Simulating LGN Response to Background Irradiance on SpiNNaker

Ajay Subramanian
Department of Electrical and Electronics Engineering
BITS Pilani K K Birla Goa Campus
f20170371@goa.bits-pilani.ac.in

Prof. Basabdatta Sen Bhattacharya
Associate Professor
Department of Computer Science and Information Systems
BITS Pilani K K Birla Goa Campus
basabdattab@goa.bits-pilani.ac.in

I. Introduction

Much work has been done recently through physiological experiments to investigate the role of background visual stimuli in causing fast narrowband oscillations in areas of the visual thalamus. Luminance has been found to have a direct relationship with the amplitude of these oscillations in the retina and dLGN [11], [14] and contrast is known to affect and control their gamma-band power spectra [3]. Using SpiNNaker, a neuromorphic computing platform [1], to model spiking neurons, we aim to validate these physiological LGN experiments with simulated results on a spiking neural network model of the LGN. We use the the Izhikevich neuron based sPyNNaker model developed in [12] as the building block.

II. Background

A. Lateral Geniculate Nucleus

The Lateral Geniculate Nucleus is a region of the visual thalamus that is responsible for relaying signals from the retina to the visual cortex of the occipital lobe. Like other areas of the thalamus, the cells in this region are primarily of three types - thalamo-cortical relay cells (TCR), reticular neurons (TRN) and thalamic interneurons (IN). TCR cells form an excitatory connection between TRN and cortical cells (mainly the primary visual cortex) while there exists inhibitory feedback connections from TRN and cortical IN to the TCR which regulate and stabilise the thalamocortical circuit.
From a functional perspective, the thalamocortical cells is triggered by sensory and feedback inputs from other areas of the brain and body. When depolarized to a voltage of less than -45 mV, the TCR cells exhibit gamma oscillations which lie in the gamma range of the frequency spectrum (25-40 Hz) [9], [13]

B. Spiking Neural Networks

Spiking neural networks are computational models that simulate neural behavior in biological systems. Like biological networks, they are driven by discrete spike trains whose instantaneous frequency encodes data. These spikes, by either polarisation or depolarisation, will alter the membrane potential of neurons. Dynamics of the neuron are decided by a set of differential equations that relate membrane voltage with conductance and synaptic current. If a neuron's membrane potential exceeds a certain threshold value, it spikes, passing the integrated information to the next neuron. The influence of a synapse on the membrane voltage of the next neuron can be of two types: excitatory and inhibitory. Excitatory synapses depolarise the neuron, pushing it towards the threshold voltage while inhibitory synapses polarise, thereby reducing the likelihood of a spike. Inhibitory neurons are generally useful to regulate spiking frequency and increase more information contained per spike of the postsynaptic neuron.
There are various neuron models that mimic fairly accurately, the dynamics of biological neurons. The Hodgkin-Huxley model (1952) [4] is a conductance-based model that uses a set of non-linear differential equations to explain the electrical characteristics of neurons. While it is a powerful model which is biophysically meaningful, it is very complex and computationally expensive to simulate. Hence a more popular option recently has been the Izhikevich neuron model [5] since it is both accurate and computationally efficient for simulation purposes. Below are the differential equations that the Izhikevich model uses to represent the spiking activity of a neuron.

$$\frac{dv(t)}{dt} = 0.04v^2(t) + 5v(t) + 140 - u(t) + I_{psc}(t) + I_{dc} \tag{1}$$

$$\frac{du(t)}{dt} = a(bv(t) - u(t)) \tag{2}$$

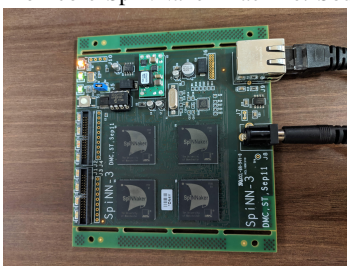
$$\textit{If } v(t) > 30\textit{, then } v(t) \leftarrow c; u(t) \leftarrow u(t) + d$$

where a, b, c, d are parameters that affect the dynamics of the model and can be chosen to obtain different kinds of spiking behavior. $u(t)$ is the recovery variable $v(t)$ is the membrane voltage. I_{psc} is the post-synaptic current and I_{dc} is the DC bias current stimulus.

C. Simulating Spiking Neural Networks

The SpiNNaker project [1] has developed a massive 1 million core parallel digital supercomputer (Fig 1a) with a low power ARM processor based architecture, inspired by connections in biological neuronal systems.

