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

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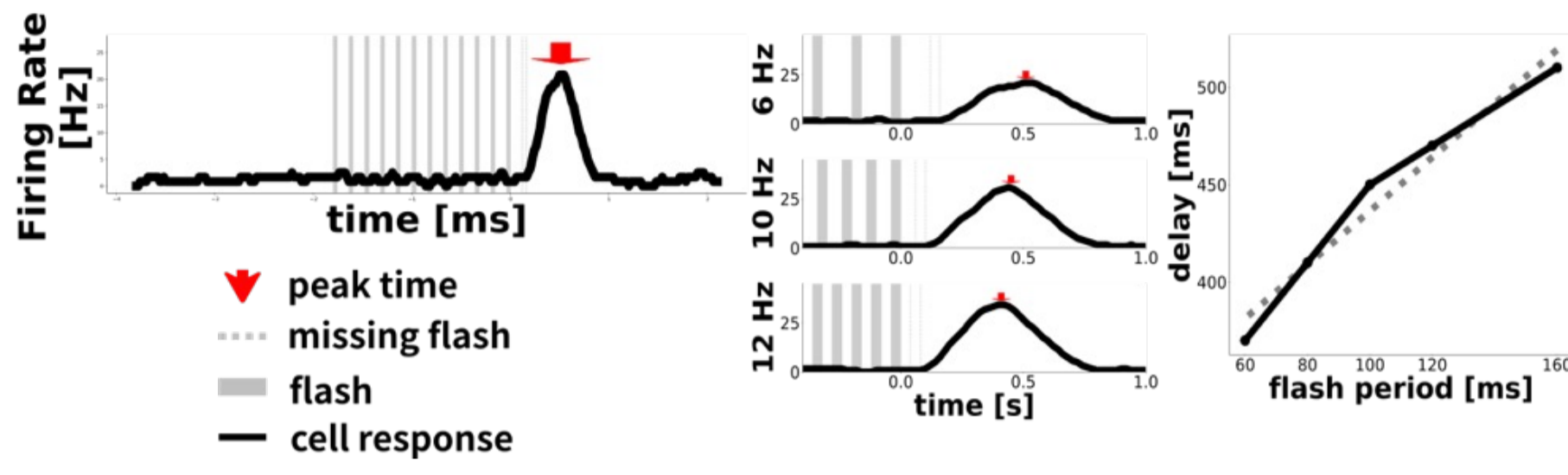
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## Abstract

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.

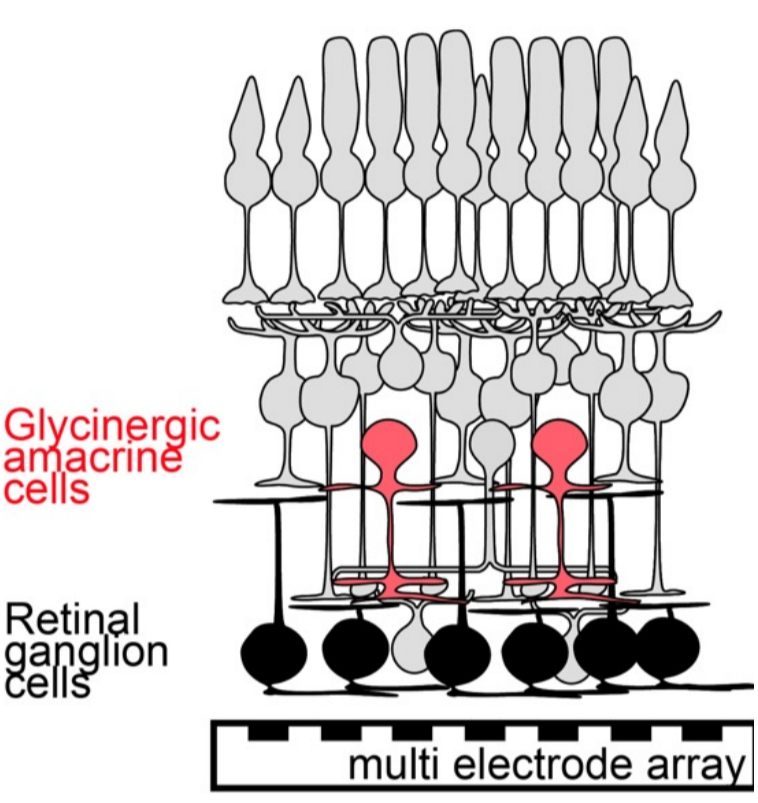


What is the underlying mechanism of the latency shift in the OSR?

