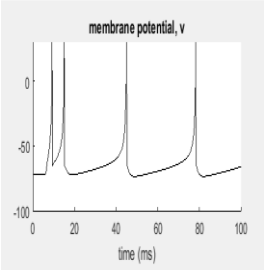
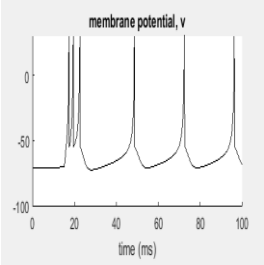
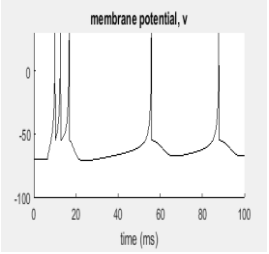
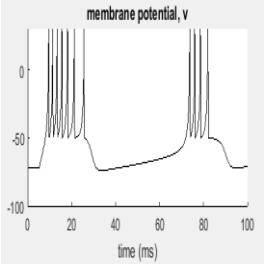
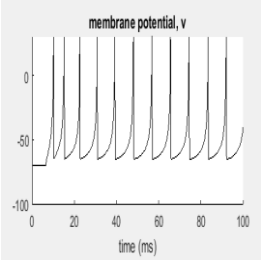
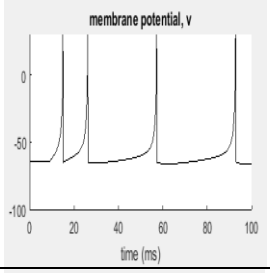
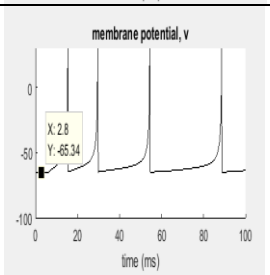
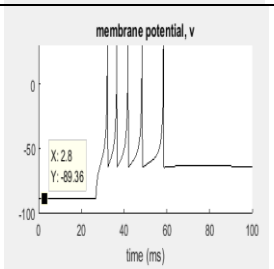
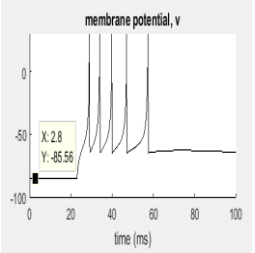
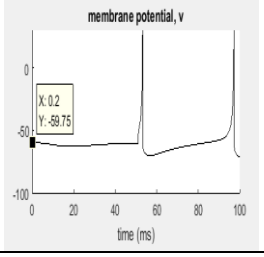
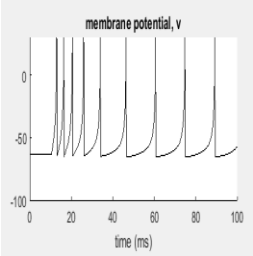
Part A: Firing Patterns

Type	Plot	Parameters	Comment
Regular spiking (RS)		*a* = 0.02 *b*=0.2 *c*=-65 *d*=8 *i*=14	• A class of excitatory neurons • Most common neurons in the cortex. • Prolonged injection of dc-current (*i*) results in few dense spikes (in this case 2) followed by an increase in inter-spike period; frequency adaptation. • Large after-spike hyperpolarization and the high Na^+ and K^+ conductance thresholds (low *c* and high *d*) regulate the spiking frequency, stopping it from becoming too fast even in the case of high injected currents.
Intrinsically bursting (IB)*		*a*=0.02 *b*= 0.2 *c*=-55 *d*= 8 * *i*=20	• A class of excitatory neurons • Characterised by an initial discrete burst of spikes. • Slow, intrinsic membrane currents in certain neurons can modulate the fast spiking activity. This is typically achieved by hyperpolarization due to the current build up during the continuous spiking. This results in the termination of the spike train and recovery of the membrane potential, finally stabilising into tonic spikes. • Found predominantly in layer 5 pyramidal neurons. • According to Izhikevich (2006), there are at least 4 theoretical mechanisms of modulation of spiking, hence (probably) the difference in parameters resulting in the same kind of spiking: ○ activation of outward current (voltage or calcium gated) ○ inactivation of inward current (voltage or calcium gated)
Intrinsically bursting (IB) (2)		*a*=0.02 *b*=0.2 *c*=-55 *d*=4 *i*=10	
Chattering (CH)		*a*=0.02 *b*=0.2 *c*=-50 *d*=2 *i*=10	• Also a type of bursting neuron • Are able to achieve high-frequency bursts of 3-5 spikes with a relatively short inter-burst period. • These are most commonly layer 3 pyramidal neurons. • in vitro, chattering activity is created due to voltage-gated potassium M-current (Wang 1999). • Chattering is created due to the presence of 2 variables of different speeds. The fast subsystem generates the spiking

