Modeling computation and plasticity for an individual pyramidal neuron in the visual cortex

Sanjana Venkatesh

13 December 2024

Faculty Advisor: Michael Berry (Princeton Neuroscience Institute)

Submitted in partial fulfillment

of the requirements for the degree of

Bachelor of Science in Engineering

Department of Electrical and Computer Engineering

Princeton University

I hereby declare that this Independent Work report represents my own work in accordance with University regulations.

I hereby declare that this Independent Work report does not include regulated human subjects research.

I hereby declare that this Independent Work report does not include regulated animal subjects research.

Sanjana Venkatesh

Modeling computation and plasticity for an individual pyramidal neuron in the visual cortex

Sanjana Venkatesh

ABSTRACT

Pyramidal neurons are the most common cell type in the cortex, making up 70-85% of the cell population (Bitanihirwe & Woo, 2021). Despite their prevalence, however, their unique properties–ways of learning patterns, integrating inputs from other cells–are yet to be well understood. Furthermore, similarities in cell population diversity and physiological properties across the cortex suggest some uniformity in the overall computing architecture; therefore, understanding pyramidal neuron computation in V1 can provide insight into the function of pyramidal neurons in other parts of the cortex (Hosoya, 2019). We model a pyramidal cell in V1, incorporating three understudied features of pyramidal neurons: dendritic plasticity updates, an expanded time window for plasticity, and sigmoidal summing of inputs. Using plasticity rules with physiologically relevant foundations, the modeled L4 complex neuron develops a spatially-invariant preference for inputs of a particular orientation.

ACKNOWLEDGEMENTS

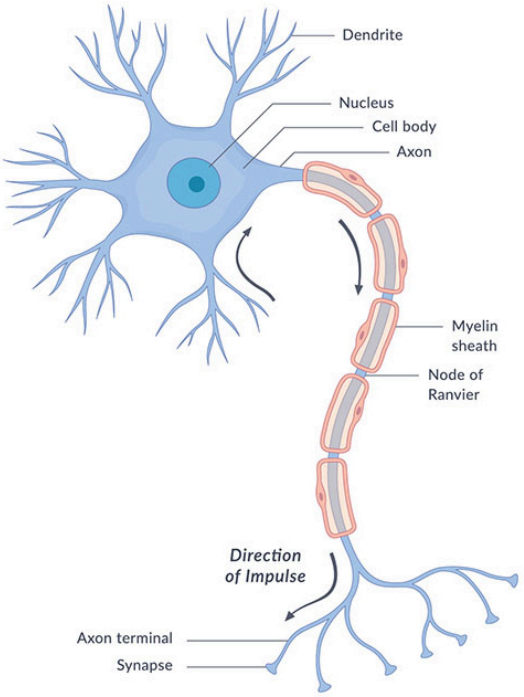
Thank you to Prof. Michael Berry (Princeton Neuroscience Institute) for advising me this semester, and to Prof. Niraj Jha (Department of Electrical Engineering) for co-reading this work.

I. BACKGROUND

IA. Anatomy of a neuron

A neuron consists of:

1. a cell body or soma, which contains the nucleus and other organelles;
2. a dendritic tree projecting from the soma that is made up of branches with synapses, each of which receive input from other neurons;
3. and an axon: a projection extending from the soma that ends in terminals that output information to other neurons (Nicholls, 2012).



(Office of Communications, 2018)

IB. Signaling

Neurons transmit information through electrical signals. The transmitting neuron is referred to as the presynaptic neuron, while the receiving neuron is called the postsynaptic neuron. Information is transmitted at synapses, where an axon terminal of the presynaptic neuron meets a dendrite of the postsynaptic neuron.

Transmitting information is dependent on a potential difference between the inside and outside of the neuron. Input signals result in an increase or decrease in membrane voltage, which can trigger activation of the postsynaptic neuron (see below). The potential difference across the membrane is defined as $\Delta V = V_{in\ cell} - V_{outside\ cell}$ (Nicholls, 2012). Ordinarily, the membrane potential ranges from -20 to -200 mV depending on cell type and organism–this is known as the resting potential (Alberts, 2002). Neurons maintain the resting potential by moving ions

(particularly Na+, K+) across the cell membrane via ion channels (passive transportation across membrane electrochemical gradient) and transporters (active transport against gradient) to establish an equilibrium electrochemical gradient (Chrysafides et al., 2024). While there is an overall membrane potential, the individual equilibrium membrane potential of each ion will vary due to differences in the neuron's membrane permeability and other characteristics of transmembrane transporters for each ion (Webb, 2017).

Electrical signaling in neurons is split into local graded potentials, which pass information across synapses, and action potentials, which send signals down the length of the neuron. Graded potentials are small changes in membrane potential (a few millivolts) generated in dendrites in response to sensory input or neurotransmitter release at the synapse. These potentials, whose magnitude depends on the duration and concentration at which neurotransmitters are present, can be excitatory (increasing the membrane potential) or inhibitory (decreasing the membrane potential) (Alberts, 2002). Excitatory postsynaptic potentials (EPSPs) are a subset of local graded potentials that consist of depolarization (increase in membrane voltage) caused specifically by binding of a neurotransmitter to the postsynaptic neuron (Moini et al., 2024). Due to the leakiness of the plasma membrane and the small size of graded potentials, these signals tend not to spread along the length of the neuron. The graded potentials generated at the synapse travel along the dendrites and soma to the beginning of the axon ("axon hillock"). If, at the axon hillock, the combined graded potentials exceed a threshold membrane potential, this triggers the opening of voltage-dependent ion channels that allow positive ions to enter. This begins a positive feedback loop in which the rising membrane potential causes more voltage-dependent ion channels to open, causing the membrane potential to spike in an action potential–a large, fixed-size pulse that lasts for 1-2ms (Byrne, 2023). The action potential

propagates down the soma to the axon terminal, where it triggers the opening of Ca2+ channels that promote the release of neurotransmitters at the synapses. In this way, the signal continues to propagate to downstream neurons.