## Glycinergic amacrine cells are necessary for latency shift

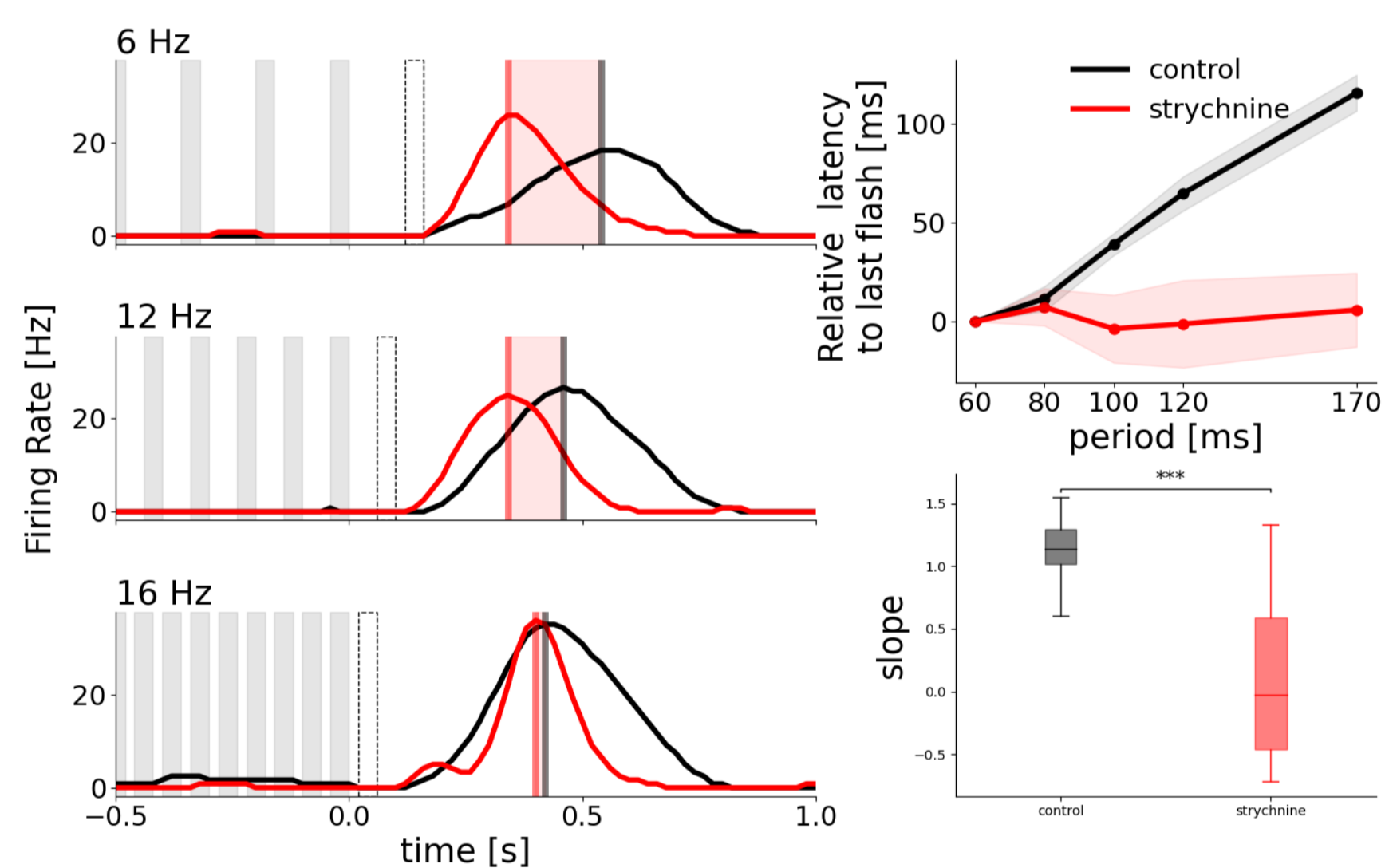
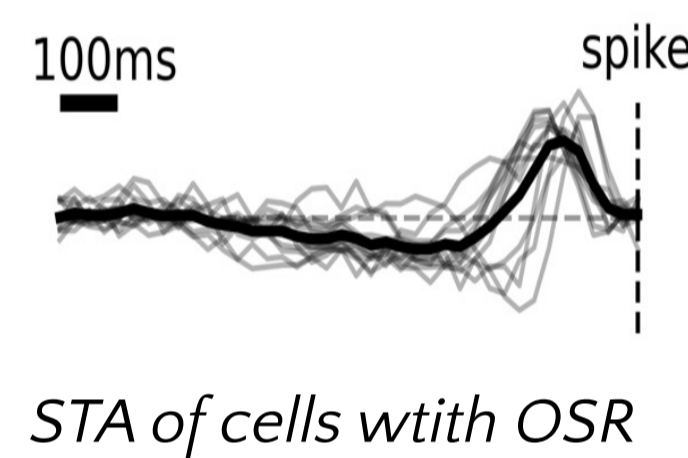
### Experimental Setup

- 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
  - inhibitory
  - dynamic
- 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  $n$

