



**HAL**  
open science

# Temporal Pattern Recognition in Retinal Ganglion Cells is mediated by Dynamical Inhibitory Synapses

Simone Ebert, Thomas Buffet, Berat Semihchan Sermet, Olivier Marre, Bruno  
Cessac

► **To cite this version:**

Simone Ebert, Thomas Buffet, Berat Semihchan Sermet, Olivier Marre, Bruno Cessac. Temporal Pattern Recognition in Retinal Ganglion Cells is mediated by Dynamical Inhibitory Synapses. ERM 2023 - European Retina Meeting 2023, Sep 2023, Tuebingen, Germany. , 2023. hal-04351175

**HAL Id: hal-04351175**

**<https://hal.science/hal-04351175>**

Submitted on 18 Dec 2023

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

# Temporal Pattern Recognition in Retinal Ganglion Cells is mediated by Dynamical Inhibitory Synapses

Simone Ebert<sup>1</sup>, Thomas Buffet<sup>2</sup>, B. Semihcan Sermet<sup>3</sup>, Olivier Marre<sup>2</sup>, Bruno Cessac<sup>1</sup>

<sup>1</sup> Université Côte d'Azur, Inria Biovision Team and Neuromod Institute, Biot, France

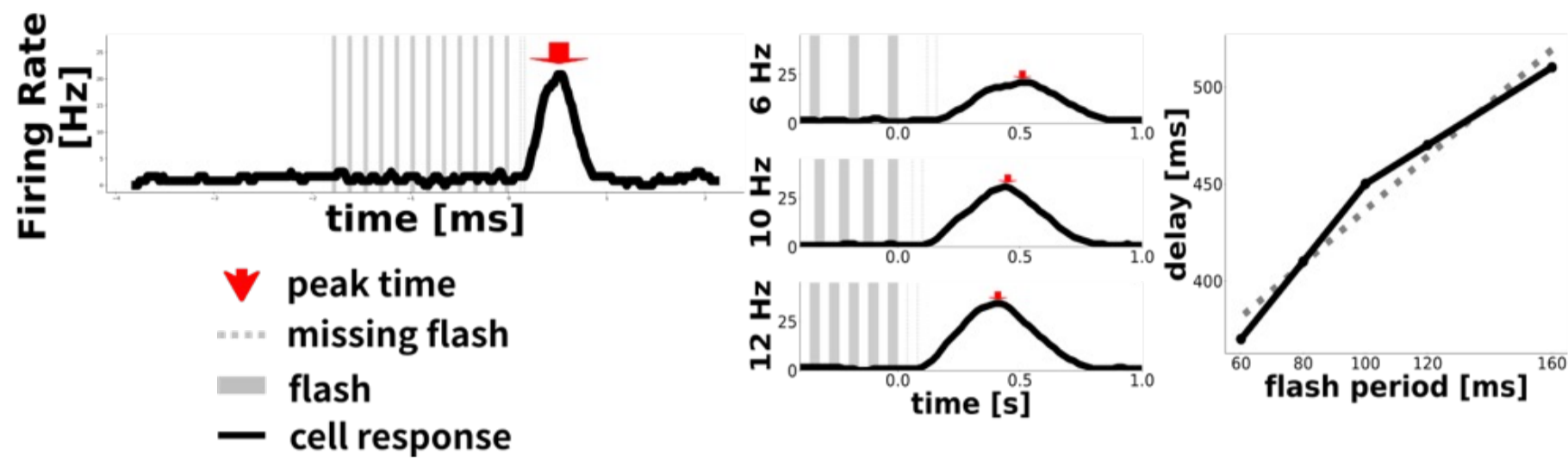
<sup>2</sup> Sorbonne Université, INSERM, CNRS, Institut de la Vision, Paris, France

<sup>3</sup> Institut Pasteur, Paris, France



## Abstract

The retina emits an Omitted Stimulus Response (OSR) [2] when an expected flash in a sequence is missing. The latency of this response scales with the period of the stimulus 1-to-1, thus signalling the time-point when the missing stimulus was expected. This is an example for predictive coding in the retina, with a temporal expectation of future stimuli.

