Rasmussen, Jonathan
#20552019

Project Evaluation for Rasmussen, Jonathan (#20552019)

The retina's neural microcircuit and light intensity adaptation: A neurobiological model using the NEF

Introduction **Okay**

Your introduction section is okay. You review the state of the art of retinal modelling and state your overall goal. However, it is unclear what exact part of the retina you'd like to model, or which specific model your model is inspired by. There is a wealth of literature on modelling the retina (see the review paper you cite), so it is unclear to me why don't take one of those models and turn it into a NEF model (at least as a starting point).

Methods **Poor**

Your methods section is poor. While you review the low-level retinal microcircuitry, I'm missing any mathematical abstraction that goes beyond these details. Your model seems to crudely follow the biological connectivity—however, you seem to mostly "abuse" Nengo as a "bad" dynamical system simulator (without ever describing the dynamical system you are building). For example, you don't describe at all how a large ensemble of LIF neurons can stand for the compartemental details of a single neuron (there are reasons why they may). Furthermore, you fail to describe key components of your NEF model. What are the variables that are being represented by the individual components (saying that they all represent "light intensity" is not helpful; you at least have filtered versions of the light intensity)? What are the functions that are being computed in the connections?

Experiments and Results **Weak**

Your result section is weak. While your model—to my surprise—seems to qualitatively match biological data, you don't even describe where the data you are comparing to comes from. I'm also missing quantitative measurements, or an exploration of different model parameters (neuron counts, time constants).

Discussion **Weak**

Your discussion section is weak. You mostly state that your model "did not work" and that you need "evolutionary algorithms" to solve the problem. Regarding the first point, I would have liked to see some discussion why your model does not work. I.e., what assumptions that you put into your model are violated? Regarding the second point, this seems to be a little early, given that your model is in a very rough state.

Overall

Overall, this is a weak project. Modeling the retinal microcircuit is an interesting topic with many opportunities for using the NEF. However, I cannot see any effort on your side to actually embrace the methodology we discussed in the course; in the very least, I cannot see you thinking about the system as a dynamical system. Furthermore, many of your claims about biology are without references, including the source of the data you compare to.

Since there are multiple cases of plagiarism in your report (see **orange** annotations), I've for now marked this report with 50% of the original grade (otherwise the grade would have been 14/30). I've informed the associate dean of this case who decides what the final penalty will be.

Mark: 7/30

Rasmussen, Jonathan
#20552019

Grading Scheme

In order to evaluate the final project, I assessed how well you did in four broad rubrics: *introduction/related work*, *methods*, *experiments and results*, and *discussion*.† I assigned one of six grades to these sections: A^+ (excellent), A (very good), B (good), C (okay), D (weak), D^- (poor). See the table below for a translation to numeric grades.

In general, my evaluation is based on how rigorously you uphold scientific standards in your report and project. I was less focusing on the complexity of your model, but whether you are able to clearly motivate your goal, explain your methods, relate them to relevant literature, perform a series of interesting experiments, as well as your ability to analyze and discuss your results.

The mark for your final project is roughly based on a 20%/30%/30%/20% weighting of the grades in the four rubrics, although I reserved the right to slightly adjust the grade up or down to account for the quality of your project relative to other students.

†: Multiple sections in your report may correspond to one rubric in the grading scheme, or vice-versa.

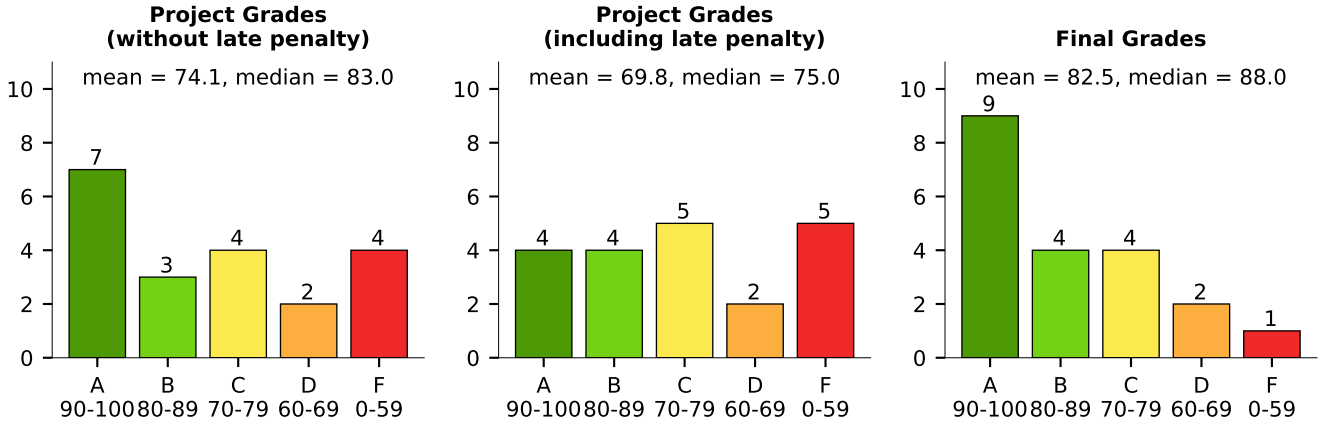
Annotations

You can find an annotated version of your project report below. I use two colors for the annotations:

- **Red** is related to the actual content. Note that check marks (✓) do not per se indicate that what you wrote is correct, but merely that something makes sense to me and that I believe you.
- **Blue** is related to style, language, and typography. This does *not* affect your mark in most cases.