			while the slow subsystem transitions from spiking to resting state (Izhikevich, 2006)
Fast spiking (FS)		*a*=0.1 *b*=0.2 *c*=-65 *d*=2 *i*=10	• Inhibitory cortical cells which fire at very high frequency without showing any adaptation. • Some neurons contain express voltage-gated potassium channels (Kv3 family). This allows a prompt response to incoming synaptic input and a sustenance of high-frequency spiking activity in response to strong input. (Freund and Kali, 2008) • In this model this is achieved by high *a* which in turn reduces membrane recovery time.
Thalamo-cortical (TC)*		*a*=0.02 *b*=0.25 *c*=-65 *d*=1* *i*=2.5	• Thalamo-cortical neurons provide the major input to the cortex. • The first kind of firing occurs when the neurons are at rest (*v* ~-60mV) and are depolarised resulting in tonic firing, very similar to RS. • similar spiking patterns were achieved with different parameters as shown by (*), however at *d*=0.05, the model could also recreate the characteristic TC pattern of rebound bursts of action potentials after neuron had been hyperpolarized to -90mV.
Thalamo-cortical (TC)		*a*=0.02 *b*=0.25 *c*=-65 *d*=0.05 *i*=1.5	
Thalamo-cortical (TC)*		*a*=0.02 *b*=0.25 *c*=-65 *d*=0.3* *i*=-36(and released)	
Thalamo-cortical (TC)		*a*=0.02 *b*=0.25 *c*=-65 *d*=0.05 *i*=-26 (and released)	
Resonator (RZ)		*a*=0.1 *b*=0.26 *c*=-65 *d*=2 *i*=0.2 (excitatory pulse after	• Characterized by the low amplitude subthreshold oscillations (damped or sustained) and low threshold spiking. This is created due to the high *b* parameter which couples *v* and *u* more strongly. • The high value of *a* also signifies that the

		small waves)	recovery period after depolarization is very fast.
Low-threshold spiking (LTS)		*a*=0.02 *b*=0.25 *c*=-65 *d*=2 *i*=10	• Also a class of inhibitory cortical cells • These are often triggered after an IPSP due to the fast recovery of the T-type Ca^{2+} channels during IPSP. • Similar to fast spiking neurons, however, they do reach a point of adaptation. • The high *b* parameter gives them a low firing threshold, thus the fast spiking pattern

*shows deviation from parameters indicated on Izihikevich's (2003) article.

The above spiking patterns were produced by a model using bifurcation methodologies reduced to 2 equations:

$$v' = 0.04v2 + 5v + 140 - u + I$$

$$u' = a\,(bv - u)$$

where *v* is the membrane potential in mV

u is the membrane recovery variable

' is *d/dt* where *t* is time in ms

model also included a simulation of auxilliary after-spike resetting, mimicing repolarization due to the opening of voltage gated K^+ channels

if $v \geq 30mV$, then $v = c$ and $u = u + d$

where *a, b , c* and *d* are variable dimensionless parameters.

