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

Claudia Bachmann^{*1}, Tom Tetzlaff^{†1}, Susanne Kunkel^{‡2,1}, Abigail Morrison^{§2,1,3}

¹Inst. of Neuroscience and Medicine (INM-6) and Inst. for Advanced Simulation (IAS-6), Jülich Research Centre and JARA, Germany

²Simulation Laboratory Neuroscience - Bernstein Facility Simulation and Database Technology, Institute for Advanced Simulation, Jülich Aachen Research Alliance, Jülich Research Centre, Germany

³Inst. of Cognitive Neuroscience, Faculty of Psychology, Ruhr University Bochum, Germany

⁴Bernstein Center Freiburg, Albert-Ludwigs University, Freiburg, Germany

Recurrent circuits of simple model neurons can provide the substrate for cognitive functions such as perception, memory, association, classification or prediction of dynamical systems [1,2,3]. 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 spiketrain patterns. We find that the loss of excitatory synapses on excitatory neurons 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 an up-scaling of the remaining excitatoryexcitatory synapses. By studying the stability of the linearized network dynamics, we show how homeostasis can simultaneously maintain the network's firing rate, sensitivity to small perturbations and its computational performance.

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c.bachmann@fz-juelich.de

[†]t.tetzlaff@fz-juelich.de

[‡]s.kunkel@fz-juelich.de

[§]a.morrison@fz-juelich.de