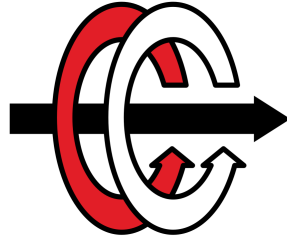**ORTA DOĞU TEKNİK ÜNİVERSİTESİ**
MIDDLE EAST TECHNICAL UNIVERSITY

EE400 Summer Practice Report

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2.1   Institution Name

Max Planck Institute for Dynamics and Self-Organization.
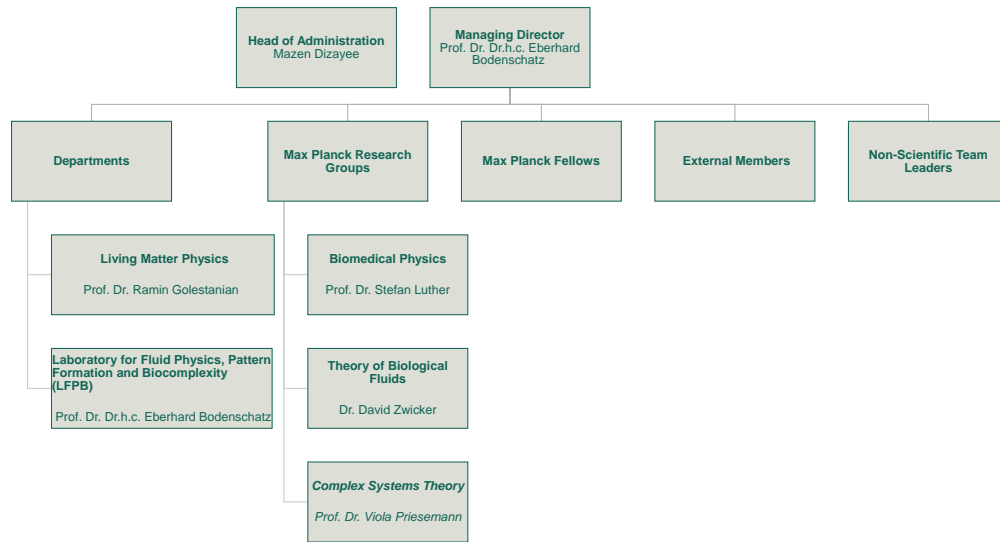
2.2   Institution Location

Max Planck Institute for Dynamics and Self-Organization
Am Faßberg 17
37077 Göttingen Germany

2.3   General Description

The Max Planck Institute for Dynamics and Self-Organization, located in Göttingen, Germany, is a prominent research institution primarily focused on the investigation of complex non-equilibrium systems, particularly within the fields of physics and biology. Its historical roots trace back to 1911 when Ludwig Prandtl initiated the establishment of a Kaiser Wilhelm Institute dedicated to the study of aerodynamics and hydrodynamics. This initial effort led to the formation of the Aeronautische Versuchsanstalt in 1915, which later evolved into the Kaiser Wilhelm Institute for Flow Research in 1924. In 1948, it became a part of the Max Planck Society. In 2003, it underwent a name change and became the Max Planck Institute for Dynamics and Self-Organization. Presently, it stands as one of the 80 institutes under the auspices of the Max Planck Society, contributing significantly to the understanding of intricate dynamic systems.

2.4   Organization Structure

The organization structure of the institute is given in Figure 1. I was part of the group led by Prof.Dr. Viola Priesemann which is indicated as italic on Figure 1.

Figure 1: Basic organizational structure of MPI-DS.

3 Project Background and Motivation

The research project for the internship is constructed upon the following set of previous research. In the field of neuroscience [6], the balance between excitation and inhibition was argued to play a central role to ensure experimentally observed irregular spiking activity. This was further supported by analytical results on the dynamics of a sparsely connected random network with fixed parameters [1]. In this model network dynamics can be grouped into three states(Fig. 2). Synchronous regular (SR) is the state where the whole network is blinking in a synchronized way. So, the neurons are synchronized with each other, exciting each other together, and regular activity is observed. Synchronous irregular (SI) is the state where the individual neurons show irregular activity, but the global activity is still synchronized. Therefore, the rate of the random network is still blinking in some sense. SI-fast and SI-slow characterize this as two different regimes. The asynchronous irregular (AI) state is the desired stable state where both the individual (in the neuron) and global activity show irregularity, that is, small average overall activity in a not-blinking global network. Reference [1] has been considered as a guiding baseline to promote that inhibition is a crucial factor for stability. It is important to note though, that reference [1] employs a fixed set of parameters on a completely randomly connected network.
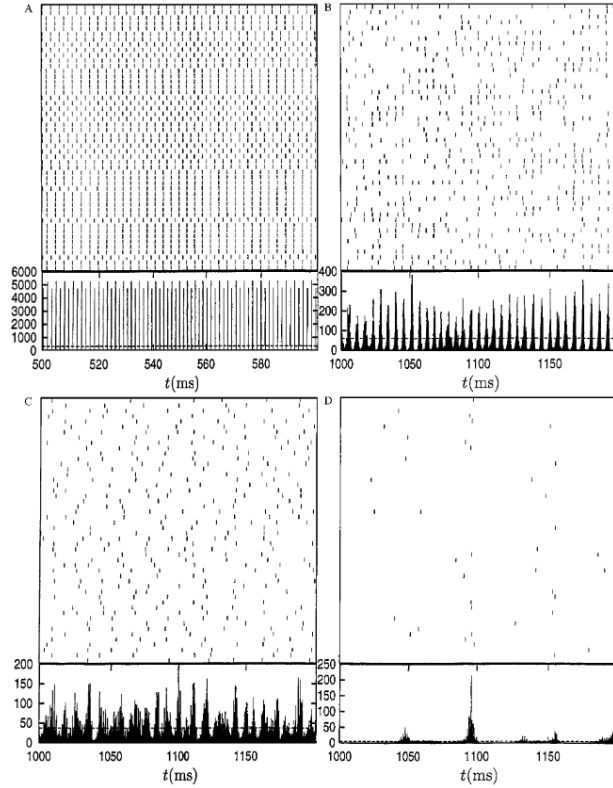
Figure 2: The states of the network. (A) Synchronous Irregular. (B) Synchronous Irregular-fast. (C) Asynchronous Irregular. (D) Synchronous Irregular-slow.

