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



Simone Ebert¹, Thomas Buffet², B.Semihchan Sermet³, Olivier Marre², Bruno Cessac¹

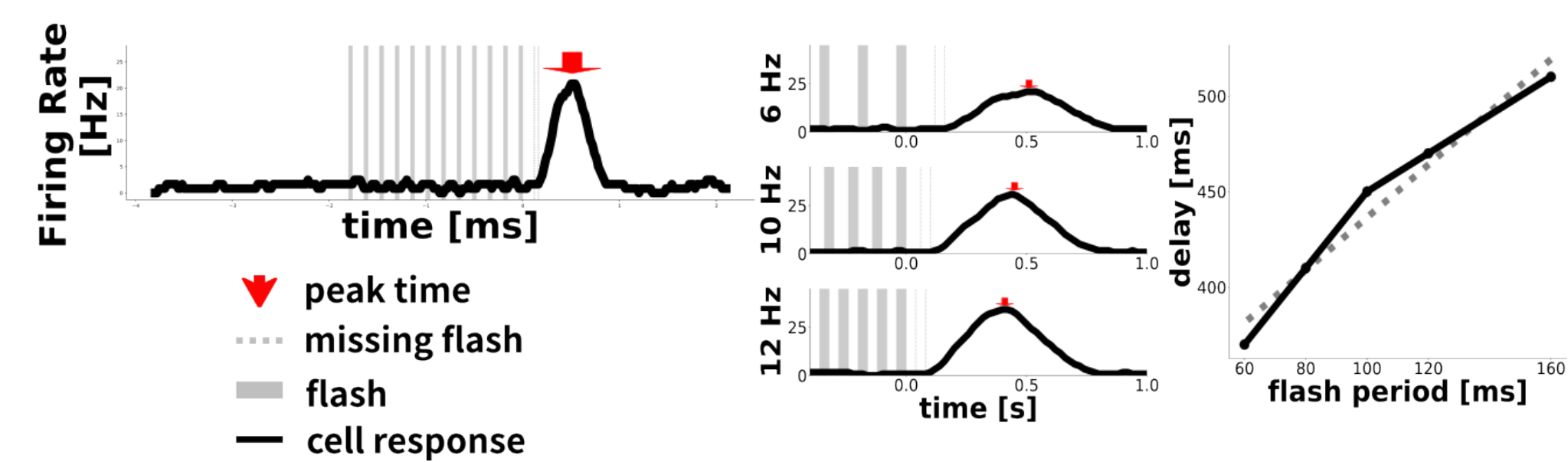
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Abstract

