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Fading Memory in Balanced Neural Networks

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Bachelor of Science

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Balanced Networks

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Abstract

Work in Progress

- Numeric implementation of balanced network following Vreeswijk and Sompolinsky [1998]
- How long can Balanced Networks remember External Input - not very long
- Look at chaotic nature of Balanced Network
- Rough overview also in chapter 1.4 Outlook

To Dos:

- Discussion
- Joining of Subsections in Introduction
- Improve in-text-description of Figures in Chap 3
- Check Figure Descriptions
- General rewriting to make more sense out of presented content
- Spell Checking

Contents

1 Introduction

We strive to understand how we think and how the brain works. We always have. Only about 200 years ago, the head of a recently deceased Joseph Haydn was removed from his grave to investigate what enabled him to become a musical genius. Back then, they resorted to measuring size and shape of the skull to gain insights to the intellectual capabilities. Our methods have come a long way since. The questions we ask, have also changed. However, being arguably the most complex organ of the human body, the brain still largely eludes understanding.

How memory can be maintained is an especially vexing question. We are able to remember some events, which happened decades ago, yet other things slip our mind in an instant. Also as a living organ, the brain changes over time. Every neuron is involved in a multitude of processes. Those reasons, combined with a multitude of more, explain why this problem has not been yet explained. Here we will use a numeric implementation of a model for cortical balanced neurons. We will investigate how this model fares when applying memory collection techniques from the field of reservoir computing. As this work has been solely computational, to verify my results no dead musicians are needed.

1.1 About the cortex and neurons

The human brain is the central organ of the nervous system. It consists of the cerebrum, the brainstem and the cerebellum. Of those, the cerebrum is the largest, taking up 4 quintils of the brains mass. The cerebrum is split into a left and a right hemisphere. Each side controls the corresponding other side of the body. On the outer layers of the cerebrum lies the cortex. It plays an important role in attention, language, perception, thought and memory. Even though the cortex is only a couple of millimeters thick (in the human brain), it represents half of the mass of the cerebrum or two fifths of the whole brain's mass. The cortex is structured in folds, which increase its surface area. Larger folds signify the borders between lobes. The cortex contains about 15 billion neurons or roughly ten percent of all neurons in the brain. Neurons are cells with specialized features. They allow them to receive and facilitate nerve impulses (also called action potentials) to the next neuron. A neuron consists of a cell body (soma), dendrites and an axon. Most neurons receive signals via the dendrite and transmit signals via the axon. The dendrite branches out widely in an area of hundreds of micrometers. This means that a neuron can receive inputs from a very large quantity of other neurons. The axon, mostly, remains in one strand but, in the human brain, can become as long as a meter. An Axon, at the very tip, can then transmit a signal to another neurons dendrite via a synapse. The signaling process is in part electrical and in part chemical. Neurons can be excited electrically. If the voltage reaches a certain threshold over a short time period, it generates an all-or-nothing electrochemical pulse (the above mentioned action potential). This travels through the axon and activates a synapse. The synaptic signal can be either excitatory or inhibitory.

This means, it can either lead to either an increase or decrease, respectively, in the net voltage reaching the next neuron's soma.

1.2 Research leading to balanced networks

In the last decades, our understanding of cortical neurons has advanced since Burns and Webb [1976] looked at spiking behavior of active and alive cats. In the early 90s several research groups (such as Abeles [1991] and Softky and Koch [1993]) found evidence of highly irregular spiking patterns of cortical neurons over time. The time between spikes are distributed in a very broad manner. In fact, the Interspike Interval Histograms (ISI) of cortical neurons resembles a Poisson distribution. However, isolated neurons fire in a predictable and regular fashion when injected with a constant current. This means, fluctuations measured in active cortices happen due to fluctuations in input [Holt et al., 1996, Mainen and Sejnowski, 1995].

This can be the case for a variety of reasons such as changes in intensity over time or stochastic patterns of synapses. However, stochastic fluctuations would be expected to disappear for large numbers of connections between neurons. Indeed, in the cortex, cells have thousands of connections [Vreeswijk and Sompolinsky, 1998]. A high level of correlation between neurons could explain this. This is only true for a small fraction of neurons [Vreeswijk and Sompolinsky, 1998].

Bell et al. [1995] first explored the idea of "balancing" the input by introdcucing excitatory and inhibitory inputs which adjust themselves to maintain the mean input tightly around the input level. This was further explored by Tsodyks and Sejnowski [1995] and Van Vreeswijk and Sompolinsky [1996]. Later on, we will mostly work with the system as described in Van Vreeswijk and Sompolinsky [1996] and expanded on in Vreeswijk and Sompolinsky [1998] This makes it a likely assumption that the brain receives balanced input. They also display a stable asynchronous state.

1.3 Memory and balanced networks

Some neural Networks can act as memory devices. Memory can be embedded in the synaptic connections of a network. An external input can then recreate a given part of the memory [Aviel et al., 2005]. This is called associative memory model, as the external input determines which memory is recalled.

A network memory model should allow for more than one remembered information. This means memory has to be stored in a distributed manner. Also background activity in th absence of any memory should be possible.

One method is the attractor neural network by Amit and Amit [1992]. There an initial input leads to a dynamical flow in one of several attractors All attractor models use hebbian cell assemblies. Neuronal ensembles of

In general, it is not possible to embed memory in the connections of a balanced network, without disregarding the random connectivity assumption in balanced networks. [Aviel et al., 2003]The introduction of some kind of order to the otherwise random connectivity matrix leads to a new critical point at which the asynchronous model becomes unstable.

We will therefore look into the merit of using external input as a way to induce information. We find out that the chaotic nature of the system hinders severely and look into how long plausibly information can be kept.

1.4 Reservoir Computing

Reservoir Computing does not supervise interconnection weights and is instead relies only certain generic properties. A memoryless supervised readout is used to collect information from the network. One of the first applications of reservoir computing in the field of theoretical neuroscience was through liquid state machines (LSM) developed by Maass and Markram [2004]. The LSM framework describes a spiking neural network with a large amount of neurons which are randomly connected. They receive time varying external input. Through the inherent recurrent nature of the connections, external input leads to spatio-temporal activation patterns in the network. Those patterns can be read out and used to extract information from the system.

Unlike other models, information does not have to be hard-coded in the network connections. Also, computations on various time scales can be performed at the same time.

1.5 Outlook

In this bachelor thesis, we will look into how one specific model fares in retaining memory. First, we will show the characteristic behavior of a balanced network as described by Vreeswijk and Sompolinsky [1998] and how this numeric implementation fulfills this behavior. Therefore, we will look at how the mean activation changes over time, examine the behavior of one neuron as well as look at the distribution of spiking intervals. Then, we will look at whether we can use techniques of reservoir computing to determine past external input. We will find that in a general balanced network, retaining memory is difficult due to the chaotic nature of the model. However, adjusting some parameters can slightly improve the networks precision in the short term.

2 Methods

2.1 Mathematical Concepts

2.1.1 Statistics and Randomness

The coefficient of variation (c_v) is defined as the ratio of standard deviation s to the mean μ, so

$$c_v = \frac{s}{\mu}$$

It describes the variability in relation to the mean of the population. The larger the coefficient, the less precise is the result at hand.
A Poisson point process is an event which occurs continuously and independently at a constant average rate. This means the occurrence of one event does not influence the happening (or not happening) of an event one increment of time later. The name giving Poisson distribution describes the probability of the point process occurring k times in a given time frame. The exponential distribution is the probability of the time interval between events in a Poisson process. It depends on the time t and the rate parameter γ.

$$f(t,\gamma) = \begin{cases} \gamma e^{-\gamma t} & t \geq 0 \\ 0 & t < 0 \end{cases}$$

Furthermore, the coefficient of variation of an exponential distribution is 1. This relation is not so clear cut the other way. However, $c_v = 1$ can still indicate a certain likelihood that the present result was drawn from a exponential distribution.

2.1.2 Boolean Networks

A Boolean Variable is a variable which only has two possible values, such as True and False, 1 and 0 or Active and not active. A Boolean Function is a function which takes in a certain amount of Boolean variables as arguments and outputs a value from a two element set. A Boolean network contains a discrete set of Boolean variables. Each of those is updated by a Boolean Function taking in a subset of the Boolean variables. There are several updating mechanisms. For a synchronous update all variables are updated at the same time. This means in each step, all variables can potentially change their value. The asynchronous update mechanism only evaluates one function at a time. The order of updates can either be constant or change over time. Boolean networks have many application throughout the sciences, for example in modeling genetic regulatory networks.