3.1   Hypothesis

In neurobiology, one can classify neurons as excitatory and inhibitory. A typical excitatory neuron is called a pyramidal neuron, whereas there are more than 20 types of inhibitory neurons. While it is known that inhibition is relevan for circuit function [3], it is also commonly considered crucial to stabilze the network [4]. Here, we hypothesize that stable network dynamics can be achieved with purely excitatory neurons. To achieve the stability, the structural connectivity of the network should be reconsidered, especially the 2D layered structure of the cortical networks in the brain [2].

4   Project Description

The project is built upon the hypothesized idea frame. First, the tools for the project are determined. For simulation implementation, Python programming language and a brain simulator package called Brian2 [5] are used. The simulations run on a HPC (High-performance computing cluster.), which is set to be used with the industry standard SGE (Sun Grid Engine). The project timeline aligns with the report format presented in this document.
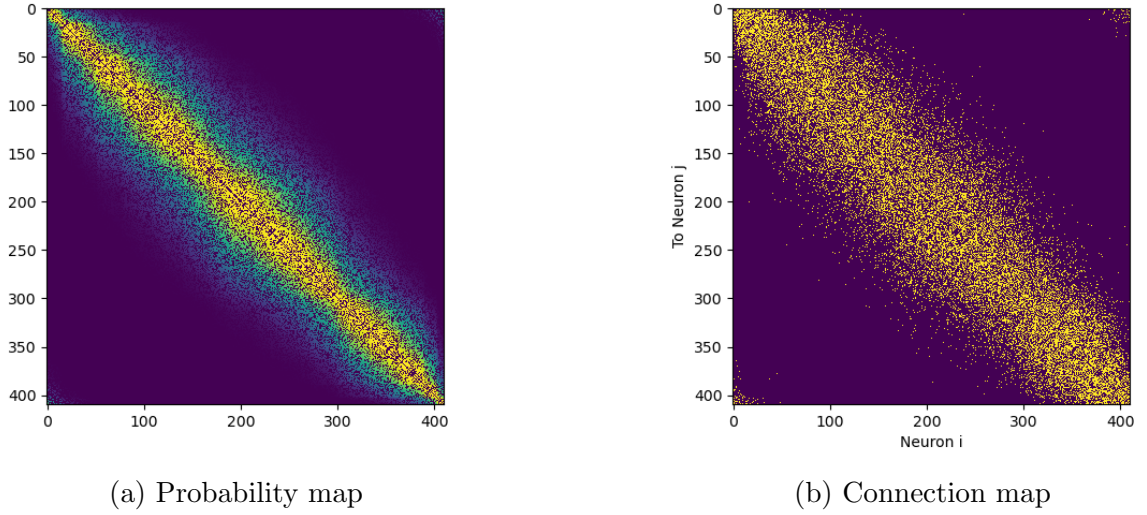
(a) Probability map

(b) Connection map

Figure 3: Sample maps for $N = 410$

4.1   2D Connectivity

The 2D locality of the network is somewhat intuitive as the neurons in the cortical networks have a higher probability of connection to nearby neurons. In order to model this phenomenon, first, N number of neurons are randomly placed in. Then, according to the "smallest distance" between each pair, the probability map is constructed by zero-mean Gaussian with variance σ^2. The density is regulated by the $\#N$ and the spatial scale and by the interaction scale σ figure 3a gives an example probability map for $N = 410$. In this formulation, the outdegree K of the neurons is fixed. That is to say, one neuron has a fixed number of outgoing connections. In other words, one axon has a fixed number of synapses. According to the probability map, the connection assignment process is conveyed as picking K number of neuron j's to connect neuron i without replacement. A sample is given in Figure 3b.