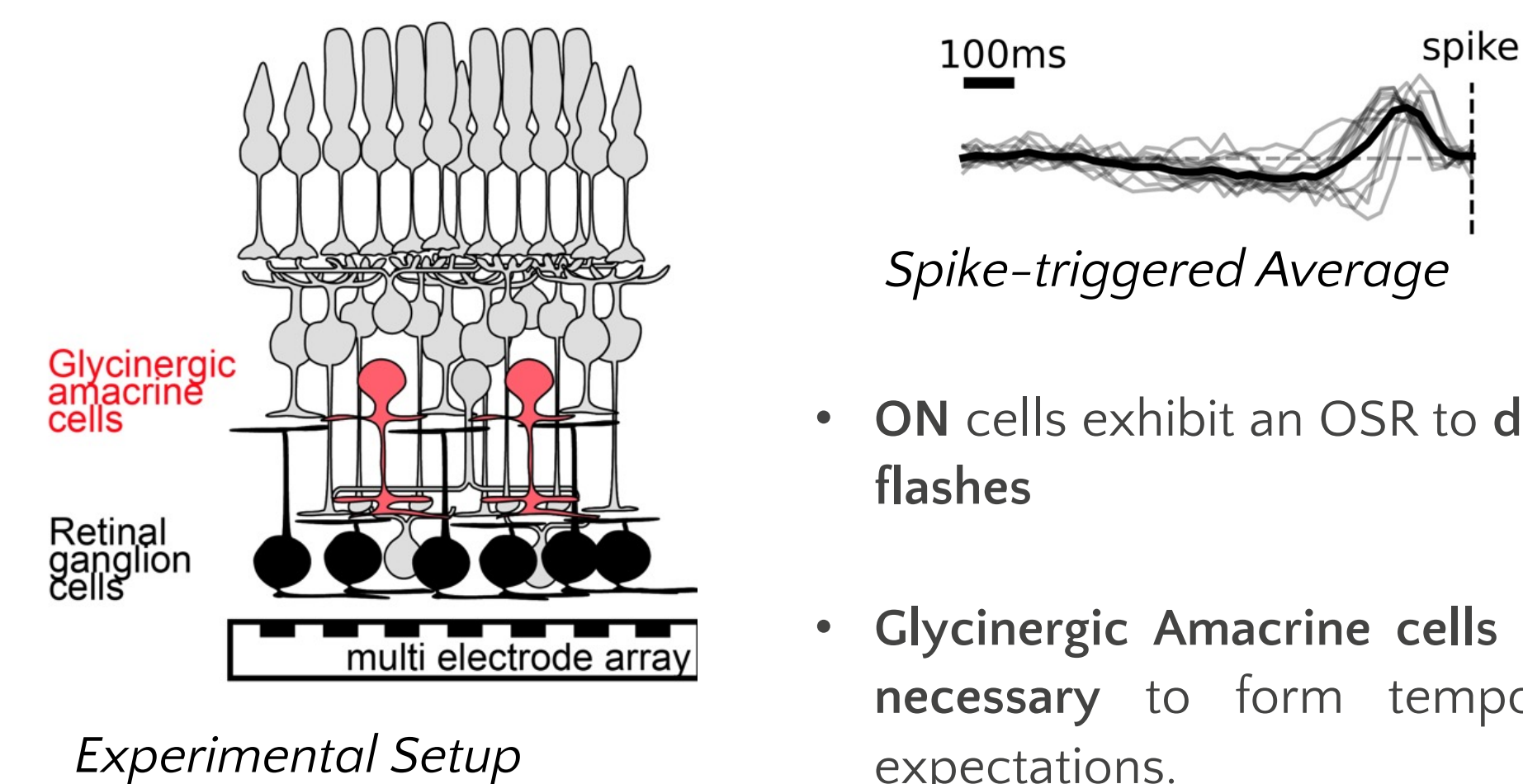
A fundamental task for the brain is to generate predictions of future sensory inputs, and signal errors when these predictions are not matched. To this end, a long-standing hypothesis is that retinal ganglion cells (RGCs), the retinal output, do not signal the visual scene per se, but only surprising events [1].



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.