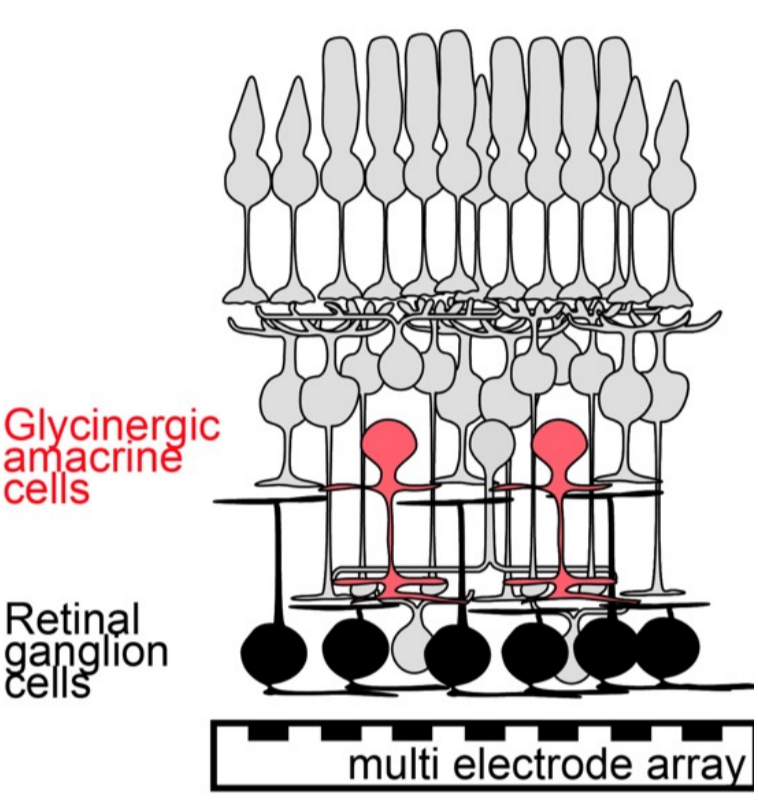


What is the underlying mechanism of the latency shift in the OSR?

## Glycinergic amacrine cells are necessary for latency shift

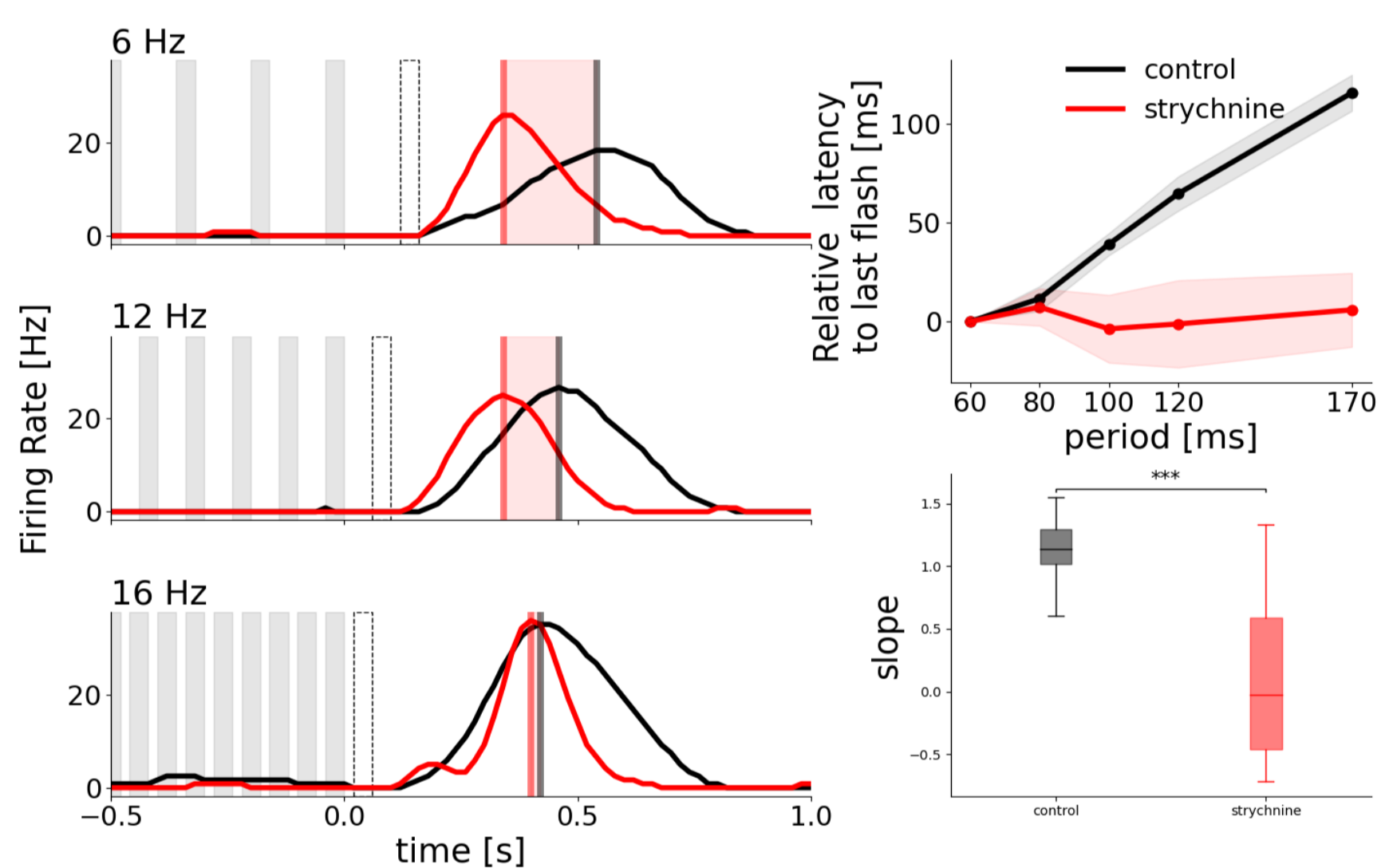
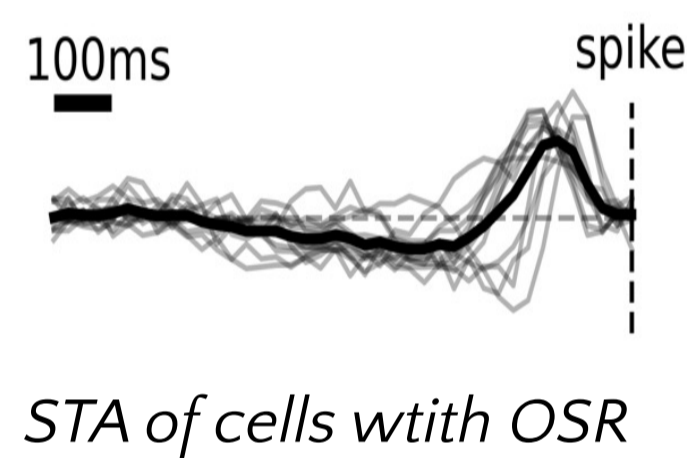
### Experimental Setup

- Electrophysiological recordings to stimulation with dark flashes of different frequencies
- pharmacological blockade of glycinergic transmission.