While the intensity of the input signal affects the magnitude and duration of local graded potentials, action potentials are fixed in size and length. Instead, intensity is expressed through the neuron's firing rate (the rate of action potentials), where a more intense input triggers a faster train of spikes (Byrne, 2023).

IC. Brain and visual cortex anatomy

The primary visual cortex (V1), which is involved in visual processing, is located in the occipital lobe of the neocortex. The visual processing system is supported by photoreceptors, bipolar cells, and ganglion cells. Horizontal and amacrine cells make links across individual pathways in the visual cortex. When presented with a visual stimulus, photoreceptor cells in the retina are activated by light that falls within a specific wavelength spectrum (Nicholls, 2012). The output of the photoreceptors is sent to the lateral geniculate nucleus (LGN), located in the thalamus. The LGN pre-processes visual information, preserving the content of the signal while adjusting response gain and increasing the signal-to-noise ratio, before sending it to the primary visual cortex (V1) (Usrey & Alitto, 2015). V1 contains 'simple,' 'complex,' and 'hypercomplex' neurons. Simple cells respond to edges of a particular orientation at a certain location in the visual field. They connect convergently on complex cells, which can respond to oriented edges, regardless of position. Finally, complex cells converge upon hypercomplex cells, which respond to oriented edges of a *fixed length* at any position in the field. V1 is split into six layers, with simple cells mainly found in Layer 4 (L4) and complex/hypercomplex cells in Layers 2 and 3

(L2/3). Visual information from the thalamus is sent first to simple cells in L4, which connect to complex cells in L2/3 (Hubel & Wiesel, 1968).

ID. Plasticity

Plasticity can be defined as "the ability of the nervous system to respond to intrinsic or extrinsic stimuli by reorganizing its structure, connections, and function" (Von Bernhardi et al., 2017). Plasticity can take place on many levels, from tweaks to existing synapses to complete rewiring of a neural circuit by eliminating unhelpful synapses and forming new ones. It is also used to explain learning, and thus, is particularly relevant for study of sensory systems such as the visual system (Feldman, 2009).

During plasticity, synapse strength–the magnitude of the EPSP produced by a synaptic input–is upweighted (long-term potentiation (LTP)) or downweighted (long-term depression (LTD)). This is accomplished by adding or removing receptor ion channels from the part of the membrane at that synapse. Changing the number of ion channels regulates the amount of ions that can enter the neuron upon stimulation, and therefore, the amount by which the membrane potential can be changed (Sumi & Harada, 2020). Effectively, LTP means that the synapse has an increased conductance to ions, while LTD means a lower conductance.

A framework for synaptic plasticity was introduced by Donald Hebb in 1949: Hebb's rule claims that a synapse is strengthened if there is activity at both the presynaptic and postsynaptic neuron; "neurons that fire together, wire together." In other words, if an input signal is present and the receiving cell is activated, the connection between those cells is strengthened. The amount by which a synapse is upweighted or downweighted can be described by a function of the presynaptic and postsynaptic activity (Zhao & Willing, 2018).

In mammals, most excitatory synapses reside on spines: small protrusions from the dendrites that help localize signaling molecules of machinery. During LTP, spines grow to accommodate the increased number of ion channels. On the other hand, neural circuit remodeling can also occur by way of spine elimination and generation. LTD can drive spine shrinkage and eventual elimination–in other words, the neuron deletes connections that are used infrequently (and as a result, unhelpful for learning). At the same time, spine generation allows for new synaptic connections that can enhance the effectiveness of learning (Stein & Zito, 2019).

The most widely-studied rule to determine whether a synapse undergoes LTP or LTD is synaptic time-dependent plasticity (STDP). STDP is a time-dependent Hebb rule that determines whether LTP or LTD happens depending on the order of presynaptic and postsynaptic activity within a narrow time window (~20ms) (Paulsen, 2000). Generally, it is thought that LTP is caused by presynaptic activity, followed 10-20 ms later by a postsynaptic spike. On the other hand, LTD is thought to be caused by a postsynaptic spike followed by presynaptic activity. Postsynaptic depolarization (a spike) can be provided by an NMDA spike or a backpropagating action potential (BAP). BAPs occur when an action potential propagates backward from the axon hillock to the dendrites (Paulsen, 2000).

IE. Pyramidal neurons

Pyramidal cells are the most common cell type in the cortex, making up 70-85% of the cortex (Bitanihirwe & Woo, 2021). Notably, their dendritic trees consist of one apical dendrite and many basal dendrites. In thin dendrites, a subset of dendrites in the pyramidal cell's dendritic tree, an additional form of electrical signals called NMDA spikes are also present (Antic et al., 2010). NMDA spikes occur within dendrites (individual branches) when 10-50 neighboring synapses are activated together (Antic et al., 2010). NMDA spikes have been shown to be

necessary for long-term potentiation (LTP), which allows plasticity to occur (Brandalise et al., 2016). Additionally, NMDA spikes have an amplitude of 40-50 mV (much larger than an EPSP) and a duration of several hundreds of milliseconds (Antic et al., 2010).