Grade Distributions

The histograms below show the grade distribution of the final project, as well as the grade distribution of the final course grade. The discretization is based on the letter-grades from the table below.



	Excellent ↓	Very Good ↓		Good ↓			Okay ↓			Weak ↓			Fail ↓
Letter Grade	A^+	A	A^-	B^+	B	B^-	C^+	C	C^-	D^+	D	D^-	F
Report Grade	30	28	26	25	24	22	21	20	18	17	16	14	0
Numeric Grade†	100	96	92	89	86	82	79	76	72	69	66	62	0

†: Equivalent grade out of one hundred for the *entire* project (i.e., relative to 40 marks, including the interim report).

The retina's neural microcircuit and light intensity adaptation: A neurobiological model using the NEF

A Report Submitted in Partial Fulfillment of the Requirements for SYDE 750

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20552019

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April 15, 2020

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Contents

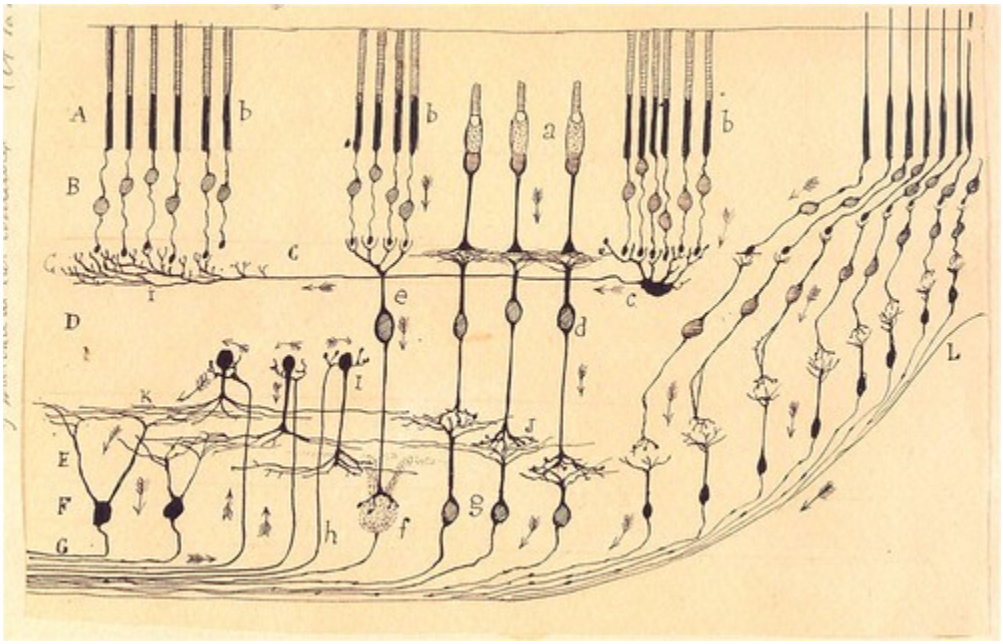


Figure 1: "Cells in the retina of the eye" (1904) a drawing found in the notebook of the Spanish neuroanatomist Santiago Ramón y Cajal, considered to be the father of neuroscience. A.Photoreceptors B.Outer Nuclear Layer C.Outer Plexiform Layer D.Inner Nuclear Layer (Amacrine, Bipolar, and Horizontal Cells) E.Inner Plexiform F.Ganglion Cell Complex G.Retinal Nerve Fibers.

1 Introduction

The retina is a complex neural system that has been extensively studied from the first findings of Santiago Ramón y Cajal to the present day. The retina is a tissue found lining the back of the eye composed of several layers of interconnected neurons. The purpose of the retina is to receive light that the lens of the eye focused, convert the light into neural signals, and send these signals on to different parts of the brain for visual recognition. Several aspects of processing of the light occurs in the retina, specifically through the complex connections between the various types of cells found in the retina. Although well studied, the interactions between the cells of the retina and their mechanisms are not completely understood. The complexity of this structure is highlighted in *Figure 1, "Cells in the retina of the eye" (1904, R. Smith, 2018).* Of particular interest is the way the retina's cells begin to compute and filter out various aspects of light. Two aspects of this that will be investigated in this report will be the retina's response to light intensity and it's ability to adapt to light intensity over time.

