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Presentation Abstract

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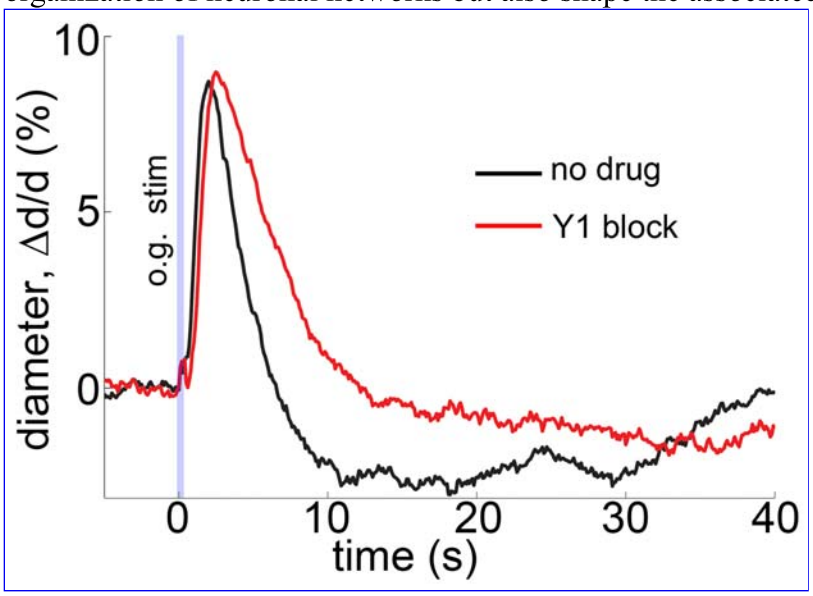
Abstract: Cortical inhibitory interneurons (IN) are known to release a repertoire of neurotransmitters and neuropeptides and pattern activity of cortical circuits. Some of these signaling molecules have vasoactive properties suggesting that IN activity may also regulate neurovascular coupling. Consistent with this idea, experiments in cortical brain slices have demonstrated that stimulation of IN can cause dilation or constriction of arteriolar segments embedded in the sliced tissue with the polarity of the effect depending on the IN cell type. Nevertheless, the role of IN in generation of functional hyperemia in vivo remains elusive. This is because excitatory and inhibitory neurons are wired together in normal cerebral circuits preventing experimental isolation of the inhibitory activity without confounding excitatory effects. To overcome this constraint, we employed in vivo 2-photon imaging of single-vessel diameters in transgenic VGAT-ChR2(H134R)-EYFP mice expressing optogenetic actuator Channelrhodopsin 2 (ChR2) in all GABAregic cortical neurons. Similar to sensory stimulation, activation of ChR2 in these mice elicited a biphasic diameter change in diving arterioles and their branches consisting of the initial dilation followed by a transient constriction throughout the imaging depth down to

cortical layer 5. Dilation latency, amplitude, and peak varied as a function of depth with the largest amplitude and the fastest onset and time-to-peak observed at the deepest locations. The decrease in dilation onset and time-to-peak with increasing depth mimicked that in response to sensory stimulation. The constriction phase of the response was abolished by blocking vascular Y1 receptors for Neuropeptide Y (NPY) (Figure 1, the red curve). Thus, IN not only play a major role in functional organization of neuronal networks but also shape the associated functional hyperemia.



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