Fixational eye movements increase acuity in a retinal Bipolar-Amacrine-Ganglion circuit model

Jason S. Sherfey[1,2], Arash Yazdanbakhsh[1,3]

[1]Center for Comp Neuro & Neural Technology; [2]Graduate Program for Neuroscience; [3]Cognitive and Neural Systems, Boston University

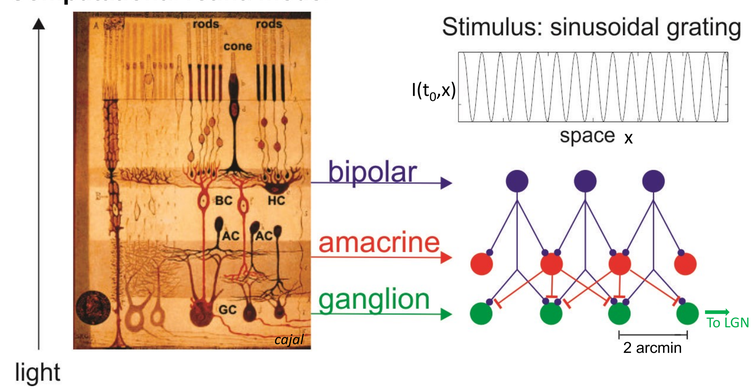
Introduction

The mammalian retina contains at least 11 Bipolar (B), 25 Amacrine (A), and 20 Ganglion (G) cell types. While the retina has been widely considered a simple interface between environment and brain with filters, relays, and image compression, the diversity of anatomical and physiological features present in the retina raises an important unanswered question: is retinal complexity an epiphenomenon of natural selection's lack of design or does it serve a purpose, endowing the retina with unforeseen computational capacities, previously masked by technological and experimental limitations. To address this question, we developed a mathematical model of a B-A-G network with eccentricity-dependent receptive fields and focused on the poorly understood issue of how retinal processing differs in the presence and absence of involuntary fixational eye movements (FEyeM = microsaccades and drift).

Methods

Drift was modeled as a continuous random walk at 10 arcmin/sec, and microsaccades were modeled as discontinuous, random events correcting for fixational deviations from fovea. Our model consists of three interconnected populations with continuous dynamics and Gaussian kernels: bipolar cells relay visual information to amacrine and ganglion cells, and amacrine cells inhibit ganglion cells. The retina model's ability to process spectral content was tested using sinusoidal gratings varying spatial (fs=2-12cpd) & temporal (ft=0-20Hz) freqs.

Computational retina model

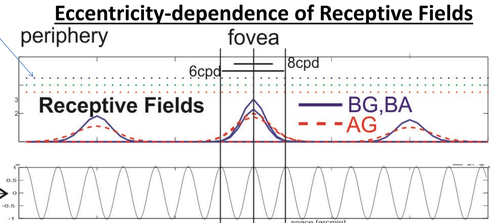


Population	Dynamics	Output
Bipolar cells (B)	$\dot{B} = a_B[-B + (2-B)I],\ \dot{z} = a_z(1\text{-}z\text{-}100Bz)$	$\hat{B} = Bz$
Amacrine cells (A)	$\dot{A} = -a_A A + (b_A - A)\left[\hat{B} * K^{BA+}\right] + GJ$	$\hat{A} = \max(0, A)$
Ganglion cells (G)	$\dot{G} = -a_G G + (b_G - G)\left[\hat{B} * K^{BG+}\right] - (G + d_G)\left[\hat{A} * K^{AG-}\right]$	G

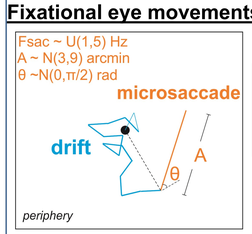
Receptive field kernels (connections): $K^{XYs} = c_{XYs} e^{-\left\|\frac{x-\gamma}{\sigma_{XYs}}\right\|^2}$, $s \in \{+,-\}$. (c and σ may vary across the retina)