Computational modelling is a simulating approach used to help further the understanding of the retina and its neural microcircuits. There are many mathematical and computational models in the literature that has provided insights into retinal physiology and

biochemistry. There are 6 typical types of computational models that are typically used in retinal neuron modeling. Single-compartment models have been used to simulate nearly all retinal neuron types. These models approximate the structure of the neuronal excitable membrane using capacitance to model the membrane phospholipid bi-layer, in parallel with several several conductors. Morphologically realistic models have been used to create models that represent their biological counterparts more closely. These types of models typically are used to simulate axons and dendrites of neurons. These are ideal to study cell morphology and how non-uniform distributions of ionic channels contribute to neuronal response dynamics. Block compartment models represent a few neuronal regions and compromises between efficiency and realism. These are typically simplified versions of the morphologically realistic models. Continuum models are used to represent broad responses of a bulk tissue without explicit representation of the constituent neurons. Block structure models captures statistical relationships between light stimuli and firing rates with minimal cellular details. A typical approach, known as cascading models, represents the retina as a series of linear and nonlinear temporal filters. These types of models typically reproduce the response of the retina to simple laboratory light stimuli. Discrete neuronal networks are retinal networks simulated by grouping discrete retinal neuron elements, with excitatory and inhibitory interactions. These models take into consideration the neural properties and stimulus parameters while also considering the influence of feed-forward and feed-backward connection neurons that are heavily prevalent in the retina (Guo et al., 2014).

This project seeks to develop a retina model using the Neural Engineering Framework (NEF) as proposed by (Eliasmith and Anderson, 2003). The model will be a discrete neuronal network modelling the retina and is heavily influenced by block-structured models to recreate the retina's response to various light intensities and it's ability to adapt to constant light over time. The NEF consists of three steps to further develop the model. A System Description where as much information about the system is collected as possible and mapping this information to the NEF. Design Specification where it is specified what the real world constraints on the system are and determine the limitations of the framework. Finally, Implementation is where the two previous steps are combined to produce a functioning model. The model will be programmed using Python and the Nengo Python package and the model will seek to validate two aspects of the retina, it's response to light and adaptation to noise in the light source.

Do NOT copy entire sentences from your source without putting them in quotes!

I assume that the entire paragraph is based on this source. To make this clearer, I would move this to the beginning. E.g, "A rec review of ... by... describes the following neuron mode used in comp utational models of the retina"

Eliasmith and Anderson propose three steps for using the NEF to develop

Use active: ...real world constraints are specified and limitation ... are determined.

2 Neurobiological Model

This section will go in-depth in the different aspects of the NEF, that was describe in brief previously. During each section, the three different parts of the NEF will be applied to the current project of developing a model of the retina neural microcircuit to investigate the retina's response to light.

mentioned

2.1 System Description

The retinal system consists of multiple neural tissue layers in the back of the eye connected to the visual cortex. The model presented encompasses several of these neural tissue layers, however not all are represented in the model. In this section the representations used by the system are specified.

The retina's microcircuits are derived from the elaborate architecture of multiple layers of neurons found in the retina. When incident light is focused onto the retina from the lens of the eye, the cells that initiate the response are the cone and rod photo-receptors. The photo-receptors encode and filter the stimuli light in a process known as phototransduction. This signal is transmitted to the retina ganglion cells via the retina's bipolar cells, a typical type of neuron that operates as a signal path for sensory operation. Retina ganglion cells transmits the visual information via the optic nerve to several regions of the thalamus, hypothalamus, and mesencephalon (Marshak, 2009). It is important to distinguish the inhibitory components present in the retina at two stages. Horizontal cells are physically horizontal cells connecting multiple photo-receptors. They help integrate and regulate the input from the photo-receptors as a form of feed-backward input. Amacrine Cells are a similar component to the bipolar cells, however they act as a feedback input to the bipolar cells (Bloomfield, 2009).

This exact wording is from Wikipedia. The Marshak, 2009 article does not mention the thalamus and mesencephalo

The project will breakdown the model into two stages, the second one building upon the previous one. Initially, a functioning photo-receptor ensemble with a feed-backward horizontal cell ensemble will be created as a model of the photo-receptor and horizontal cell complex. This stage will introduce the retina's ability to respond to light intensity. Second, the photo-receptor and horizontal cell model will be connected to a bipolar ensemble with an amacrine inhibitory feedback loop that is in turn connected to a ganglion cell ensemble. This stage introduces the retinas ability to adapt to varying light contrast over time.

2.1.1 Light Intensity

Light intensity as a form of visual stimuli varies greatly in real life environments. The retina adapts to this intensity contrast early in the visual processing stage. This adaptation could be measured from the response of the horizontal cells (V. C. Smith et al., 2001).