Also, the connections for one neuron are illustrated in Figure 4 as a part of the whole network for $N = 10000$ and $K = 100$, which is the standard network size through the rest of the study.
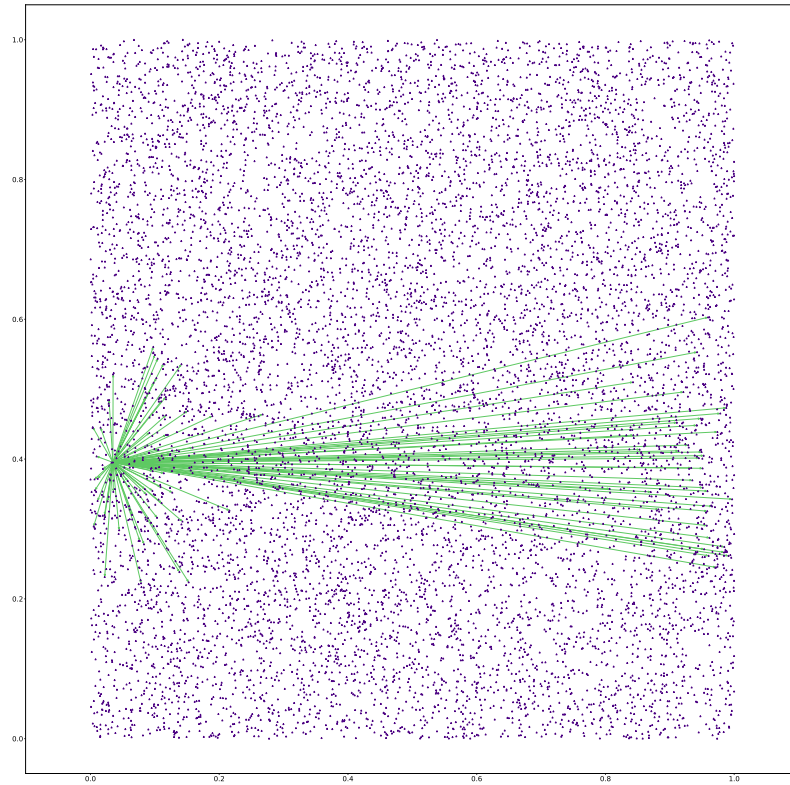
Figure 4: Connections of a neuron on the bigger network size.

There are two important points which should be stated. First, the "smallest distance" is being calculated for a 2D plane periodic boundary conditions, creating a 3D torus structure. Second, as the outdegree is fixed the σ value, which is the standard deviation of the Gaussian function has two limitations. One dependent on the system size, the other one is dependent on the outdegree. Since the periodic boundary conditions apply, larger σ loses its effectiveness. On the other hand, as the σ goes smaller and smaller it becomes impossible todistinguish between two different values because of the fixed number of selections, so one can not go more local in that sense. This is illustrated in Figure 5 where we plot the *effective* σ, which is the average distance of the connected neurons, versus the real σ value. This plot allowed us to choose a small and useful enough σ value.
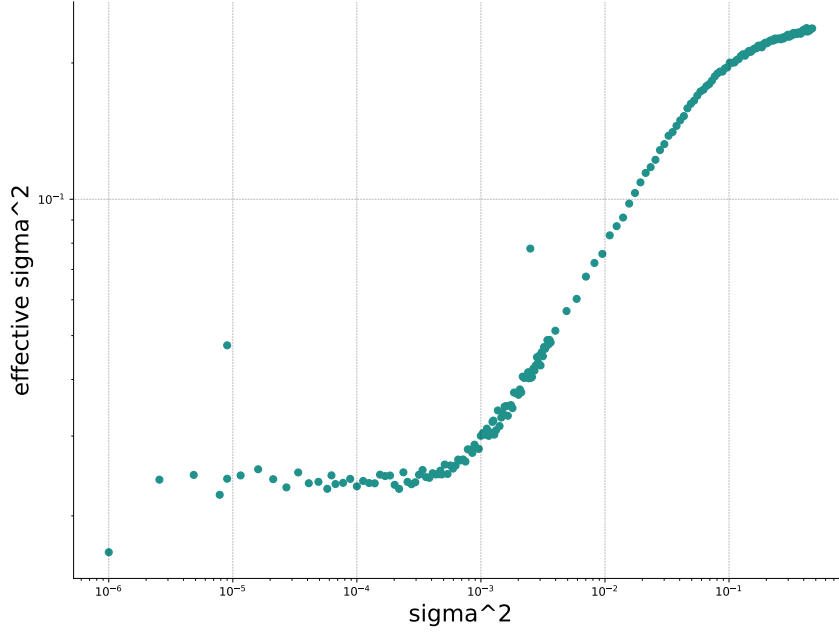
Figure 5: effective σ^2 vs σ^2.

4.2   Baseline

First, the simulations that form a basis are conveyed before introducing regulative mechanisms. Let us build our model one by one. The differential equations that determine the behavior of a neuron are

$$
\begin{aligned}
\frac{dv}{dt} &= -\frac{(v - v_{leak_i})}{\tau_{mem_i}} + \sigma_\eta \times \sqrt{(\frac{2}{\tau_{mem_i}})} \times \eta + I \\
\frac{dI_i}{dt} &= \frac{I_i}{\tau_{exc_i}} + \Sigma J_{ij} \Sigma \delta(t - t_j^k - d_{syn,i})
\end{aligned}
\tag{1}
$$

The main parameters are taken from [1] for consistency. The parameters in Table 1. This is the model for spiking neurons as the v_{thres} exceeded the neuron spikes and the membrane voltage returns to the reset value. The d_{syn} is the axonal delay parameter where the spikes take their time to reach the destination neuron. The τ_{ref} corresponds to the refractory period in which the neuron cannot spike for that amount of time after spiking. The reversal parameter is implemented in order to scale the code easily in case of introduction inhibition. So, if a neuron gets too much inhibition in a time interval, the reversal parameter sets a lower limit. That is biologically realistic since, in such a situation, the synaptic interface gets saturated.

It should be noted that, different from [1], temporal and parameter noise is involved in our model in order to make it stochastic. Also, to make sure that introducing five percent

Parameter	Value	Maximum Noise
τ_m	20.0 ms	1.0
$\tau_{\text{syn\_exc}}$	5.0 ms	0.2
v_{leak}	0 mV	0
v_{thres}	20 mV	0.5
v_{reset}	10 mV	0.5
τ_{ref}	2 ms	–
d_{syn}	1.5 ms	–
v_{rev}	-10.0 mV	reversal

Table 1: Parameter Values

of noise does not make the system deviate from where it should be, the simulations are also run in no-noise settings, and it is verified that the variability does not make the system go crazy.