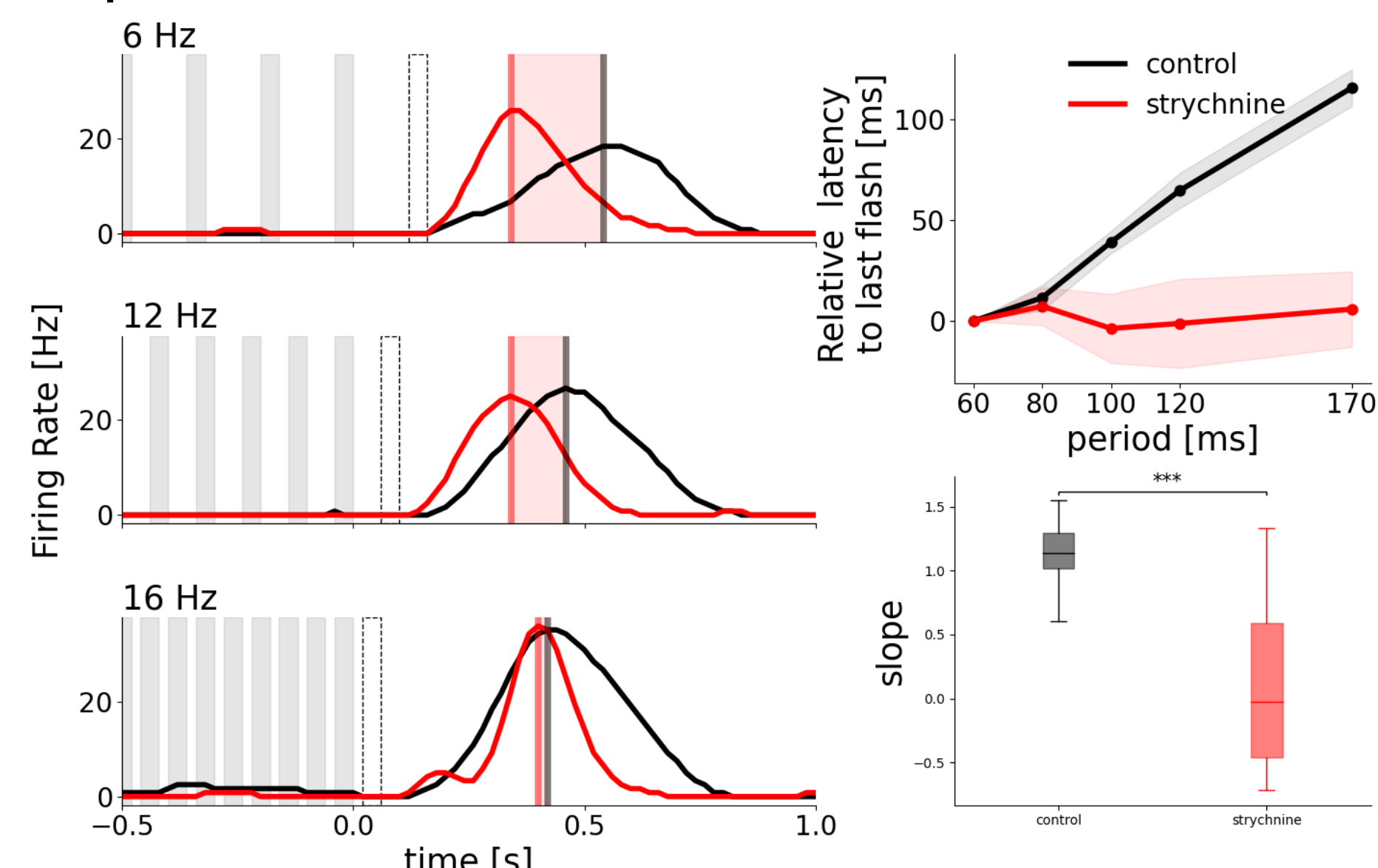
Experimental Approach

We electrophysiologically record of RGCs during stimulation with dark flashes of different frequencies. Pharmacological compounds selectively block transmission onto glycinergic amacrine cells (inhibitory).