The photo-receptor and horizontal cell complex can be broken up into 4 separate components; photo-transduction, calcium-feedback, inner segment, and horizontal cell feedback. Photo-transduction is the first part of the process where the photo-receptor converts the light stimuli to a neural response. The second stage needs to take into account the calcium feedback loop of the photo-receptor. The calcium feedback loop is where an increase in cyclic guanosine monophosphate concentration (cGMP) opens cyclic nucleotide-gated ion channels in the outer segments of the photo-receptors. This results in an inward current consisting partly of Ca2+. This concentration increase counteracts the initial rise of cGMP and acts as an inhibitory feedback loop to the response. The next step of the photo-receptor is the inner segment, which chief function is to provide ATP for the sodium potassium pump of the photo-receptor. Finally, the response from the inner segment is regulated by horizontal cells. Horizontal cells are depolarized by the release of glutamate from photo-receptors, which occurs in the absence of light. Depolarization of the horizontal cell causes hyper polarization to nearby photo-receptors. The opposite happens when photo-receptors are activated by light, where the horizontal cells are hyper polarized from the lack of glutamate release by the photo-receptors. Hyper polarization is where a change in a cell's membrane potential occurs that makes it more negative. This inhibits action potentials by increasing the stimulus required to move the membrane potential to the action potential threshold. Essentially this is describe the inhibitory feedback loop the horizontal cells exhibit to the photo-receptors' signal. Source missing.

2.1.2 Temporal Contrast

Adaption to the variance of light intensities is known as temporal contrast adaptation. It is particularly used for the retina's ability to reduce and ignore the noise or variance of light in the environment. Retinal cells exhibit two types of adaptive mechanisms. A fast adaptation occurs within the first ten milliseconds and a slow change occurring over ten seconds following the change in contrast. Initially, when there is a high contrast, the average response will increase. However, when a high contrast pattern is maintained over time, the response decays over time. This contrast adaptation does not occur in the photo-receptors nor the horizontal cells, but in the bipolar cells. The slow adaption mechanism has been linked to the bipolar-to-ganglion synapse. Both of these methods of adaptation

is integral for the ability of the retina to adapt to the surroundings and provide important visual stimuli to the brain. Source?

2.2 Design Specification

This section will address issues of precision, noise and range of the representation.

2.2.1 Light Intensity

In the case of this project, the response from the light is set to a range of incident light from 0 to 1. This value is a normalized value of the contrast used by in literature (Warren et al., 2016). This is order to adhere to the simplifications employed by Nengo. The incident light will be adjusted to reflect different light intensities, of 1, 10, 100 td, while also maintaining the contrast ratio of 8:1 as described in the literature. The amount of noise expected can be derived from recent results regarding the transmission of single by individual neurons. The precision of the representation is unspecified as the purpose of the model is to determine the precision of the model compared to neurobiological data.

What is this unit?

Contrast between what?

Source? I can't find this number in the prevously cited material.

I don't really understand the last point. If you're not interested in replicating the precision (i.e., variance) of your data, then you can just use a perfect mathematical model. The nice thing about the NEF is that you can build a model that may reproduce properties such as the variance.

2.2.2 Temporal Contrast

For the temporal contrast, it was important to show the retinas ability to mitigate the response of an increase in contrast over time. Therefore, the incident light to be tested was a Gaussian white noise signal. This signal would represent the typical noise found in the environment that the retina reduces. The range of 1 to -1 of the signal represents arbitrary degrees of contrast that the retina would adapt to and reduce the ganglion cell response to it. As the signal gradually increase or decreases, the ganglion cells will have a mitigated response.

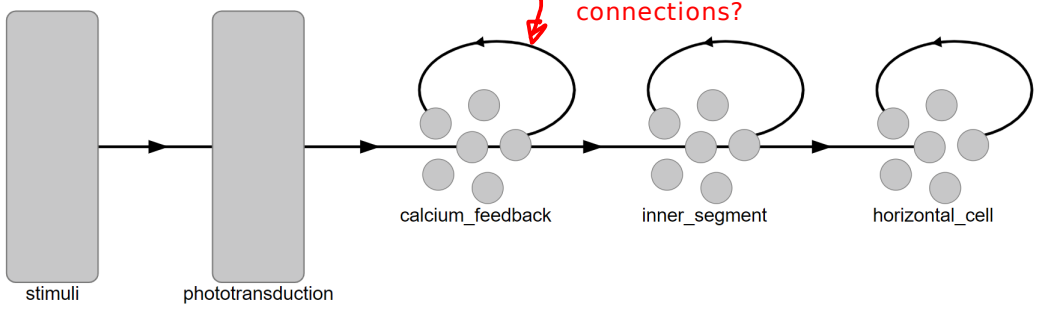
I don't see how this follows.

2.3 Implementation

2.3.1 Light Intensity

The implementation of the photo-receptor and horizontal cell complex is as described. This is done using a polynomial non-static linearity and a low-pass filter to convert and filter the light into a response in the photo-transduction stage of the model. To represent the calcium feedback loop a leaky integrate-and-fire neuron ensemble is used to represent

What do you mean by non-static? What is a polynomial linearity?



stimulus. stimuli is plural.

