Simulating Layout and Connection Pattern Development of Retinal Horizontal Cells

Final Report

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Introduction

Lateral inhibition in which a group of cells suppresses the activities of their surrounding cells, firstly discovered in the eyes of *Limulus*[2], has been found in various systems of multi-cellular organisms[16]. Primary visual processing of visual animals, from insects to mammals, involves lateral inhibition to enhance sharpness and modify color discrepancy of visual scenes[16]. In mammals, retinal horizontal cell, an interneuron playing a critical role in primary retinal lateral inhibition, receives inputs from surrounding photoreceptors and provides negative feedback to photoreceptors, facilitating the formation of center-surround receptive fields[15].

It has been found that different types of horizontal cells hold different connection patterns and may be beneficial in various environments[13]. Regardless of cell types, branching distances, receptive field overlapping, and cell densities vary in different positions of a retina, depending on the densities of photoreceptors, and among different species, depending on the species' habitats[12]. Together, retinal horizontal cells collaborate with each other and modulate space arrangement, forming mosaic that allow uniform coverage and efficient process of visual input[6][9][14].

However, why do the arrangements and dynamics of horizontal cells, the pre-processing interneurons in retinas, not show significant differences among investigated species? In other words, how this elaborate structure evolves and whether there are any alternatively effective structures remain unclear. This project hypothesizes that there are alternative structures that lead to local maxima of fitness (minima of survival cost), and, if there is any, will attempt to find the reason to explain why those alternatives do not exist or have not been discovered in real world. Looking for and analyzing these alternatives help us to

understand retina and evolution better. To explore those alternatives, genetic algorithm, a random-based searching algorithm optimal for identifying multiple local extreme values in a fitness surface[10], is used and analysis of these structures will be performed.

Method

Representation of Retina

Retinal lateral inhibition is commonly modeled as either directed graphs, i.e. artificial neural network, where horizontal cells are interneurons in hidden layers, or image filtering, where horizontal cells are filters convoluted with visual input. The latter is much less computationally intensive and easier to implement, so it is used in many simulations or models where the pre-processing of visual input is modelled as convoluting the input with difference of Gaussian (DoG) or the similar[4][5][11][17]. However, filter approach does not support complex network dynamics. Thus, the former is used since it enables flexible alteration of horizontal cell properties and dynamics. The amount of receptors are fixed and receive constant input continuously, and their states are modulated by interneurons; retinal ganglion cells (RGCs), the output neurons, are also fixed in its amount which is smaller than that of receptors. The general retinal processing could be described as a set of three differential equations:

$$\tau \frac{\mathrm{d}}{\mathrm{d}t} V_r = -V_r + I + \sum_{i \in \{1,2,\ldots\}} \sigma(W^{(i)^T} V_i)$$
$$\tau \frac{\mathrm{d}}{\mathrm{d}t} V_j = -V_j + \sum_{i \neq j, i \in \{r,1,2,\ldots\}} \sigma(W^{(i)^T} V_i)$$
$$\tau \frac{\mathrm{d}}{\mathrm{d}t} V_g = -V_g + \sigma(W^{(r)^T} V_r)$$

where V is the internal states of a type of cells, W is the connection matrix, I is the input, τ is the time constant assumed to be the same for all types of neurons, and σ is the activation function, chosen to be sigmoid. Subscripts r and g stand for receptors and RGCs respectively, and j and i are interneuron types labelled as positive integers. Preliminary experiments were done to find the appropriate total simulation time.