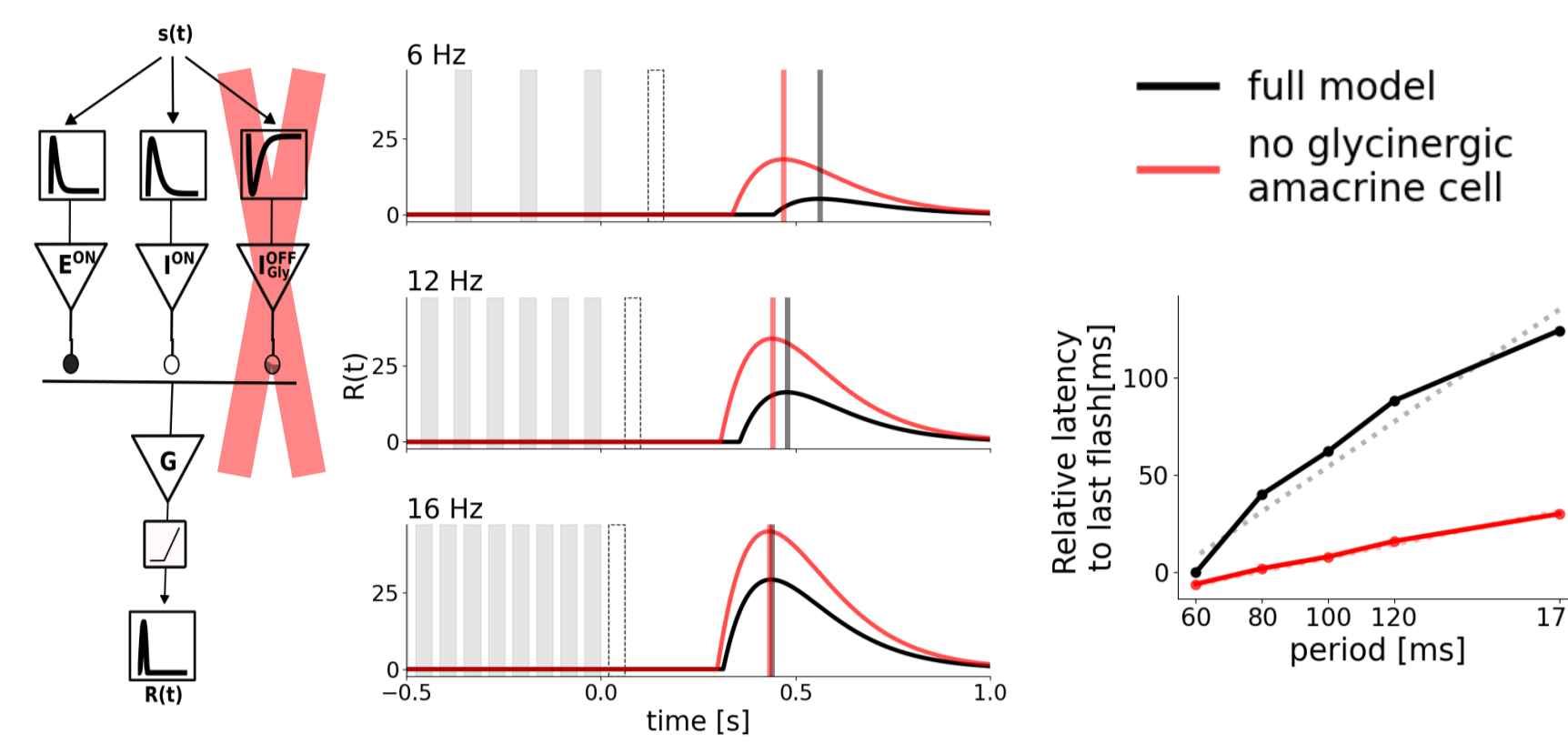
$$w_{Gly}^{OFF}(t) = n(t) \bar{w}_{Gly}^{OFF}$$

which obeys the kinetic equation [3]

