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Light-induced regulation of Ras and cdc42 proteins in the mouse visual cortex

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Using synRas animals expressing permanently activated Ras in neurons, we have previously shown that intracellular neuronal Ras induces morphological changes, such as increase in the diameters of axons, associated with an enhancement of the size and ramification of dendritic trees and elevated dendritic spine density. However, the *in vivo* intracellular signaling pathways mediating spine plasticity are not fully known and will be investigated here. Furthermore, we ask about the possible function of normal neuronal Ras activity in the brain in response to light.

In the visual cortex (VC) of mice, Ras activity oscillates over the day/night cycle with high activity during light phase. This correlates with the circadian changes in levels of Trk neurotrophin receptor tyrosine phosphorylation. By applying light-pulses to the animal during night a short-term photic regulation was not detected. However, keeping mice in the dark for 7 days resulted in a dramatic decrease of Ras activity, which was accompanied by decreases in activities of the actin cytoskeleton regulating proteins cdc42 and Rac1. Acute exposure of mice to light for 1 h re-increased all small GTP-binding protein activities (Ras, cdc42, Rac). In addition, 7 days dark treatment decreases the relative levels of phosphorylation of cyclic-AMP response element binding protein (pCREB), which was re-increased following a 1 h light exposure. However, in 7 days dark treated synRas mice expression of constitutive activate Val12Ha-Ras transgene persisted and prevented the drop in cdc42 activity and pCREB levels.

In conclusions, Ras activity is regulated in circadian manner and by acute light stimuli in 7 days dark treated animals. The lack of cdc42 down-regulation in the VC of 7 days dark treated synRas mice suggests that Ras might be an upstream modulator of this Rho-family protein.