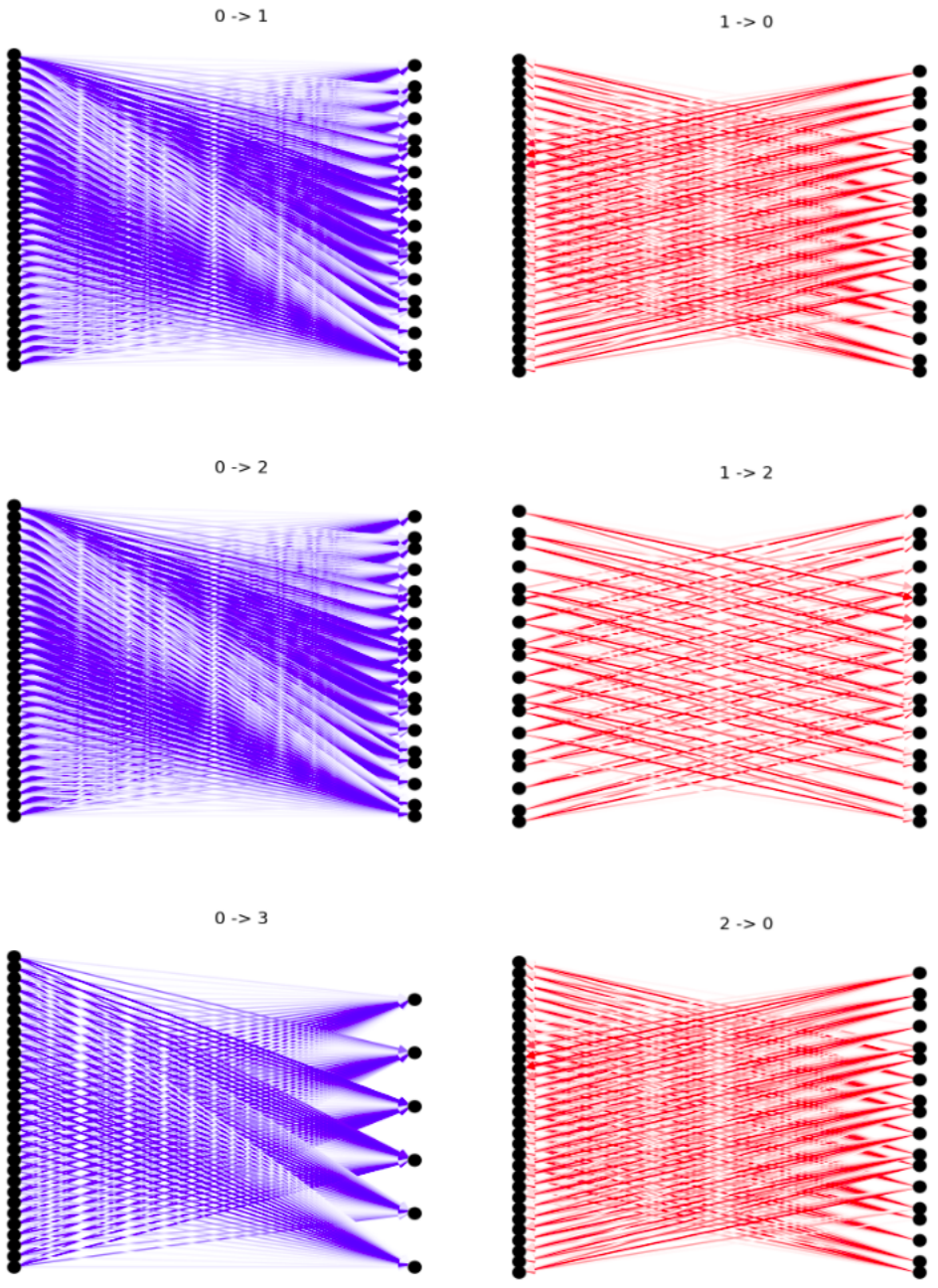

Figure 1: an example of the retinal connection. At the top of each sub-graph is a label "$i \to j$" representing "from type i (left black circles) to type j (right black circles)." In this graph, type 0 is receptor, and type 3 is RGC. Red arrows indicates excitatory projections and blue indicates inhibitory projections. The lighter the arrows, the weaker the connections. Weights below 10^{-2} are not shown but used in the computation.

Genome

A retina is built from a set of descriptors to be optimized, termed as genome. Each retina has an integer specifying the number of interneuron types it can have, referred as capacity. Each type of interneuron, i, has 1) an axon descriptor $x_{i,a}$ and a dendrite descriptor $x_{i,d}$, encoded in 32-bit binary strings, that abstractly represent their axon and dendrite properties respectively; 2) an integer specifying how many cells are for this type; 3) the polarity of this type $p \in [-1, 1]$; and 4) scalar ϕ and β that controls the scale and preferred range of projection.