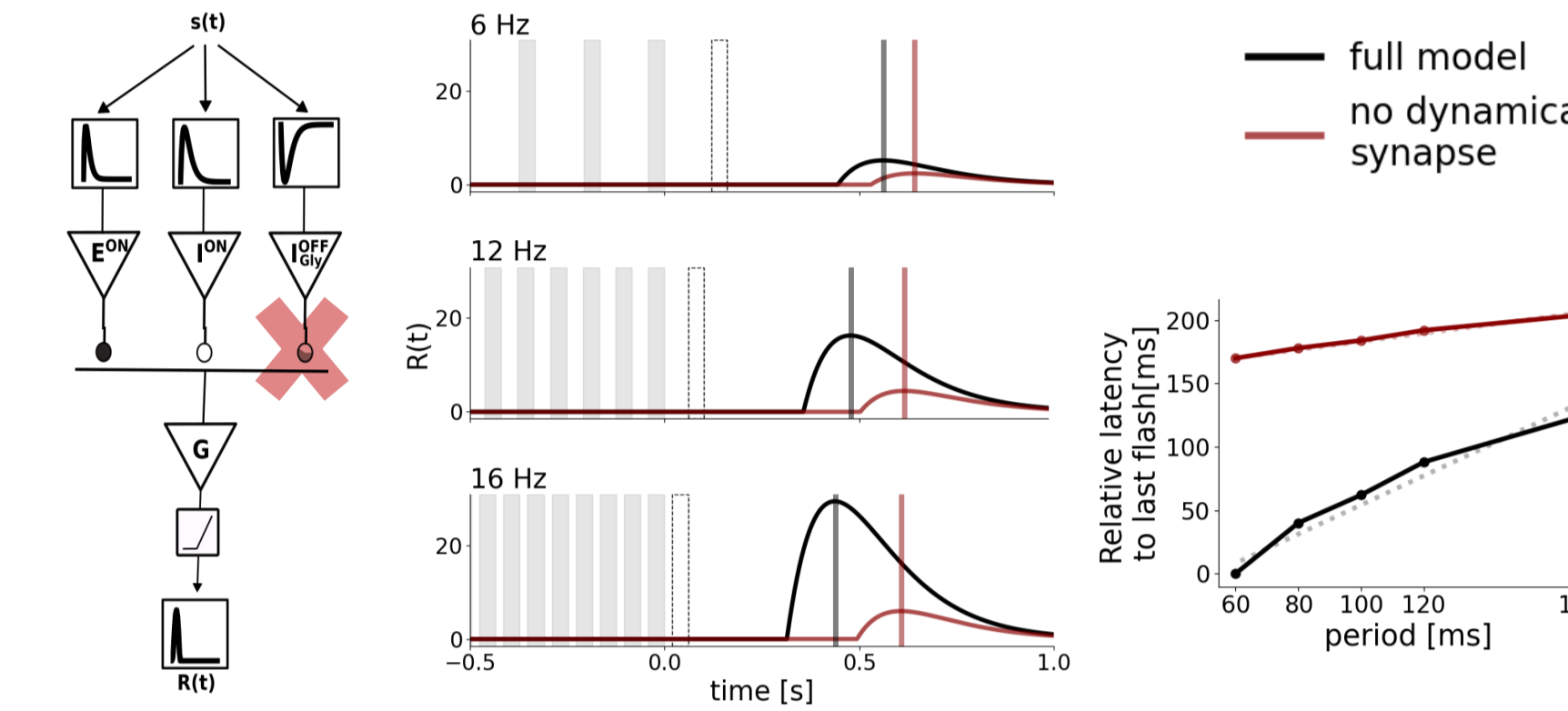
$$\frac{dn}{dt} = (1-n) k_{rec} - \beta k_{rel} p(V_{Gly}^{OFF}, \theta_{Gly}^{OFF}) n.$$

