The Role of Dynamical Synapses in Retinal Surprise Coding

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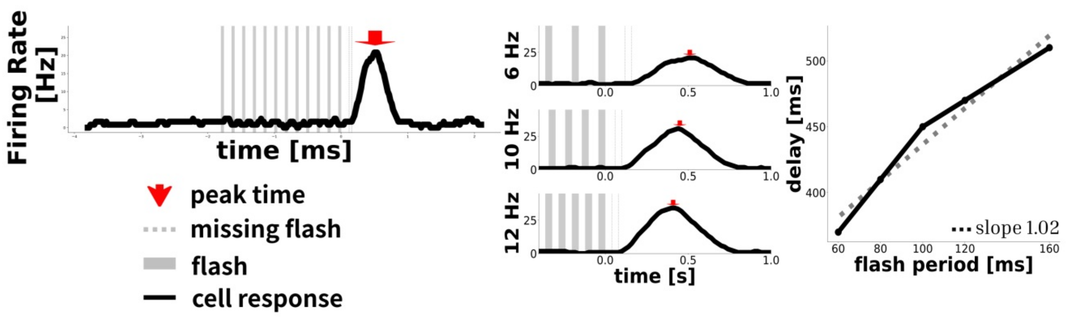
Abstract

The retina is the first stage of visual processing. The efficient coding theory postulates that it compresses relevant visual information before sending it to the brain. A long-standing hypothesis is that retinal ganglion cells, the retinal output, do not send signals about the visual scene per se, but signal only surprising events [1], such as mismatches between observation and expectation formed by previous inputs. A striking example of this is the Omitted Stimulus Response (Box 1) [2]. In this context, the main question of this study is :

How can a local circuit detect 'surprise' and which cellular mechanisms enable these computations ?

Box 1 : The Omitted Stimulus Response



The retina emits an Omitted Stimulus Response when an **expected flash** in a periodic sequence is **missing**. The time-point of this response scales linearly with the period of stimulus, signaling the **precise time when the next flash was expected.**

Experimental Approach

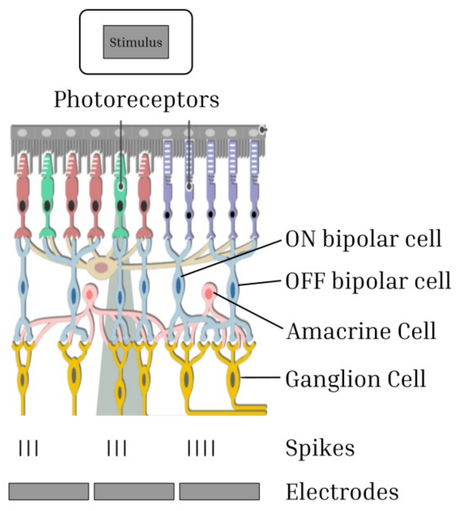
Electrophysiology

The retina is stimulated with sequences of periodic dark flashes. Ganglion Cell spiking is recorded via multi-electrode arrays. Shown below are the firing rates of individual cells, averaged over multiple trials

Pharmacology

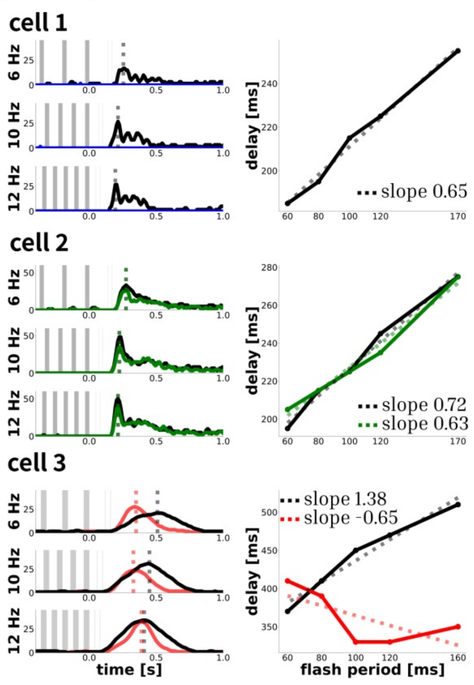
Pharmacological compounds selectively block synaptic transmission:

- onto ON bipolar cells
- onto OFF bipolar cells
- from glycinergic amacrine cells



Retinal architecture and Recording Setup

Results



Omitted Stimulus Responses in 3 different retinal ganglion cells before (black) and after (colored) application of pharmacological compounds.