## Results

- ON cells exhibit an OSR to dark flashes
- Glycinergic Amacrine cells are necessary to form temporal expectations.



## Computational model

- Convolution of the stimulus  $s(t)$  and a temporal kernel  $\alpha_X$

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

- excitatory (filled circle)
  - inhibitory (open circle)
  - dynamic (circle with dot)
- where  $X = E^{ON}, I^{ON}, I_{Gly}^{OFF}$ .

- Signal integration via a dynamical system where  $V_X$  is the voltage of cell  $X$  with characteristic time  $\tau_X$

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

- A RGC pools over the network where each input has a synaptic weight  $w_X$

$$\frac{dV_G}{dt} = -\frac{V_G}{\tau_G} + \sum w_X p(V_X, \theta_X).$$

- Short-term depression in glycinergic amacrine cells by modulation of  $w_{Gly}^{OFF}$  via a dynamic variable  $n$

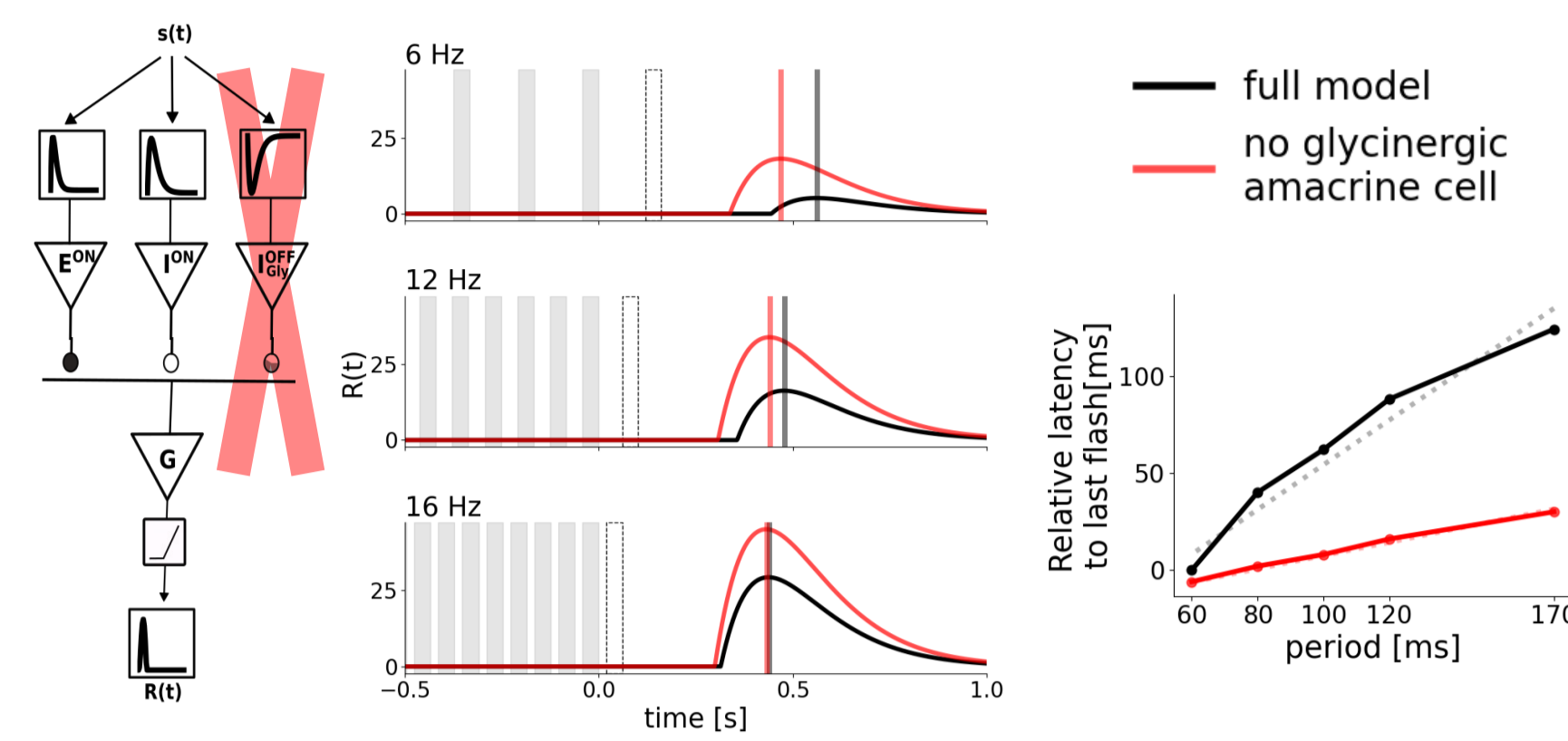
$$w_{Gly}^{OFF}(t) = n(t) \bar{w}_{Gly}^{OFF}$$

which obeys the kinetic equation [3]