Figure 2: Diagram of the photo-receptor model implemented in Nengo. An initial stimuli from the environment is applied to the photo-receptor. This initial signal is filtered in a process known as phototransduction. The effect of calcium feedback of the neuron's cell membrane is taken into account. The inner segment's non-linearity function of the signal is applied. Finally the feedback of the horizontal cell is applied to the signal, which would be transmitted to the bipolar cell.

the membrane conductance effect. This membrane has a feedback loop of a polynomial non-static linearity and a low pass filter to complete the feedback component. In the next section of the model, the inner segment is modelled as a leaky integrate-and-fire neuron ensemble that represents the range of ion channels that in literature is typically modelled as a Hodgkin compartment model. The feedback stage of this component is modelled as a rectified non-static linearity is used with the low pass filter. Finally the horizontal cell component of the model is composed of ~~an~~ a leaky integrate-and-fire ensemble with a feedback loop consisting of a polynomial non-static linearity transform and a final low pass filter. The resulting model can be seen in *Figure 2*.

I don't really understand this. You have to write down the equations that describe the model you try to implement. I don't know how a LIF neuron model can represent ion channels.

As I said, I cannot understand what you were trying to accomplish without giving a mathematical description of what the connections in this diagram are supposed to compute.

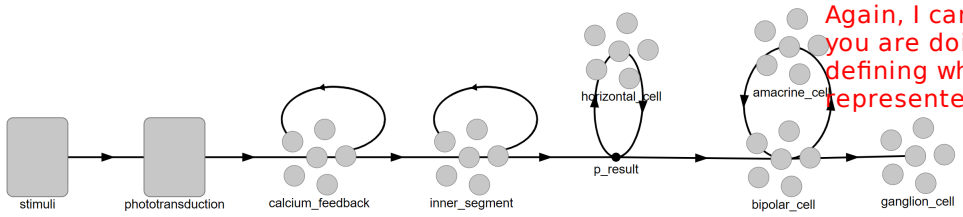
2.3.2 Temporal Contrast

The implementation of the bipolar, amacrine and ganglion cells are as described. Firstly, the model of the photo-receptor as described above is implemented. One specific change was to turn the horizontal cell feedback loop into a feed forward loop in order to better represent the interaction between the photo-receptor connection with the bipolar cells. The resulting response is then passed to a bipolar cell ensemble. Bipolar cells implement a contrast gain control based on a divisive feedback loop containing an amicrine cell ensemble of leaky integrate-and-fire neurons. This feedback loop consists of a rectified non-static linearity and a low pass filter. This will amplify the neural signal of the bipolar cells. This response is then transferred to the ganglion cell ensembles. At this stage it is necessary to take into account the short term synaptic plasticity. Short term synaptic plasticity is a phenomenon in which synaptic strength changes over time that reflects the

Unclear, what is a feed-forward loop?

source?

see above?



Again, I can't understand what you are doing here, without defining which variables are represented by the populations, and what functions are computed between the populations. Your code is also only mildly helpful

Figure 3: Diagram of the proposed whole retina model implemented in Nengo. An initial stimulus from the environment is passed through the photo-receptor model as previously described. Unlike the previous implementation, the horizontal cell response is treated as a feed-forward loop as it is more representative of the interaction between the horizontal cell, photo-receptors and bipolar cell connection. This signal passed thought the bipolar cell ensemble with an amicrine cell feedback loop connected. Finally, the response is passed into a ganglion cell ensemble using an adaptive leaky integrate-and-fire ensemble to represent the synaptic dynamics.

source?

history of pre-synaptic activity. For the purposes of this model, it was determined using the Nengo neuron type adaptive leaky integrate-and-fire neurons would suffice. A final low-pass filter will minimize any noise that occurs during the transformations and the resulting signal should be a representation of the retina's response to light. The resulting model can be represented as seen in *Figure 3*.

3 Experiments and Results

Two sets of experiments were conducted to investigate the accuracy of the retina model.

3.1 Light Intensity

It was necessary to investigate the initial response of the retina to the intensity of the light stimuli. As previously described, it is thought that the initial response to light, the adaptation of light intensity, is conducted in the photo-receptor and horizontal cell feedback complex. To investigate the response of the light intensity in the model, the following experiment was conducted. An incident pulse of light is introduced as a stimuli to the modelled photo-receptors. The length of time this light pulse persisted was varied from 10ms, 100ms, and 160 ms and is referred to as the width of the light pulse. The contrast of this light pulse was varied from 1 troland (td), 10 td, and 100 td. Troland, is a unit of conventional retinal illuminance. It is meant as a method for correcting photo-metric measurements of luminance values impinging on the human eye by scaling them by the effective pupil size. Using the troland allows us to assume the incident light is unaffected

I don't understand what contrast refers to here

You should have mentioned what this unit is earlier.

Thanks, but I read the definition of "td" on Wikipedia the first time you mentioned that unit.