Gordon et al. demonstrate experimentally that the rules for inducing LTP and LTD in pyramidal cells vary across parts of the dendritic tree (Gordon et al., 2006). Under typical STDP conditions (an input followed ~10 ms later by a postsynaptic action potential), LTP was induced as expected in basal dendrites near the soma; however, this caused LTD in basal dendrites further from the soma. These distal dendrites were shown to require NMDA spikes and the presence of a cofactor to induce LTP. Due to the longer duration of NMDA spikes (100 ms) compared to action potentials, the temporal window within which pre- and postsynaptic activity can occur is dramatically lengthened (~150 ms). This increases the opportunities for plasticity in basal dendrites. Furthermore, LTP was shown to occur regardless of the temporal order of presynaptic input and NMDA spike; however, the time window is asymmetrical, and postsynaptic before presynaptic activity (opposite order of STDP) most frequently produced LTP.

Upon stimulation, EPSPs resulting from inputs to a dendrite or neuron are combined; if the total exceeds a particular threshold, a spike is triggered (NMDA spike or action potential, respectively). While many studies report that EPSPs are summed linearly throughout a neuron, Polsky et al. find that in basal and oblique dendrites of pyramidal neurons, a sigmoid function is applied to synaptic weights (i.e. EPSP values) before summing them (Polsky et al., 2004).

II. MOTIVATION

Despite the prevalence of pyramidal neurons in the cortex, their unique properties–local plasticity, an increased time window for LTP/LTD, and nonlinear integration of inputs–are understudied. Furthermore, understanding visual processing can provide insight into other

functions of the cortex. One hypothesis about the cortex is that small groups of diverse neurons form “unit circuits” that are repeated throughout. Although different parts of the cortex perform different functions, similarities in cell population diversity and physiological properties across the cortex suggest that the computing architecture is similar. Thus, an understanding of the function of pyramidal neurons in visual processing can be at least partially generalized to other sensory perception processes, motor control, and language processing (Hosoya, 2019).

Three key characteristics are used to model a pyramidal cell: local plasticity dynamics and a prolonged time window for LTP/LTD (both are consequences of NMDA spike-dependent plasticity), and the sigmoid summation of inputs to produce a spike.

III. MODEL DESCRIPTION AND PROGRESS

IIIA. Setup and training

Foldiak proposes a 2-layer ANN to model complex V1 neurons, in which plasticity is moderated by a modified Hebbian rule. The size of the synaptic strength update is proportional to a combination of presynaptic activity and a time average of postsynaptic activity. Using the time average allows the neuron to learn spatial invariance to oriented bar stimuli (Földiák, 1991). Taking inspiration from Foldiak, a single layer 2/3 pyramidal neuron in the visual cortex is modeled as a two-layer ANN. The sensory input is represented as a bar, 4 units long, with one of four orientations (0°, 45°, 90°, 135° to horizontal), that scans across a visual field, represented as an 8-by-8 grid of squares.

This stimulus is sent to the input layer, which consists of Layer 4 neurons–the first to receive visual information from the thalamus (the thalamus preprocesses visual information and relays it to the visual cortex). (Miller, 2003; Usrey & Alitto, 2015). Layer 4 neurons are simple

cells by Hubel and Wiesel's classification and are activated by line or edge stimuli of a particular orientation.The model contains 256 Layer 4 neurons–one for every possible location and orientation. Upon receiving a stimulus, the four stimulated L4 neurons are activated (activation status is represented as 0 (inactive) or 1 (active)).

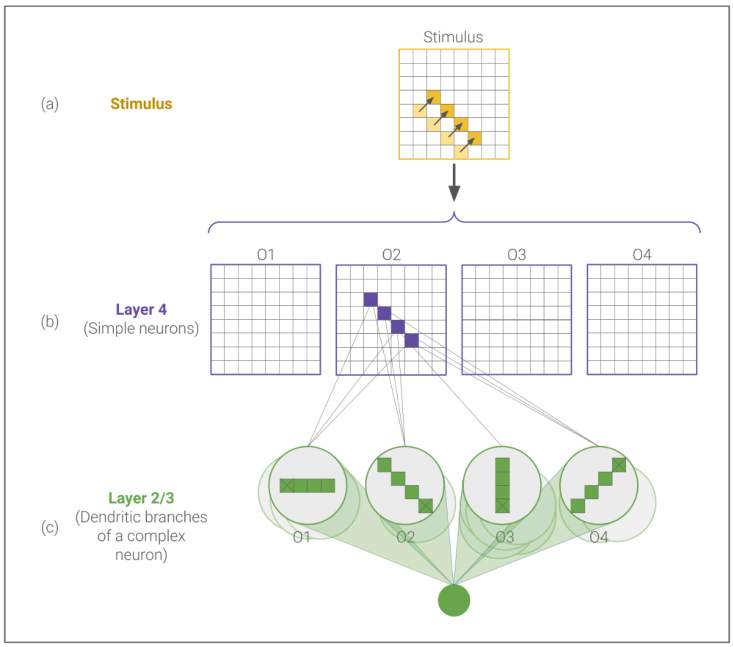


Figure 1. Model schematic. **(a)** The receptive field is an 8x8 grid of squares; stimuli are bars of 4 possible orientations (0˚, 135˚, 90˚, 215˚) that scan across the receptive field. **(b)** There are 256 layer 4 (L4 or simple) neurons (one for every orientation at every position). Only neurons which correspond to the stimulus bar orientation are activated. **(c)** Neurons are initially connected randomly to branches of a layer 2/3 complex neuron. As more stimuli are passed to the model, the strength of these connections is modulated. As a result, individual branches of the complex neuron develop *spatially-invariant* receptive fields that correspond to each of the four oriented bars.

