for bridging the gap between network simulations and electrophysiological population activity data from human recordings, further improvements in both, network models and conversion procedures, will be needed.

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## References

[1] L. de Arcangelis, H.J. Herrmann, C. Perrone-Capano, Activity-dependent brain model explaining EEG spectra, arXiv:q-bio.NC/0411043 v1, 23 Nov 2004.
[2] V. Braitenberg, A. Schüz, Cortex: Statistics and Geometry of Neuronal Connectivity, second ed., Springer, Berlin, 1998.
[3] N. Brunel, Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons, J. Comput. Neurosci. 8 (3) (2000) 183–208.
[4] O. David, J.K. Friston, A neural mass model for MEG/EEG: coupling and neuronal dynamics, NeuroImage 20 (2003) 1743–1755.
[5] W.J. Freeman, Mass Action in the Nervous System, Academic Press, New York, 1975.
[6] A. Kumar, S. Schrader, S. Rotter, A. Aertsen, Dynamics of random networks of spiking neurons with conductance-based synapses, in: Computational and Systems Neuroscience (Cosyne), 2005, p. 153.
[7] D.T. Liley, D.M. Alexander, J.J. Wright, M.D. Aldous, Alpha rhythm emerges from large-scale networks of realistically coupled multicompartmental model cortical neurons, Network 10 (1) (1999) 79–92.
[8] R. Meier, H. Dittrich, A. Schulze-Bonhage, A. Aertsen, Automatic detection of different seizure morphologies in surface EEG without use of prior information, in: 44th Conference of the German Section of the International Liga against Epilepsy, 2004.
[9] A. Morrison, C. Mehring, T. Geisel, A. Aertsen, M. Diesmann, Advancing the boundaries of high-connectivity network simulation with distributed computing, Neural Comput. 17 (8) (2005) 1776–1801.
[10] P.L. Nunez, Neocortical Dynamics and Human EEG Rhythms, Oxford University Press, New York, 1995.
[11] A. Rotterdam, F.H. Lopes da Silva, J. van der Ende, M.A. Viergever, A.J. Hermans, A model of the spatio-temporal characteristics of the alpha rhythm, Bull. Math. Biol. 44 (1982).
[12] The Neural Simulation Technology Initiative (NEST), software available at ⟨www.nest-initiative.org⟩.

**Ralph Meier** was born in Germany in 1976. He obtained his Diploma in Biology at the Albert-Ludwigs- University of Freiburg in 2003. Then he obtained his Ph.D. in cooperation with the Center for Epilepsy, Freiburg and the Neurobiology & Biophysics Department at the University Freiburg, Germany in 2006. Currently he is a post-doctoral fellow at the Bernstein Center for Computational Neuroscience, Freiburg. His research is focused on understanding the dynamics of large scale neuronal networks, emergence of epileptiform activity and the development of scientific software.

**Arvind Kumar** was born in India in 1976. He did his M.E. (Electrical Engg.) from Birla Institute of Technology and Science, Pilani, India in 1999. After a short association with Indian Institute of Technology, Delhi, India, as a senior research fellow, he moved to the University of Freiburg, Germany, where he obtained his Ph.D. in 2006. Currently he is a post-doctoral fellow at Deptartment of Neuroscience, Brown University Providence, USA. His research is focused on understanding the dynamics of neuronal networks and modeling of cortical activity.

**Andreas Schulze-Bonhage** was born in 1960 in Berlin, Germany. He studied medicine at the University of Münster and consecutively worked there at the Institute of Neuroanatomy and Neurophysiology. After his training as a neurologist, he joined the Epilepsy Centre at the University of Bonn as a research fellow and consultant. After visits at the Montreal Neurological Institute and of the Cleveland Clinic Foundation, he became head of the newly founded epilepsy centre at the University of Freiburg. His research interests focus on clinical epileptology, brain imaging and EGG time series evaluation.

**Ad Aertsen** was born in 1948 in Holland, where he obtained his M.Sc. (University Utrecht) and Ph.D. (University Nijmegen) degrees in Physics. After associations with the University of Pennsylvania (Philadelphia), the Max-Planck-Institute for Biological Cybernetics (Tuebingen), the Hebrew University (Jerusalem), the Ruhr-University (Bochum), and the Weizmann Institute of Science (Rehovot), he is now Professor of Neurobiology and Biophysics at the Albert-Ludwigs-University in Freiburg, Germany (www.brainworks.uni-freiburg.de) and Coordinator of the Bernstein Center for Computational Neuroscience (www.bccn-freiburg.de). His research interests focus on the analysis and modeling of activity in biological neural networks and the associated development of neurotechnology.