There are, in addition, three "biological limits" regardless of genome types: the maximum number of cells each type of neuron can ever reach, the maximum number of interneuron types a retina can ever reach, and the width of the retina. Moreover, it is assumed that cells are uniformly distributed in the space, and the thickness of the retina is ignored.

From the genome, the connection W_{ji} from neuron i to j is the product of affinity, a, polarity, p, and decay, d.

$$
\begin{aligned}
W_{ji} &= apd \\
a &= \frac{1}{32}\text{count0}(x_{i,a} \otimes x_{j,d}) \\
d &= e^{-[(\Delta-\beta)/\phi]^2}
\end{aligned}
$$

where $\otimes$ denotes XOR operation, function count0 counts the number of zeros, and Δ is the lateral distance between the two cells. The whole weight matrix is then normalized to be in $[0, 1]$ or $[-1, 0]$ depending on its polarity.

Selection

Some portion of "elites", those with the greatest fitness, are preserved, and the rest are replaced by "children", so that after one generation, some good results that the simulation achieves so far could be preserved.

Children are produced by two "parents" generated from binary tournament selection, in which each of the two parents is selected from two rivals drawn from the whole pool without replacement (Figure 2). The rival with better fitness evaluation has higher chance (> 0.5) to be one of the parents, while the one with worse fitness evaluation has lower

chance to be the parent. Different from the conventional approach where the chance is fixed, the chance in this simulation depends on the ratio of the two survival cost, s_1, s_2.

$$p(s_1, s_2) = 1 - 0.5 \times \frac{\min\{s_1, s_2\}}{\max\{s_1, s_2\}}$$

If two rivals are equally competent, the ratio between their scores is close to 1, and the chances for both are close to 0.5. Otherwise, the more competent one is more likely to be the parent. In case of comparably good retinas, this dynamical procedure potentially prevents premature convergence.

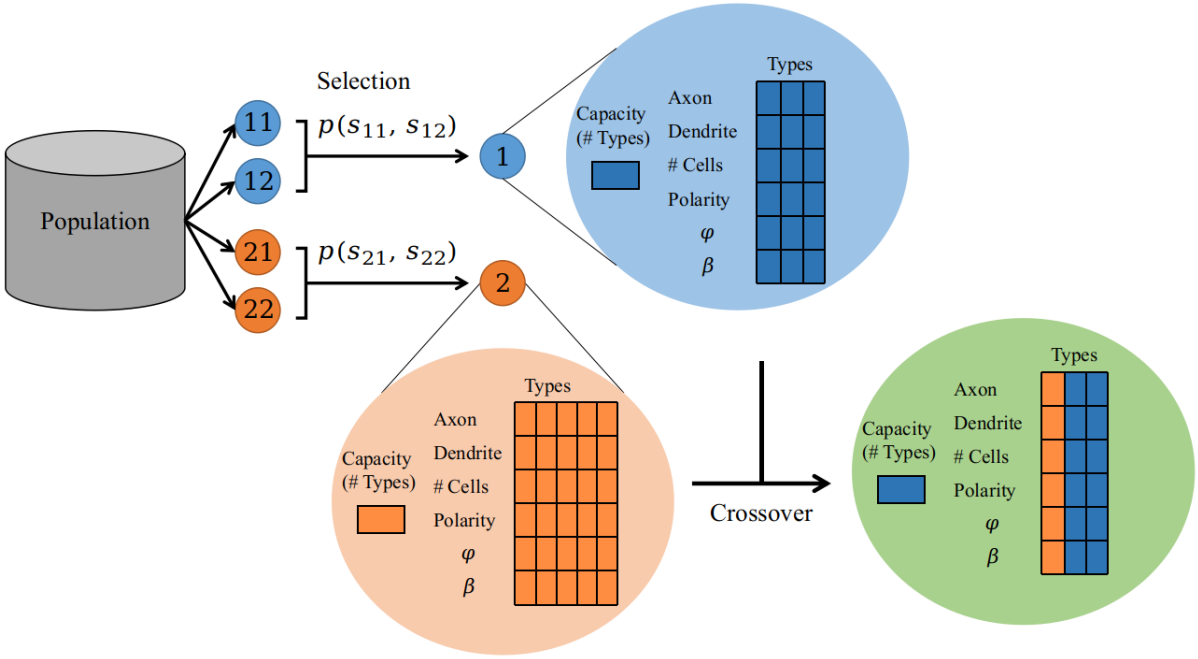

Figure 2: selection and crossover performed in the simulation. A parent x is selected with probability $p(s_{x1}, s_{x2})$ from two rivals with tag $x1$ and $x2$. The two selected parents, unlabelled blue and orange circles, crossover and produce a child in green. In a magnified circle, rectangles represent genes named in the figure.