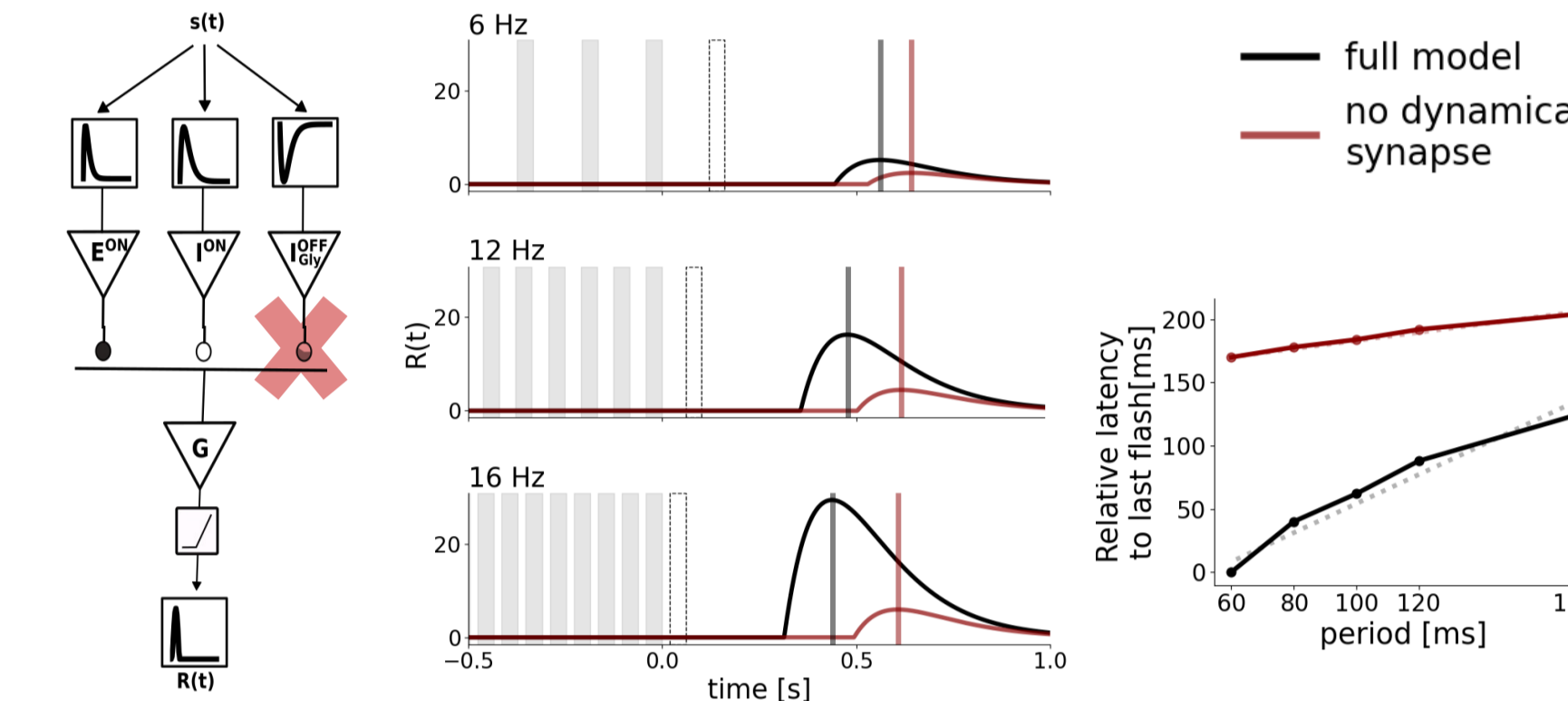
$$\frac{dn}{dt} = (1-n) k_{rec} - \beta k_{rel} p(V_{Gly}^{OFF}, \theta_{Gly}^{OFF}) n.$$

