NMDA receptor-dependent molecular plasticity in dendritic spines of the cerebral cortex after somatosensory stimulation

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The F-actin capping protein CapZ accumulates more in dendritic spines within regions where a long-term potentiation (LTP)-inducing stimulus has been applied. With the goal of developing an in vivo synaptic plasticity marker, we produced a transgenic mouse line, called AiCE-Tg, in which CapZ tagged with enhanced green fluorescence protein (EGFP-CapZ) is expressed in some spines. Twenty minutes after somatosensory stimulation under inactivation of the unilateral sciatic nerve in the AiCE-Tg mice, EGFP-CapZ signals were brighter in a subset of dendritic spines in the sensory cortex that receive preserved projections than those in the other hemisphere. That difference was abolished by an NMDA receptor blocker, MK801. Immunolabeling of α-actinin, a PSD-95 binding protein that can recruit AMPA receptors to postsynaptic sites, showed that α-actinin localization was more frequent/more accumulated in the brightest EGFP-CapZ spines (top 100) than in less bright spines (top 1000). This input-dependent redistribution of EGFP-CapZ may reflect LTP-like changes in vivo and thus may provide a useful tool for synaptic plasticity research.