(a) 1-million core SpiNNaker machine. Source: [2]



(b) 18-core SpiNN3 hardware with 4 SpiNNaker chips. Can be used for smaller scale simulations.

Fig. 1: SpiNNaker machines

It uses a bio-inspired architecture that was built keeping in mind the following design philosophy:

- **Scalability**: A system that attempts to model the brain should be scalable to large networks of neurons and connections. Therefore, a generalizable low level architecture is essential.
- **Energy efficiency**: To ensure wider investments and adoptability, a large scale system such as SpiNNaker should be energy efficient and economical.
- **Bio-inspired hardware**: Arguably the most important requirement. The SpiNNaker architecture uses ¿1 million ARM9 cores that communicate via packets through a custom fabric. In order to ensure that packets are similar to neuron spikes, the packet size is very small but transmitted at a very high rate of 5 billion packets per second.

It can be used for large scale simulations of spiking neurons at biologically plausible time-scales. It can be interfaced with software models using the sPyNNaker [10] a PyNN-based interface to SpiNNaker. It enables conversion of a PyNN script into a form suitable for execution on a SpiNNaker machine. A few advantages of using PyNN as a description language are:

- A single code can be run on different simulators without modification
- High level abstraction
- API versioning

It is possible to access the large SpiNNaker machine through the Human Brain Project portal in order to run large-scale simulations. However, remote access is slow and some model might not require such a large machine for simulation, for example, simulating a 140 neuron single node LGN. For such use cases, we make use of a much smaller hardware with only 18 ARM cores and 4 SpiNNaker chips, called SpiNN3 (Fig 1b). This can be accessed locally via an Ethernet connection and hence speeds up small scale simulations. Even for larger models, it can be used for debugging since doing so with the large machine would be a very long feedback loop.

III. Related Work

A. LGN Response to Irradiance

The response of LGN cells to varying background light intensity was first studied by Storchi et al. in [14] via physiological irradiance experiments on mice. Some of the relevant and important results established in the work are as follows:

- Irradiance results in a higher dLGN firing rate and fast periodic spiking predominantly in the β-γ range of the frequency spectrum i.e. from 25 - 50 Hz.
- Power is modulated by varying contrast of background light at lower luminance.
- In bright conditions, the oscillations were centred at a frequency of 60 Hz and exhibited a gradual dependence on irradiance.
- At a certain level of luminance called L0, varying the contrast results in amplitude modulation of the collected SSVEP signals. When we increase the brightness to a higher level L1, the same variation in contrast causes both amplitude and frequency modulation. Finally at a higher value L2, we observe only frequency modulation for the same stimulus.

B. SpiNNaker LGN Model

Sen-Bhattacharya et al. in [12] proposed and developed a sPyNNaker model of the dLGN that mimics its properties using Izhikevich neurons [5] and 3 neuron populations representing TCR, TRN and IN cells. The three populations are connected to each other as shown in Fig 2.

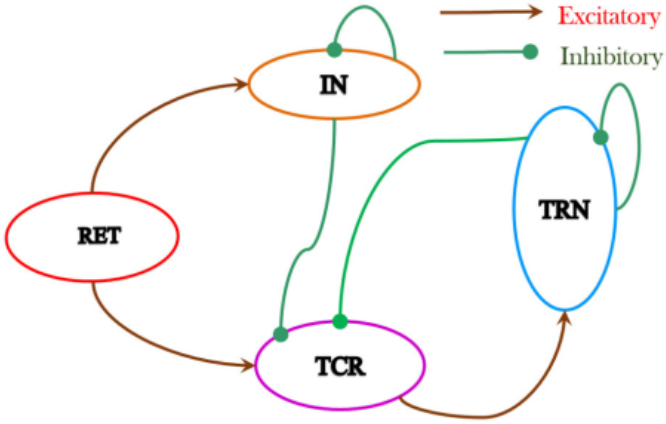


Fig. 2: The single node LGN consists of 3 kinds of cells: TCR, TRN, IN. TCR connects the cortex to the thalamus with excitatory synapses while IN and TCR inhibit TCR to regulate the circuit. Source: [12]