As we have compiled our basic neuronal model, the coupling parameter J is swept through different values in order to find the threshold where the neuronal activity jumps. The activity is measured as a rate per second per neuron from a few milliseconds of as a factor of time. Therefore, the resulting rate gives necessary information about sustaining activity. In this baseline, the external drive can be summarised as follows. For 5 seconds, each neuron gets a very high external Poisson rate of excitation. So, the network gets charged up. Then, the external drive was suspended, and the simulation continued for 15 seconds. As a result, when we zoom in on the time development of the four closest points to the jump point, as presented in 6, one can see that even though each network settles for a small amount of time to an intermediate non-full-bursting state, two of them dies out afterward. Also, the $J(mV)$ vs activity plot presented in Figure 7 is obtained by averaging over stationary past time instants.
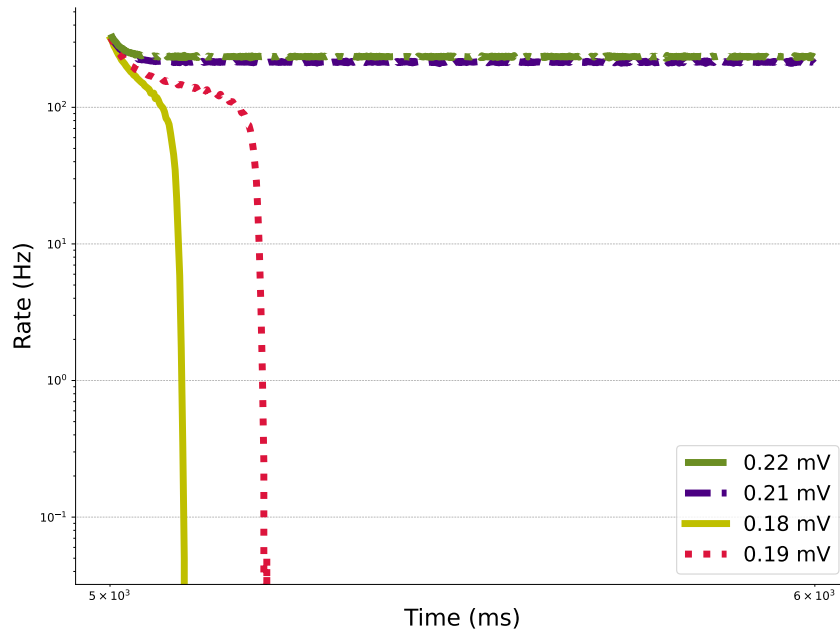
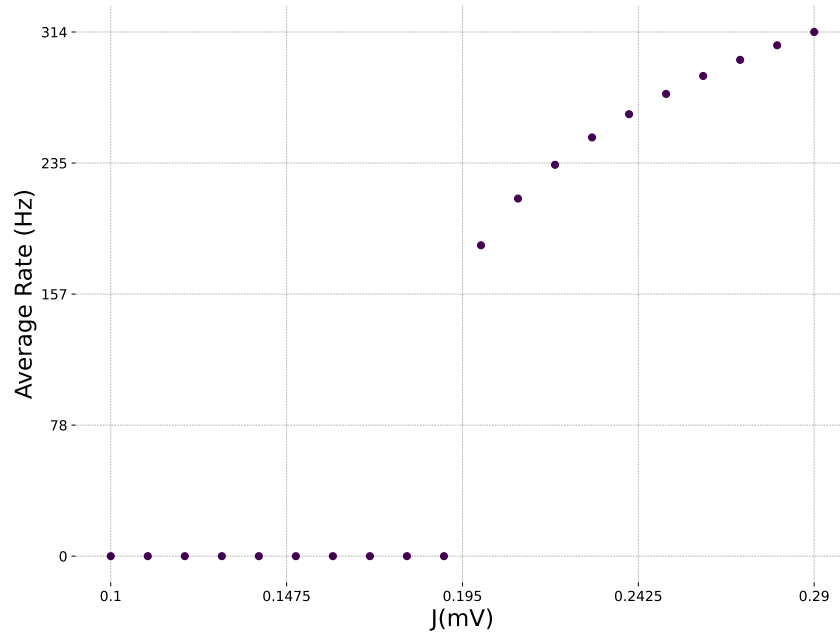Figure 6: Rate (Hz) vs time (ms) for baseline samples in logscale.

Figure 7: Rate (Hz) vs J(mV) for baseline.

The project is steered towards employing regulatory mechanisms for coupling parameter J by forming a baseline.

4.3   Homeostastic Regulation

Homeostatic regulation is a basic mechanism that is rooted in neurobiology. The idea is to use a local mechanism for each neuron to adjust its synaptic couplings (weights) according to a target firing rate.

Here we model each coupling J_{ij} as dynamical variable. The synapse model is constructed as given in 2. So then, each J_i gets regulated.

$$\frac{dJ}{dt} = A_c \times \frac{J - A_c}{\tau_{hp}}$$

when a spike arrives at the postsynaptic neuron

$$J = \frac{(J - A_c)}{\tau_{hp}} \tag{2}$$

when a spike arrives at the presynaptic neuron

$$I_{exc} = I_{exc} + J \text{ mV}$$

As can be observed from the baseline step, the system needs some level of energy in order to start spiking at a considerable rate. Therefore, different external Poisson excitation rates are applied. The target rate is set to 10 Hz.