The nodes in the input layer are connected to the second layer, whose nodes represent the collection of synapses across the distal dendrites of a single Layer 2/3 neuron. (Distal dendrites follow the NMDA spike-based plasticity rule determined by Gordon et al., as opposed to traditional STDP (Gordon et al., 2006). Synapses are grouped by the dendritic branch they are located on.

To run and evaluate the model, test and train stimuli sets are generated. The untrained model is first evaluated using the test set, then trained with the train set, and re-evaluated with the initial test set.

During plasticity (training), the model is trained by making changes to several variables that govern weights on each branch or synapse. The plasticity rules are as follows:

4. **Somatic plasticity only**: if an action potential is generated, increase the inhibitory conductance at the soma. Else, decrease it.

This is done to keep the action potential rate at a reasonably consistent level. Increasing inhibition in the soma reduces the amount of ions that can enter and depolarize the cell, and therefore, decreases the likelihood of spiking with high frequency. Similarly, decreasing inhibition increases the probability of a future spike.

5. **Adding in dendritic plasticity**: If there is an NMDA spike in a branch, the inhibitory conductance is incremented. If not, it is decremented.

   Similarly to somatic plasticity, this is done to keep the NMDA spike rate at a reasonable level.

6. **For additional synaptic plasticity**: If there is a branch NMDA spike, open a time window for plasticity.

   Although Gordon et al. show that the time window for a presynaptic input to occur extends before and after the NMDA spike, the model was simplified to include only the latter, given the strong asymmetry of the time window (Gordon et al., 2006).

   If a time window is open:

   i. If a synapse has input, potentiate it by incrementing the size of the EPSP triggered by the input.
   ii. If a synapse is not receiving input, depress it by decrementing the size of the EPSP that would be triggered by an input.

   If a time window is *not* open:

   iii. Depress all active synapses (weight is reduced because the synaptic input was not associated with a branch spike).

iv. Increment all inactive synapses by a baseline value. This is done to prevent spine recycling from happening too quickly.

Lastly, if any synapse is depressed below a set threshold, it is recycled and a new one is randomly created.

v. While this can take up to a day to happen physiologically, it happens within one timestep in the model for speed purposes.

The model can be adjusted to apply plasticity at only the somatic level, somatic and dendritic, or somatic, dendritic, and synaptic plasticity together.

IIIB. Parameters

The model accepts parameters on the neuron, including thresholds for NMDA spikes and action potentials, the size of the synaptic weight changes for each case listed above, and spine recycling threshold (Appendix I). Parameters were selected by my advisor, Michael Berry (Princeton Neuroscience Institute) and tuned to match experimental data.

IIIC. Results and contribution

I spent the semester learning about the neuroscientific basis for the model and attempting to reconcile that understanding with the model code. I translated the model, written in IGOR Pro (a programming language for numerical computing) into MATLAB so as to be able to work on it in future semesters. I am currently in the process of debugging the model and attempting to recreate the results captured in the IGOR Pro version.

Below are the outputs of my code (right) in comparison to my advisor's (left):

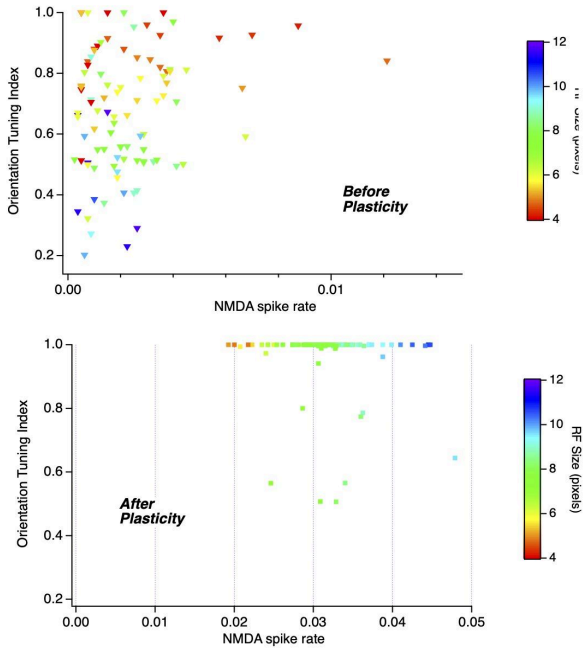
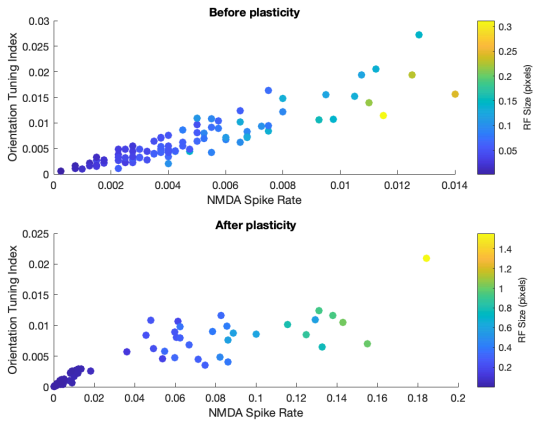
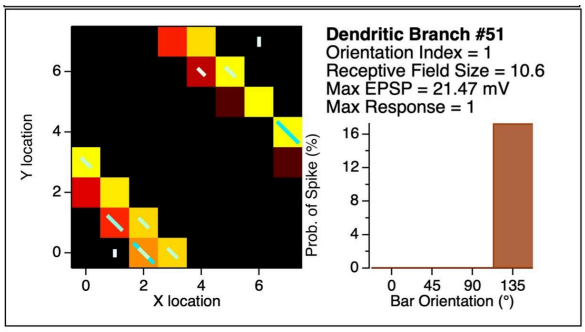
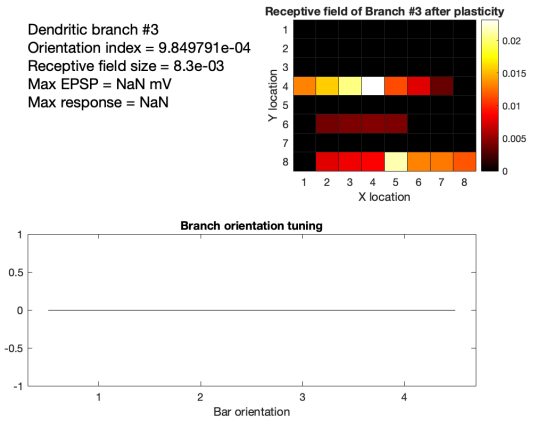
IGOR Pro	MATLAB
Figure 2a. A scatter plot of orientation tuning (how strongly a branch prefers one orientation) vs. NMDA spike rate for all branches (n=100).	
	