The Hamming Distance is a measure of how different two Boolean networks are. A Hamming Distance d_H of 0 means that the systems are identical and a d_H of 1 means all values are flipped. A value of d_H of 0.5 means therefore that the two systems are independent of each other. It is calculated as follows:

$$d_H(t) = \frac{1}{N}\sum_{i=1}^{N}\left(\sigma_i(t) - \sigma_i'(t)\right)^2$$

2.2 Model

2.2.1 Definition and Parameters

In the following, we will follow the definition of a a network in a chaotic balanced state, as described by Vreeswijk and Sompolinsky [1998]. As described above, the system consists of excitatory and inhibitory neurons. They, thus, have a positive or negative impact on the activation function respecively. Unless otherwise specified, all neurons of a given type are interchangeable and share all characteristics. Neurons have Boolean values and are updated in a random order. They receive external input as well. All neurons have Boolean values. This means, they are always in one of two states: The Null state will also be referred to as inhibited state and the One state will also be referred to as activated or excited.

The network is to behave the same in different size configurations. The balance of the network is achieved by having the sum of all excitatory and all inhibitory to oscillate tightly around the threshold.

2.2.2 Connection Matrix

The connection matrix J only distinguishes two things. Whether a connection exists between two neurons and the type of both, the outgoing and receiving, neuron. Only the average amount of connections per population between neurons K is specified. This means one neuron has on average K inhibitory and K excitatory inputs. K is a lot smaller than total size of neurons N meaning this is a sparsely connected network. As only the average amount of connections is determined, some neurons can have more connections than others. Also because of that, it is possible to have more connections with one population type than the other one. This leads to a consistent differences within a population and increases differences in the behavior of neurons.

The actual weight is thus calculated based on how many connections K the average neuron has and a prefactor J_{kl} depending on the type of neurons involved as we show in Equation 1:

$$J_{kl}^{ij} = \begin{cases} \frac{J_{kl}}{\sqrt{K}} & \text{if connected} \\ 0 & \text{otherwise} \end{cases} \tag{1}$$

Here j and k indicate the population type (represented either by 1,2 or E,I) while i,j show the position inside the respective population. This means the average input for every neuron from each population looks roughly like this:

$$K \cdot J_{kl}^{ij} = \sqrt{K} J_{kl}$$

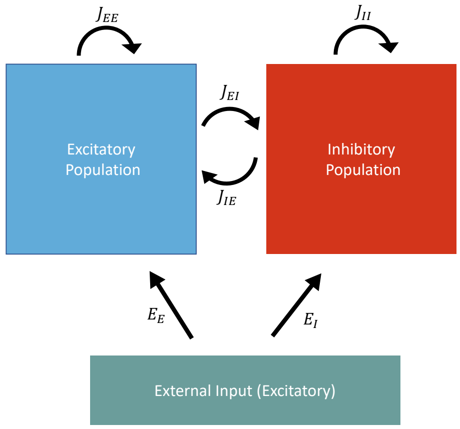


Figure 2.1: Relations between population of neurons

This shows a proportional relationship between the cumulative gross inputs into a neuron and $\sqrt{K}$.

2.2.3 External Neurons and Thresholds

Unless otherwise specified, we will focus on constants thresholds. The threshold is chosen to be one or close to one. This is possible because it only matters that all inputs oscillate around the threshold

The input to the neuron is chosen to be a lot larger than the threshold. So both external and internal neurons have a weight of roughly $\sqrt{K}$. The external neurons, furthermore, consider the external input strength factor E and the mean activation measure m_0. E_k has value of roughly one, while the mean activation can have a value between 0 and 1. (In fact, in our tests we only consider $m_0 \leq 0.3$.) Thus, the total value of external input u_0 is calculated as follows:

$$u_k^0 = E_k m_0 \sqrt{K} \tag{2}$$

2.2.4 Activation Function and Update Mechanism

As stated above, all neurons are binary, meaning the value of a given neuron σ_k^i is either one or zero. Here k indicates the population type and i which position the neuron has within this population. The value of the neuron is calculated with the input of the other neurons as well as external input and subtracting the threshold. The result is then plugged into a Heaviside function to yield a

Boolean value.

$$u_k^i = u_k^0 + \sum_{l=1}^{2}\sum_{j=0}^{N_l} J_{lk}\sigma_k^i \qquad \rightarrow \qquad \sigma_k^i = \Theta(u_k^i) \tag{3}$$

The neurons are updated asynchronously meaning that one neuron at a time is changed. The neurons within a population are chosen at random. Between the populations the update times can differ. This means there are two mean update times τ_E and τ_I for the excitatory and inhibitory part of the population respectively. We will measure time t in units of τ_E. This means effectively τ_E will be fixed to 1. With only τ_I being variable, this will be our measure of the relative update time.

2.2.5 Measures

Next, we will discuss some measures used to convert the recorded signals into valuable information. The vector $\vec{\sigma}_k(t)$ contains the Boolean values of population k (with $k \in \{E, I\}$) at time step t. The activation rate $m_k(t)$ is the share of excited neurons, meaning those whose value is one.

$$m_k(t) = \langle \sigma_k^i(t) \rangle_i = \frac{1}{N_k}\sum_{i=1}^{N_k} \sigma_k^i(t)$$

N_k represents the size of the population, while i represents the individual members of the given population. The network finds a stable state. The mean activation rate, on which the network settles, is defined as m_k. The mean activation rate of external neurons m_0 is handled similarly. However, contrary to m_k, m_0 is a parameter set by the model, not result of some form of dynamics.

Meanwhile, the firing rate $\vec{r}_k(t)$ describes the share of neurons, which turn active in a given time step. This means, it doesn't record a one for a neuron which was already active but instead a zero.

$$r_k(t) = \langle r_k^i(t) \rangle_i \qquad\qquad r_k^i(t) = \begin{cases} \sigma_k^i(t) - \sigma_k^i(t-1) & \sigma_k^i(t) = 1 \\ 0 & \sigma_k^i(t) = 0 \end{cases}$$

From this immediately follows, that $m_k(t)$ is allows larger or equal to $r_k(t)$. As long as $m_k(t)$ is low, the likelihood of a neuron to be activated for two consecutive timesteps is low. This means that for sufficiently small $m_k(t)$ holds:

$$m_k(t) \approx r_k(t)$$

2.2.6 Stable States

As defined in Equation 1, there is on average input coming from K neurons of each population and each of those connections has a value of $\frac{J_{kl}}{\sqrt{K}}$. External input, as defined in Equation 2, has a value of $E_E m_0 \sqrt{K}$. Thus, the mean input to each population is as follows:

$$u_k = (E_k m_0 + J_{Ek} m_E + J_{Ik} m_I)\sqrt{K} - \theta_k$$

In a balanced network, m_k can neither be one or zero. If the network was completely active (or completely inactive) for longer periods, the name giving balance would not exist. This means, u_k has to be finite and, thus, in the large K limit, this must hold.

$$E_k m_0 + J_{Ek} m_E + J_{Ik} m_I = \frac{(\theta_k - u_k}{sqrtK} = \mathscr{O}(\sqrt{K}) = 0 \tag{4}$$

Vreeswijk and Sompolinsky [1998] always set the prefactor of input originating from excitatory neurons (i.e. J_{EE} and J_{EI}) to 1. This is possible as there are several degrees of freedom. As we, for the most part, adhere to this as well, we will for our next steps have set J_{EE} and J_{EI} to 1, too. The connections originating from inhibitory neurons are negative. To avoid confusions when talking about the absolute value, it will always be displayed with absolute value bars. With this, we can transform our results and show the mean network activity for excitatory m_E and inhibitory neurons m_I.

$$m_E = \frac{|J_{II}|E_E - |J_{IE}|E_I}{|J_{IE}| - |J_{II}|} m_0 = A_E m_0 \tag{5}$$

$$m_I = \frac{E_E - E_I}{|J_{IE}| - |J_{II}|} m_0 = A_I m_0 \tag{6}$$

A_E and A_I have to be positive. This means one of the two following cases has to hold.

$$\frac{E}{I} > \frac{J_{IE}}{J_{II}} > 1 \qquad \text{or} \qquad \frac{E}{I} < \frac{J_{IE}}{J_{II}} < 1$$

The latter one, however, allows - for cases outside of the stable, longterm solution - to lead to a solution of m_k being 1 or 0. Therefore, the first condition must hold.

