Tonic inhibition sets the state of excitability in olfactory bulb granule cells

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Abstract:

Granule cells are the main source of inhibition in the olfactory bulb (i.e. the first station of odour processing in the mammalian brain), but very little is known about the inhibition that acts upon them. Using in vivo whole cell patch clamp recordings in anaesthetized mice we report the following new findings: We found odour-evoked responses to be rare (seen only in 18% of the odour presentations, and only in cells that showed also evoked excitatory responses to odours). We report for the first time the presence of tonic inhibition in the olfactory bulb. We show that tonic inhibition dominates over phasic synaptic inhibition evoked by odours, thereby being the key regulator shaping the granule cells spike output. Preliminary (in vivo) evidence suggests that sensory evoked phasic inhibition onto granule cells is provided by deep short axon cells in the olfactory bulb. GABAergic granule cells (GCs) regulate, via mitral cells, the final output from the olfactory bulb to piriform cortex and are central for the speed and accuracy of odour discrimination. However, little is known about the local circuits in which GCs are embedded and how GCs respond during functional network activity. We recorded inhibitory and excitatory currents evoked during a single sniff-like odour presentation in GCs in vivo. We found that synaptic excitation was extensively activated across cells, whereas phasic inhibition was rare. Furthermore, our analysis indicates that GCs are innervated by a persistent firing of deep short axon cells that mediated the inhibitory evoked responses. Blockade of GABAergic synaptic input onto GCs revealed a tonic inhibitory current mediated by furosemide-sensitive GABAA receptors. The average current associated with this tonic GABAergic conductance was 3-fold larger than that of phasic inhibitory postsynaptic currents. We show that the pharmacological blockage of tonic inhibition markedly increased the occurrence of supra-threshold responses during an odour-stimulated sniff. Our findings suggest that GCs mediate recurrent or lateral inhibition, depending on the ambient level of extracellular GABA.

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