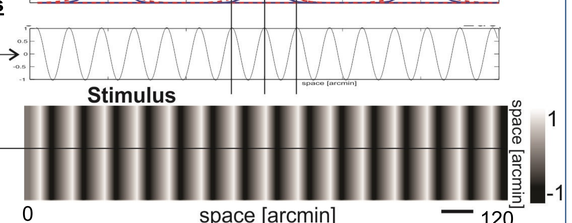
kernel	σ	c
BG+	2-3	3-.5
BA+	2-3	3-.5
AG-	3-4.5	2-.1

Eccentricity-dependence of Receptive Fields



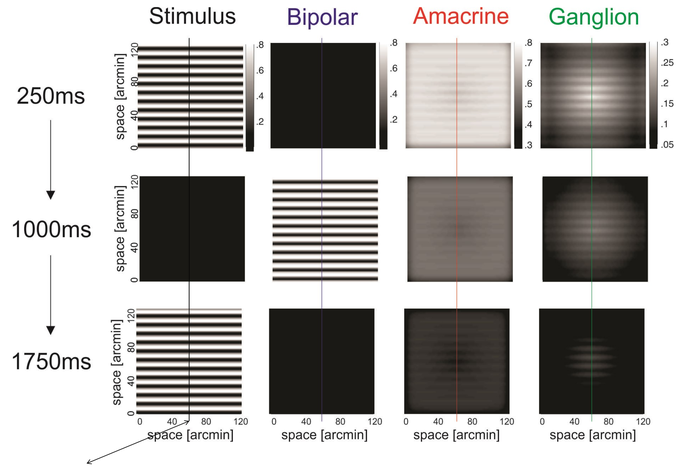
Fixational eye movements



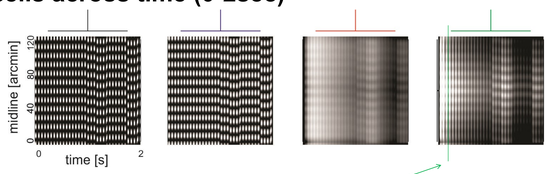


Model performance

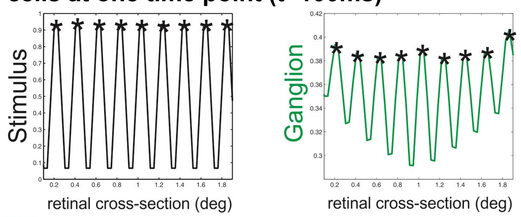
Example simulation



Midline cells across time (0-2sec)



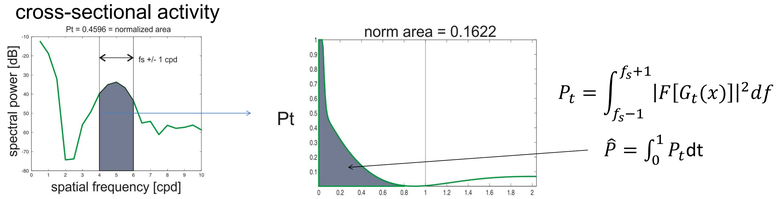
Midline cells at one time point (t=100ms)



Ganglion performance for a given stimulus

At t=100ms | Across time

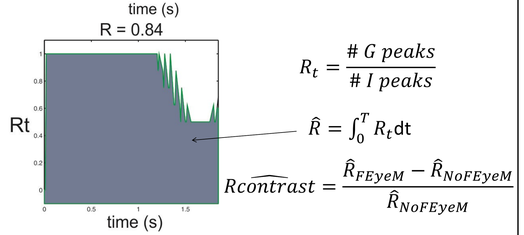
(1) Power spectrum of cross-sectional activity



