Building a Computational Model of Aging in Visual Cortex

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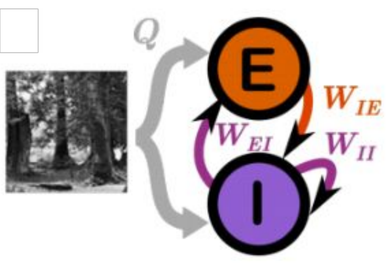
Motivation

The mammalian visual system has been the focus of countless experimental and theoretical studies designed to elucidate principles of sensory coding. To our knowledge, few computational studies have attempted to mechanistically investigate **how the computational properties of a network change as a healthy organism ages.**

We show that a spiking network model trained on visual inputs can qualitatively replicate experimentally observed features of aging by a simple change to its learning rules.

Model

In this work we use E-I Net [1], a spiking network model of primary visual cortex that has been shown to reproduce the visual response properties seen experimentally and modify it to reflect biological changes observed in animal studies of senescence.



Excitatory network

Inhibitory network

Learning Rules

Hebbian-Oja (HO)	$\Delta Q_{ik} \propto n_i X_k - n_i^2 Q_{ik}$
Correlation Measuring (CM)	$\Delta W_{ij} \propto n_i n_j - \langle n_i \rangle \langle n_j \rangle (1 + W_{ij})$
Threshold Adaptation	$\Delta \theta_i \propto n_i - p$

The input weights, synaptic weights, and firing thresholds evolve according to these learning rules that promote image reconstruction, uncorrelated firing, and sparse firing with target rate *p*, respectively. Here, n_i are the total spikes in response to an image.

We age the network by allowing the excitatory target spike rate *p* to increase linearly with continuous training.

Network model: activity of the neurons is modeled using a leaky integrate-and-fire model:

$$u_i(t+\delta t) = e^{-\delta t/\tau} u_i(t) + \sum_k Q_{ik} X_k(t) + \sum_j W_{ij} y_j(t) + I$$

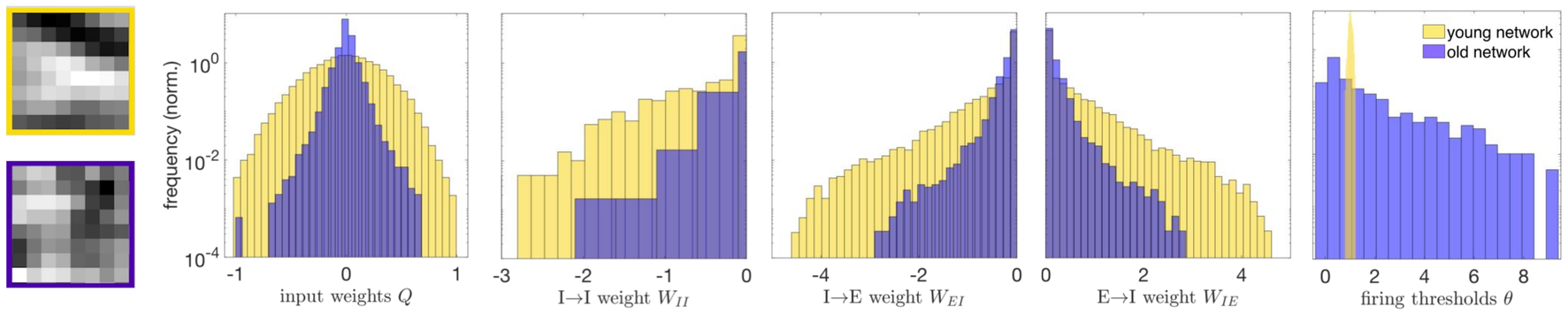
$$y_j(t+\delta t) = \begin{cases} 1, & \text{when } u_i(t+\delta t) \geq \theta_i \\ 0, & \text{otherwise} \end{cases}$$

$$u_i(t+\delta t) \to 0 \text{ when } y_i(t+\delta t) = 1$$

where $u_i(t)$ is the membrane potential of the neuron at time *t*, δt is the time step, τ is the membrane time constant, θ_i is the firing threshold of neuron *i*, Q_{ik} is the input weight from pixel *k* to neuron *i*, $X_k(t)$ is the k^{th} pixel value at time *t*, W_{ij} is the synaptic weight from neuron *j* to neuron *i*, *I* is a constant current input, and y_i is the spike train of neuron *i*.