As mentioned in II-A the TCR population connects the retina to thalamocortical with excitatory synapses and is regulated by inhibitory feedback connections from the TRN and IN cells. Beyond that, IN and TRN also have inhibitory connections to themselves.
The model consists of 80 TCR, 20 IN and 40 TRN cells making a total of 140 neurons. It uses fixed probability connectors with connection probability and weights derived from biological systems. Since the TRN is known to have a considerably lower inhibitory effect than IN, its probability is initialized to only 0.077 while for IN it is 0.232. Even weight follows a similar pattern; 2 for TRN to TCR and 8 for IN to TCR. Another detail is that the SpiNNaker model simulates with a timestep of 0.1 which means that 1 second for the model takes 10 seconds to actually run.
However, a 140 neuron system like this will not be enough to simulate a robust biological behavior for physiological system. For such a case, we will need a much larger model that uses this one as a building block. Sen-Bhattacharya et al. [12] have also explored a way to combine these to model a multi-node LGN, though they only use three LGN nodes for the purpose of illustration. As shown in Fig 3, it consists of three LGN nodes arranged in a circular topology and connect to each other with TRN-TCR and TRN-TRN inhibitory connections and TCR-TRN excitatory connection between nearest neighbour nodes. This creates an interdependence of spiking activity between LGN nodes, thereby simulating large networks of cells. Ideally, for biorealistic simulations we would require a large number nodes and more variability in the initialization parameters of the model. Both of these issues will be addressed in our modified model in future sections.

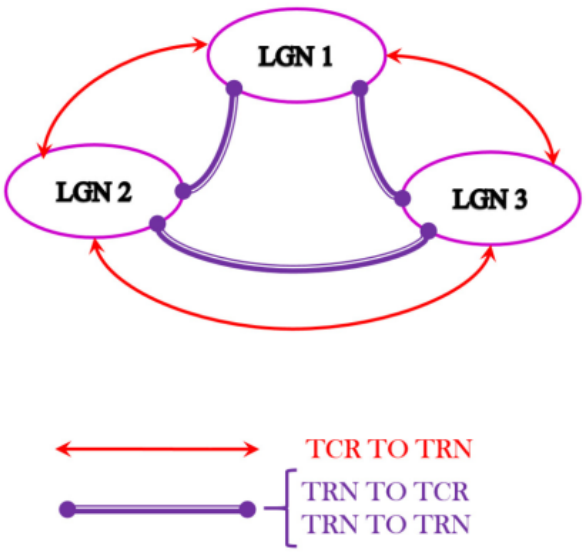


Fig. 3: A three-node, 420 neuron LGN model with inhibitory interconnections for TRN-TRN and TRN-TCR and excitatory TCR-TRN connection. Source: [12]

IV. Our Work

Having given a concise background of prerequisite concepts and previous work relevant to this project, we move on to describing the objectives, setup and procedural details of our work.

A. Objectives

Through this work we aim to validate the results of [14] using a sPyNNaker model of the LGN. We perform the following simulations in this regard:

1) **Shifting luminance:** Observe variation of firing rate and spike amplitude with variation in luminance. This acts as a sanity check for our model following which we can begin our simulations.
2) **Synchrony for single node LGN:** Synchrony is one of the properties observed in most biological neuron populations, including the LGN, and also contributes to feature extraction in the visual cortex [6]. So we will use synchrony as a metric to validate our single node LGN model before moving on to larger populations.
3) **Finding L0, L1, L2 luminance:** We will then carry out experiments to find out the L0, L1 and L2 luminance values after introducing variability to the initial voltage and time delay values for each population.

We will now discuss each of these steps in more detail. Until now, we have completed step 1 and are working on step 2 currently.

B. Experimental setup

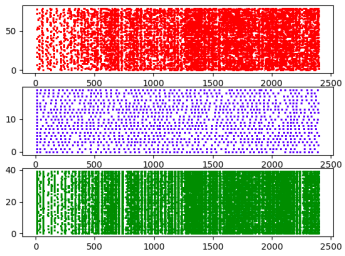
The single node LGN has already been described in detail in III-B. It has 140 neurons consisting of 80 TCR, 40 TRN and 20 IN cells. In our experiments, we combine 200 of these with nearest neighbour connections to form a large circular string of LGN nodes. It consists of $200 * 140 = 28000$ Izhikevich neurons. The DC bias value for these neurons represents the level of luminance (or brightness) and is initialized to 4.0. Apart from the TCR, TRN and IN cells, we include Poisson noise inputs to each of these populations. These 3 populations $noise2TCR$,$noise2TRN$ and $noise2IN$ simulate the contrast in brightness. There are several measures for contrast, the most popular of them being Michelson contrast [8] which is defined as

$$\frac{I_{max} - I_{min}}{I_{max} + I_{min}}$$

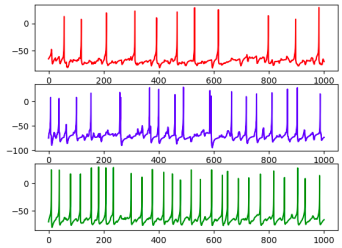
The Poisson rate for these noise populations is initialized to 30. For the irradiance experiments, we make use of a *loom* luminance input. This entails selecting a few nodes to irradiate strongly and move the light bar from left to right. In our experiments we provide irradiance to 40 consecutive 'central' nodes. The spiking activity is expected to be transferred from these to the other nodes since their TCR and TRNs are connected through excitatory connections.