On top of that, if both $|J_{Ik}|$ are smaller than their respective J_{Ek}, there is a solution for u_k with $m_E = m_I = 1$ and $m_0 = 0$.

$$u_k = (J_{Ek} m_E + J_{Ik} m_I)\sqrt{K} = (J_{Ek} m_E - |J_{Ik}| m_I)\sqrt{K}$$

This is not desired as we don't want stationary solutions with m_k = 0,1.

As long as those two conditions are met, no stationary solution emerges with either populations activity rate $m_k = 0, 1$:

- $|J_{IE}| > 1$
- $\frac{E}{I} > \frac{J_{IE}}{J_{II}} > 1$

If the above stated conditions hold, the system will regulate itself and not fully activate itself nor fully shut off at any given time point (i.e. $m_E, m_I = 0$ or $m_E, m_I = 1$, respectively). If through external action brought in such a situation, the following will happen.

For the case of a total shutdown of the network, all internal input would be 0. Since the update mechanism is asynchronous and random, only one neuron at a time is updated. The external input in our model is of magnitude $\sqrt{K}$ and the threshold of magnitude 1. The next neuron to be updated therefore has to be excited. Only when a sufficient number of inhibitory neurons (roughly of order $\sqrt{K}$) has been excited, a neuron can be updated and be below the threshold. The state therefore cannot be maintained. The same mechanics pushes the mean activation $m_k(t)$ upwards as soon as it shifts too far below the balanced state.

In the case of complete activation, by definition, the input from internal inhibitory neurons is a lot stronger than the input from both, internal and external, excitatory neurons. Therefore, if the whole network is excited, the next neuron has to be switched off. Only after enough inhibitory neurons have been deactivated, an update can again turn into a positive value and thus a neuron can be activated again. Again this means, that a state of complete activation cannot be maintained and that the same mechanics nudge the mean activation $m_k(t)$ towards the balanced state.

3 Results

3.1 Input Parameters

In this section, we will look at concrete numeric implementation of the above described balanced network. The Size parameter N refers to the size of one population and the connection number K refers to the average number of connections one neuron has to each population. Both apply to both populations. We kept the size of both populations and the connection number the same for both populations and in all tests. Therefore we did not add subscripts. Time is measured in units of τ_E or the average amount of cycles it takes to update all excitatory units once. The mean external activation m_0 is constant for the most part. However in Chapter 3.8 we also look at a case for a m_0 which randomly oscillates between two different values. There we also vary the values for J_EE and E_E. All other parameters are the same throughout this thesis. Where possible and appropriate, we chose the same parameters as Vreeswijk and Sompolinsky [1998] did for their model. This allowed us to compare our results with those of them and verify the validity of both results. The parameters are listed in Table 1.

The update order is random within a population and the choice between the populations depends on the ratio between τ_E and τ_I. However, we also take a look at more deterministic update orders.

N	10,000
K	1,000
m_0	0.1*
t	200*

Effect on:	J_{Ek}	J_{Ik}	E_k	θ_k	τ_k
Excitatory E	1*	-2.0	1.0*	1	1
Inhibitory I	1	-1.8	0.7	0.7	1.1

Table 1: Paramter Overview
Left hand side shows all parameters for the whole simulation. Right hand side shows all parameters which are different for each population.
* for parameters that have different values in some figures.
J_{hk} strength factors of connection matrix weights (first subscript shows originating population, second target population), E_k strength factor of external input weight, θ_K threshold, τ average time of updates in a time step, K average number of connections per population, N Size per population, m_0 External excitation rate, t Timesteps of simulation (times an average excitatory neuron has been updated)

3.2 Output Parameters

Technically speaking, we extract four arrays containing information from the system. Firstly we extract the time stamps, when a given neuron is activated. The time stamp is calculated on the basis of the average update times of excitatory neurons. It is taken the cumulative number of updates of excitatory neurons and divided by the amount of excitatory neurons. Effectively this is an array with the amount of all neurons N on the first axis and the individual events on the second axis.

This way of saving information was more memory efficient than an array of size N x time t . This is the case because m_k in both cases is a lot smaller than 0.5 for all tests. Furthermore, it is theoretically possible, in the random regime, that a neuron is activated twice in one time step. At the same time, this means, in the random regime, there is no concrete information whether a cell was updated at all during a given time step.

We also record the neuronal firing separately. All events contained within the measure of neuronal firing can be found in the the measure of neuronal activation as well. However, as explained in the definition of the firing rate, if the neuron was already active, it cannot fire. Thus, in this case, no time stamp is recorded.

For some plots on the individual neuron level, more information is needed. For a (customizable) subset of neurons, more information is recorded. Specifically:

- Cumulative Excitatory Input (positive)
- Cumulative Inhibitory Input (negative)
- Net Input (except Threshold)
- Threshold
- Internal Excitatory Input
- External Excitatory Input

Lastly, changes in the mean activation of the external input m_0 are recorded. This is relevant in Chapter 3.8, as there the m_0 is varied between a high and a low level. For this reason, only the label high or low is recorded (in Boolean notation). On top of that, there is also a graphical interface available to interact with the code. This will let you create many of the plots displayed in the following chapters.

3.3 Individual Neuron

First, we will look at a single neuron and see how the balance looks in practice. An example is displayed in Figure 3.1. In the upper part we see all relevant inputs to the neuron. The fictive current is measured in units of the excitatory threshold. Both cumulative excitatory and inhibitory inputs are large compared to the threshold. They also scale with the connectivity, as we discussed in Chapter 2.2($\propto \sqrt{K}$). As the system is balanced, the net input is of the order as the threshold. The external input is constant as it should be (see Equation 2). Both internal excitatory and inhibitory input fluctuate roughly with the same intensity in comparison to their mean. However, the inhibitory internal neuron has a mean which is a lot higher. This is due to the higher weights in the connection matrix J. This, also, leads to its fluctuations being larger than those of the excitatory input. The net input fluctuates slightly below the threshold for the most time. In the lower part we see the output, namely, when the neuron is activated. This is marked as a black stripe. There we see the irregular spike train of the neuron.

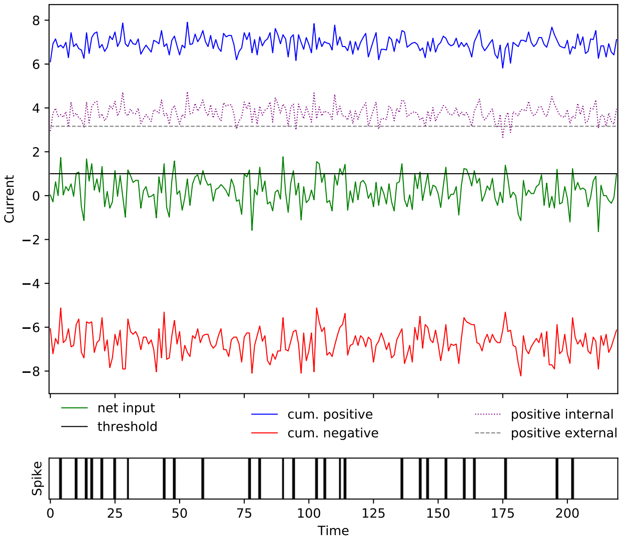


Figure 3.1: Temporal structure of all input streams of an excitatory neuron. Displaying the Balance of excitation and inhibition in neurons. Upper part: Dashed line: input from external neurons. Blue dotted line: input from internal excitatory input to the neuron. Solid blue line: input from all excitatory(i.e. positive) inputs to the neuron. Red line: Inhibitory (i.e. negative) input to the neuron. Solid black line: Threshold for excitation. Solid green line: sum of all excitatory and inhibitory (i.e. net) inputs to the neuron.
Lower part: When the total sum crosses the threshold, the function is excited or activated and a spike is recorded. If other neurons recorded for a longer period of time, this will be marked by a grey area in the spiking area. Time in units auf tau_E and current in units of (excitatory) thresholds θ_E Einheiten!!!! All Parameters as defined in Table 1

In Figure 3.2, the update order within a population is held static. This means, it is still random (with likelihoods determined by the values of τ_I) whether a neuron of the excitatory population or inhibitory population is chosen. But if, say, σ_E^{17} once is followed by σ_E^{254}, anytime σ_E^{17} is updated, the next time an excitatory neuron is to be updated, it will be σ_E^{254}.