Before plasticity, branches show varied orientation tuning with a low NMDA spike rate. After plasticity, both the orientation index and NMDA are drastically increased.	This plot does not resemble the figure from the original model: neither the tuning index nor the NMDA spike rate follows the same trend; the data's range also does not match that from the original model. The orientation index calculation produces some NaN values, so there are likely divide-by-zero errors to be corrected. The discrepancy in the NMDA spike rate (see "After plasticity") is likely due to an error within the simulation itself.

Figure 2b. A map of a branch's receptive field, showing stimuli that it responds to most strongly, and a histogram of the orientations of bars that activated this branch.





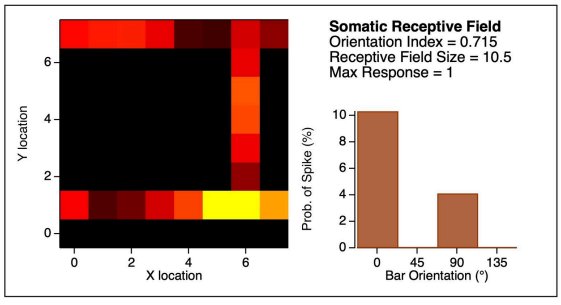
This branch reacts most often to bars with 135˚ orientation. The appearance of diagonal bars in multiple parts of the receptive field is a consequence of the long plasticity window that opens after an NMDA spike. Synapses corresponding to future locations of the scanning bar continue to be strengthened as a single bar stimulus scans across the visual field; this promotes spatial invariance to stimuli.

According to the heatmap, this branch is activated in response to horizontal bars. The calculated orientation index is $9.9 * 10^{-4}$ when it should be close to 1; when calculating orientation index, it appears that there is an error in scaling some of the numbers.

Figure 2c. A map of the somatic receptive field, showing stimuli that most commonly trigger an action potential (a spike at the soma).

Comparison: Soma RF vs. Sum over all Branch RF

Note: I used some different parameters here...

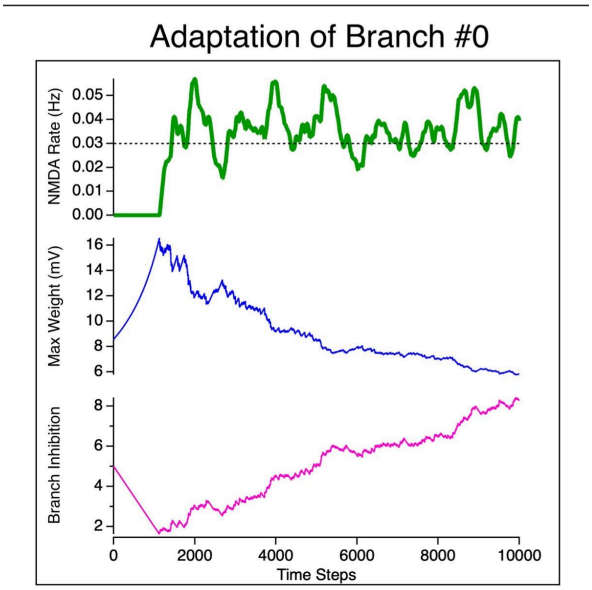


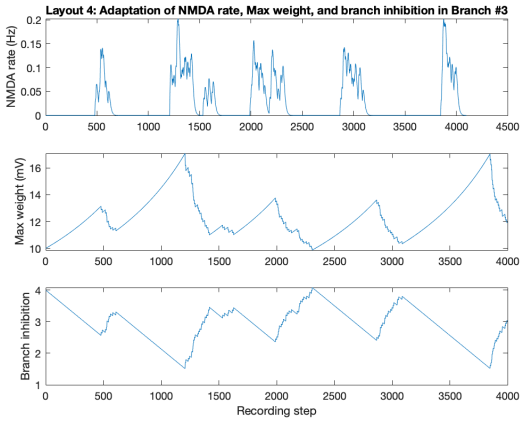
Have not written code to generate this figure.

The heatmap and histogram both suggest that

action potentials were typically triggered by horizontal or vertical bars.	