## Model simulations show latency shift

### Computational model replicates experimental finding

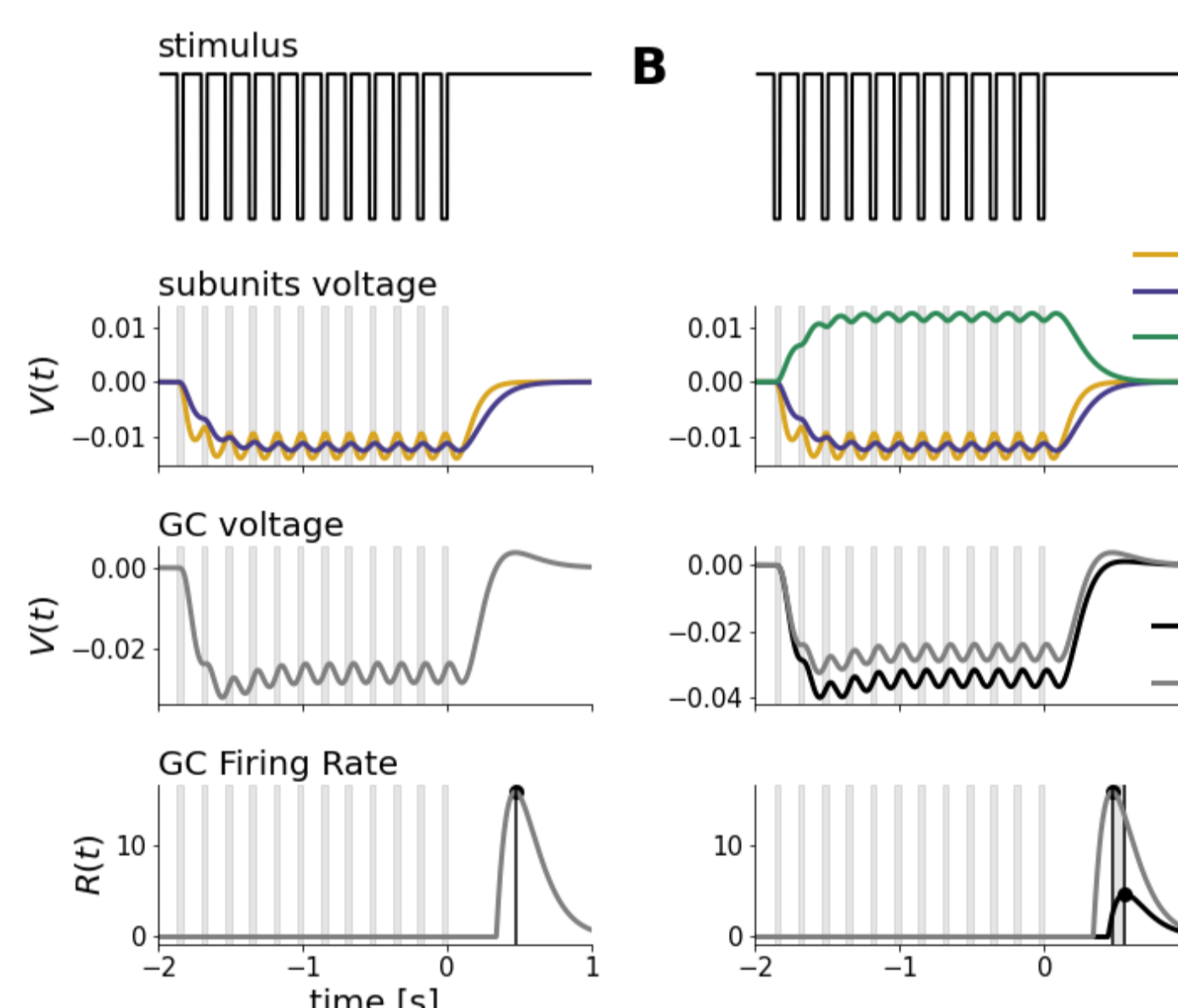


### Removing synaptic plasticity removes latency scaling



## Peak generation via disinhibition