Now that we have formulated our homeostatic steps, let's look at the results obtained. First, notice that there are more than one set of points. The reason is to be able to keep the 2D locality as a comparison element. The two simulations are run simultaneously. The simulations are named by the aviation alphabet to keep track of them more manageable when we have more than a few setups. The "Charlie" simulations (indicated by the color yellow) have a 2D Gaussian. The "Sierra" simulations (indicated by the color indigo.) are the ones with random network. However, the fundamental setup measures, such as a number of external inputs and outdegree, were kept identical. Figure 8 shows the plot that illustrates external input versus average rate. It can be said that the system is primarily input-driven. However, the target rate is approximated around the point $\nu_{ext} = 76$, creating a small plato.
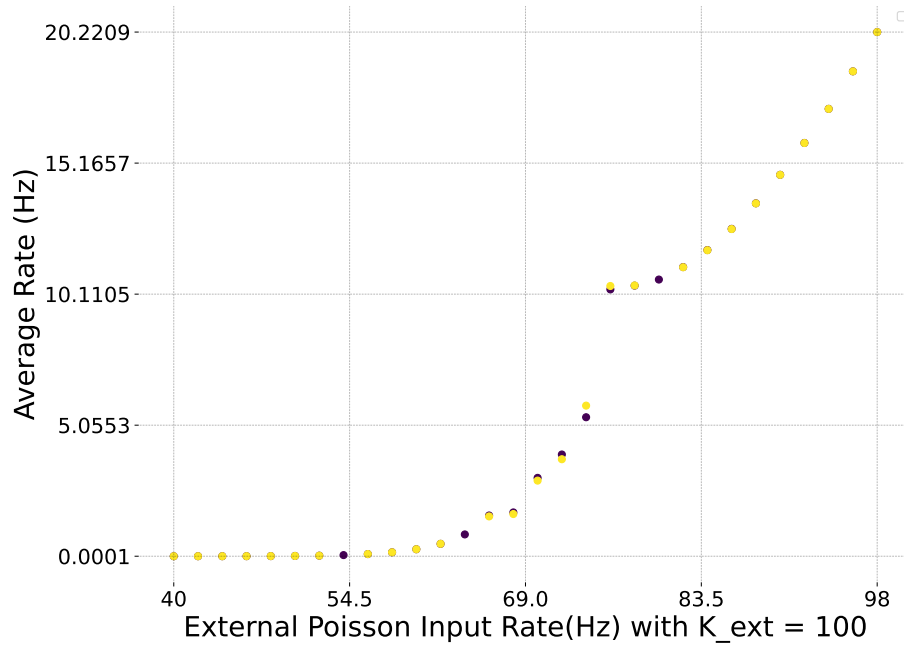
Figure 8: ν_{ext} vs Rate (Hz).

In figure 9, the rate versus time plot for $\nu_{ext} = 76$ is shown. It can be seen that even though some bursts occur after 100 seconds, the system tries to settle for 10 Hz.
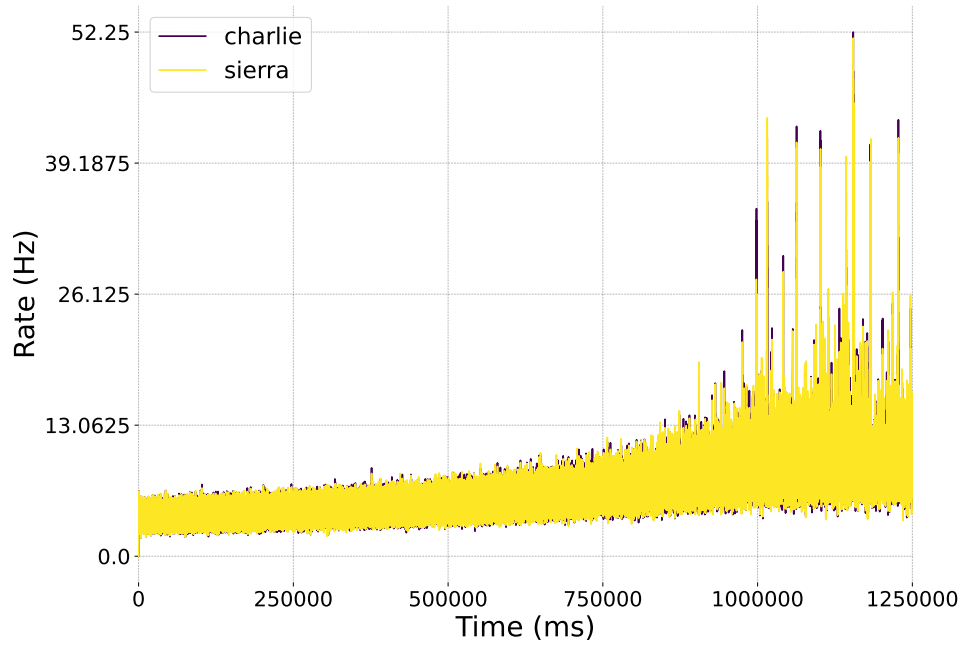
Figure 9: Rate (Hz) vs Time (ms)

