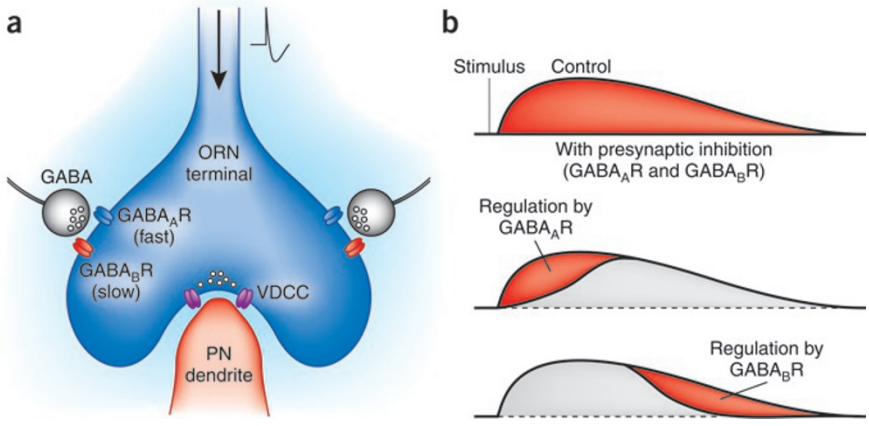

Katie Ris-Vicari

Figure 2 Both ionotropic and metabotropic GABARs regulate olfactory signal transfer. (**a**) Receptor neurons activate antennal lobe PNs by releasing acetylcholine. Presynaptic GABARs can depress acetylcholine release either by shunting the terminal via a transient increase in chloride conductance from activated $GABA_A$Rs, or by activating G proteins coupled to $GABA_B$Rs. The effector mechanisms for $GABA_B$R-mediated presynaptic inhibition are not known in this system, but could include activation of presynaptic potassium channels, modulation of voltage-gated calcium channels that control acetylcholine release or inhibition of the transmitter release machinery itself. VDCC, voltage-dependent calcium channel. (**b**) Presynaptic GABAergic inhibition is composed of two processes with different kinetics. Presynaptic $GABA_A$Rs rapidly, but transiently, inhibit input from receptor cells (middle), whereas $GABA_B$Rs attenuate late input (bottom). Activated together, presynaptic $GABA_A$Rs and $GABA_B$Rs can completely suppress signal transfer from receptor cell to PN (top).

How generalizable are these results? One step to answer this question will be to determine whether the specific glomerular-layer inhibitory circuit that Olsen and Wilson[2] found in *Drosophila* has a parallel in the mammalian olfactory bulb. This study also prompts a long list of behavioral questions. Are there different behavioral consequences of blocking presynaptic receptors that mediate lateral inhibition and postsynaptic GABARs? What are the behavioral implications of multiple presynaptic GABAR subtypes with different kinetics? How can we still perceive differences in odor concentration if much of the concentration-specific information is removed at the glomerular layer?

At a more general level, one implication of this study is that gain control and decorrelation are tightly linked processes, and therefore may appear together elsewhere in the brain. In *Drosophila*, it is not clear whether separate populations of interneurons carry out these apparently diverse functions. Olsen and Wilson[2] show that attenuating lateral presynaptic inhibition decreases the response specificity of projection neurons. This implies that, at least in insects, one type of inhibitory local circuit may simultaneously regulate input strength and differentiate responses to overlapping input patterns. In mammals, gain control and decorrelation are probably mediated by different inhibitory circuits. In particular, the high synapse specificity achieved by reciprocal dendrodendritic connections between mitral and granule cells[14,15] seems well suited for decorrelating glomerular activation patterns. The Olsen and Wilson study[2] may help to prompt a search for linked gain control and decorrelation circuits in other brain regions that operate on diffuse input patterns.

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Learning outside the song system

Young birds learn to sing during a critical period of development. Although the song system circuits have been identified, the molecular systems responsible for song template memorization have remained unknown. On page 579, London and Clayton reveal a critical role for the ERK signaling pathway in auditory cortex during normal song learning.

Male zebra finches copy the songs of older tutors when learning to sing. Auditory memories are initially formed, and through sensorimotor correction, the young bird eventually adopts a song that mimics the template derived from the tutor. In the new paper, the authors pharmacologically disrupted ERK signaling by injecting inhibitors into the auditory lobule during song learning exposure, presumably as the young bird was creating its template. Although the injection site was located outside the song system, these animals produced poor copies of the tutor song as compared to controls. Therefore, a brain region distinct from the areas controlling song output is integral to song learning and development.

There are efferent connections between the disrupted auditory area and the song system, suggesting that error feedback mechanisms between sensory and motor areas may be deficient when ERK signaling is disrupted. These results emphasize the importance of looking beyond the 'simple' circuits involved in species-specific actions to determine how multiple interacting systems in the brain shape behavior.

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