Luminance (DC bias)	Time (in ms)
4	200
30	400
90	800
270	800
4	200

TABLE I: Simulation details for shifting luminance.



(a) Spike raster plot for luminance shifting.



(b) Membrane voltage plot averaged across a single population for the luminance shifting experiment.

Fig. 4: Luminance shifting experiment

C. Shifting luminance

In this step, we ran the 200-node LGN model on varying luminance values for a total of 2400 ms split up into the sequence shown in Table I

The results for the simulation was as expected with neuron firing rate being proportional to luminance as seen in Figure 4a. Also since the contrast is kept constant during the duration of the experiment, we do not observe any change in the amplitude of spikes (Figure 4b).

D. Synchrony for single node LGN

This step involves returning to the single node LGN model and calibrating its weights in order to observe the property of synchrony that is commonly seen in the physiological model of the LGN and other areas of the thalamus [6], [7]. The task will be to gradually increase the synaptic weight values until we observe neurons oscillating synchronously. We expect results similar to that shown in Fig 5. However, before simulation we must introduce variability to the initial membrane voltage, Poisson noise rates and time delays between neuron populations to ensure that the default spiking activity of the populations is not synchronized. A good way to implement this would be to select these values from a uniform random distribution with common mean and a fixed variance.

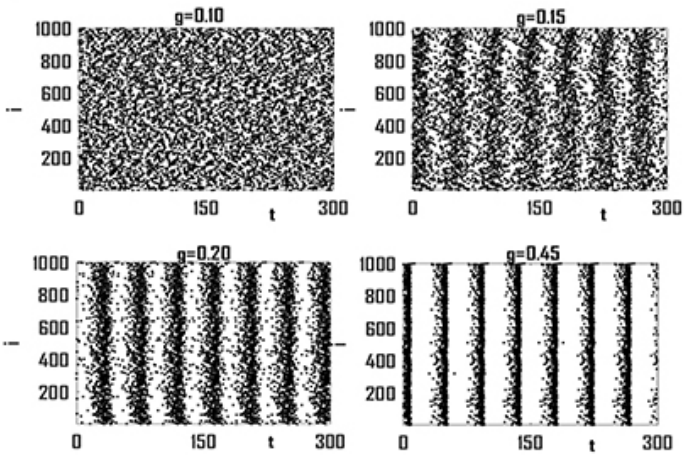


Fig. 5: As weight g is increased, neurons start spiking at similar times thereby exhibiting synchrony. Source: [7]

E. Finding L0, L1, L2 luminance

Once again, we move back to the multi-node LGN string model. In this step, our task is to find the values of L0, L1 and L2 luminance. As described earlier, at L0, the neuron voltage oscillations are amplitude modulated by varying contrast. Similarly L1 (at slightly higher irradiance, typically 10 times L0) causes both frequency and amplitude modulation and L2 (very high irradiance) causes only frequency modulation. We shall conduct the experiment in the following way.

1) Fix the value of luminance (DC bias) for all neuron populations at a selected constant value.
2) Simulate for a total of 2400 miliseconds divided into time intervals of 200. Each interval operates under a different contrast value (Poisson rate). However, for a given interval, contrast provided to all populations is the same.
3) Repeat until L0 luminance is found.

Once L0 has been found, we can repeat the same steps to find L1 and L2 luminance as well. Typically, L0, L1 and L2 luminance values are separated linearly as follows

$$i_{L0} = 0.1 * i_{offset}$$

$$i_{L0} = 1.0 * i_{offset}$$

$$i_{L0} = 10.0 * i_{offset}$$

and hence the others can easily be computed once one of them is found.

V. Conclusions and Future Work

Therefore, we have laid the framework for experiments that we have already conducted and plan to conducted on the spiking neural network of the LGN. In summary, we are interested in validating physiological results in studying the response of LGN to varying background light intensity or irradiance, achieved by Storchi et al. [14] by making use of

an extended version of the single node dLGN sPyNNaker model proposed by Sen-Bhattacharya et al. [12] wherein we construct a circular string of LGN nodes with nearest neighbour excitatory and inhibitory connections. We perform three phases of experiments on this model, namely:

1) Observe variation in oscillation frequency in the string LGN when subject to varying luminance and constant contrast.
2) Demonstrate the property of synchrony in an LGN initialized with noisy values, by changing the synaptic weight values.
3) Finally, finding L0, L1 and L2 luminance via controlled experiments performed in constant luminance and varying contrast.