## Model simulations show latency shift

### Computational model replicates experimental finding

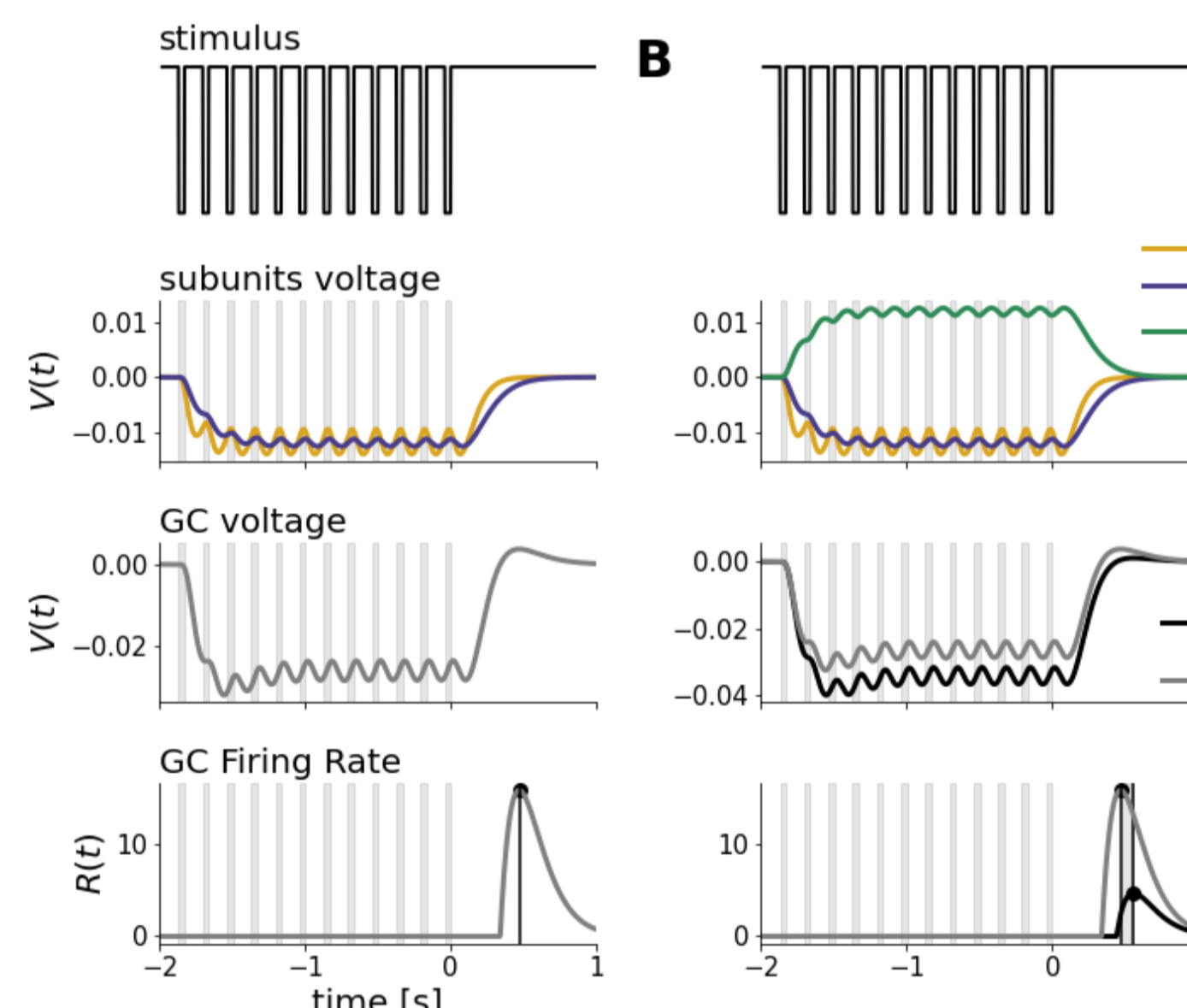


### Removing synaptic plasticity removes latency scaling



## Peak generation via disinhibition

