Synapticplasticity in the mouse somatosensory cortex driven by paralemniscalpathways

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In the somatosensory cortex mapplasticityisassociatedwith long termpotentiation (LTP) and depression (LTD). We have characterizeddifferentforms of sensory-evoked LTP in cortical pyramidal cells. Usingwholecellrecordings in vivo, wefoundthat LTP canreadilybeevokedusingspiking-dependentparadigms but alsousingsensory stimuli that do not evokespikes. Spiking-independent LTP relies on dendritic NMDA receptor conductances that are in part driven by the activity of paralemniscalsynapticpathways. This studysuggeststhat the repeatedcoincidentactivity of a paralemniscal feedback circuitrymayincrease L2/3 neurons' sensitivity to future sensory stimuli. Indeed, preliminary data indicatethatsensory stimuli thatevoke LTP also cause changes in subsequentsensory-evoked calcium dynamics in L2/3 cells. Furthercharacterization of the synaptic circuits underlying feedback-drivenplasticity in brain slices suggeststhat direct and repeatedco-activation of paralemniscal and lemniscalsynaptic inputs on L2/3 pyramidal cellsissufficient to evoke LTP in the absence of somaticspikes. This is in part dependent on paralemniscalpathway-driven activation of disinhibitory microcircuits. Interestingly, we have previouslyshownthattrimming of all excepttwowhiskersrapidly opens the possibility to drive STD-LTP by the sparedsurroundwhisker, a processthatalso relies on mechanisms of disinhibition. In thiswhisker-trimmingparadigmwefound a concomitant increase in NMDA receptor conductance. Altogether, these data indicatethatsensorymapplasticitymaydepend on differentforms of LTP, which are facilitated by disinhibition and an increase in dendritic NMDA receptor-mediatedevents. Thesephenomena are potentiallydriven by paralemniscalsynapticpathways.