a --> timescale of *u*

b --> sensitivity of *u*

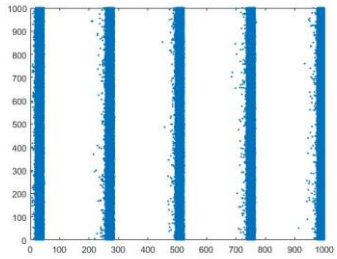
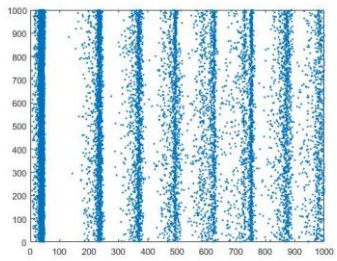
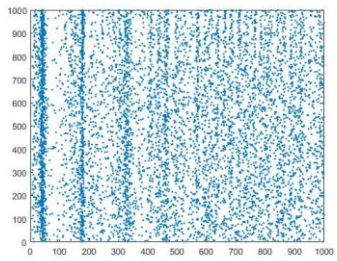
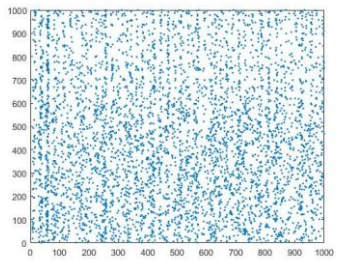
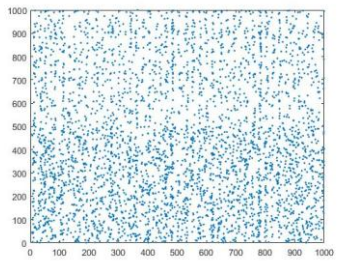
c --> after spike reset value of *v*

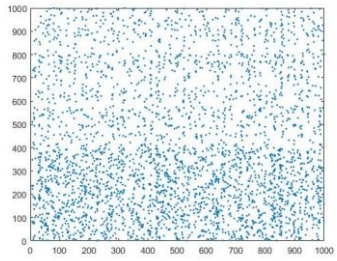
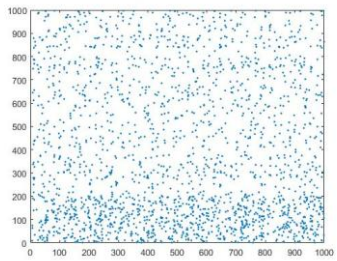
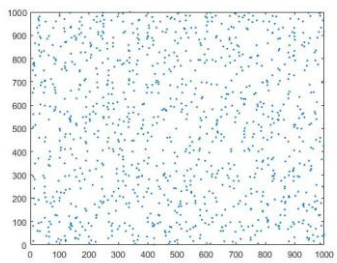
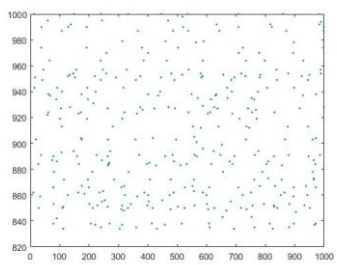
d --> after-spike reset of *u*

and *i* is the variable DC induced current

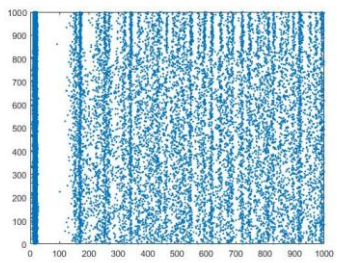
Part B: Network of Neurons

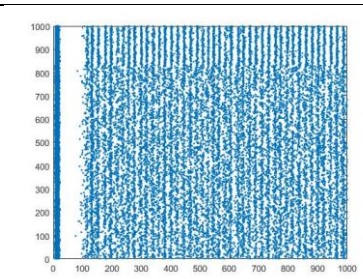
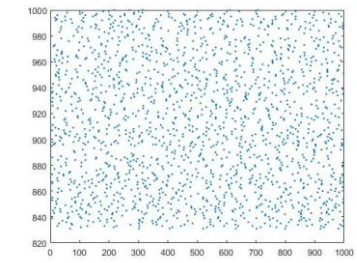
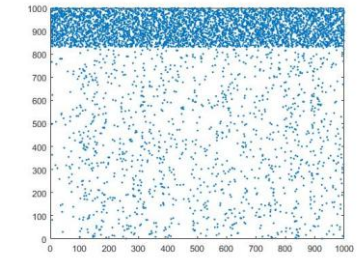
a)