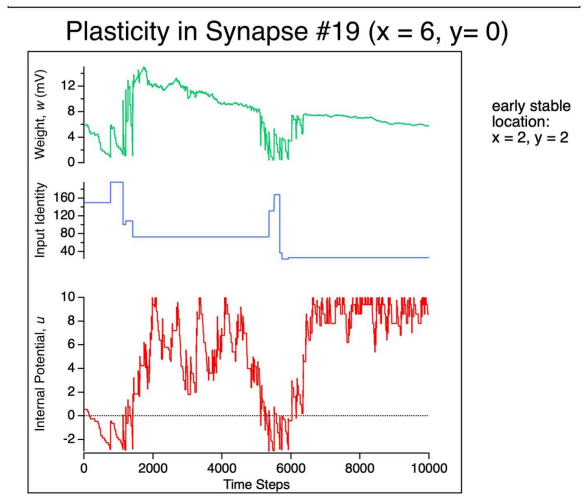
Figure 2d. Plot describing how variables influencing synaptic strength (branch inhibition, maximum possible synaptic weight, NMDA spike rate) changed during plasticity (training).

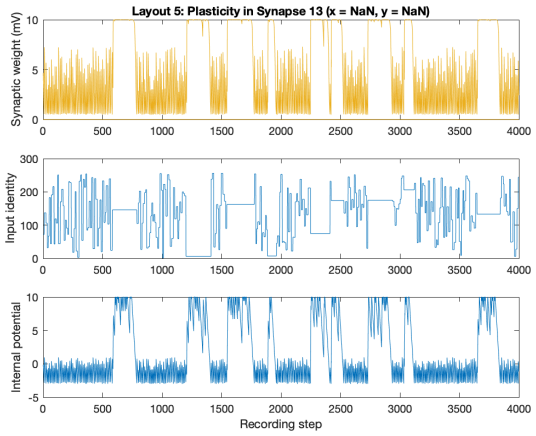




All three variables appear to approach a steady state by the end of the model runtime.	Dynamics of the fluctuations of the three variables appear similar (branch inhibition has sharp drops and gradual linear increases, while max weight has sharp increases and gradual decreases). However, the frequent recycling of synapses leads to a cyclical-looking graph rather than one that resembles the figure from the original model.

Figure 2e. Plot describing a selected synapse over the training period: synaptic weight, the identity of the presynaptic neuron (this changes during spine recycling), and magnitude of the EPSP that would occur if an input was present. Dips in the EPSP magnitude (corresponding to LTD) below the threshold (-3 mV) line up with spine recycling events.

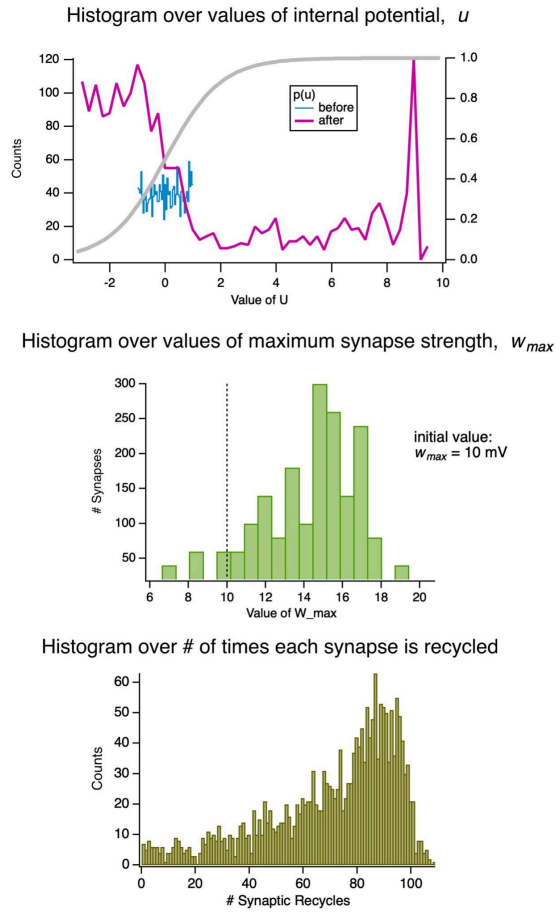


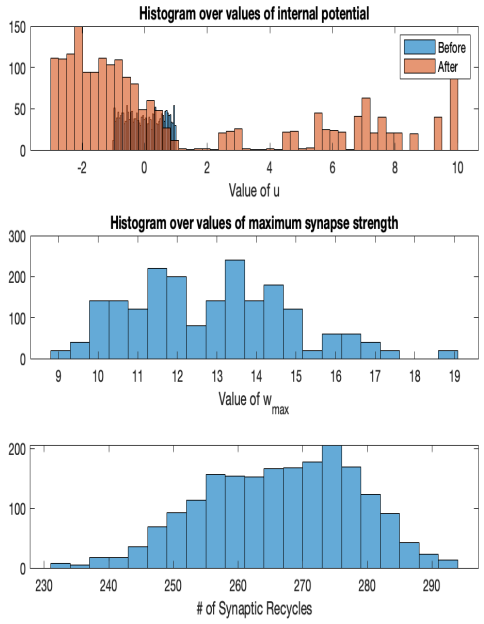


Some spine recycling events result in increases in weight and EPSP magnitude of the synapse, showing that the newly-connected spine contributes to learning a particular oriented bar.

This figure is also affected by the high rate of spine recycling. However, the fluctuations between recycling events appear similar to the original model and fall within the same range.

Figure 2f. Histograms of internal potential values before/after plasticity, maximum synaptic strength values, and number of synaptic recycles.



