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# The Role of Dynamical Synapses in Retinal Surprise Coding

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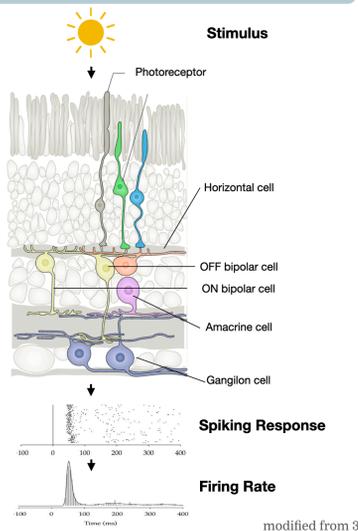
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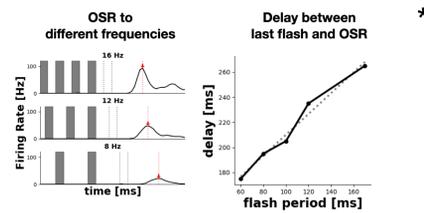
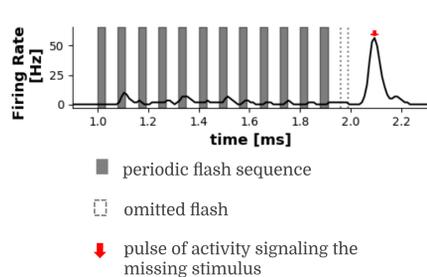
# The Role of Dynamical Synapses in Retinal Surprise Coding

## 1 Background

- The Retina transforms visual information into spike trains transmitted to the rest of the brain.
- It processes information in **parallel pathways** selectively responding to specific features of the visual input:
  - The **ON pathway** responds to bright light,
  - The **OFF pathway** responds to dark stimuli.
- The retina also contains **predictions** of its future input and detects **violations of its expectation** with high precision.
- An example of this 'surprise signal' is the **Omitted Stimulus Response (OSR)** (box 1).



### Box 1: Omitted Stimulus Response (OSR)



## 2 Motivation

The Retina can recognize patterns and signal its 'surprise' when these patterns are violated, with a high level of precision.

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

We combine **electrophysiological experiments** in which we pharmacologically **inactivate specific cell types** in the retina, to identify key components of a dynamic response to changes, with **computational modelling** to investigate the potential role and location of **short-term plasticity**.

## 3 Experiments

### Methodology

- Microelectrode Array Recordings:



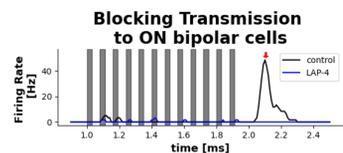
- Stimulation of a dissected retina with a sequence of periodic dark flashes.
- Extracellular spike recordings.

- Identification of necessary components via pharmacological inhibition of synaptic transmission onto:

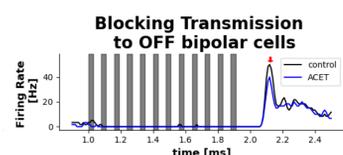


- ON bipolar cells via mGluR6 receptor antagonist LAP-4.
- OFF bipolar cells via AMPA receptor antagonist ACET.

### Results



ON BC inhibition cancels the OSR.



OFF BC inhibition has no direct effect on the OSR.

### Conclusions

- The ON pathway is required for the OSR to dark flashes whereas the OFF pathway is not directly required, but could still be activated by lateral input
- This asymmetric contribution of ON and OFF pathway could be caused by:

- a network effect via **directed lateral connections** (amacrine cells),
- an **intrinsic mechanism** specific to ON bipolar cells.

## 4 Modeling surprise coding in the Retina

### Existing Models

Gao et al., 2007, *Computation in Neural Systems*

- Existing Models explained the OSR via two antagonistic (ON-OFF) parallel pathways and a resonance mechanism in ON bipolar cells;
- This mechanism could not be experimentally verified and cannot account fully for the predictive timing to flash frequency (box 1).

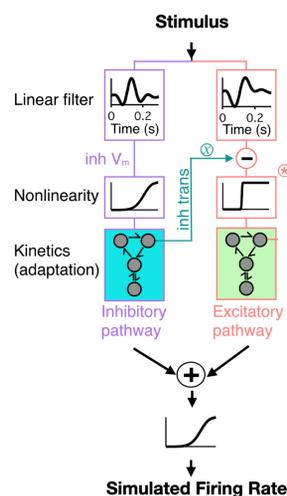


We will test a model of synaptic adaptation *in silico* to explore the hypothesis that dynamical synapses cause the rapid frequency adaptation.

### Linear-Nonlinear-Kinetic (LNK) Model

Kastner et al., 2017, *Current Biology*

- Two parallel pathways that together provide input to one ganglion cell,
- Directed lateral connection before summation,
- Short-term plasticity in bipolar cell and amacrine cell synapses.



Processing Steps:

- Step 1:** Convolution of stimulus and a temporal kernel simulates the integration of the stimulus into bipolar cell voltage.
- Step 2:** Applying a nonlinearity simulates rectification within bipolar cells.

$$u(t) = N_{LNK} \left( \int F_{LNK}(t - \tau) s(\tau) d\tau \right). \quad (1)$$

- Step 3:** Kinetics of synaptic vesicle release are simulated as a dynamical system of Markov type (box 2).

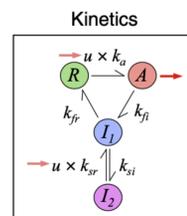
- Step 4:** Summation and second nonlinearity simulates transformation of the response into a ganglion cell output firing rate.

### Box 2: Short-term plasticity (STP)

- Synaptic vesicles can occupy 1 out of 4 different States:

- R**: readily releasable
- A**: active (released)
- I1**: inactive (empty)
- I2**: inactive (refilled)

- $P = (P_R, P_A, P_{I1}, P_{I2})$  are probabilities to occupy each state



- Transitions** between states are controlled by **rate constants**:

- $k_a$ : vesicle activation
- $k_{fi}, k_{si}$ : fast/slow inactivation
- $k_{fr}, k_{sr}$ : fast/slow recovery

- fixed or **vary in time**, scaled by bipolar cell voltage  $u(t)$  (1).

$$\frac{dP^T(t)}{dt} = P^T(t) * \begin{pmatrix} -u(t) * k_a & u(t) * k_a & 0 & 0 \\ 0 & -k_{fi} & -k_{si} & 0 \\ k_{fr} & 0 & -(k_{fr} + k_{si}) & k_{si} \\ 0 & 0 & u(t) * k_{sr} & -u(t) * k_{sr} \end{pmatrix}$$

The change in probability is described by a set of **first-order differential equations**

The **output** of the synapse will be determined by the probability of the **active state**,  $P_{A(t)}$

## 5 Perspectives

Can the predictive latency of the OSR be explained via the model of STP?

Can a similar mechanism, embedded in a **large scale network** account for **more complex pattern recognition**, such as motion reversal?

We aim to build new hypothesis at the limits of biological experiments & understand computational paradigms at work.

In future we will experimentally test if:

- A lateral amacrine connection is involved in the OSR
- The OFF pathway contributes indirectly to the OSR

## 6 Acknowledgements

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- All experiments were performed in the team of Olivier Marre at Institut de la Vision.

## 6 References

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