In order to observe the evolution of J_i, it is given in Figure 10. As one can see from the plot, the averaged J value converges to a fixed value.
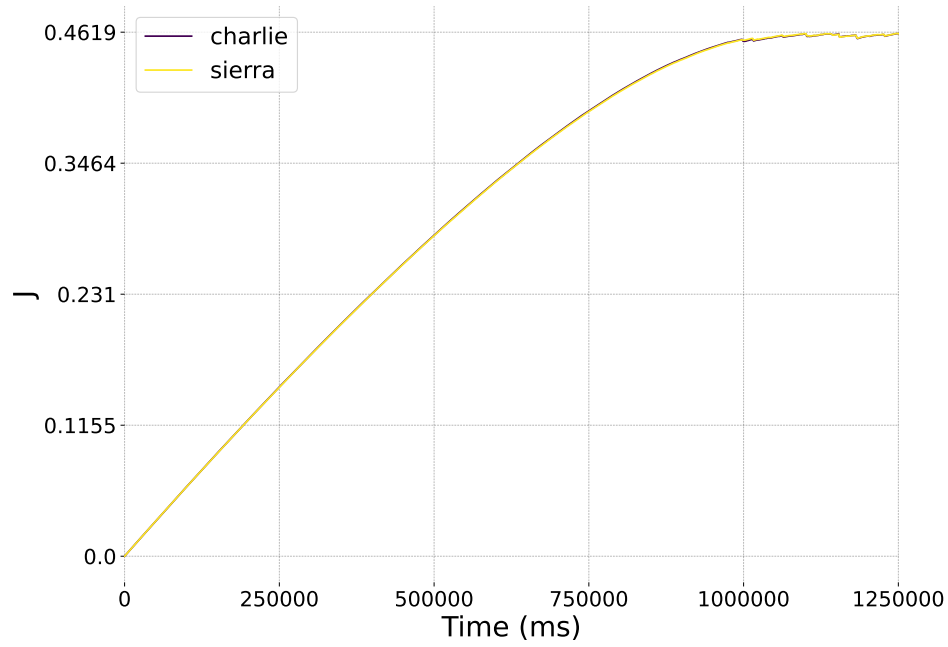
Figure 10: J (mV) vs Time (ms)

Also, the Figure given in 11 illustrates the final distribution of J_i's. This plot tells us that a considerable amount of connnections die out, and there is a Gaussian-like distribution of coupling strengths.
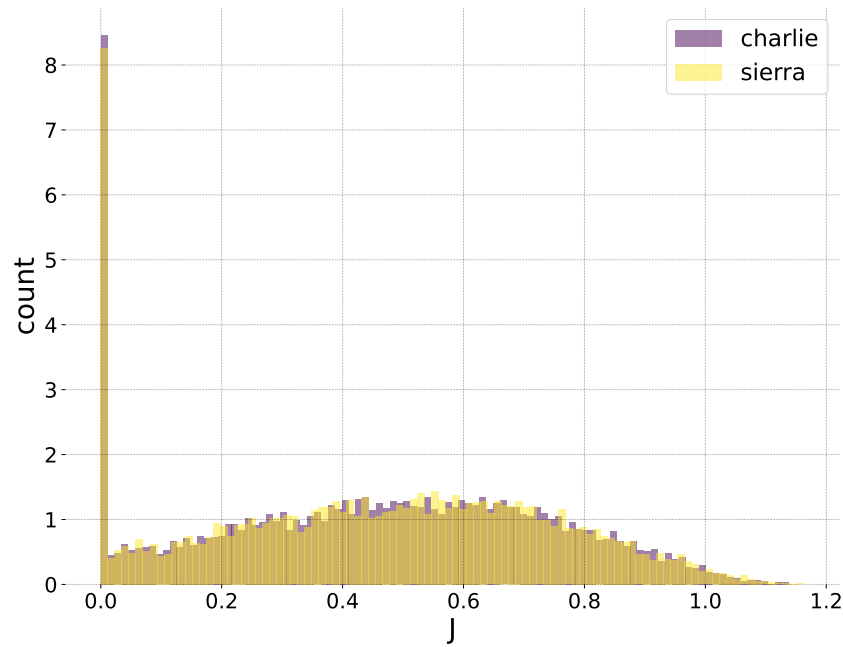
Figure 11: Distribution of J's

To be able to understand the steadiness of the rate signal, the power spectral density is calculated and plotted as shown in Figure 12. However, this plot is considered a preliminary result since we are skeptical about the accuracy. This is because of the ramp part having a few data points.
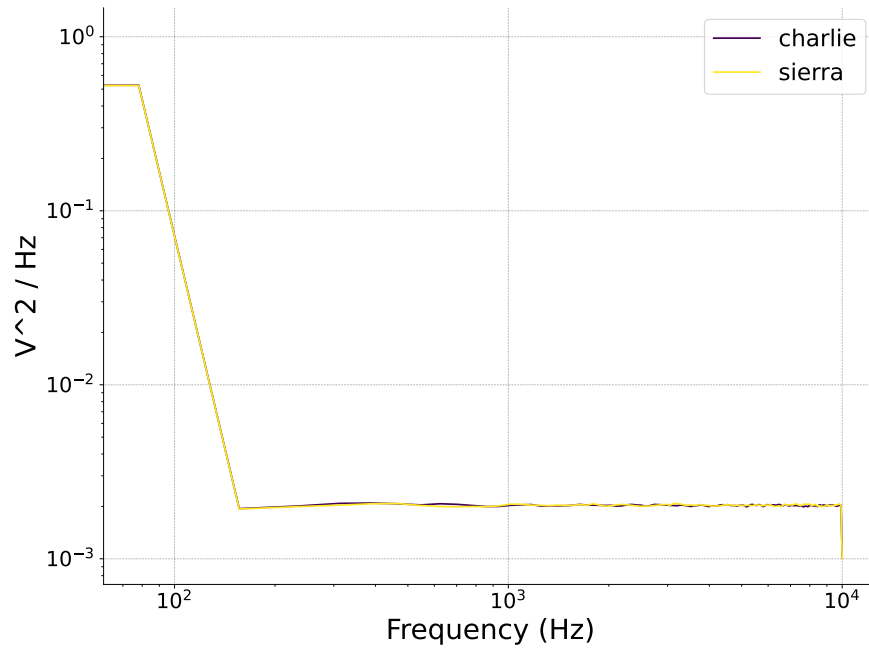
Figure 12: Power spectral density

