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The selective role of cortical inhibitory interneurons in functional hyperemia

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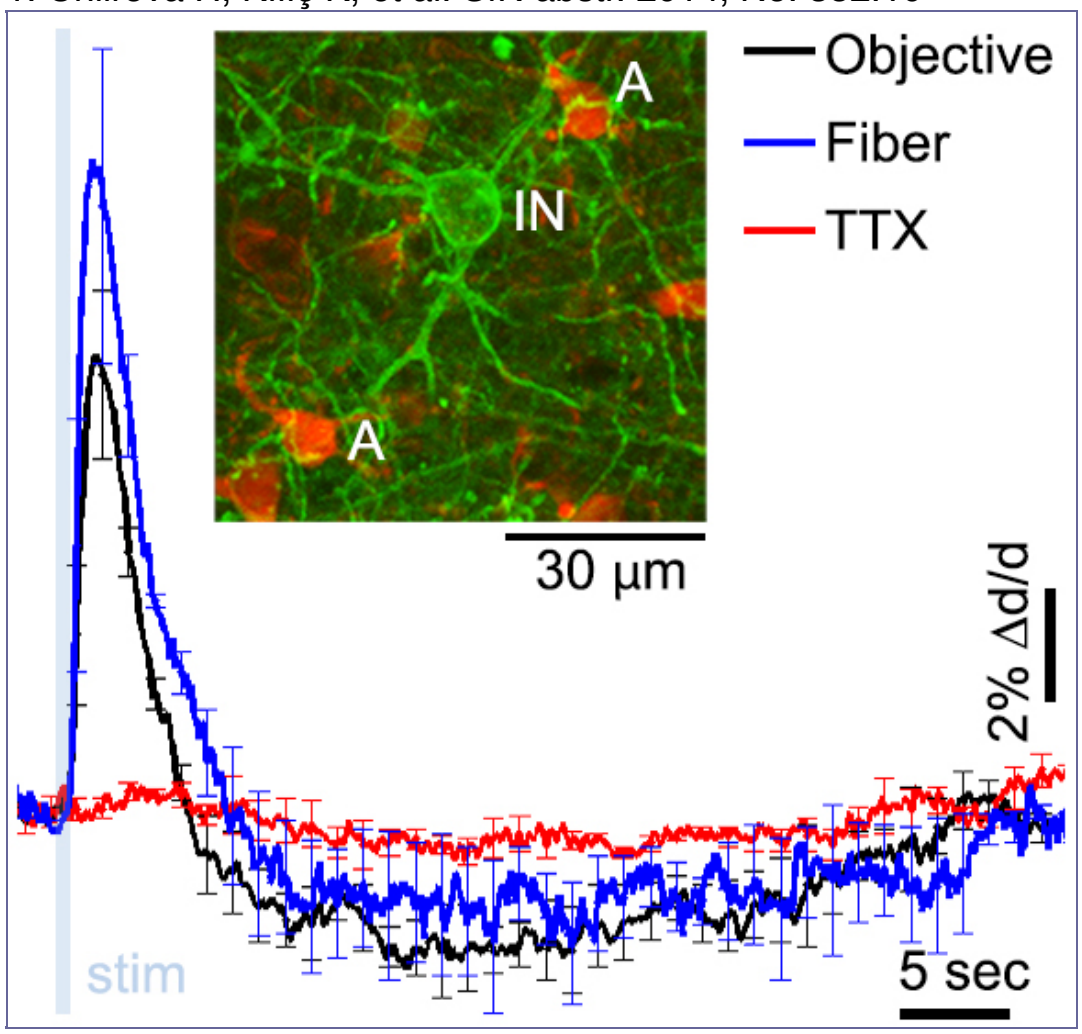
Abstract:
Identification of the cellular players that communicate neuronal activity to the vasculature is important for the basic understanding of cerebrovascular regulation. Previously, we reported that arteriolar dilation in response to optogenetic (OG) activation of all cortical inhibitory neurons (INs) started in deep cortical layers [1]. There, we delivered 473 nm-laser light for OG stimulation to the cortical surface via the objective. Since light at this wavelength does not penetrate deep into the tissue, we reasoned that INs that were driving the early dilation onset in deep layers have superficial axons or dendrites. In principle, OG-induced depolarization of superficial axons may trigger antidromic action potentials (APs)

that couple OG stimulation at the surface to the release of vasoactive messengers in deep layers. In this case, the vasoactive effect would be abolished by blocking APs and rescued by delivering OG light directly to layer V. To test this hypothesis, we used a tapered optical fiber positioned with its light-emitting tip in layer V for OG stimulation. Under control conditions, OG stimulation through objective and fiber induced similar arteriolar responses, but upon AP blockade with TTX, the response was lost in both cases (Figure). These results suggest that spiking is required to enable release of vasoactive agents, and that depolarization along dendrites rather than axons may communicate superficial excitation to vasoactive messenger release in deep layers. Supporting this, computational simulations of INs spanning across cortical layers revealed that depolarization of superficial dendrites could drive the soma, located as deep as in layer V, above its firing threshold. Thus, INs mediating dilation may have dendrites, which extend to the surface, and axons that reach into deep layers. One IN type meeting these morphological criteria are which express vasoactive intestinal peptide (VIP). However, further studies with more selective expression of OG actuators are required to test this prediction.

1. Uhlirova H, Kılıç K, et al. SfN abstr. 2014, No. 352.10



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