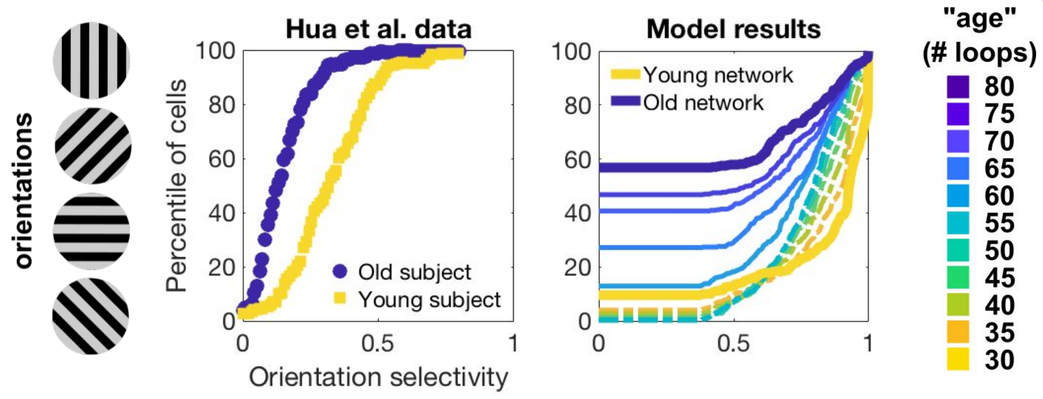
Results

Physiological changes



Input connections, lateral connections, and firing thresholds all change with age. Far left: Input weights of a particular neuron organized into a receptive field (RF) in youth (top) and old age (bottom). Histograms: Input weights & lateral weights weaken in magnitude with age, while firing thresholds become more broadly distributed.

Performance changes



Firing selectivity to oriented gratings decreases with age, in qualitative agreement with experimental data from [5]

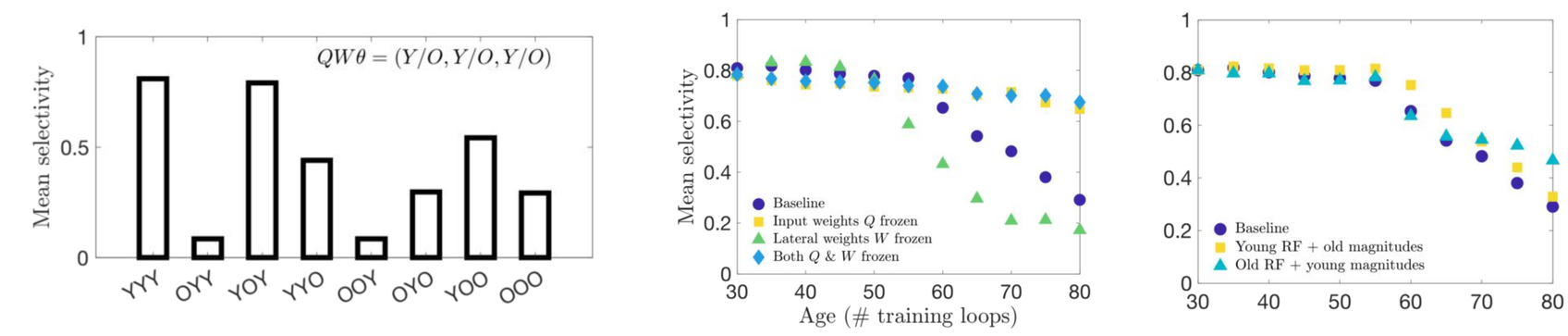
Neurons with Gabor-like RFs

Young (30 loops)	Old (80 loops)
123/400	26/400

	Gabor in age	Not Gabor in age
Gabor in youth	6/123	117/123
Not Gabor in youth	20/277	257/277

The fraction of neurons with Gabor-like receptive fields drastically drops in age

Methods to identify which physiological changes have greatest effect on selectivity



Swap young parameters with old

Freeze parameters during aging

Mix & match young/old RF magnitudes & structure

Methods

We train the model on black and white video of natural scenes [2]. To mimic the role of the retina and lateral geniculate nucleus in preprocessing visual input, we first whiten the frames before feeding them to the network [1].

We train the network for 30 loops at the baseline excitatory target spike rate of *p* = 0.01 expected spikes per image presentation to obtain our mature "young" network. To "age" this network, we then increase *p* by 0.02 every subsequent loop, culminating in the "old" network after 80 total loops.

To test orientation and direction selectivity, independent of training, we present whitened grating stimuli of various directions of motion to the networks. The orientation index **(OI)** for each neuron is calculated from the response ($n_j(\Phi_k)$) to each of the gratings (angles Φ_k = 0, $\pi/4$, …, $3\pi/4$) [4]:

$$OS_i = \frac{\left|\sum_{\text{trials}} \mathbf{r}_i^{\text{trial}}\right|}{\sum_{\text{trials}} \left|\mathbf{r}_i^{\text{trial}}\right|}$$

We can decouple the effects of the changing RF structure and magnitude on network selectivity by examining, independent of the training procedure, the selectivity after remapping the young input weights onto a later distribution of input weight magnitudes, for the networks after 30, 35, 40...80 loops:

$$Q_{\text{re}} = \Phi_{\text{old}}^{-1}\left(\Phi_{\text{young}}\left(Q_{\text{young}}\right)\right)$$

where $\Phi_{\text{young}}(\cdot)$ and $\Phi_{\text{old}}(\cdot)$ are the cumulative distribution functions for the young and old input weight distributions and Q_{re} are the remapped input weights.

We quantify how Gabor-like RFs are by the fitting procedure performed in [7], except that we use a less strict rejection condition (0.8 vs. 0.5).

Conclusions

- **Dysregulation of neuron firing may** be a **cause** of **age-induced changes** in brain physiology and performance.
- **Deterioration of input strength & structure** is the primary cause of performance deficits in our model.
- **Computational modeling holds promise** as an approach to forming a mechanistic understanding of aging.

Acknowledgments & References

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