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

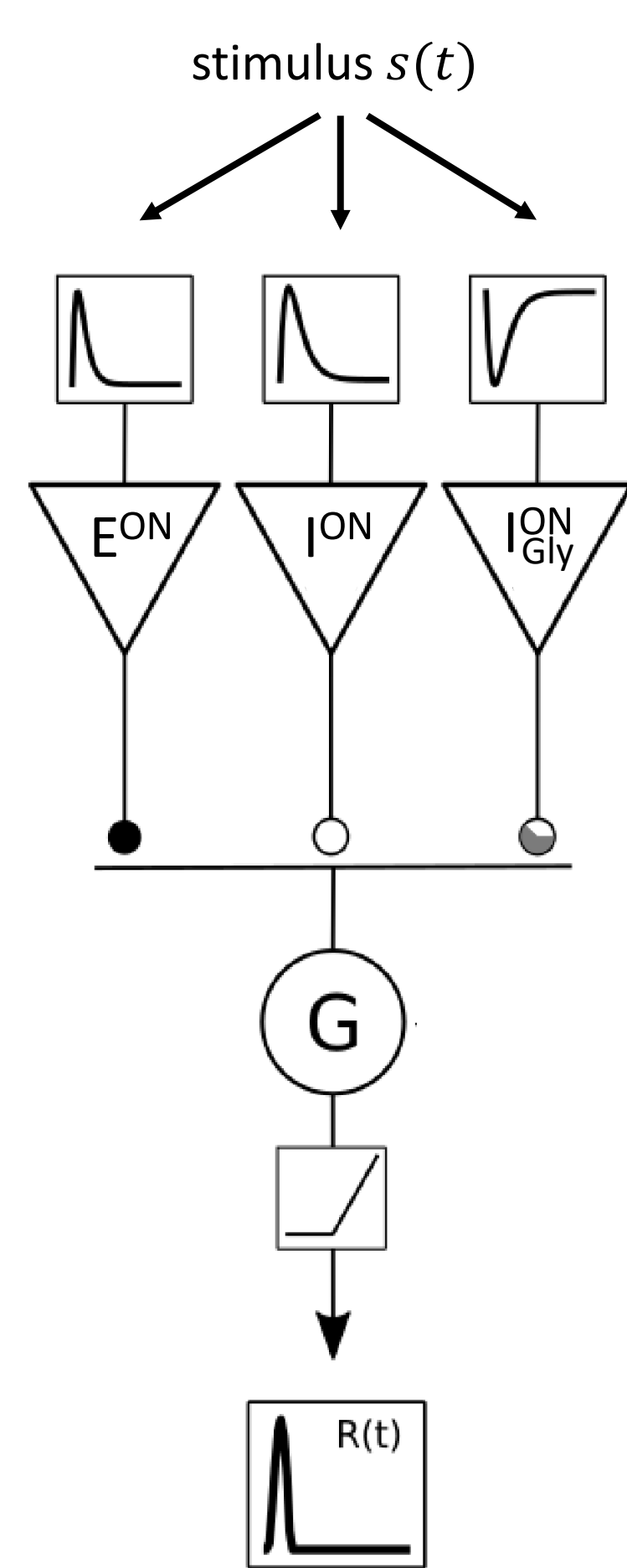
Experimental Result



Glycinergic amacrine cells are necessary for latency scaling of the OSR.

Mechanistic Model

Model Description



Schematic description of the model

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