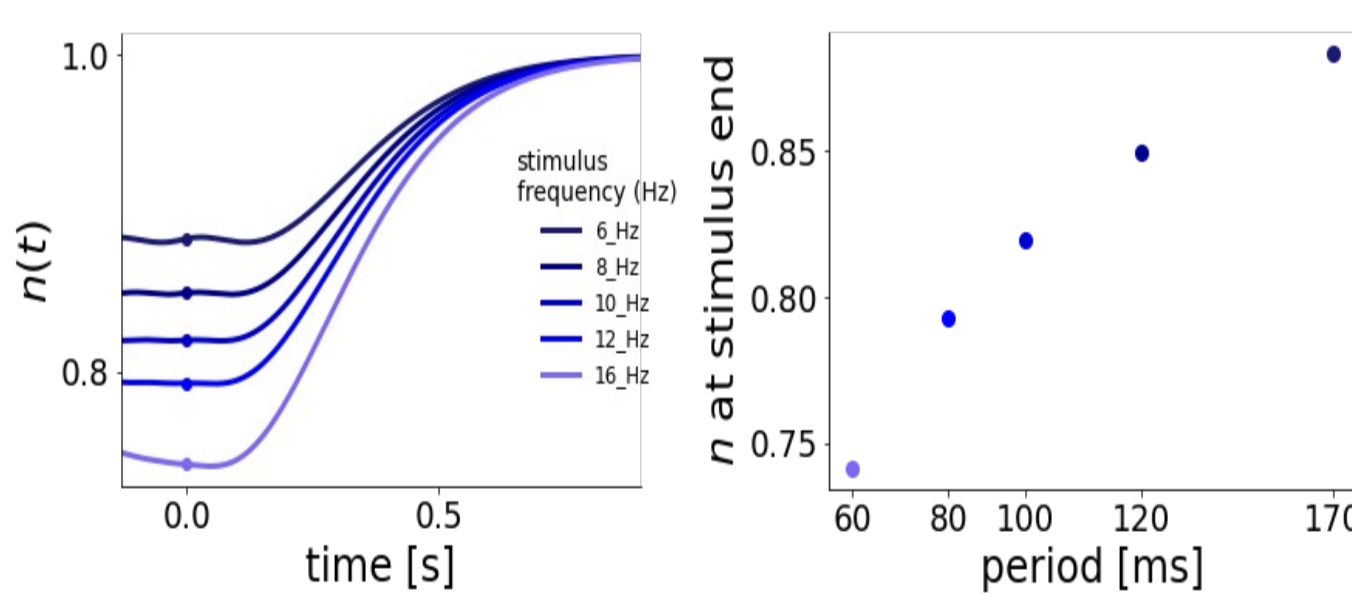
### Internal variables of the model



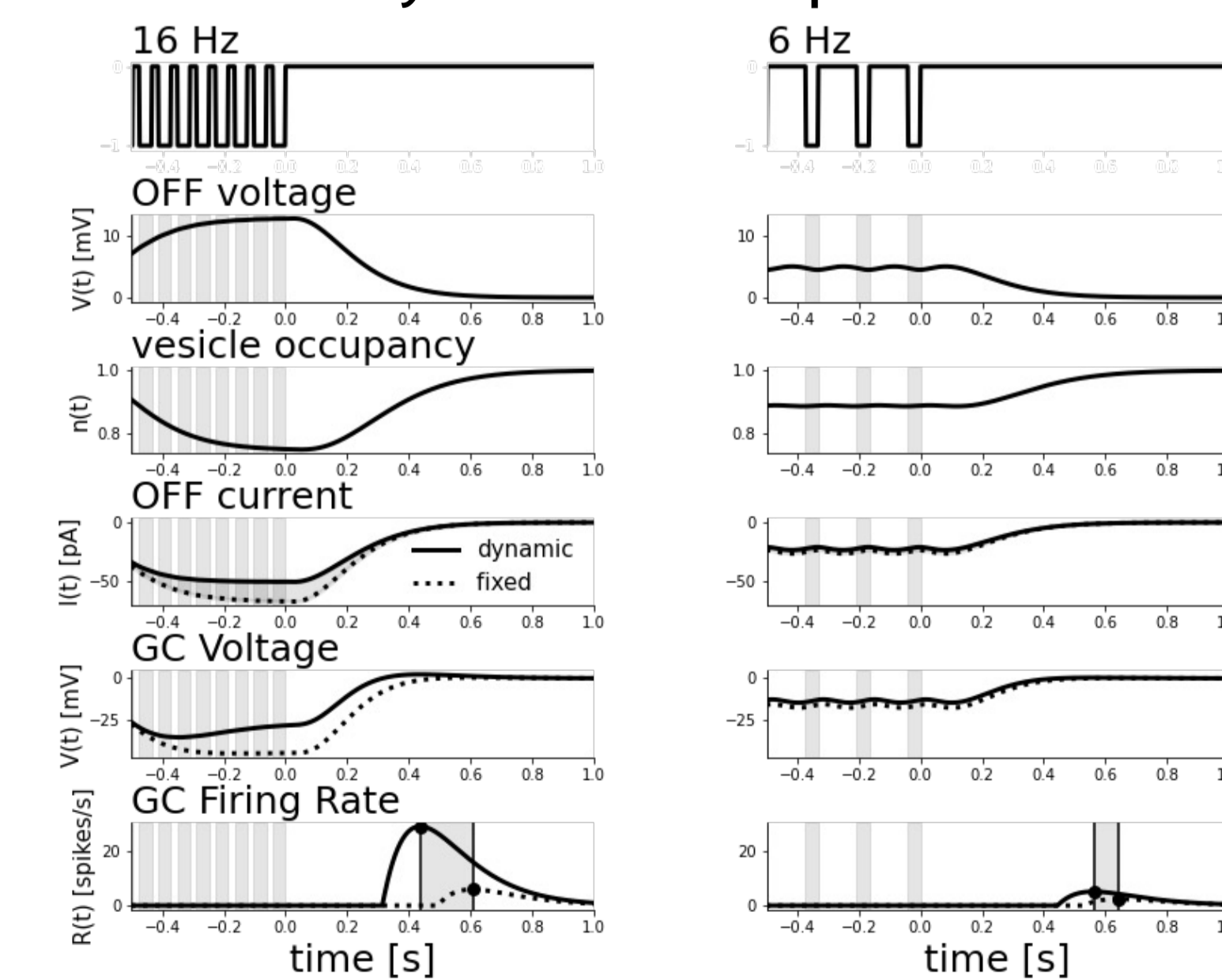
- error signal via disinhibition from ON components
- glycinergic OFF input shifts the latency backwards