Another set of comparisons is presented in Figures 13 and 14, where the difference between the 2D structured one and the random one is more distinct. There the external excitation rate is set as $\nu_{ext} = 72$.
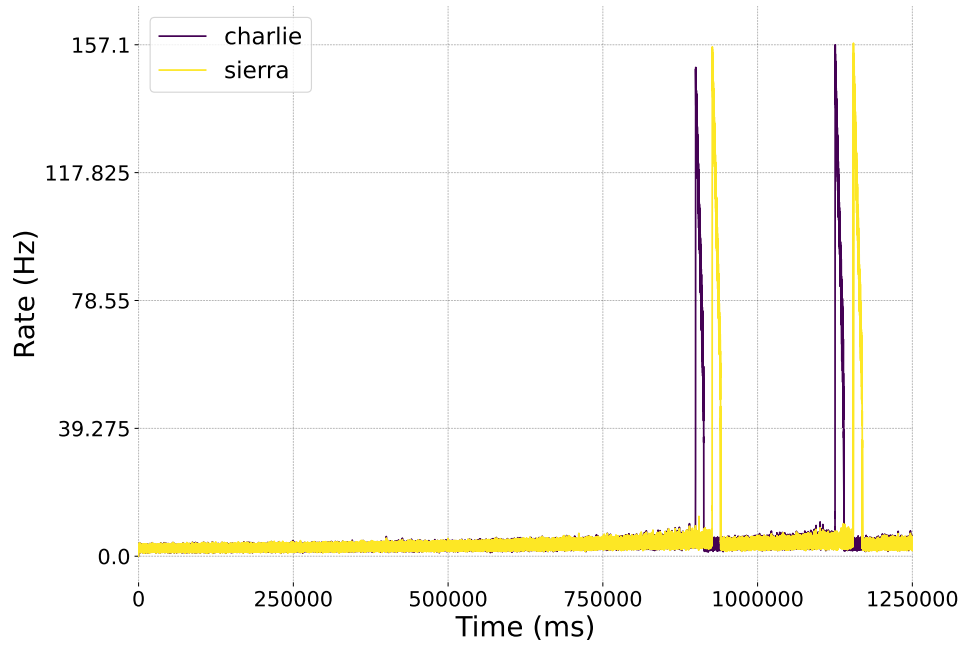
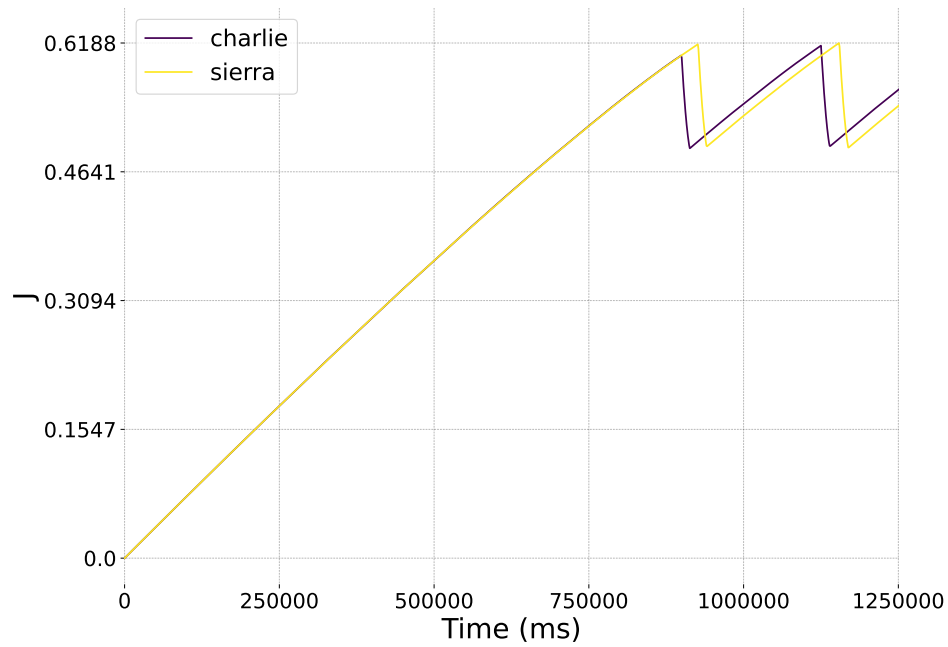Figure 13: Rate (Hz) vs Time (ms) for $\nu_{ext} = 72$.

Figure 14: J (mV) vs Time (ms) for $\nu_{ext} = 72$.