Image	Ne	Ni	Thalamic noise	Comment
	1000	0	5-2	As displayed in the figures on the left, going down the column, there is a pattern going from 100% excitatory neurons to 100% inhibitory neurons. The horizontal axis of the plots represent time in ms while the vertical axis represent neuron numbers from 1 to 1000 (ratio of excitatory to inhibitory as specified in columns 2 and 3). The inhibitory neurons are represented in the top portion of the plot while excitatory neurons are represented in the bottom portion. At the default thalamic input of 5:2 (excitatory : inhibitory), varying the ratio of the number of excitatory to inhibitory neurons, results in a synchronization pattern of distinct spike trains in the 100% excitatory to a completely random spiking pattern in 100% inhibitory neurons. It is interesting to note that the frequency of the oscillations also changed. At 100% excitatory, neurons formed 5Hz oscillatory patterns, at 83:17, it showed an 8z oscillatory pattern (alpha rhythm) while at 4:1 ratio (the natural ratio of the mammalian cortex), the frequency was seen to change within the same second, from alpha rhythm to gamma rhythm. Also, oscillations are propagated equally through the inhibitory and excitatory neurons. The gamma rhythm remained visible
	830	170	5-2	
	800	200	5-2	
	600	400	5-2	
	500	500	5-2	

	400	600	5-2	down to 2:3 ratio after which, synchronization of neurons appears to be absent. The boundary between excitatory and inhibitory neurons was not very evident at ratios above 3:2. However below that, the boundary became more and more evident as inhibitory neurons showed increasingly reduced activity. In conclusion, it appears that at ratios above 2:3, all conditions (such as coupling strength, time delay and excitatory to inhibitory ratio) are met, for synchronized oscillations to form. Since plasticity is not really included in this model, one cannot really attribute this to a neural ensamble-type behaviour, however, it shows that even without special plastic connections between specific neurons, it is possible for oscillatory patterns to emerge. Therefore, while the two phenomena could be closely connected in function and even causational to one another, they are two distinct processes which can appear independent from eachother.
	200	800	5-2	
	0	1000	5-2	
	830	170	2-2	

b)

Image	Ne	Ni	Thalamic noise	Comment
	830	170	7-2	For this part, I chose to keep the ratio of 83:17 (excitatory : inhibitory) as it showed only one oscillatory pattern and thus, other changes could be more easily identified when changing a secondary variable in my opinion.

	830	170	10-2	The thalamic noise from the excitatory part was first varied. Increasing the parameter from 5 to 10, showed a distinct increase in frequency. oscillations changed from alpha to gamma frequencies and spike trains remained equally distinctive. On the other hand, increasing the inhibitory thalamic noise disrupted the synchronization and created random spiking patterns. The role of thalamic noise in cortical neurons is still not thoroughly understood, however, through this model it is evident that at least theoretically, it is possible that its prevalence in excitatory neurons, aids in the synchronization of neural spiking resulting in oscillations.
	830	170	0-5	
	830	170	5-10	

References:

- Eugene M. Izhikevich (2006) Bursting. Scholarpedia, 1(3):1300., revision #150167
- Izhikevich, E. M. (2003). Simple model of spiking neurons. *IEEE Transactions on neural networks*, *14*(6), 1569-1572.
- Tamas Freund and Szabolcs Kali (2008) Interneurons. Scholarpedia, 3(9):4720., revision #89023
- Wang X.-J. (1999) Fast burst firing and short-term synaptic plasticity: a model of neocortical chattering neurons. Neuroscience, 89:347-362.
- Zhan, XJ; Cox, CL; Sherman, SM (2000). "Dendritic depolarization efficiently attenuates low-threshold calcium spikes in thalamic relay cells". The Journal of Neuroscience. 20 (10): 3909–14. PMID 10804230.