## Latency scaling via synaptic depression

### Synaptic strength $n(t)$ scales with stimulus frequency



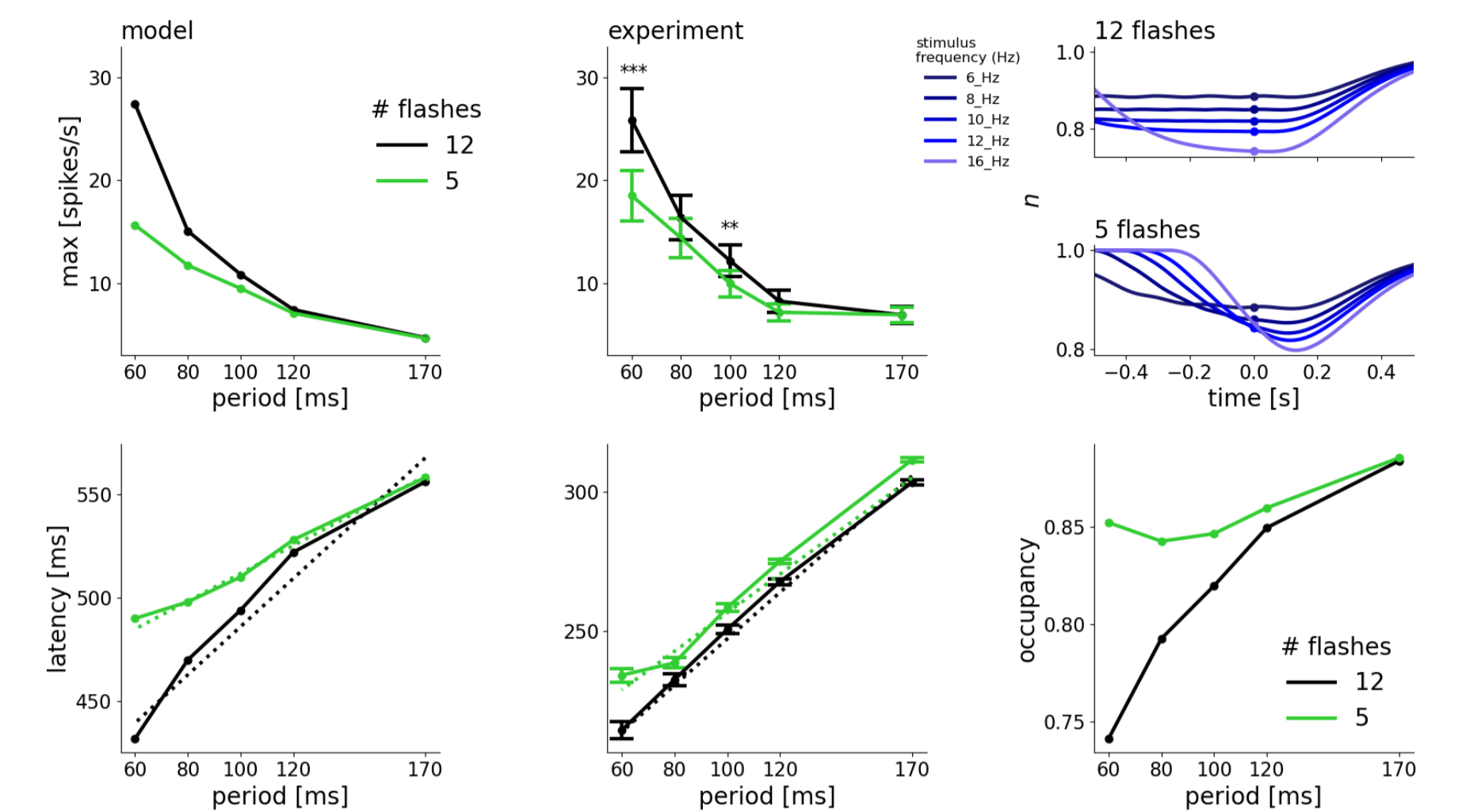
### $n(t)$ scales latency shift of the response



- Depression reduces inhibition and thus latency shift
- Higher depression for fast frequency yields shorter a latency
- Little depression for slow frequency yields longer latency