We believe that achieving good results on these objectives will influence more neuroscience research to use computational models due to the convenience and accessibility thay they offer. Also, more work along similar lines will enhance our understanding of biological neuronal networks and also contribute to propelling fields like AI and neurological research.

Until now, we have satisfied the first of our three objectives and plan to continue our work on our other aims.

VI. Acknowledgements

Firstly, I would like to thank my advisor Prof. Basabdatta Sen Bhattacharya for her constant support, patience and motivation throughout the duration of my work. Her knowledge and tips were great resources for me while exploring and learning new concepts.

I would also like to thank Dr. Riccardo Storchi for his valuable inputs and providing direction for our research. Finally I thank the SpiNNaker team, University of Manchester and the Human Brain Project for enabling us to run simulations of the SpiNNaker machine through the HBP platform.

References

[1] S. B. Furber, F. Galluppi, S. Temple, and L. A. Plana. The spinnaker project. *Proceedings of the IEEE*, 102(5):652–665, May 2014.

[2] Steve Furber. Large-scale neuromorphic computing systems. *Journal of Neural Engineering*, 13(5):051001, aug 2016.

[3] J Andrew Henrie and Robert Shapley. Lfp power spectra in v1 cortex: the graded effect of stimulus contrast. *Journal of neurophysiology*, 94(1):479—490, July 2005.

[4] A. L. Hodgkin and A. F. Huxley. A quantitative description of membrane current and its application to conduction and excitation in nerve. *The Journal of Physiology*, 117(4):500–544, 1952.

[5] E. M. Izhikevich. Simple model of spiking neurons. *IEEE Transactions on Neural Networks*, 14(6):1569–1572, Nov 2003.

[6] Sean T. Kelly, Jens Kremkow, Jianzhong Jin, Yushi Wang, Qi Wang, Jose-Manuel Alonso, and Garrett B. Stanley. The role of thalamic population synchrony in the emergence of cortical feature selectivity. *PLOS Computational Biology*, 10(1):1–13, 01 2014.

[7] Mahsa Khoshkhou and Afshin Montakhab. Beta-rhythm oscillations and synchronization transition in network models of izhikevich neurons: Effect of topology and synaptic type. *Frontiers in Computational Neuroscience*, 12:59, 2018.

[8] Albert Michelson. Studies in optics. *University of Chicago Press*, 1927.

[9] null null, R. Llinás, U. Ribary, D. Contreras, and C. Pedroarena. The neuronal basis for consciousness. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 353(1377):1841–1849, 1998.

[10] Oliver Rhodes, Petruţ A. Bogdan, Christian Brenninkmeijer, Simon Davidson, Donal Fellows, Andrew Gait, David R. Lester, Mantas Mikaitis, Luis A. Plana, Andrew G. D. Rowley, Alan B. Stokes, and Steve B. Furber. spynnaker: A software package for running pynn simulations on spinnaker. *Frontiers in Neuroscience*, 12:816, 2018.

[11] Aman B. Saleem, Anthony D. Lien, Michael Krumin, Bilal Haider, Miroslav Román Rosón, Asli Ayaz, Kimberly Reinhold, Laura Busse, Matteo Carandini, and Kenneth D. Harris. Subcortical source and modulation of the narrowband gamma oscillation in mouse visual cortex. *Neuron*, 93(2):315 – 322, 2017.

[12] Basabdatta Sen-Bhattacharya, Teresa Serrano-Gotarredona, Lorinc Balassa, Akash Bhattacharya, Alan B. Stokes, Andrew Rowley, Indar Sugiarto, and Steve Furber. A spiking neural network model of the lateral geniculate nucleus on the spinnaker machine. *Frontiers in Neuroscience*, 11:454, 2017.

[13] M Steriade. Synchronized activities of coupled oscillators in the cerebral cortex and thalamus at different levels of vigilance. *Cerebral Cortex*, 7(6):583–604, 09 1997.

[14] Riccardo Storchi, Robert A. Bedford, Franck P. Martial, Annette E. Allen, Jonathan Wynne, Marcelo A. Montemurro, Rasmus S. Petersen, and Robert J. Lucas. Modulation of fast narrowband oscillations in the mouse retina and dlgn according to background light intensity. *Neuron*, 93(2):299 – 307, 2017.