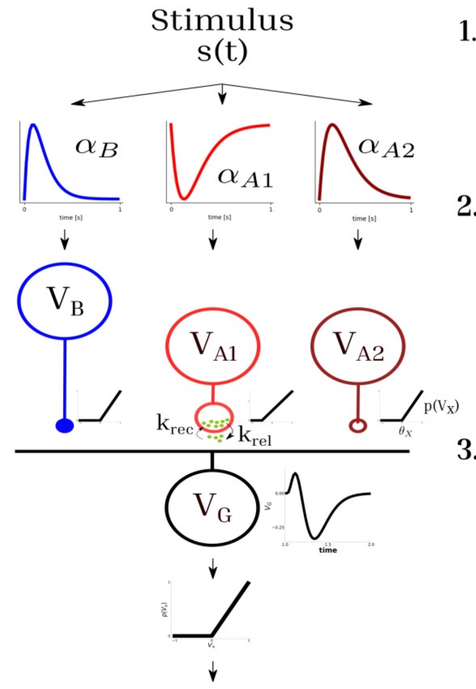
➤ ON bipolar cell inhibition **cancels** the OSR

➤ OFF bipolar cells have **no direct effect** on the OSR

➤ glycinergic amacrine inhibition **cancels predictive timing**

Modeling Approach

Network model with short-term plasticity



1. Convolution of stimulus and temporal kernel $X = B, A1, A2$

$$F_X(t) = [\alpha_X * s](t)$$

2. Bipolar and amacrine cells integrate the signal into their membrane potential

$$\frac{dV_X}{dt} = -\frac{V_X}{\tau_X} + F_X(t)$$

3. Ganglion cells pool over the network.

$$\frac{dV_G}{dt} = -\frac{V_G}{\tau_G} + \sum_{X=1}^{N} w_X p_X(V_X(t), \theta_X)$$

4. Ganglion cell voltage response is transformed into a firing rate

A Simple circuit with one excitatory (B), one delayed inhibitory (A2) and one adapting inhibitory unit (A1) to form predictive responses .

Box 2 : Dynamic Synapse

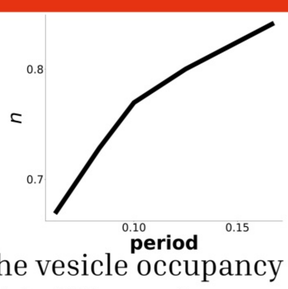
The occupancy n of the vesicle pool follows the kinetic equation:

$$\frac{dn_{A1}}{dt} = \underbrace{(1-n)k_{rec}}_{\text{replenishment}} - \underbrace{\beta k_{rel} p_{s\ n}(V_{A1}(t), \theta_{s\ n}) n_{A1}}_{\text{release}}$$

The synaptic weight is scaled by n

$$w_{A1}(t) = \bar{w}_{A1} n_{A1}(t)$$

[3]

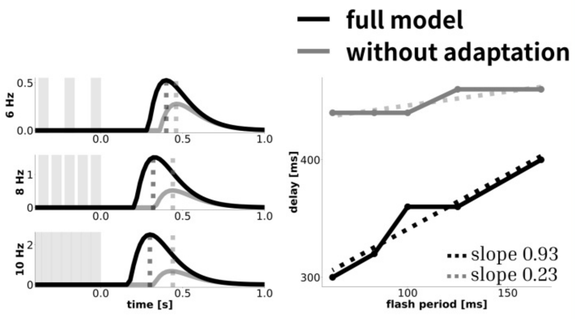


The vesicle occupancy varies with different frequencies of the stimulus.

Mechanistic Explanation

1. Delayed inhibitory input via A2 causes rebound response after flash end.
2. Dynamically adapting synapse of A1 enables the circuit to have a fine temporal prediction by
   - Adapting the weight of inhibitory inputs to the stimulus frequency
   - Changing the amplitude and peak-time of the rebound response.

Simulation



Simulated response of the mechanistic model can reproduce an Omitted Stimulus Response including its predictive timing. Simulations without the A1 unit predict a peak but do not capture the scaling with period.

Conclusions & Perspectives

The retina encodes complex and rapidly moving visual scenes via its hierarchical, multilayered structure where most neurons respond by a graded variation of their membrane potential without spiking (except ganglion cells). Here, we show that inhibition via amacrine cells can enable the retina to encode surprise by

(A) evoking a **response to a change** in the stimulus input vial **delayed inhibition** and

(B) adapting the **timing of this response** to the stimulus via **short-term plasticity**.

Besides temporal predictions on a spatially uniform stimulus, the retina has been shown to anticipate motion and to respond to changes in trajectories. Can we extrapolate the mechanism proposed here to a more general setting, where amacrine cells enable the anticipation of a moving object [4] while short-term plasticity allows to detect fast changes in its trajectory ?

References

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