In this case, we see that there is a clear pattern repeating itself. The frequency of which is roughly of ten to fifteen time steps. There are random elements as well, leading to a certain level of noise. If one has a completely unchanging update order the frequency becomes even smaller. It is also possible to update simply all excitatory and then all inhibitory neurons in a row. Then the system simply oscillates between excitation and inhibition every other timestep.

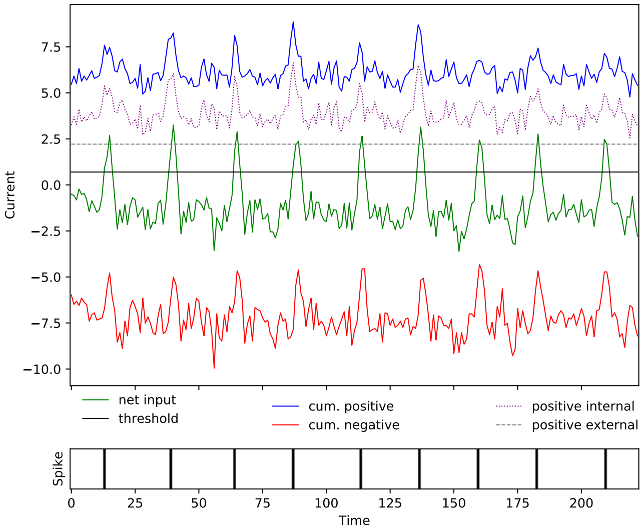
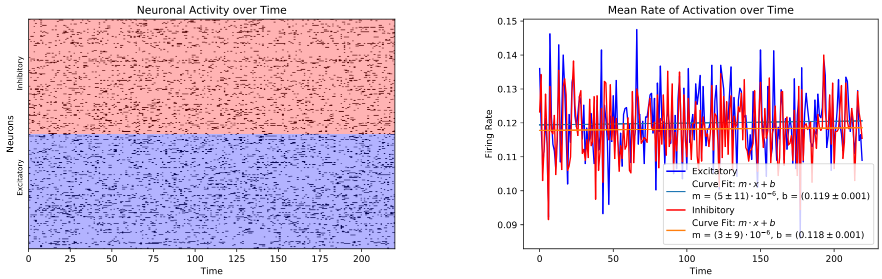


Figure 3.2: Rigid update order leads to oscillations. Here, the update order within a population is fixed. There is still a randomized decision whether a member of the excitatory or inhibitory population is updated. All other conditions are the same as in Figure 3.1.

3.4 Behavior of each population

Now, we will look at the system as a whole. An overview over the activation of the whole system over time is given in Figure 3.4. There seems to be no apparent structure or synchronization between different neurons spiking behavior.

This is Figure 3.4 The mean activation is constant in time for unchanging external input. until their is an equilibrium (or balance) is reached between We test this and plot mean activation over time in Figure 3.5. We see that both mean inhibitory and mean excitatory activation fluctuate heavily. As the linear regression shows, the slope of the trendline is only negligibly different from 0.



(a) Overview over all spike trains

(b) Mean Activation over time

Figure 3.3: On the left hand side, there is an overview over neural activity of the whole network over time. Every point on the y-axis represents one neuron with excitatory neurons being in the lower half and inhibitory neurons in the upper.
The right hand side shows the mean firing rate over time together with a linear fit. All parameters as defined in Table 1

3.5 External Rate vs Network Rate

In Figure 3.6 we look at how the mean activation rates behaves, when the external mean activation range is changed. In the Calculations in Chapter 2.2.6 we already showed that the overall activation of the network is dependent on the external input. We ran our model for 20 time steps at many levels of external activation m_0 and measured the mean activity for each population(m_E and m_I). The linear relationship is very strong. Our Fit shows that for the case of K = 1000 it similar to what [Vreeswijk and Sompolinsky, 1998] predicted (see also Figure A.4).

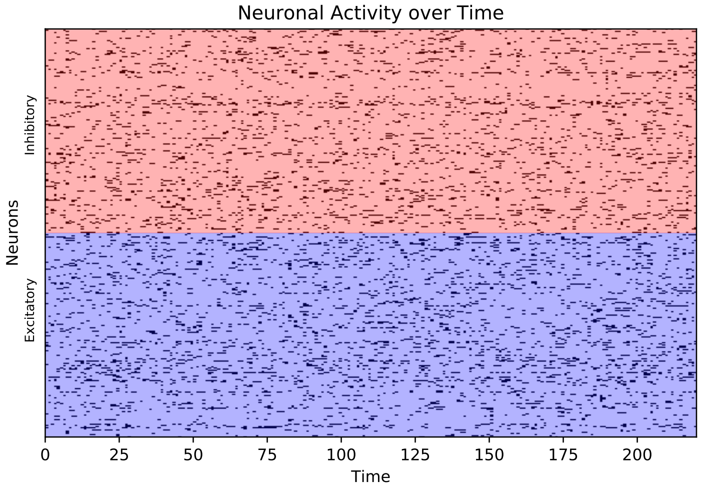


Figure 3.4: Overview over neural activity of whole network over time. Some neurons are highly active, others not at all. Each process seems to be seemingly independent of other processes. All Parameters as defined in Table 1

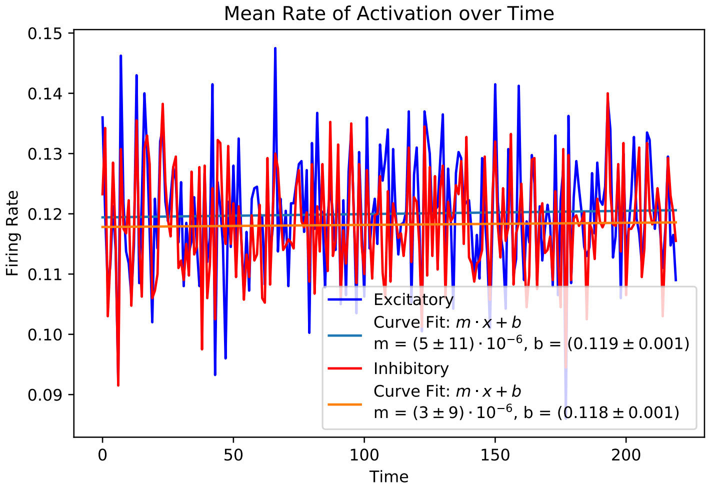


Figure 3.5: High Fluctuations without trending in a direction. Mean firing rate over time. All parameters as defined in Table 1

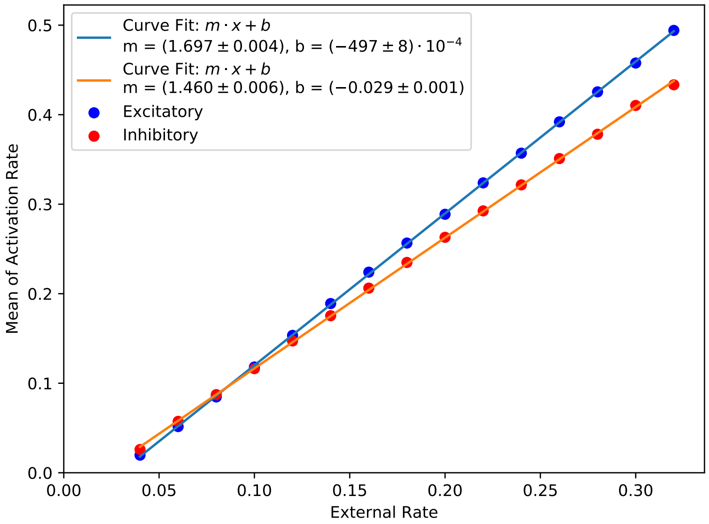


Figure 3.6: Linear Relation between external mean activity m_0 and excitatory and inhibitory mean activation m_E and m_I. Measuring mean activity over 20 timesteps for each datapoint. Other parameters as defined in 1.

3.6 Differences between neurons

Next we look at the distribution of firing rates in the population. This is displayed in Figure 3.7. The firing rate is normalized. It rapidly increases and increases and peaks at roughly half the average firing rate. Then it falls rapidly in a nonlinear fashion. As discussed above, each neuron has a distinct number of connections. This means each neuron has a distinct composition of inputs. Which in turn allows for some neurons to be highly active while others are unlikely to fire at all. Exactly this is here on display. We are able to replicate the predictions by Vreeswijk and Sompolinsky [1998](see also Figure A.5).

