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The effects of modulatory systems in sensory thalamic nuclei

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Thalamic relay cells receive two types of afferents: **drivers**, considered responsible for relay cells' receptive field properties, and **modulators**, thought not to contribute significantly to the receptive field properties, but, rather, to modulate thalamic relay properties. Modulators often work by slowly modifying the resting potential of the cell and thus determining the mode of response: if sufficiently depolarized, tonic (more linear) or if sufficiently hyperpolarized, burst (highly non-linear but capable of larger signal to noise ratios and cortical activation).

Thalamic drivers have two origins. Some relay cells receive their drivers from subcortical areas, including those in the lateral geniculate nucleus (LGN), the ventral posterior nucleus (VP), and the ventral portion of the medial geniculate body (MGBv). These have been called **first order relays (FO)** since this is the first time that a particular information type is relayed to cortex. Other relay cells receive drivers from layer 5 of cortex, including those in the lateral posterior nucleus (LP), the posterior medial nucleus (POm), and the dorsal portion of the medial geniculate body (MGBd). These are known as **higher order relays (HO)**. Both FO and HO relays receive modulatory inputs, mainly from brainstem areas (e.g., cholinergic input from the parabrachial region, noradrenergic input from locus coeruleus, serotoninergic input from the dorsal raphé nucleus, etc) and from layer 6 of cortex.

We sought to determine the effects of modulators in the two types of relays, using current and voltage clamp recordings of rat (P12–P18) thalamic cells in the whole-cell, patch-clamp configuration. We bath-applied general agonists for muscarinic and serotonergic receptors and determined their effects on relay cells of six sensory nuclei, three FO relays (LGN, VP, and MGBv) and three HO relays (LP, POm, and MGBd).

We have recently shown that cholinergic input (by activating muscarinic M2 receptors) hyperpolarizes about 17% of the HO relay cells, whereas it depolarizes all FO relay cells through M1 and possibly M3 receptors. Preliminary results suggest that serotonergic inputs also have differential effects in FO and HO relays: 3 out of 5 cells recorded in LP are depolarized by serotonin whereas all 4 cells from VP and LGN are depolarized.

Furthermore, we are finding that HO and FO cells differ in their response properties. HO cells are more likely (60% of cells vs. 25% in FO; N = 23) to show spike frequency adaptation, which is also stronger than in FO cells. Activation of muscarinic receptors modifies the response properties of some HO cells, largely reducing the spike frequency adaptation.

Our results indicate that modulatory influences are different in thalamic nuclei that process information of cortical and non-cortical origin. They suggest that HO nuclei are more likely to be hyperpolarized when brainstem centers are active (waking, attention), therefore being more likely to respond to cortical inputs in the non-linear burst mode.