Crossover

The crossover imitates the real crossover happens in organisms. The properties for a type of neuron, including axon, dendrite, number of cells, polarity, ϕ, and β, are linked, so that either none or all of them are passed to an offspring. During crossover, a child inherits its

capacity from one of the parent randomly. Similarly, the child gets the properties of receptor and RGC from one parent. Then, for each interneuron type, the properties of an interneuron type from one of the parents are copied.

Mutation

To avoid non-convergence due to high randomness, the mutation rate is low. Each axon or dendrite descriptor is flipped only two bits randomly at one generation. In one generation, for each type of neurons, random values drawn from zero-centered Gaussian distribution with very small standard deviation (0.005) are added to non-integer variables (p, ϕ, β), and the number of cells (# Cells) has very small chance (0.05) to increase or decrease by 1. Then, non-integer and integer variables are clipped in case of invalid values. The boundaries used in the simulation is

Variable	Min	Max
p	-1	1
ϕ	1	Width of Retina / 2
β	0	Width of Retina / 2
# Cells	0	Max number of Cells

Table 1: minimum and maximum values the variables can ever reach during mutation

Visual Input

The visual input is a one-dimensional array of brightness, which is simpler yet effective to promote the evolutionary process. If possible, two-dimensional scheme will be developed in the future. The reference brightness curve of prays is artificially defined to be narrow, while that of predators is wide. The inputs are perturbed brightness curves of prays and predators so as to test the retinas' capability of reducing noise and increasing contrast at the edges of the inputs (Figure 3).

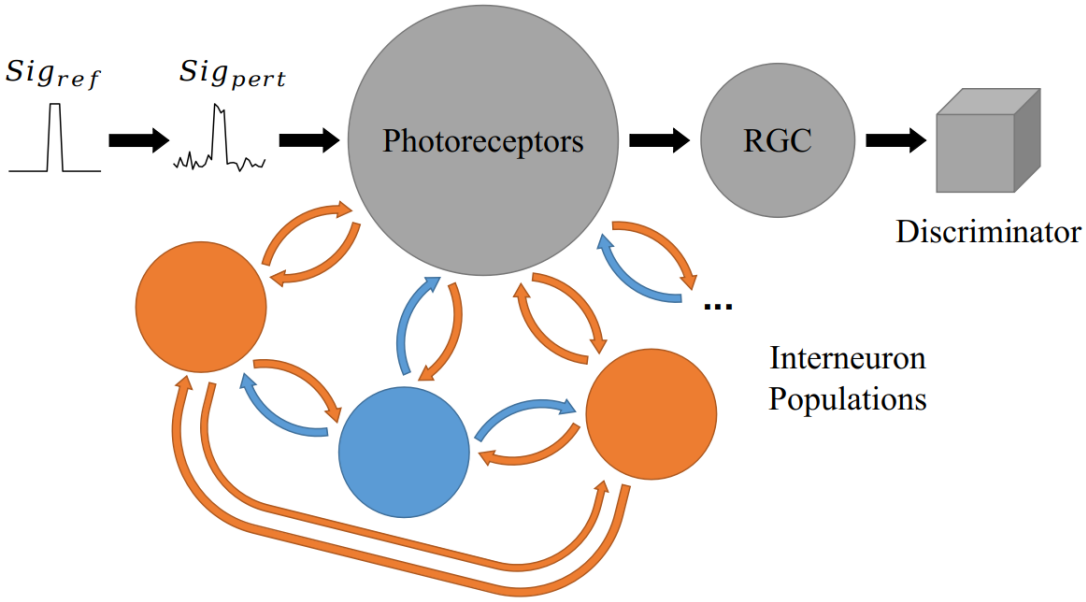

Figure 3: a diagram of the general structure of the retina. Each circle represents a group of neurons, with no inter-connection. Sig_{ref} is the reference signal, perturbed to Sig_{pert} by adding Gaussian noise. Then it is presented to photoreceptors, which make reciprocal connections with interneurons (in orange and blue) and project to RGCs. For interneurons, orange indicates excitation and blue indicates inhibition. The output of RGCs is taken by a discriminator whose performance is evaluated. The discriminator in use is a single layer perceptron.