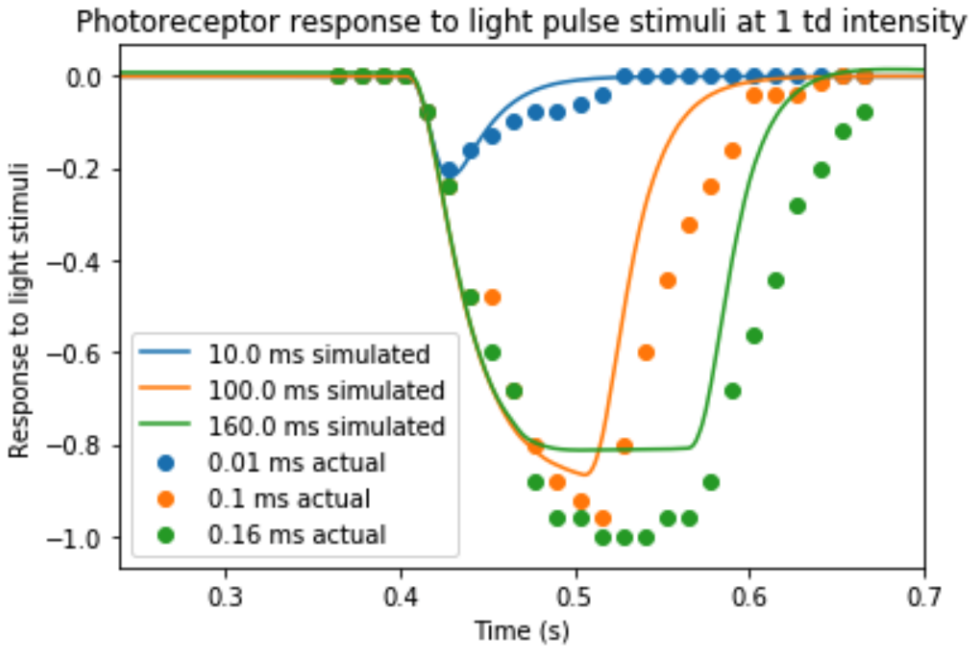


Figure 4: Result of applying three pulses of varying widths (10ms, 100 ms, 160 ms) to the photo-receptor model at an intensity of 1 td. Dots represented biological neuron data and the plotted line is representative of the simulated data.

Where is this data coming from? I also can't find any referenc in your code.

by the rest of the structure of the eye, such as the lens or cornea. ✓ See, this sentence is much clearer than the one you copied from Wikipedia.

The three different levels of intensity were applies to the model. The background of each level of intensity was reduced by a factor of 8 from the impulse strength. This is in accordance to the neurobiological data. The results or the 1 td pulse waves can be seen in *Figure 4*. The results of the simulation shows a approximate response of the biological photo-receptor. However it is noted that it lacks the specific details of the biological photo-receptor curves. It is also noted that the 160 ms pulse does not achieve a response at the same level as the biological neuron. ✓ The pulses with light intensity of 10 td is shown in *Figure 5*. As in the previous intensity it shows and approximate response to the stimuli light. Although it is noted that it appears worse. Specifically all three curves experience a spike at their maximum. This spike also occurs in the 100 td intensity light as seen in *Figure 6*. It is noted that for both intensities of 10 td and 100 td, the response of the simulated receptors lasts approximately 20 ms and 50 ms longer the expected respectively. ✓

3.2 Temporal Contrast

The purpose of this section was to investigate the retina's ability to reduce changing contrast overtime. It was determined that incident light of greater than 5 quanta will elicit a

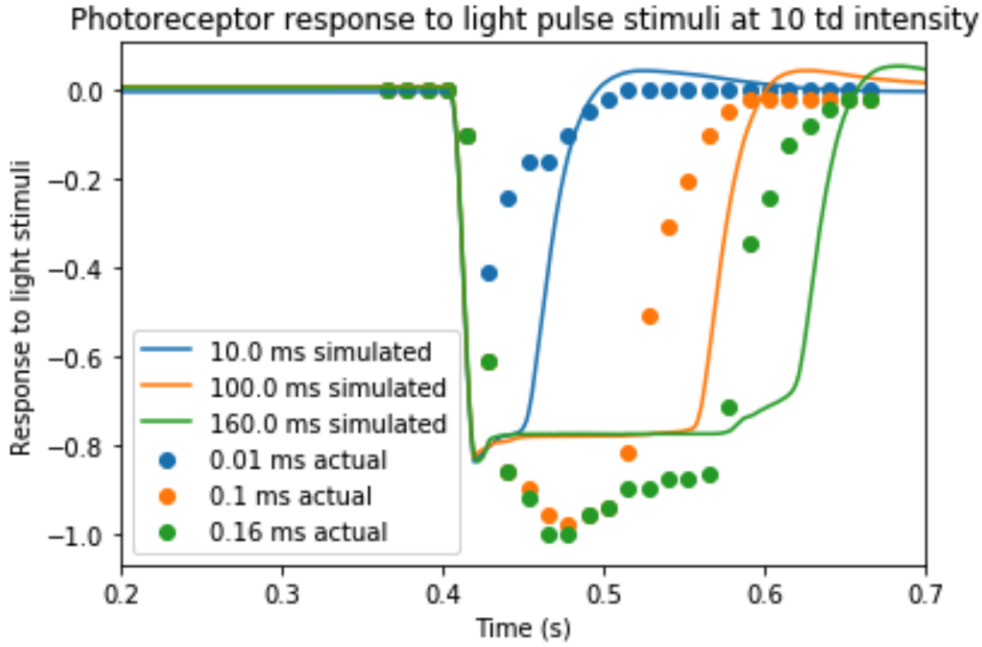


Figure 5: Result of applying three pulses of varying widths (10ms, 100 ms, 160 ms) to the photoreceptor model at an intensity of 10 td. Dots represented biological neuron data and the plotted line is Representative of the simulated data. ✓