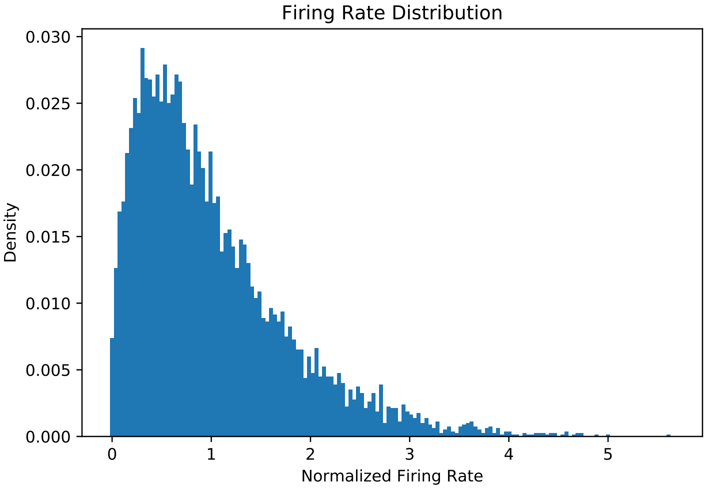
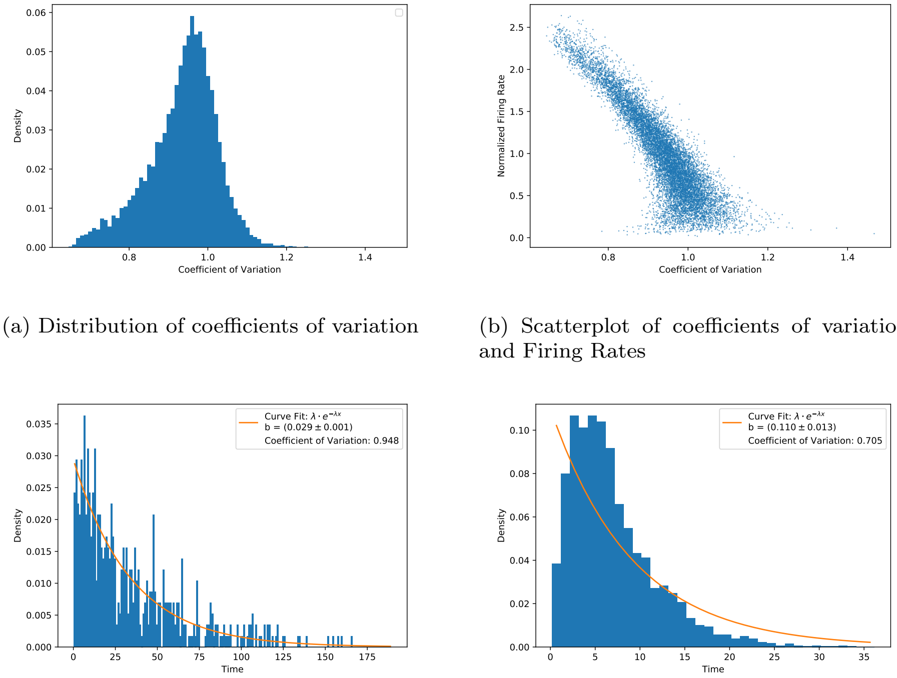


Figure 3.7: Distribution of Firing Rates. The normalized firing rate is calculated by dividing each rate through the mean firing rate.

3.7 Interspike Rate

Now we turn our attention to one of the main questions of Vreeswijk and Sompolinsky [1998]. The seemingly random behavior of neurons in vivo. For this we look at two measures: The interspike intervals and coefficients of variation.

Figure 3.8a shows the distribution of the coefficient of variation. The lowest CVs are at around 0.7, then it rises quickly. The peak is around a CV of around 0.95 and falling of quickly therafter. The highest value is measured at roughly 1.2. With a majority of neurons having a CV of close to one, this means the majority of them follow an exponential distribution. In subfigure 3.8b, the points are highly clustered implying a strong correlation between the CV and firing rate. The highest firing rates have the lowest coefficients of variation. This almost negative and linear correlation holds for almost all points. The



(a) Distribution of coefficients of variation

(b) Scatterplot of coefficients of variation and Firing Rates

(c) Interspike Interval of an irregular spiking neuron

(d) Interspike Interval of a fast spiking neuron

Figure 3.8: Simulation over 20,000 time steps. Otherwise ((a) Distribution of coefficients of variation, Firing rate and CV calculated per neuron; (b) CV vs firing rates, Firing rate and CV calculated for each neuron; (c) ISI irregular spiking neuron; (d) ISI fast spiking neuron;)

neurons with the lowest firing rates, however, spread out more widely. For very low firing rates the variance of the variance rises as events become rarer and therefore more driven by fluctuations. A CV lower than one implies a more regularly firing neuron. This seems to be the case for fast spiking neurons fire.

3.8 Memory Capabilities

In this section, we now look at how the system with changing external inputs. At random, we choose between a low or high external input m_0. For the following, we chose inputs of $m_0 = 0.1$ and $m_0 = 0.3$. Otherwise this follows the baseline assumptions of the last chapter as outlined in Table 1.

We trained a linear classifier to identify whether input was high or low in the current period. We also trained it, to identify the input level for previous periods(prediction with delay). The readout worked well for the current period and got worse over for each iteration as can be seen in Figure 3.9. With a delay of one, the readout was still relatively reliable, however there was a high level of fluctuation. For a larger delay the prediction accuracy was hardly better than chance.

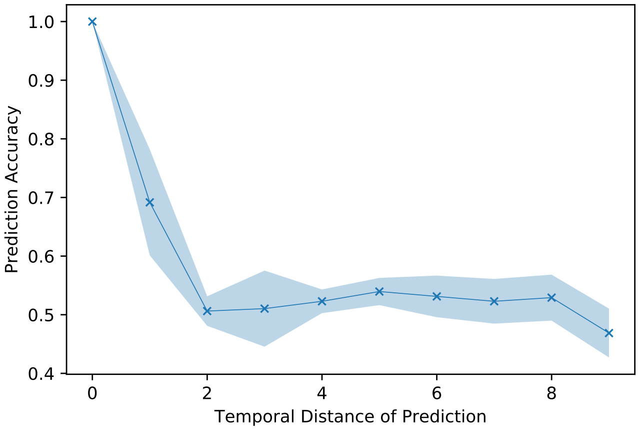


Figure 3.9: Memory Capacity of Network drops of rapidly. External mean activation m_0 randomly changes between 0.1 and 0.3. Share of times logistic regression correctly labels activation as high or low in dependency of how many timesteps have passed between labeled event and prediction. All other Parameters as defined in 1.

Next we investigated the question whether the relative strength of external and internal excitatory input has an effect on the quality of the prediction. The line of thinking goes as follows, if a higher share of input is dependent on the network input, there can be more information used from it. We varied both the strength of the external input E_E as well as the strength of the internal excitatory to excitatory input J_{EE}. Results are displayed in Figure 3.10.

Contrary to the naive assumption, a higher internal input does not directly translate to higher predictive quality. The best predicitive results seem to be achieved for high levels of external strength indicator E_E and internal excitatory strength levels J_{EE} around 1.1. It seems to be like a plateau ending around

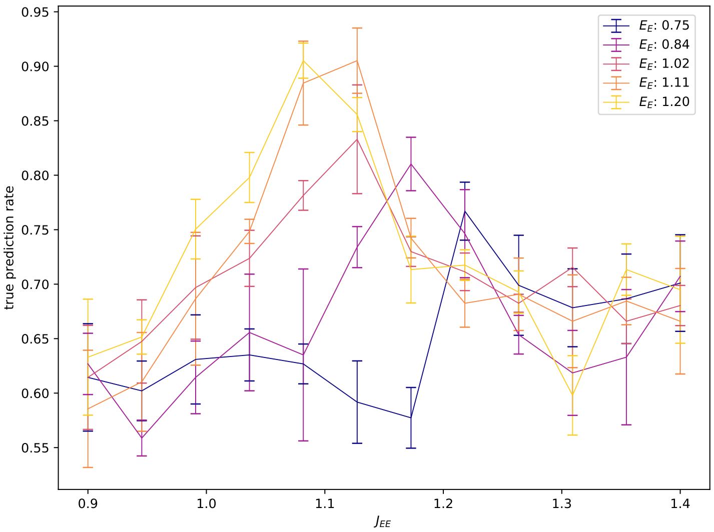


Figure 3.10: Prediction of high or low level of external activation m_0 after one time step has passed. Correctness of Linear Readout in relation to the strength of the Internal Excitatory to Excitatory connection strength J_{EE}. The colors of lines stand for different levels of strength for the External Excitatory Input. Errorbars for five independant measurements for each datapoint.