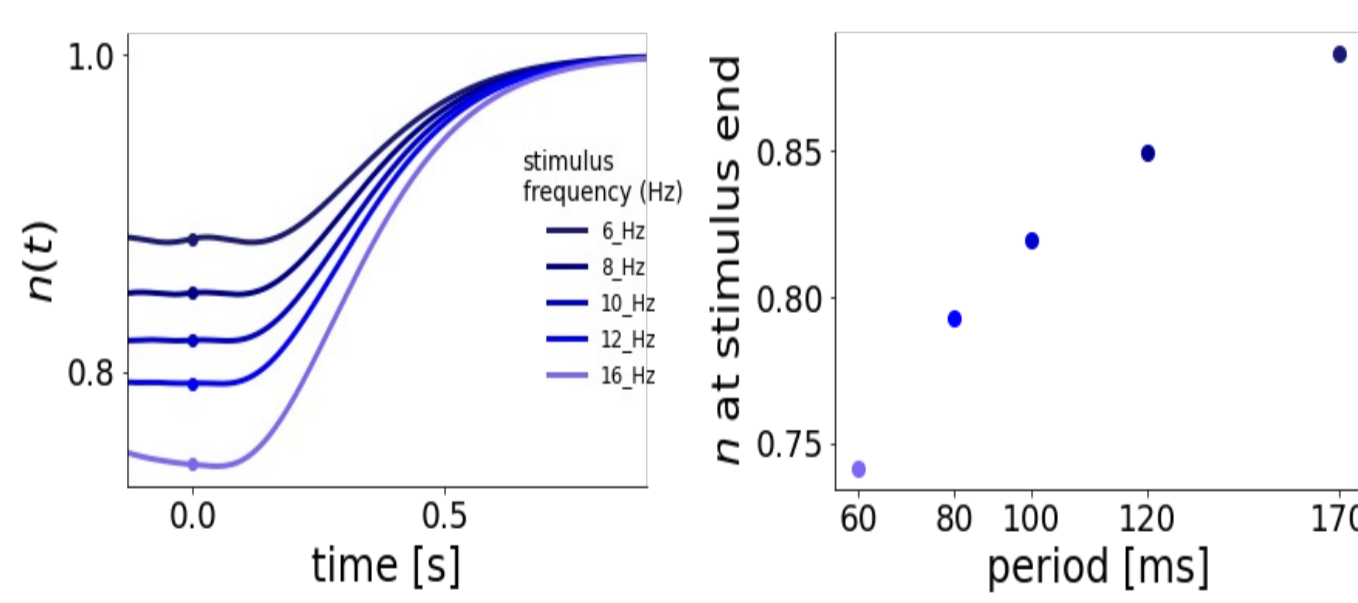
### Internal variables of the model



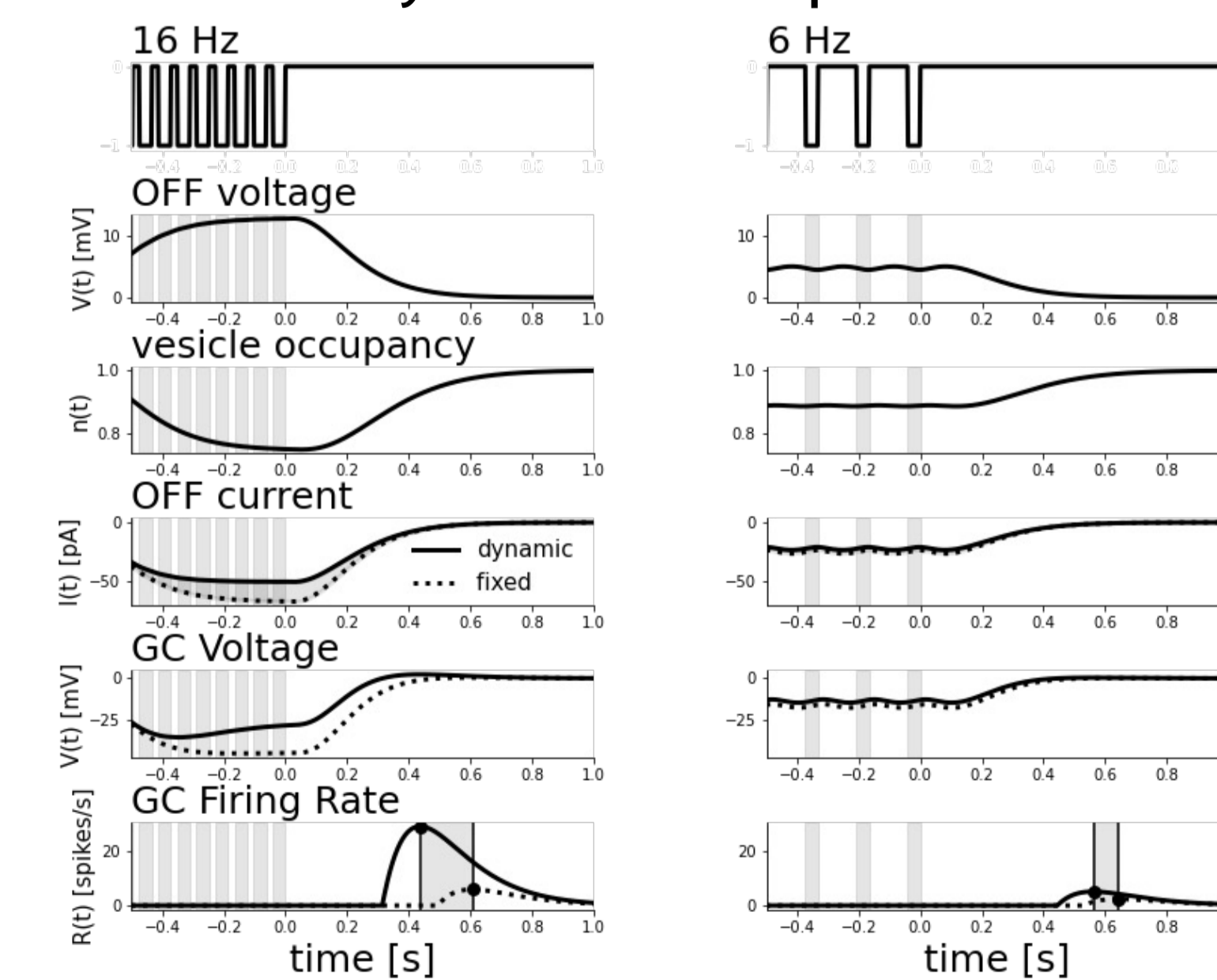
- error signal via disinhibition from ON components
- glycinergic OFF input shifts the latency backwards