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

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 τ_X

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

- A Retinal Ganglion Cell pools over the network where each input has a synaptic weight w_X

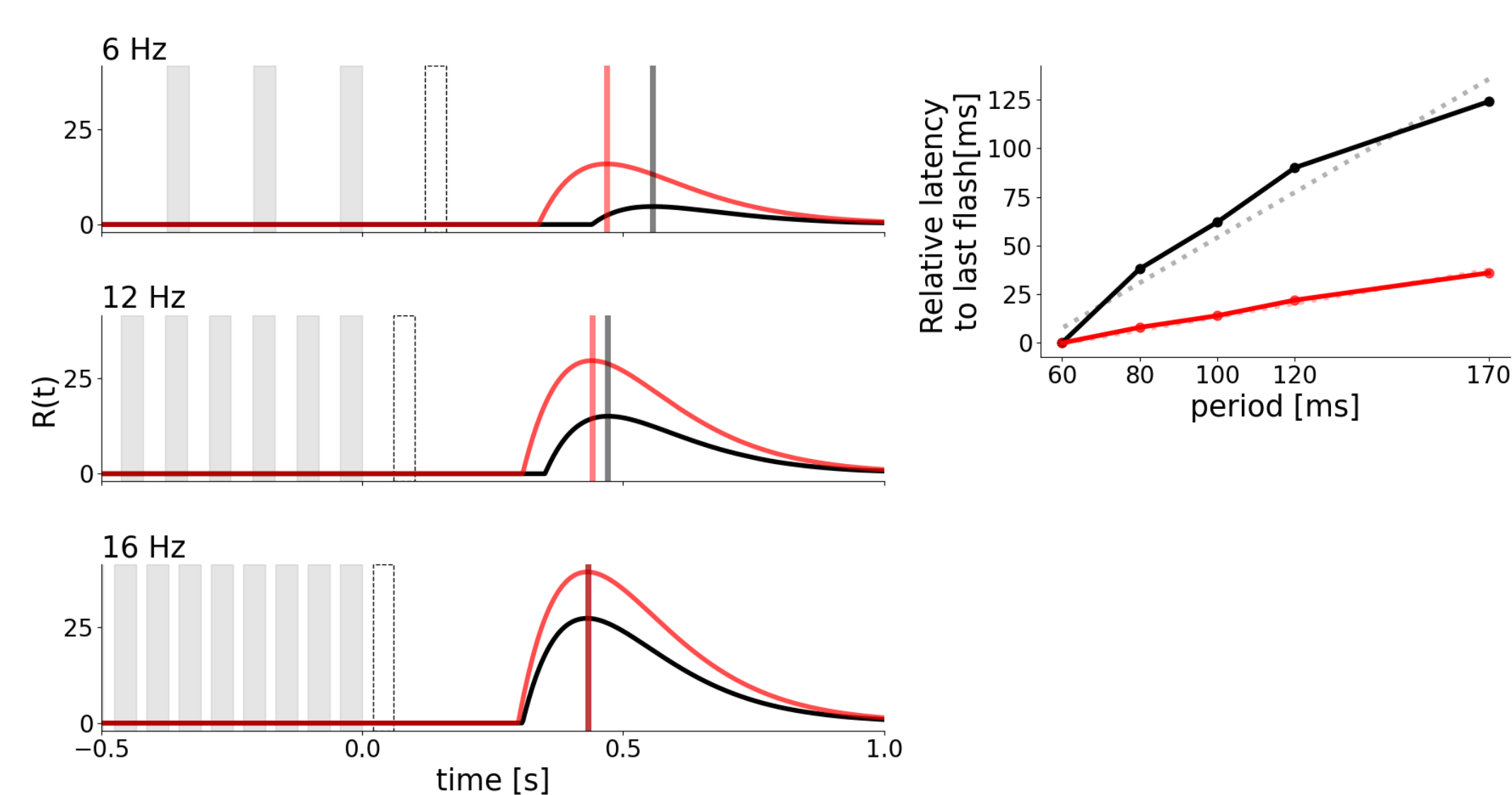
$$\frac{dV_G}{dt} = -\frac{V_G}{\tau_G} + \sum w_X p(V_X, \theta_X)$$