roughly $J_{EE} = 1.2$. High predictive values can be reached for high enough values of E_E. The larger E_E, for the lower values of J_{EE} the prediction rate remains high. There seem to be two constraints: As presented in equation 2 both m_0 and E_E are proportional to total external excitatory input u_E^0. This means, if E_E becomes too small, the high and low version of u_E^0 become to similar to be told apart after minimal amounts of time. A larger difference between the two u_E^0, on the other hand, facilitates discerning the differences. To investigate why for larger values of J_{EE} the predictive quality diminishes we take a look at Figure 3.11 What we see here is the strength of Internal excitatory input. u_E^0 (defined in Equation 2) does not depend on J_{EE}. Also the range of E_E is comparatively narrow containing only a rise of 50%. Therefore, it suffices to look at the change of internal excitatory input. It covers the vast majority of change in the ratio of internal to external neurons. We see several things: A larger J_{EE} leads to higher internal input. For high levels of J_{EE} one cannot discern between different levels of E_E. This means it is also impossible to differentiate between different levels of m_0. In the area around $J_{EE} = 1.1$ the differences of trajectories for different levels of E_E is largest. This could also explain the higher prediction accuracy. There is enogh space for all four possible combinations (high-high, high-low,...) to occupy its own activation level. Meanwhile, if the internal input is too low, the current external input dominates and information of previous time steps is overwritten.

However even using those memory efficient factors, the predictive capacity does not improve by much for a higher temporal distance. To explain this, we will look at the chaotic nature of the system.

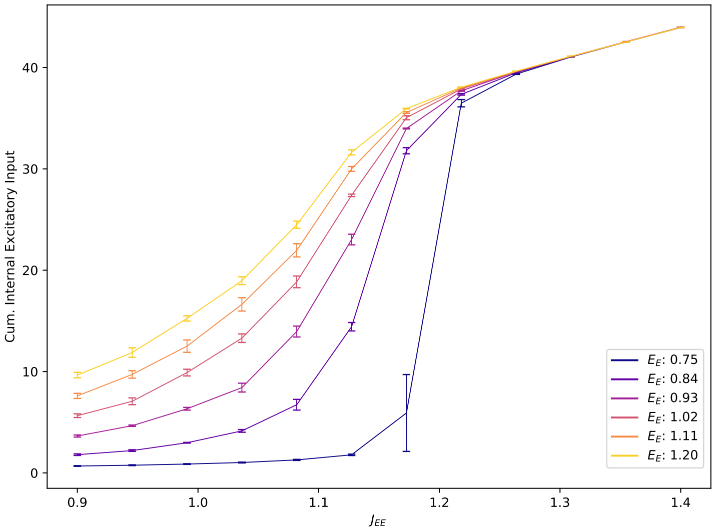


Figure 3.11: Drastic change in Input Structure. Internal Excitatory Input in dependence of Internal Excitatory to Excitatory Factor J_{EE}. In units of the excitatory threshold. The colors of lines stand for different levels of strenght for the External Excitatory Input. All parameters identical to Figure 3.9.

3.9 Chaotic Behavior

For this we take a look at two opposing initializations of the system. We take a fully activated system and a fully inhibited system, meaning starting with a vector of $\sigma_i(t = 0)$ equaling either 0 or 1 for all units. The parameters are again as described in Table 1, especially E_E and J_{EE} are both set to 1 again. We track the mean firing rate change over time. The networks need to compensate for the lack or multitude of activated neurons and counteract the current state. This explains the drastic changes in the first time step. Specifically, the fully activated system cannot fire in the first time step, as it needs the neurons to deactivate first, before it can fire again. The updating process happens randomly, so there are some neurons which still have not been set to 0. The balanced equlibrium of activation is at below 20%. Therefore, likely a larger number of neurons are still active while not firing and keep the system surpressed. At the third time step however, the long term equilibrium is approached. The dead or fully inhibited network However, even in the second time step, with most neurons turned of, it still does not fire After that, the neurons slowly approach After two time steps, however, both networks are indistinguishable. The type of neuron does not have an effect either.

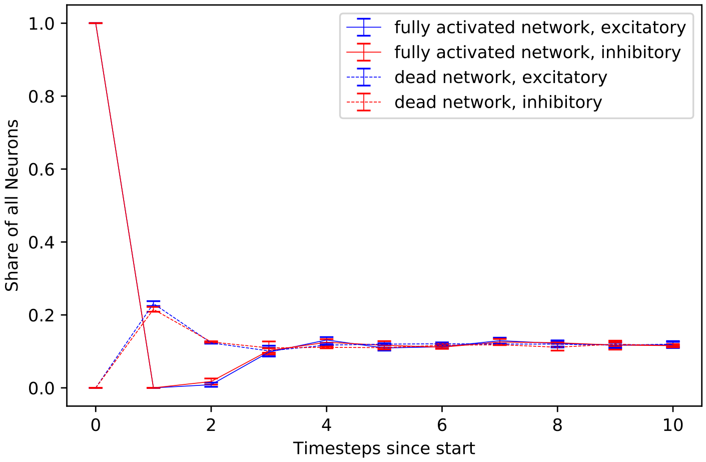


Figure 3.12: Initializing values to a fully inhibited (dead) or fully excited network. Measuring mean activation over time for both excitatory and inhibitory neurons. All parameters as in Table 1.

In our scenario we need to take into account that at any given time most of the system is not active. In Figure 3.13 we plotted two cases. We looked at the case of initializing the same starting condition and only flipping one value from 0 to 1 (or 1 to 0). We calculate the Hamming distance d_H for each time step maintaining the same update order of neurons. As a control we looked at the hamming distance to a randomly initialized starting vector $\vec{\sigma}(t = 0)$. The Hamming distance rapidly increases and after about five time steps it has reached a comparable level to the control. It remains a bit lower than the control also in the larger limit. It is possible for the difference to be extinguished in the first time step. It occurred seldomly however and has been excluded from the displayed data. **The remaining differences, I cannot explain**. The control starts out with a larger Hamming distance because the mean activation in the beginning $m(t = 0)$ does not equal the stable state mean activation m.

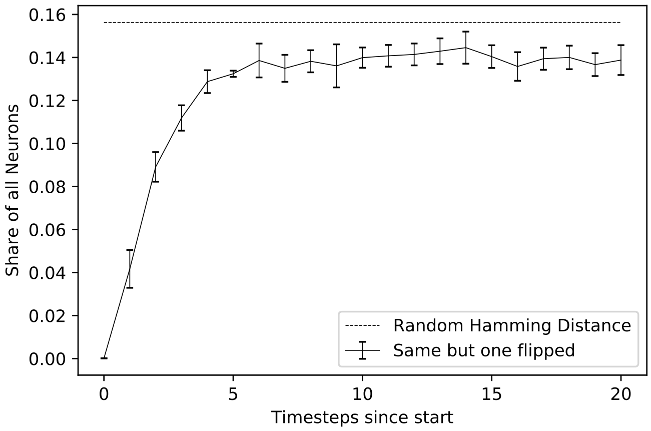


Figure 3.13: Chaotic Nature of Network. Hamming Distance of two versions of the same network (same parameters and update-order) with a single value flipped in initial vector $\vec{\sigma}(t = 0)$. Also, the Hamming distance of two unrelated initializations. Parameters as described in Chapter 2.2.

4 Discussion

4.1 Conclusion

We were able to replicate a balanced network exactly as Vreeswijk and Sompolinsky [1998] described it. We showed in Figure 3.1 that net input to a cell remains close to the threshold. In Figure A.8, we also showed the Poisson distribution of the interspike intervals. This displays the unpredictability of the system made up of deterministcally acting units. The wide range of different firing patterns is introduced by the indivdual amount connections the individual neurons posses. Some neurons can have more than average excitatory connections and a lesser amount of inhibitory connections. This leads to a high firing rate. The reverse would lead to a low firing rate.