## Latency scaling via synaptic depression

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



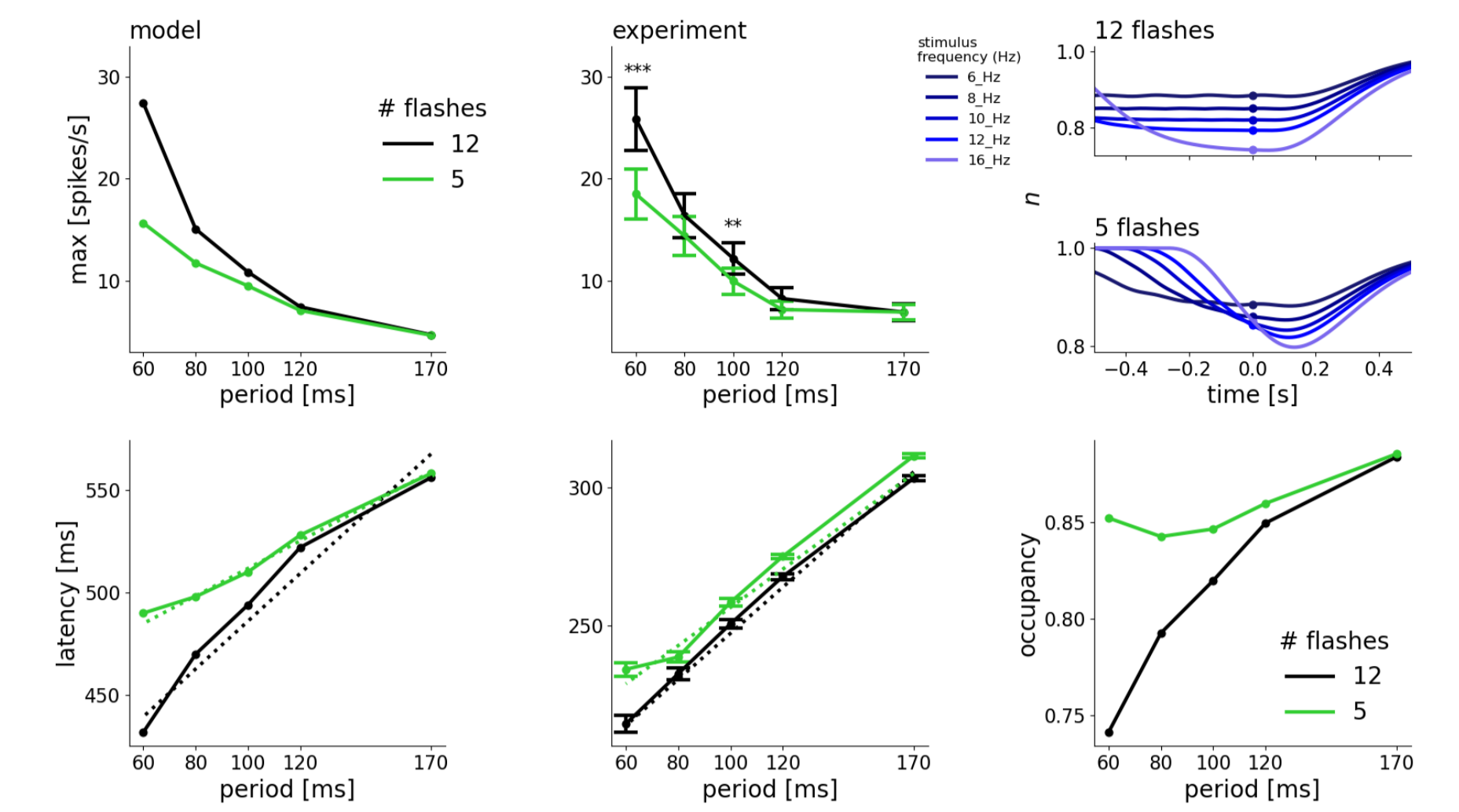
### $n(t)$ scales latency shift of the response