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

which obeys the kinetic equation [3]

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

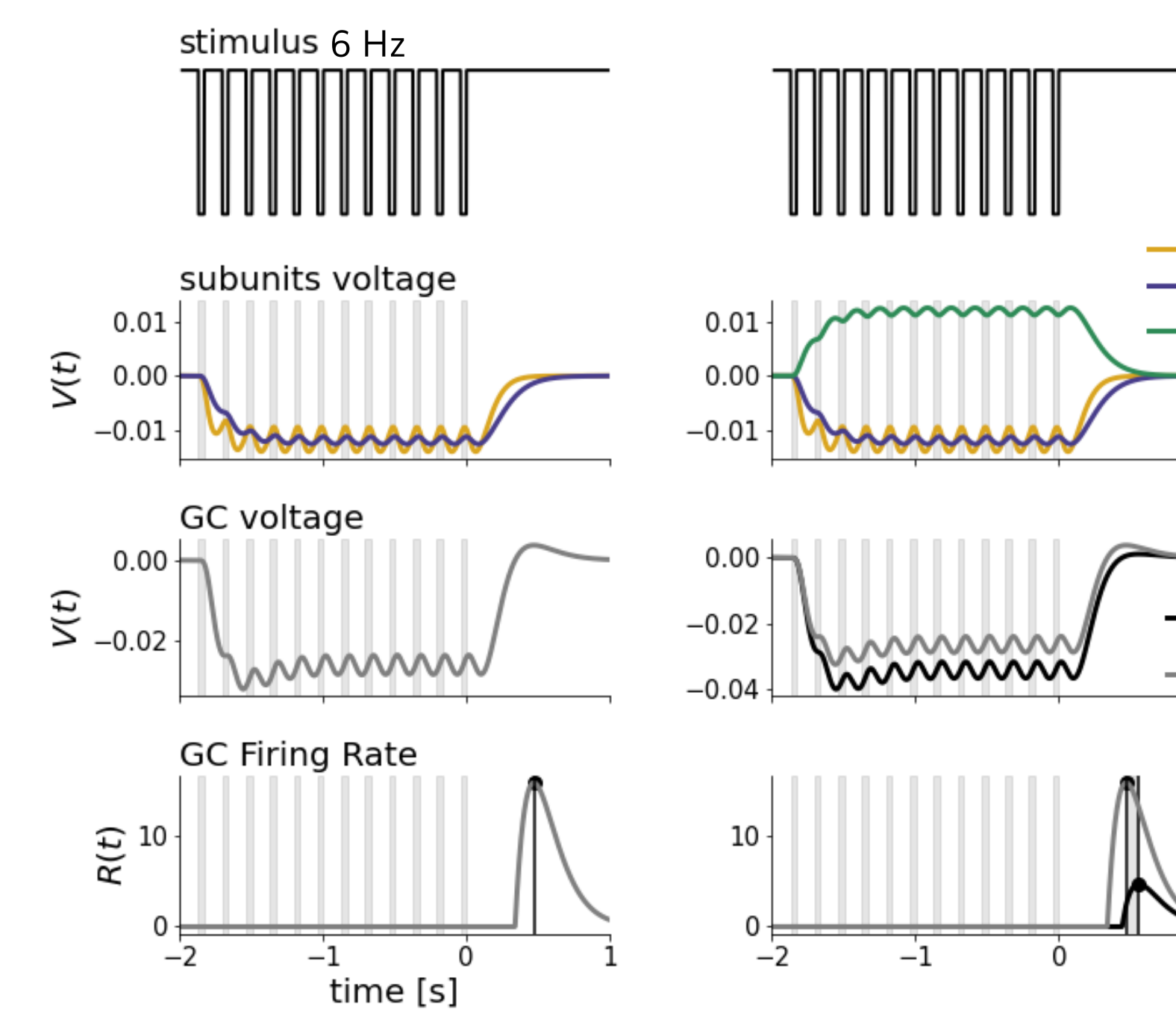
Simulated Result



Mechanistic model with depressing glycinergic amacrine cell replicates experimental finding.

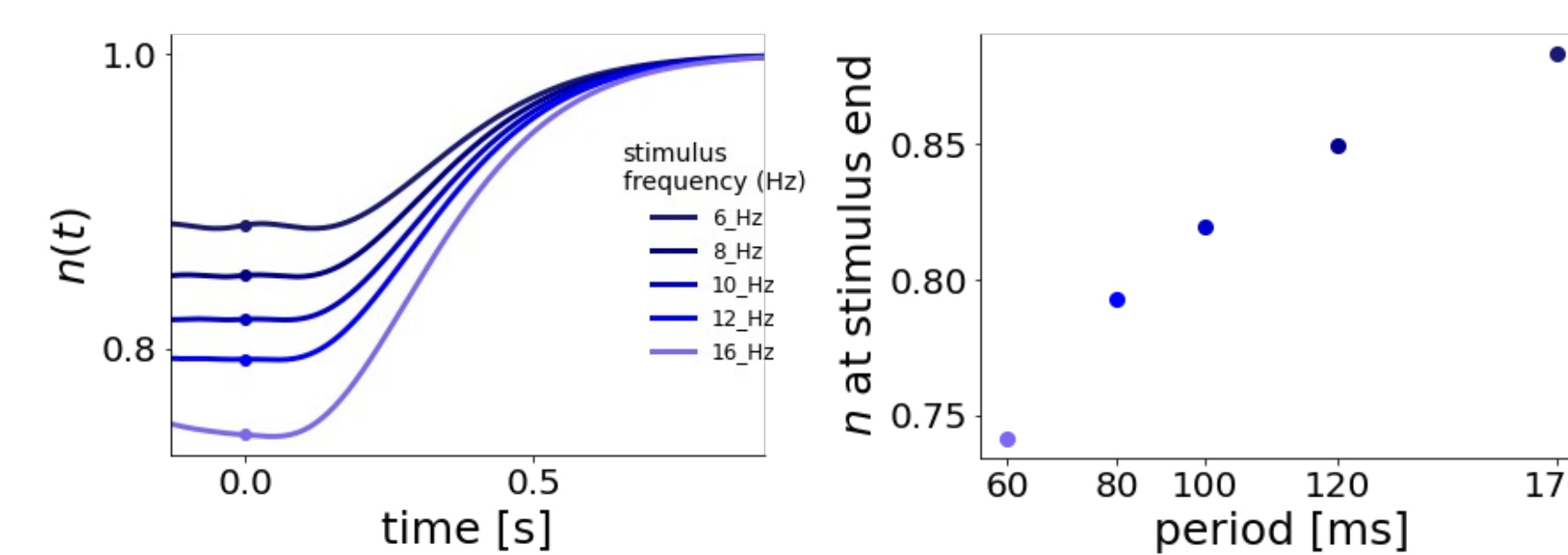
Mechanistic Explanation

To understand the origin of the error signal and its latency shift in the model it is helpful to look at the temporal evolution of its internal variables.