## OSR depends on the number of flashes in the stimulus

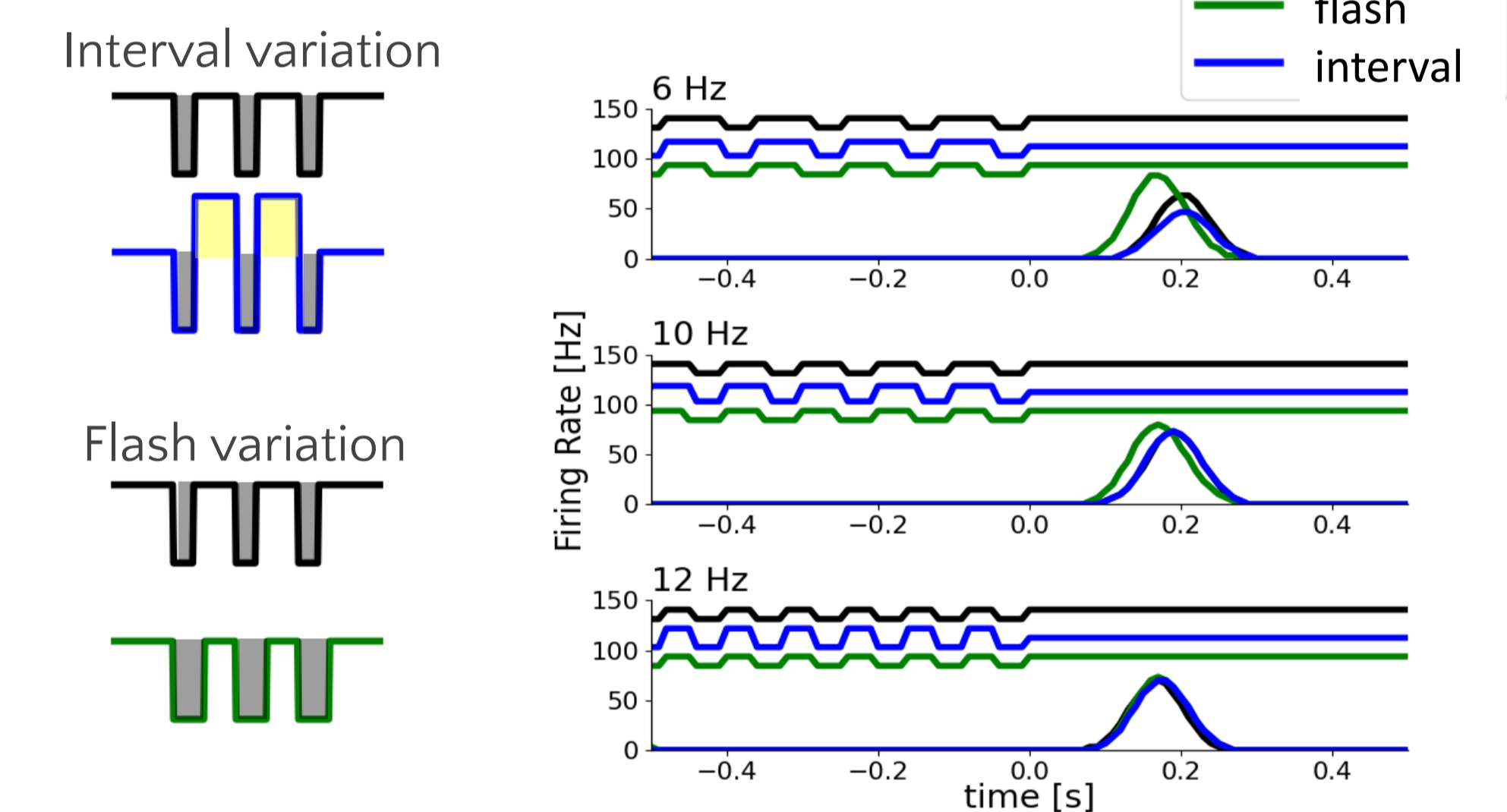
### OSR to 12 and to 5 Flashes



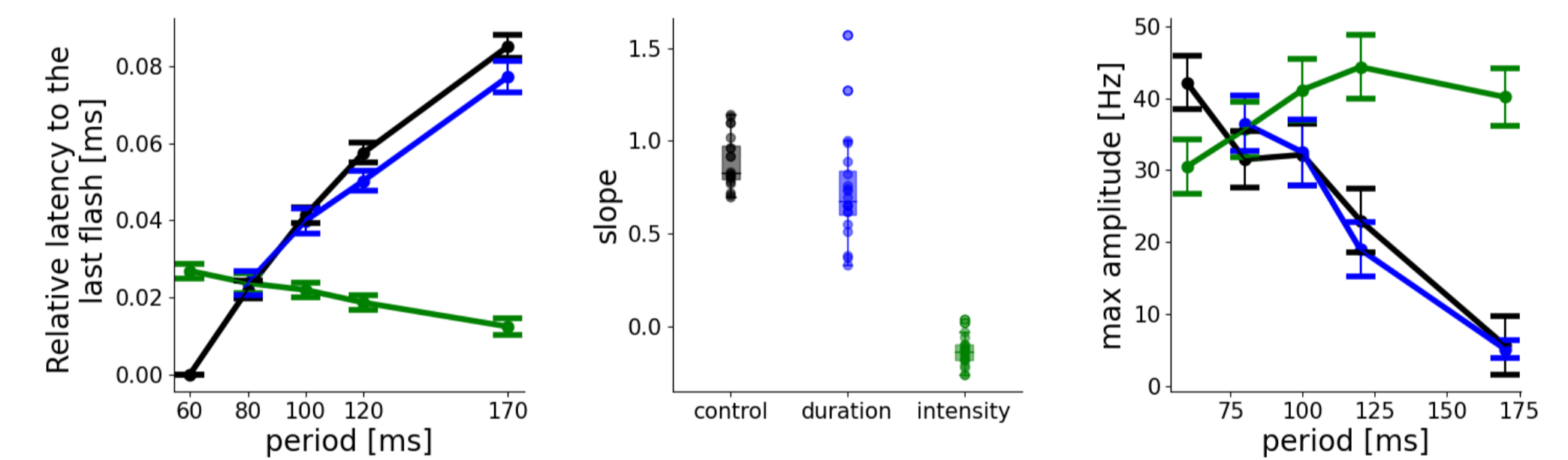
- Latency shift flattens for shorter stimuli
- In the model, this is due to lacking steady state of synaptic strength  $n(t)$

## Does average luminance effect the OSR?

### OSR to stimulus variations without average luminance decrease



- The latency shift remains unchanged when the intervals between flashes are brighter
- Latency shift is lost when the flashes change duration with frequency.



## Conclusion

The retina encodes complex and rapidly moving visual scenes via its hierarchical, multi-layered structure where most neurons respond by a graded variation of their membrane potential without spiking (except ganglion cells). Here, we show that the retina

- forms error signals via excitation and slower inhibition
- while short-term plasticity refines temporal predictions of future stimuli.

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 enables a temporal refinement of speed estimation?

## Acknowledgements

This work has been funded by the Neuromod Institute, Université Côte d'Azur.

## References

- Hosoya, T., Baccus, S. A., and Meister, M. (2005). Nature. 436:71–77.
- Schwartz, G., Harris, R., Shrom, D., and Berry, M. J. (2007a). Nat Neurosci, 10:552–554.
- Hennig, M. (2013) Frontiers in Computational Neuroscience, 10.3389/fncom.2013.00045.
- Souihel, S., Cessac B. (2021). Journal of Mathematical Neuroscience ,10.1186/s13408-020-00101-z.