- 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

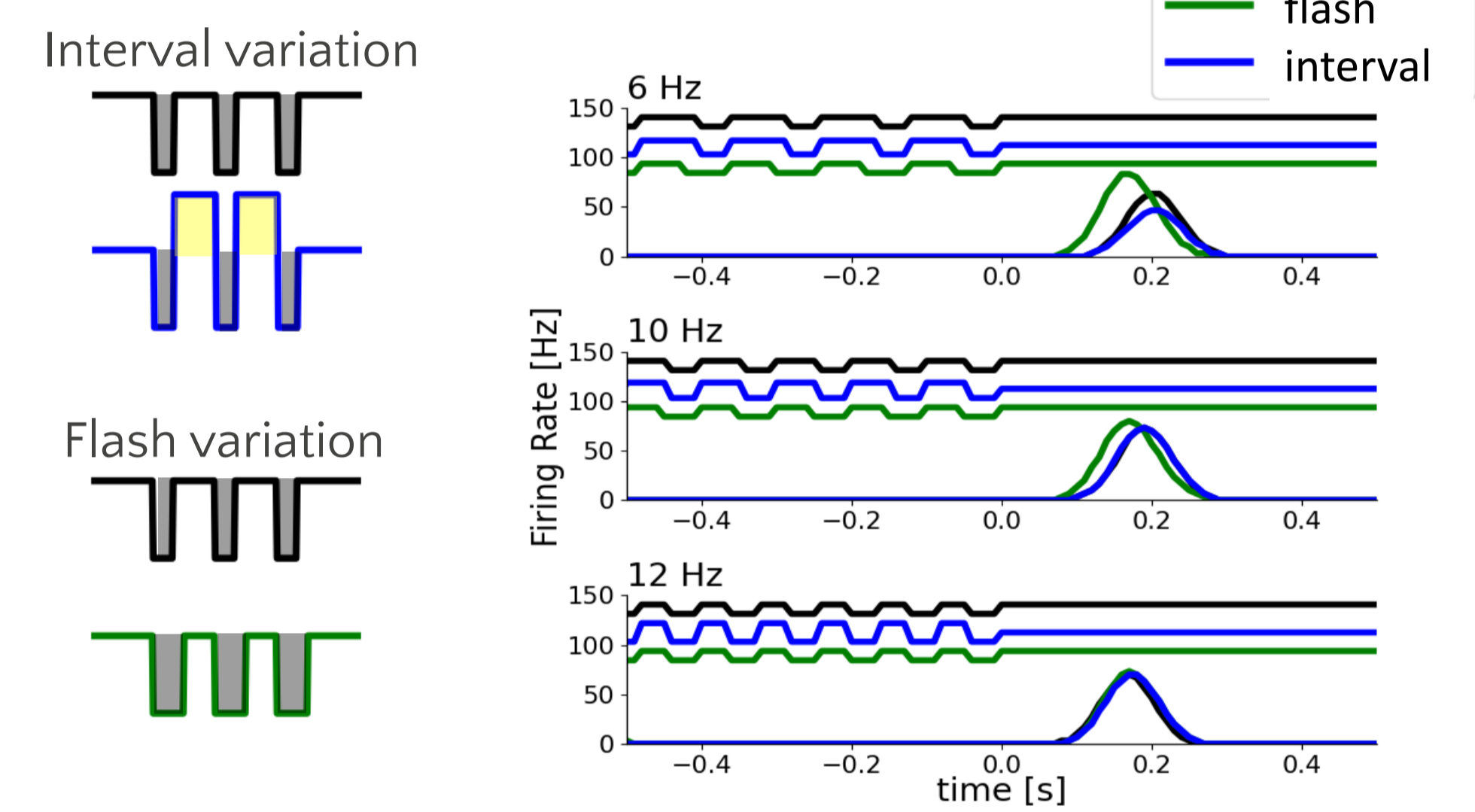
### OSR to 12 and to 5 Flashes



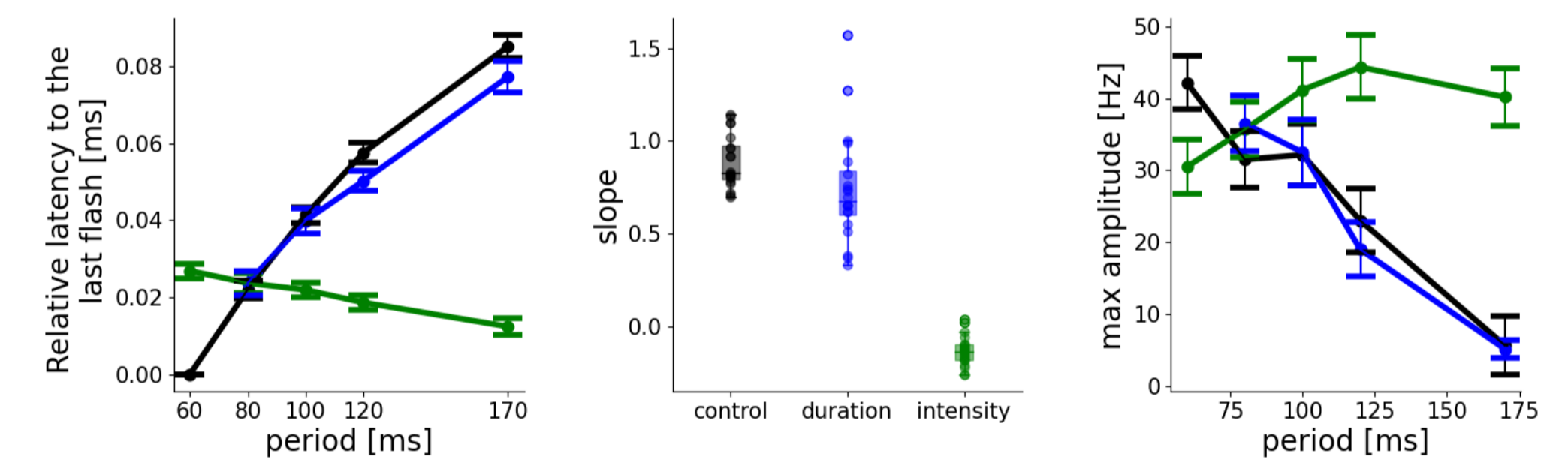
- Latency shift flattens for shorter stimuli
- In the model, this is due to lacking steady state of synaptic strength  $n(t)$

## 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

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## References

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