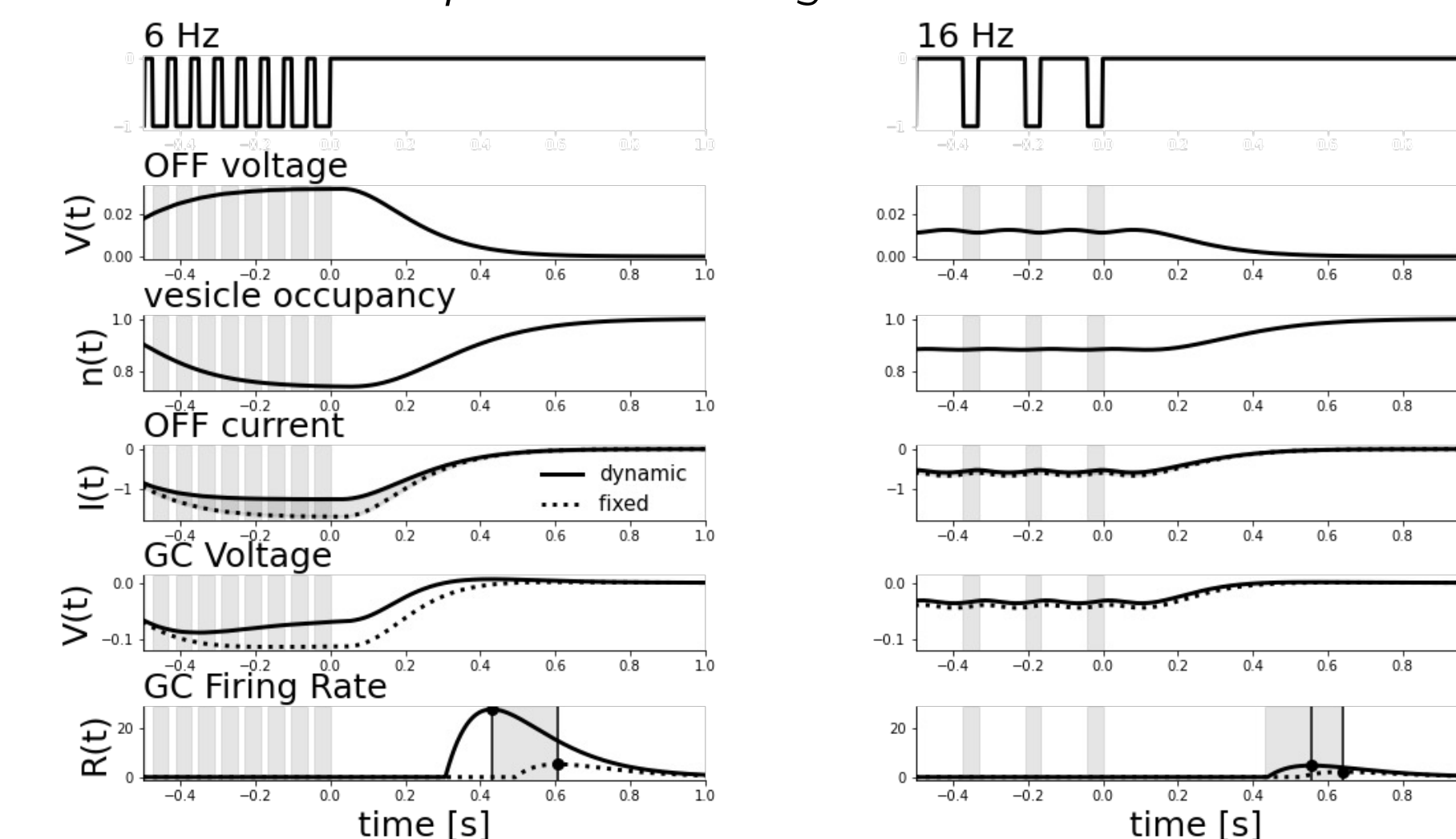


ON components of the model produce the error signal (left) while the glycinergic OFF input shifts the latency backwards (right).

The error response is produced via disinhibitory overshoot after cancellation of excitation and slower inhibition. The latency of the response is shifted by additional inhibition. STP scales the latency shift to the stimulus frequency by scaling the weight of inhibition.