4.3.1   External synapse adaptation.

The adaptation of the external synaptic coupling strengths is also implemented. The mechanism for the external regulation is identical to the inter-neuron homeostasis. In Figure, 15, the average population rate versus external Poisson input rate is presented. It can be inferred that as the external rate exceeds a specific rate of 72 in this case, the external regulation solves the problem by adjusting the couplings (weights). It can be said that for this setup, the system is purely input-driven and shows no self-regulated sustaining collective network activity.
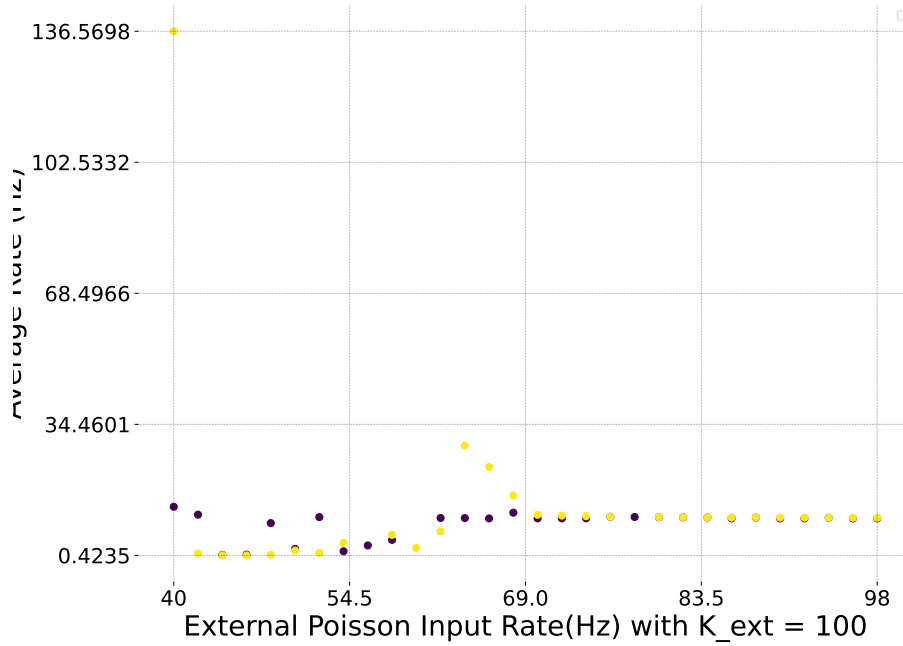

Figure 15: ν_{ext} vs Rate (Hz) for external adaptation enabled case.

Figure 16 presents the power spectral density for external rate $\nu_{ext} = 76$. Similarly, this PSD also carries a preliminary function.
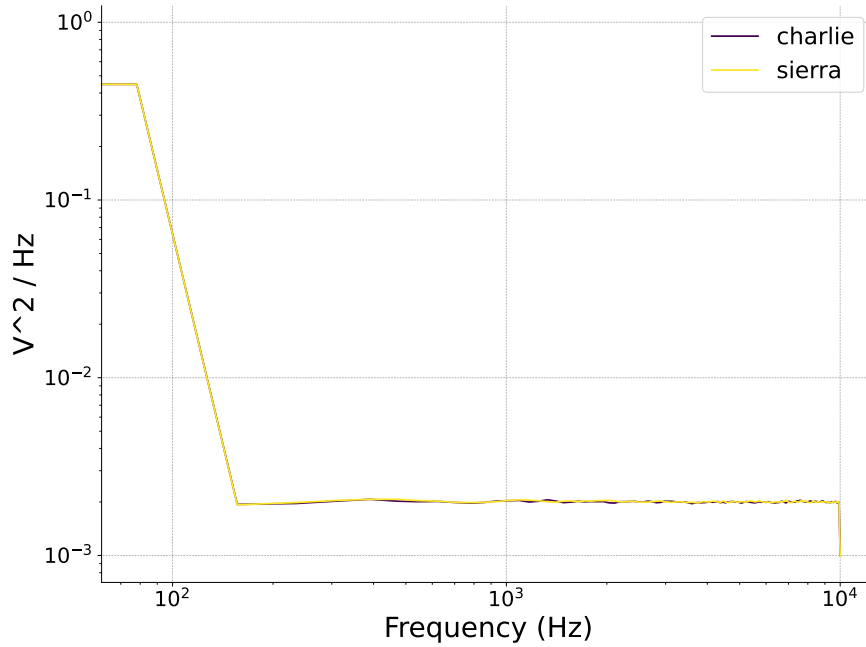
Figure 16: Power spectral density for external adaptation enabled.

4.4   Future Ideas

- Spike time dependent plasticity learning rule is a possible strategy to regulate the system dynamics. So, it can be employed as an alternative to homeostasis.

- Creating subregions on the system and externally exciting the whole system heterogeneously might be an option to decrease the possibility of synchronized state.

- Definition of connection angle for each neuron as an addition to the fixed outdegree parameter in order to develop the idea of locality further.

5   Conclusion

In this document the work done at the research internship pursued at MPI-DS as a part of Erasmus+ program and EE400 course is presented. The main focus of the internship to get hands-on experience with scientific research and HPC computing. This was achieved while studying the dynamics of only-excitatory locally structured sparse spiking neuronal networks' dynamics. The population behaviour together with stability constraints were investigated.

References

[1] Nicolas Brunel. Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons. *Journal of Computational Neuroscience*, 8, 01 2000.

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[6] C. van Vreeswijk and H. Sompolinsky. Chaos in neuronal networks with balanced excitatory and inhibitory activity. *Science*, 274(5293):1724–1726, 1996.

6 Appendix