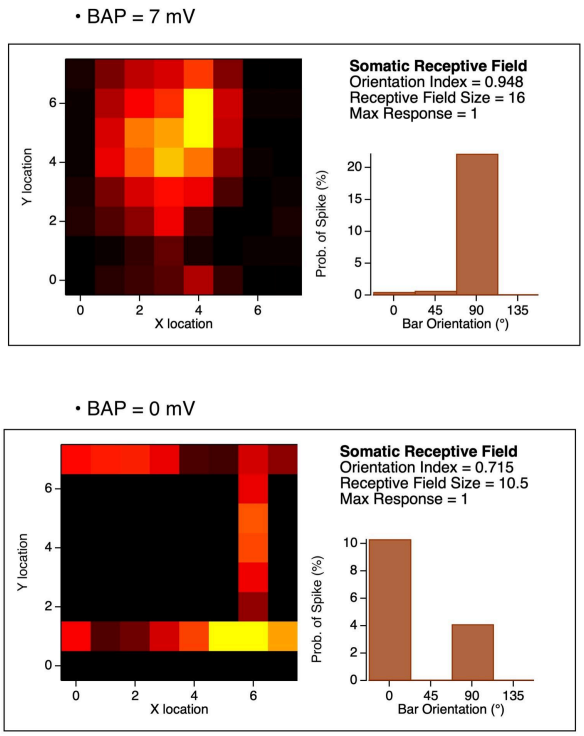


(top) The EPSP magnitude ('internal potential') of each synapse is initialized randomly between -1 and 1. After plasticity, a majority of synapses have a negative internal potential (indicative of LTD, showing that they are rarely activated) or a very high weight (branches are highly dependent on these synapses). (middle) The maximum synaptic weight increases from 10 to about 15 during plasticity. (bottom) Synapses are recycled, on average, about 85 times in a run lasting 10,000 timesteps.

(top) The range and shape of this histogram, before and after plasticity, match that of the original model. (middle) The distribution of max synapse weights is skewed further left in the original model; the extraordinarily high synaptic recycle rate (bottom) might be a contributing factor. The median number of synaptic recycles is about 275, more than 3 times as much as the original model (~85 recycles).

Figure 1g. A map of the somatic receptive field with and without the addition of a backpropagating action potential (BAP). BAPs cause a voltage increase in all branches, which helps synchronize their preference for certain stimuli.

BAP versus no BAP (replotting previous data)



Have not yet implemented BAPs in model.

(top) The somatic receptive field and histogram indicate that the cell strongly prefers vertical bars (orientation index = 0.948) at most locations in the visual field. (bottom) Without a BAP, the cell has a weaker preference (orientation index = 0.715) for horizontal bars instead, and a smaller receptive field.

IV. CHALLENGES, FUTURE STEPS, AND APPLICATIONS

It was a challenge to simultaneously understand the neurological mechanisms underlying the model while also interpreting and translating the code, which was written in a language that I was unfamiliar with, into MATLAB, with which I also had little experience. As a result, I

encountered many syntax issues in addition to gaps in conceptual understanding. Over the course of the semester, I became better acquainted with IGOR and MATLAB, as well as the neuroscience foundations needed to understand the model. I feel better equipped to add new features and compare this model to others in the literature in coming semesters. In the future, I would like to compare the efficacy of 'local' learning (dendritic plasticity) with 'global' learning (plasticity due to backpropagating action potentials). Noise in the stimuli and dendrites can also be incorporated; this would better represent the noisy signals received and transmitted in the brain. From a clarity standpoint, I plan to write comprehensive documentation for the model after ensuring it matches the IGOR Pro version. Lastly, the idea of local vs. global learning may also be used to improve neural network training. Backpropagation, the most common way to train a neural network, is expensive; implementing local learning in conjunction with backpropagation could ease the computational cost of training (Pagkalos et al., 2024; conversation 12/11/2024 with Niraj Jha).

V. CONCLUSION

This model of a pyramidal cell in V1 incorporates three understudied features of pyramidal neurons: dendritic plasticity updates, an expanded time window for plasticity, and sigmoidal summing of inputs. Using plasticity rules with physiologically relevant foundations, the modeled L4 complex neuron develops a spatially-invariant preference for inputs of a particular orientation.

V. APPENDIX

APPENDIX 1: Model parameters for pyramidal neuron

Total number of synapses on neuron	2000
Total number of basal dendrites	10
Number of synapses per dendritic branch	20
Maximum synapse strength (mV)	10
Attenuation of signal at soma	10
Threshold for NMDA spike (mV)	7
Threshold for somatic spike (mV)	20
Amplitude of backpropagating action potential (mV)	9
Fractional noise on synapses	0
Fractional noise on dendritic branches	0
Noise at soma (mV)	0
Range of initial synaptic weights	1
Potentiation: change in U	2
Depression: change in U	-0.3
Presynaptic spiking: change in U	-0.3
No spiking: baseline change in U	0
Spine recycling threshold in U	03
Maximum U	10
Time window for plasticity	4
Synaptic scaling for NMDA spike	-0.1
Synaptic scaling for no NMDA spike	0.003

G_leak for branch	1
Initial inhibitory conductance (G_inh) for branch	4
Threshold change for somatic spikes (mV)	0.1
Threshold change for no somatic spike (mV)	-0.005
G_leak for soma	0.1
Initial inhibitory conductance (G_inh) for soma	0.5

APPENDIX 2: Code

Please find all files at https://github.com/sanjana-venkatesh8/iwBerry.