The main purpose of a balanced network is as the name says to maintain a balance between excitatory and inhibitory input. This strongly reduces the space of possible states. We tried to find out whether we could determine any memory in this system. We found that for one time step you could drastically improve the predictive power of a logistic regression by adjusting the connection matrix prefactos. For larger time steps however, this approach seems futile. We showed this by both looking at very similar conditions which became more different on the level of individual neuron positions (measured by Hamming distance). And also how opposite starting position rapidly converged to the same mean activation. The system, in its current configuration, therefore does not display the right properties for reservoir computing.

4.2 Further Research Questions

Deploying a balanced network with a different parameter set. Is there a set of parameters or different composition of a balanced network that is capable

A Appendix

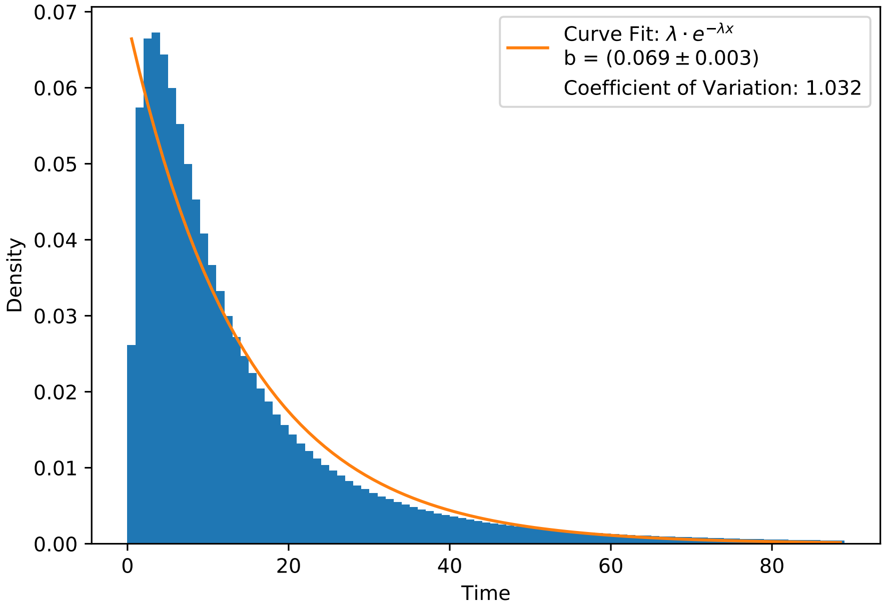


Figure A.1: Random nature of individual firing behavior. Histogram of Interspike Interval. Collecting the time differences between two spikes with time measured in τ_E. In the beginning, there is a short refractory period preventing a high frequency of spiking. For most of the data however, the likelihood of larger spiking intervals falls exponentially. Other Parameters as defined in Table 1

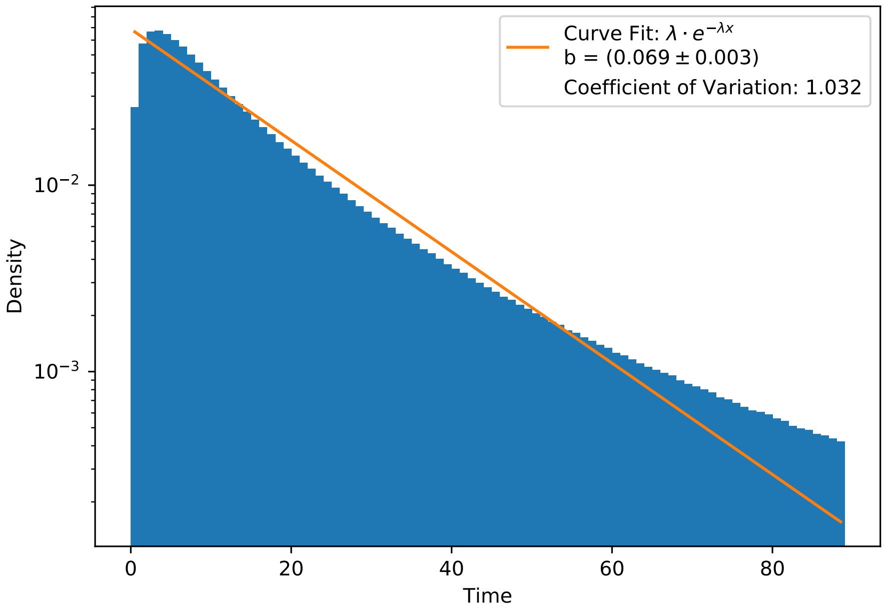


Figure A.2: Random nature of individual firing behavior. Histogram of Interspike Interval. Collecting the time differences between two spikes with time measured in τ_E. In the beginning, there is a short refractory period preventing a high frequency of spiking. For most of the data however, the likelihood of larger spiking intervals falls exponentially. Other Parameters as defined in Table 1

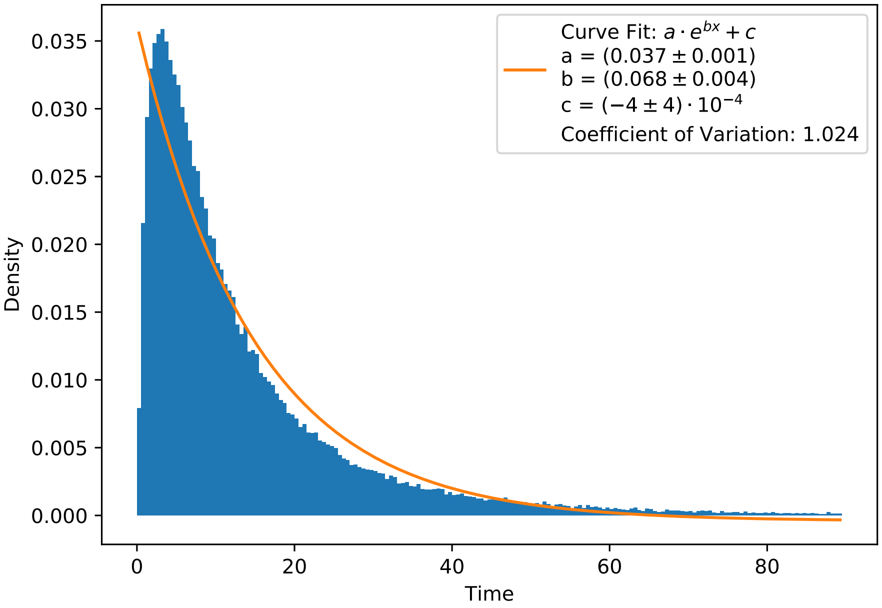


Figure A.3: Random nature of individual firing behavior. Histogram of Interspike Interval. Collecting the time differences between two spikes with time measured in τ_E. In the beginning, there is a short refractory period preventing a high frequency of spiking. For most of the data however, the likelihood of larger spiking intervals falls exponentially. Other Parameters as defined in Table 1

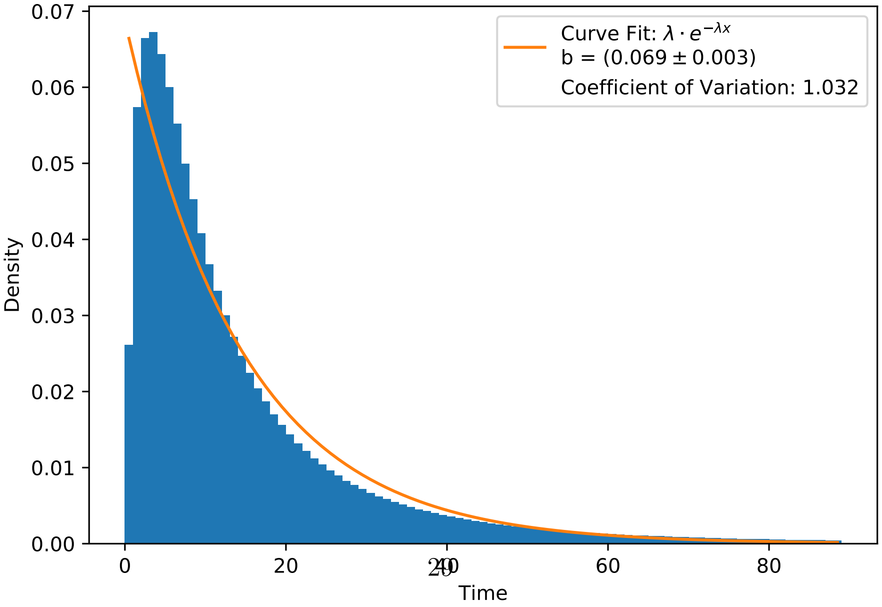


Figure A.7: Random nature of individual firing behavior. Histogram of Interspike Interval. Collecting the time differences between two spikes with time measured in τ_E. In the beginning, there is a short refractory period preventing a high frequency of spiking. For most of the data however, the likelihood