Fitness Evaluation

The project, like many other models, assumes there is no information loss due to signal transduction. Therefore, the modified visual information is considered as the input to the further visual processing, which is abstractly modeled as a perceptron that classifies predator and prey patterns. The perceptron for each retina is trained with a few processed input, i.e., the weights of a perceptron depend on the corresponding retina. The performance of the perceptron as well as space usage are considered in fitness evaluation of a retina (Table 2). The overall cost of an individual is the linear combination of the metrics in consideration.

Metrics	Reason
$-(y \log p(y) + (1-y) \log(1-p(y)))$	y is the ground truth label and $p(y)$ is the prediction. This cross entropy loss measures how well the perceptron can use the retina to learn the input.
$(\frac{1}{n}\sum_i h_i)^{-2}$	h_i is the distance between cells of type i, with n types in total. Small interval is less plausible in real retina where cell bodies occupy space.
c_{total}/c_{max}	c_{total} is the total number of synapses and c_{max} is the maximum possible number of synapses. Supporting synapses need space and nutrition.

Table 2: terms considered in fitness evaluation

Other methods, such as replacing the perceptron with bidirectional associative memory (BAM) and directly measuring the sharpness of the processed input, are also believed to be effective. Instead of train and test with labels, BAM associates two patterns by updating its weights with a rule[8], such as Hebb rule which is inspired by Hebbian theory of neural plasticity[3][7]. Sharpness measurement can be done in a variety of approaches, such as summarizing the result of fast Fourier transformation (FFT) on a processed input[1]. The methods will be tried in the future.

Results and Discussion

500 individuals are randomly generated and 100 elites are preserved in each generation. By setting the weight for all fitness evaluation metrics other than cross entropy error to zero, the genetic algorithm converges very fast, and the costs are lower bounded by approximately 0.68 (Figure 4, top). Setting non-zero weights that scales those metrics to be in similar range of cross entropy loss also results in premature convergence and similar lower bound, except that the numbers of neurons decrease due to spatial cost. In both cases, the population converges to retinal configurations that make the output nearly all zeros, which means no individuals can process the input better than being "blind" (Figure

1). Given that the dynamics of a retina is largely affected by the polarity of the receptors, which, if negative, simply causes the outputs of interneurons and RGCs to be zero, there should exist an energy well preventing the population from converging to other minima of cost that are not "blind" retinas.

To see if removing the energy well reduces premature convergence and leads to better results, the polarity of receptor cells is set to be positive, in range $(0, 1]$, for every retina. This does improve the issue of premature convergence, yet the cross entropy loss of the resulting structures are much higher, lower-bounded by about 1.3. Adjusting the parameters of genetic algorithm, such as number of generations, number of individuals, and simulation time, only slightly shifts the quantiles, minimum and maximum, but never lets the cost go below 1.3. The reason for the lower bound has not been found, but is believed to be a lack of concern in some part of fitness evaluation.

