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THALAMIC SENSORY GATING

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The thalamus is not simply a relay station for sensory information to reach the cortex, but is actively engaged in shaping and modulating the afferent signals through a process which is controlled by the intrinsic properties of thalamocortical relay (TC) neurons. For example, burst spike activities of TC neurons that are driven by low threshold Ca^{2+} currents mediated by α1G T-type channels play a critical role in this sensory gating function of the thalamus. Thus, mice lacking the thalamic burst firing due to a deletion of the α1G T-type channel show hyperalgesic responses to persistent pain, accompanied by an increase in pain-encoding tonic spikes in TC neurons. This mutant mouse also shows enhanced responses to sensory inputs of other modalities. An important question is how the thalamocortical circuit is regulated to control the intrinsic property of TC neurons and how this property controls the amount of sensory information to the cortex. We addressed this question by analyzing several mutant mouse lines that show phenotypes indicative of an altered thalamic control of the pain perception toward persistent pain. In this talk, I will briefly review the roles of the thalamocortical circuit in the context of sensory gating, and discuss new findings that are relevant to the issue.