see above

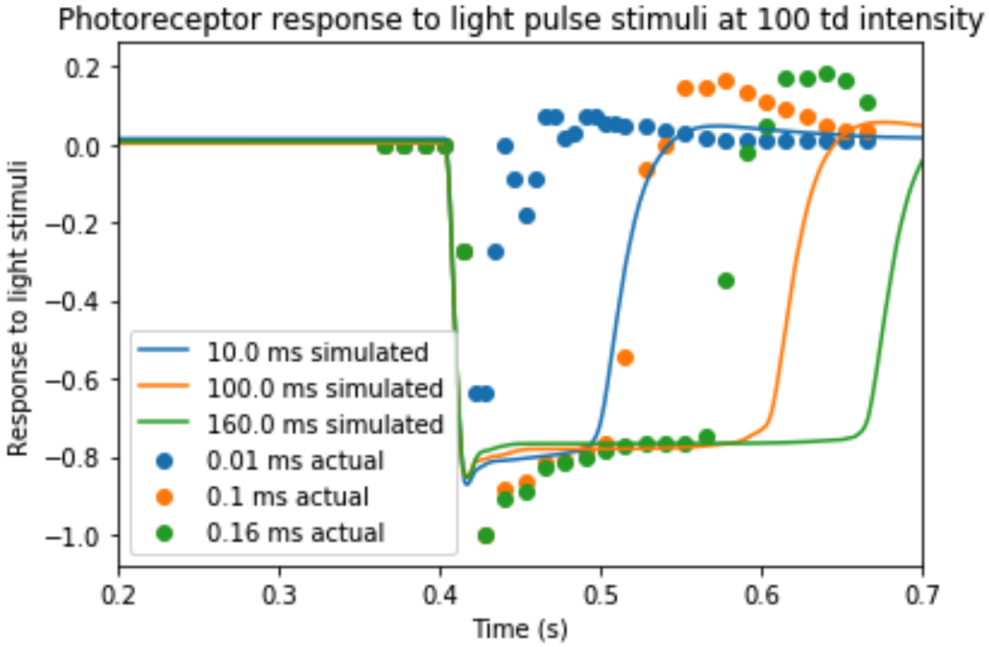
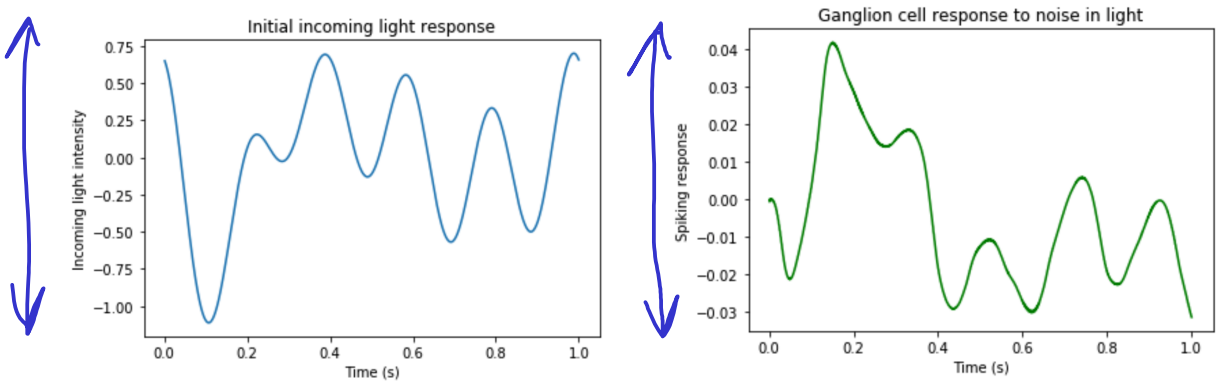


Figure 6: Result of applying three pulses of varying widths (10ms, 100 ms, 160 ms) to the photoreceptor model at an intensity of 100 td. Dots represented biological neuron data and the plotted line is Representative of the simulated data. ✓

see above

Use the same scaling, otherwise it is unclear what you are trying to show.