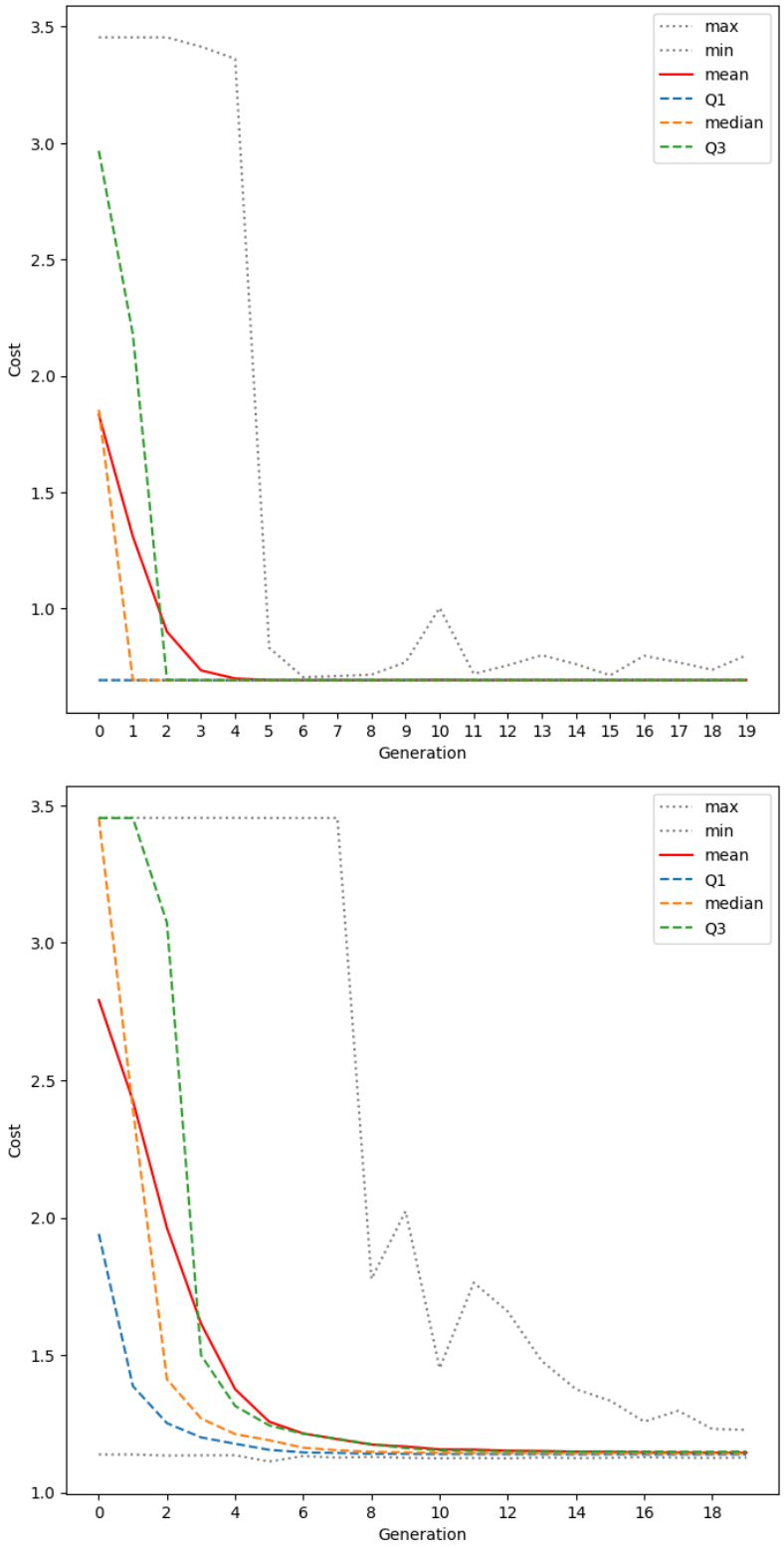

Figure 4: top: one example of the performance of the simulation without enforcing the projections from the receptors to be positive. Bottom: an example of the simulation with enforcing the projections from the receptors to be positive.

Future Plan

Based on the discussion above, the design of fitness evaluation and implementation of genetic algorithm should probably be furthermore modified, and be paid most attention in the future. Bidirectional associative memory will be tried instead of perceptron[8].
Moreover, performing regression rather than classification could be helpful and interesting to try, since labelling a perturbed narrow signal to be prey and a perturbed wide signal to be predator probably abstracts the visual cognition system too much. Instead, doing regression on input signal and reference signal width could be more straightforward.
On the other hand, other approaches that do not need a discriminator, such as measuring the sharpness of a signal, could be used. Evaluating by sharpness is a more direct method that ensures smooth outputs, such as zeros, will have high cost. However, finding a good way of measuring such sharpness is critical. A method using FFT, as briefly described in fitness evaluation, could be suitable for this simulation from some simple experiments conducted[1].
Furthermore, current construction of retina merely takes "rods" into account, while also disregarding its tuning curve. Adding cones and corresponding tuning curves into the model and assess its capability of correcting color discrepancy could make the simulation more robust, and the results may be more interesting.

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