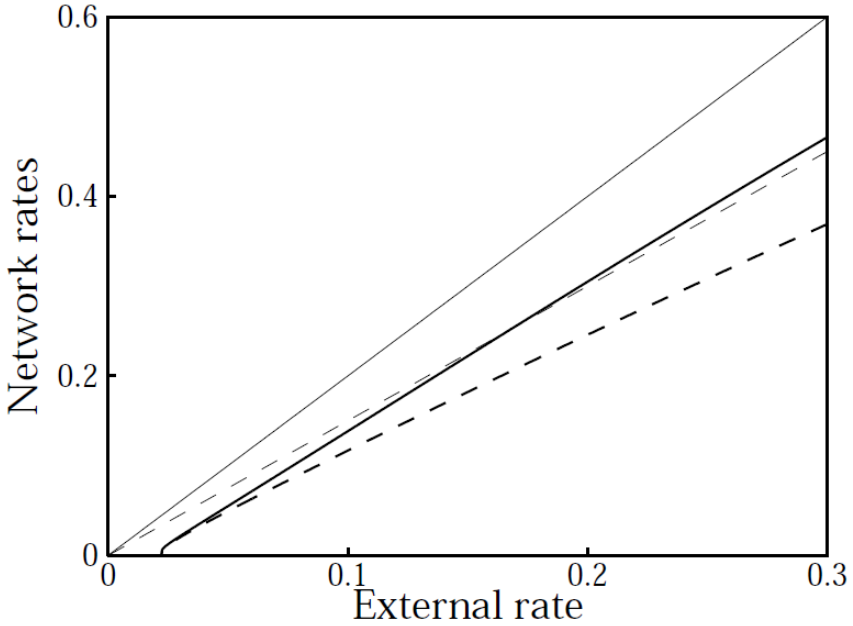


Figure A.4: Graph of Vreeswijk and Sompolinsky [1998] displaying how changes in external rate translate to changes in activation rate. Same parameters. Thin lines infinite K limit. Continuous line m_E, Dotted line m_I

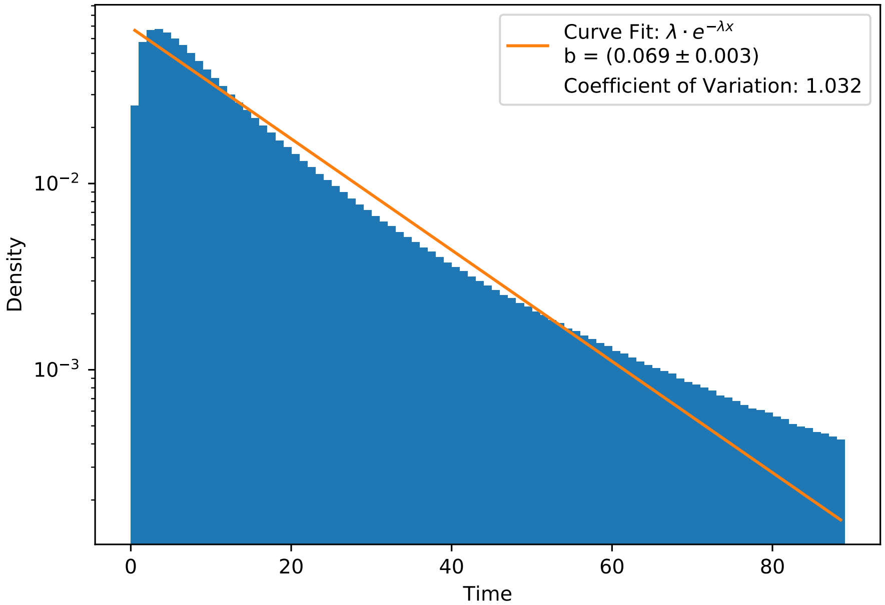


Figure A.8: Random nature of individual firing behavior. Histogram of Interspike Interval. Collecting the time differences between two spikes with time measured in τ_E. In the beginning, there is a short refractory period preventing a high frequency of spiking. For most of the data however, the likelihood of larger spiking intervals falls exponentially. Other Parameters as defined in Table 1

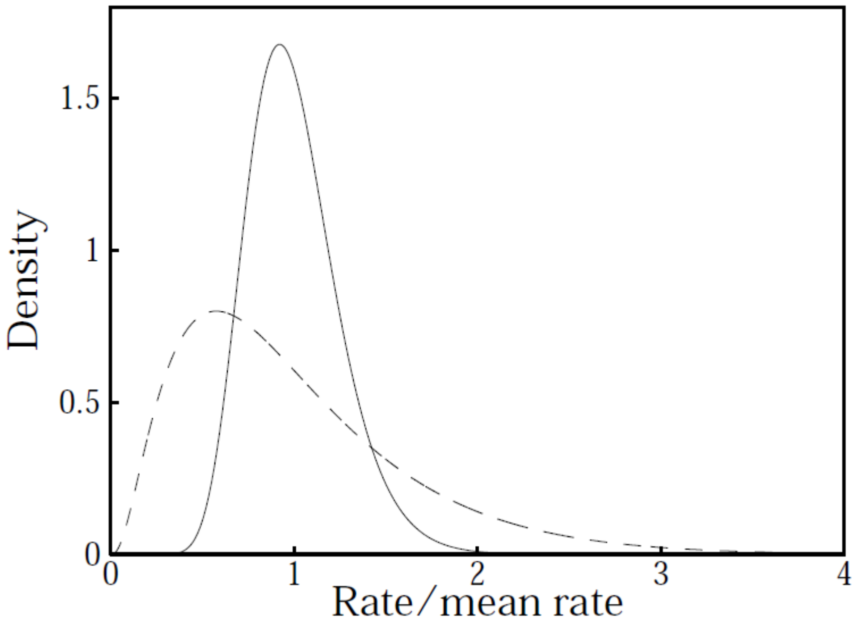


Figure A.5: Distribution of firing rates by Vreeswijk and Sompolinsky [1998]. Same parameters. dotted line for $m_0 = 0.01$, continuous line for $m_0 = 0.1$

A.1 (Short) User Manual for GUI

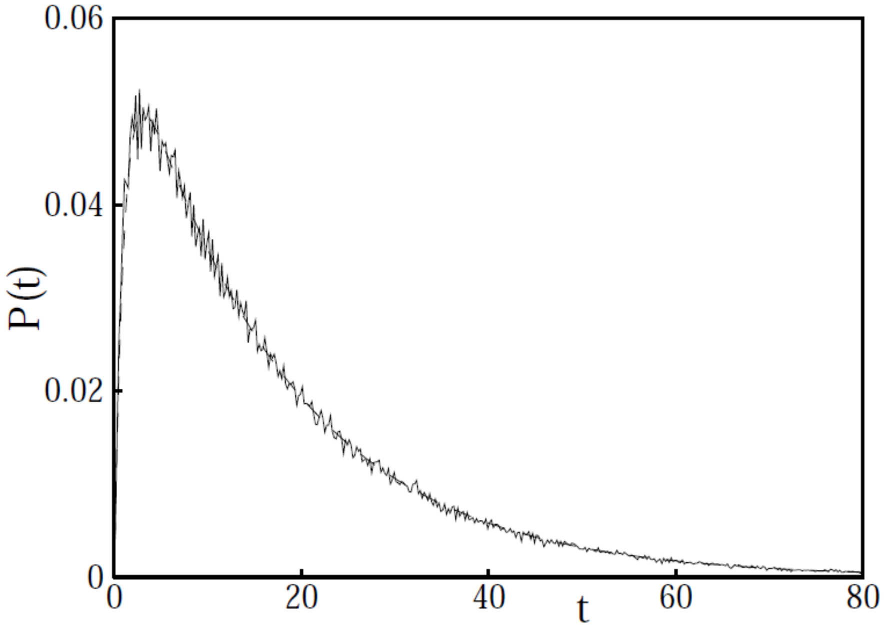


Figure A.6: Histogram of Interspike Distribution by Vreeswijk and Sompolinsky [1998], Same Parameters.

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