VI. Bibliography

Alberts, B. (Ed.). (2002). *Molecular biology of the cell* (4th ed). Garland Science.

Antic, S. D., Zhou, W., Moore, A. R., Short, S. M., & Ikonomu, K. D. (2010). The decade of the dendritic NMDA spike. *Journal of Neuroscience Research*, *88*(14), 2991–3001. https://doi.org/10.1002/jnr.22444

Bitanihirwe, B. K. Y., & Woo, T.-U. W. (2021). Pyramidal neurons. In *Factors Affecting Neurodevelopment* (pp. 433–445). Elsevier. https://doi.org/10.1016/B978-0-12-817986-4.00037-7

Brandalise, F., Carta, S., Helmchen, F., Lisman, J., & Gerber, U. (2016). Dendritic NMDA spikes are necessary for timing-dependent associative LTP in CA3 pyramidal cells. *Nature Communications*, *7*(1), 13480. https://doi.org/10.1038/ncomms13480

Byrne, J. (2023). *Chapter 1: Resting Potentials and Action Potentials*. Neuroscience Online. https://nba.uth.tmc.edu/neuroscience/m/s1/chapter01.html

Chrysafides, S. M., Bordes, S. J., & Sharma, S. (2024). Physiology, Resting Potential. In *StatPearls*. StatPearls Publishing.

Feldman, D. E. (2009). Synaptic Mechanisms for Plasticity in Neocortex. *Annual Review of Neuroscience*, *32*(1), 33–55. https://doi.org/10.1146/annurev.neuro.051508.135516

Földiák, P. (1991). Learning Invariance from Transformation Sequences. *Neural Computation*, *3*(2), 194–200. https://doi.org/10.1162/neco.1991.3.2.194

Gordon, U., Polsky, A., & Schiller, J. (2006). Plasticity Compartments in Basal Dendrites of Neocortical Pyramidal Neurons. *The Journal of Neuroscience*, *26*(49), 12717–12726. https://doi.org/10.1523/JNEUROSCI.3502-06.2006

Hosoya, T. (2019). The basic repeating modules of the cerebral cortical circuit. *Proceedings of*

the Japan Academy, Series B, *95*(7), 303–311. https://doi.org/10.2183/pjab.95.022

Hubel, D. H., & Wiesel, T. N. (1968). Receptive fields and functional architecture of monkey striate cortex. *The Journal of Physiology*, *195*(1), 215–243. https://doi.org/10.1113/jphysiol.1968.sp008455

Miller, K. D. (2003). Understanding Layer 4 of the Cortical Circuit: A Model Based on Cat V1. *Cerebral Cortex*, *13*(1), 73–82. https://doi.org/10.1093/cercor/13.1.73

Moini, J., LoGalbo, A., & Ahangari, R. (2024). Neural tissue. In *Foundations of the Mind, Brain, and Behavioral Relationships* (pp. 39–62). Elsevier. https://doi.org/10.1016/B978-0-323-95975-9.00017-2

Nicholls, J. G. (Ed.). (2012). *From neuron to brain* (5. ed). Sinauer Associates, Inc.

Office of Communications. (2018, October 1). *What are the parts of the nervous system?* National Institute of Child Health and Human Development. https://www.nichd.nih.gov/health/topics/neuro/conditioninfo/parts

Pagkalos, M., Makarov, R., & Poirazi, P. (2024). Leveraging dendritic properties to advance machine learning and neuro-inspired computing. *Current Opinion in Neurobiology*, *85*, 102853. https://doi.org/10.1016/j.conb.2024.102853

Paulsen, O. (2000). Natural patterns of activity and long-term synaptic plasticity. *Current Opinion in Neurobiology*, *10*(2), 172–180. https://doi.org/10.1016/S0959-4388(00)00076-3

Polsky, A., Mel, B. W., & Schiller, J. (2004). Computational subunits in thin dendrites of pyramidal cells. *Nature Neuroscience*, *7*(6), 621–627. https://doi.org/10.1038/nn1253

Stein, I. S., & Zito, K. (2019). Dendritic Spine Elimination: Molecular Mechanisms and Implications. *The Neuroscientist*, *25*(1), 27–47.

https://doi.org/10.1177/1073858418769644

Sumi, T., & Harada, K. (2020). Mechanism underlying hippocampal long-term potentiation and depression based on competition between endocytosis and exocytosis of AMPA receptors. *Scientific Reports*, *10*(1), 14711. https://doi.org/10.1038/s41598-020-71528-3

Usrey, W. M., & Alitto, H. J. (2015). Visual Functions of the Thalamus. *Annual Review of Vision Science*, *1*(1), 351–371. https://doi.org/10.1146/annurev-vision-082114-035920

Von Bernhardi, R., Bernhardi, L. E., & Eugenín, J. (2017). What Is Neural Plasticity? In R. Von Bernhardi, J. Eugenín, & K. J. Muller (Eds.), *The Plastic Brain* (Vol. 1015, pp. 1–15). Springer International Publishing. https://doi.org/10.1007/978-3-319-62817-2_1

Webb, W. G. (2017). Neuronal Function in the Nervous System. In *Neurology for the Speech-Language Pathologist* (pp. 74–92). Elsevier. https://doi.org/10.1016/B978-0-323-10027-4.00004-X

Zhao, L.-R., & Willing, A. (2018). Enhancing endogenous capacity to repair a stroke-damaged brain: An evolving field for stroke research. *Progress in Neurobiology*, *163–164*, 5–26. https://doi.org/10.1016/j.pneurobio.2018.01.004