$$P_t = \int_{f_s-1}^{f_s+1} |F[G_t(x)]|^2 df$$

$$\hat{P} = \int_0^1 P_t \mathrm{dt}$$

(2) Fraction of peaks resolved by ganglion cells



$$R_t = \frac{\#\ G\ peaks}{\#\ I\ peaks}$$

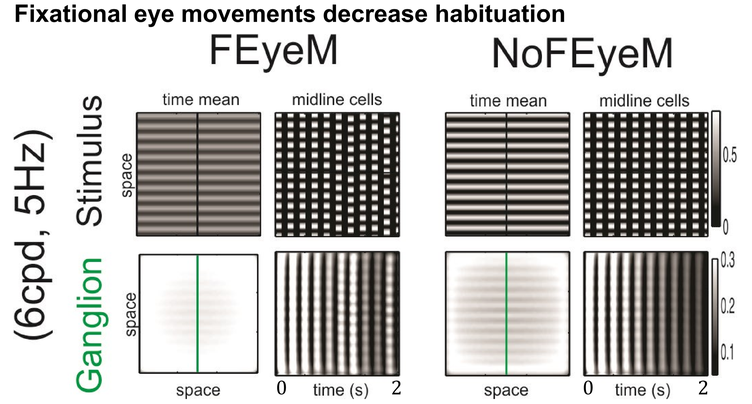
$$\hat{R} = \int_0^T R_t \mathrm{dt}$$

$$\widehat{Rcontrast} = \frac{\hat{R}_{FEyeM} - \hat{R}_{NoFEyeM}}{\hat{R}_{NoFEyeM}}$$

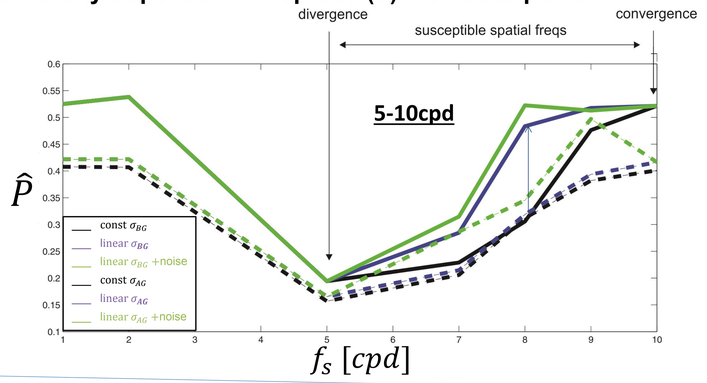
$\hat{P}$ and $\hat{R}$ represent how much of the stimulus power was relayed to LGN by the ganglion cells for a given grating frequency f_s and flash rate f_t.

Results

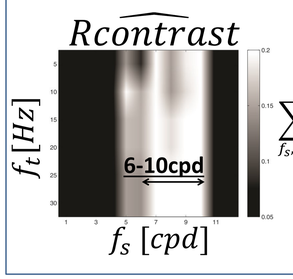
Fixational eye movements decrease habituation



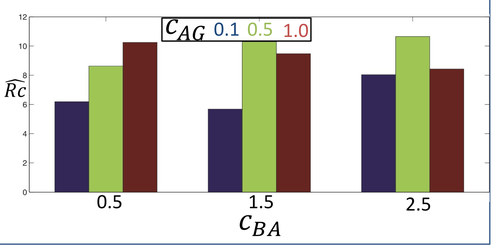
Eccentricity-dependent RF spread (σ) increases power



FEyeM increases power



Enhancement increased by Increasing peripheral inhibition (c_{AG})



Conclusions

Fixational eye movements enhance power for spatial frequencies 6-10cpd by increasing the area sampled by each ganglion cell and, on longer time scales, by delaying habituation. Adding eccentricity-dependence to bipolar-to-ganglion kernels also increased power at spatial frequencies 5-10cpd, both with and without FEyeM. In our model, these spatial frequencies have a cycle period on the order of the foveal ganglion receptive field. We hypothesize that FEyeM and receptive field gradients are two mechanisms that can increase retinal acuity for spatial frequencies on the order of ganglion receptive fields.

Future simulations will aim to disentangle drift & microsaccade effects; explore dependence on temporal characteristics, amacrine physiology, implications for neural encoding, cortical processing, & development.

Acknowledgments

Supported in part by CompNet travel grant, CELEST (NSF SBE-0354378 and OMA-0835976), Office of Naval Research (ONR N00014-11-1-0535), and AFOSR FA9550-12-1-0436