(a) **(b)**

Figure 7: Ganglion Cell response to noisy light stimuli. This experiment was conducted in order to visualize the ability of the retina model to reduce the noise of changing contrast stimuli.

response from the retina ganglion cells. Anything lower then that will not. This is in order to reduce the effect of noise in the retina's response to visual stimuli. For our representation a Gaussian white noise signal was produced to show random noise in an arbitrary stimuli. The model passes this noise thought the different layers of the retina to the Ganglion cells. The ganglion cells response adapts to the noise. As the amplitude of the noise increases the ganglion cells will have a negative response to counter act this noise. It is also noted that when two peak of similar trend are consecutive, the ganglion cells will mute this difference as seen at the two peaks approximately at 0.8 s of *Figure 7* where the two peaks are distinct in the initial stimuli, but have less prominence in the ganglion response. The issue with this representation is the units of the Ganglion response. As can be seen in *Figure 7*, the ganglion response is at 100 magnitude smaller then that of the white noise. This is due to the parameters of the transformations used in the retina computation, as they are inefficient to capture the magnitude of the ganglion cell response. Ideally if the model worked correctly, adding the white noise to the ganglion response would show light responses of greater magnitude then 5 quanta as the retina naturally does biologically.

4 Discussion and Conclusion

In this report, a retina model was developed and presented. It was used to investigate the effect of light flashes in two parts of the retina. The initial response offered by the photo-receptors of the retina and the final response sent to the brain for visualization by the retina ganglion cells. Disappointingly the results of both experiments were poor ap-

What makes you say this?

Sources?

That seems to be a last resort. It seems as if you didn't even attempt to think about this problem in a more principled/ functional way.

Oh, that is what that daya is. Why didn't you mention that before? And what is the source of that data?

And we would understand what?

proximations of neurobiological data of the same experiments conducted on Macaques photo-receptors and Cat retina ganglion cells. This is due to the transformations conducted at each section of the model retina. Upon further investigation of literature developing similar models, most groups use a genetic algorithm to determine the best values for the approximation of each step. With further time and opportunity to learn methods of genetic algorithm implementation, this simulation may produce greater results. Unfortunately, the student cannot further investigate this method of optimization. Thankfully this project has offered a lot of learning of the retina structure, the use of neurons in the retina, and the process involved in the NEF and implementation of neural systems. It is also acknowledged that the student initially started creating the model without the use of Nengo. However, when Nengo was used, it made the process far easier and improved the performance of the model. It is concluded that although the model in its current state doesn't offer an ideal replication of the retina's response to light, it shows an approximation that with further investigation of the transformations between retina layers could offer a better representation of the retina for further investigation.

References

Smith, Roberta (Jan. 2018). *A Deep Dive Into the Brain, Hand-Drawn by the Father of Neuroscience*. URL: https://www.nytimes.com/2018/01/18/arts/design/brain-neuroscience-santiago-ramon-y-cajal-grey-gallery.html.

Guo, Tianruo et al. (2014). "Understanding the Retina: A Review of Computational Models of the Retina from the Single Cell to the Network Level". In: *Critical Reviews in Biomedical Engineering* 42.5, pp. 419–436. DOI: 10.1615/critrevbiomedeng.2014011732.

Eliasmith, Chris and Charles H. Anderson (2003). *Neural Engineering: Computation, Representation, and Dynamics in Neurobiological Systems*. Cambridge, Massachusetts: MIT Press. 380 pp. ISBN: 978-0-262-55060-4.

Marshak, D.W. (2009). "Retinal Ganglion Cells: Anatomy". In: *Encyclopedia of Neuroscience*. Ed. by Larry R. Squire. Oxford: Academic Press, pp. 211–218. ISBN: 978-0-08-045046-9. DOI: https://doi.org/10.1016/B978-008045046-9.00897-4. URL: http://www.sciencedirect.com/science/article/pii/B9780080450469008974.

Bloomfield, S.A. (2009). "Retinal Amacrine Cells". In: *Encyclopedia of Neuroscience*. Ed. by Larry R. Squire. Oxford: Academic Press, pp. 171–179. ISBN: 978-0-08-045046-9. DOI: https://doi.org/10.1016/B978-008045046-9.00891-3. URL: http://www.sciencedirect.com/science/article/pii/B9780080450469008913.

Smith, Vivianne C. et al. (2001). "Primate Horizontal Cell Dynamics: An Analysis of Sensitivity Regulation in the Outer Retina". In: *Journal of Neurophysiology* 85.2. PMID: 11160492, pp. 545–558. DOI: 10.1152/jn.2001.85.2.545. eprint: https://doi.org/10.1152/jn.2001.85.2.545. URL: https://doi.org/10.1152/jn.2001.85.2.545.

Warren, Ted J. et al. (2016). "Kinetics of Inhibitory Feedback from Horizontal Cells to Photoreceptors: Implications for an Ephaptic Mechanism". In: *Journal of Neuroscience* 36.39, pp. 10075–10088. ISSN: 0270-6474. DOI: 10.1523/JNEUROSCI.1090-16.2016. eprint: https://www.jneurosci.org/content/36/39/10075.full.pdf. URL: https://www.jneurosci.org/content/36/39/10075.