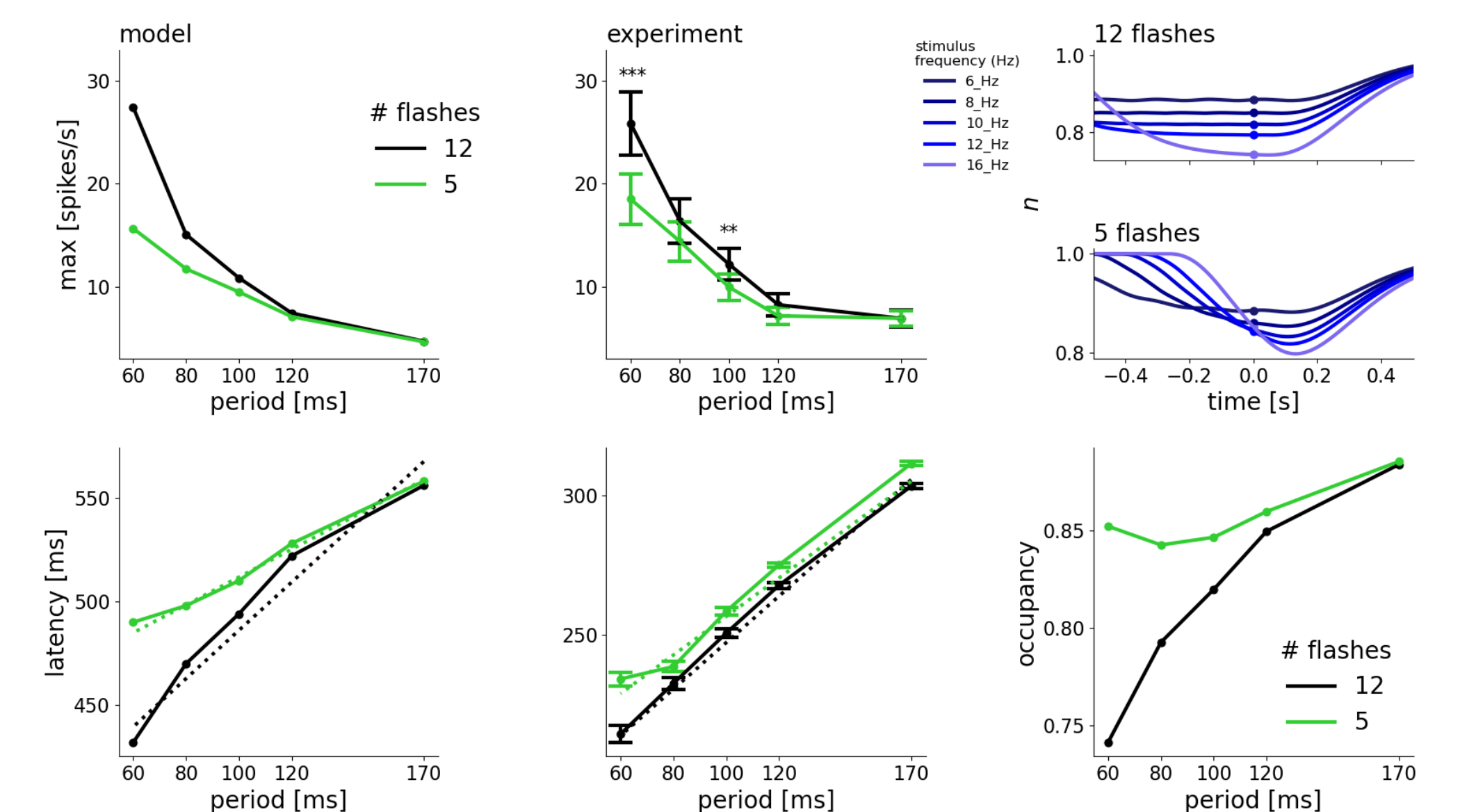
Inhibitory weight scaling with stimulus frequency over time (left) and at the end of the stimulus (right).



Impact of depression n is higher for fast (left) than for slow frequencies (right), yielding a shorter latency.

Model Confirmation

The model predicts that a sufficient number of flashes is necessary to reach a steady state for n that reliably tunes the latency shift. When the stimulus consists of only 5 flashes, the stimulus is too short to reach a steady state that scales with stimulus frequency, resulting in an inaccurate latency scaling. This effect is indeed observed in experiments.



Latency shift decreases for shorter stimuli due to lacking steady state of n .

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 allows to detect fast changes in its trajectory?

Acknowledgements

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