

Correction

NEUROBIOLOGY. For the article “Calmodulin priming: Nuclear translocation of a calmodulin complex and the memory of prior neuronal activity,” by Paul G. Mermelstein, Karl Deisseroth, Neela Dasgupta, Ann L. Isaksen, and Richard W. Tsien, which appeared in number 26, December 18, 2001, of *Proc. Natl. Acad. Sci. USA* (**98**, 15342–15347; First Published December 11, 2001; 10.1073/pnas.211563998), Figs. 3 and 4 did not appear in color due to a printer’s error. The color figures and their legends appear on this page and the facing page.

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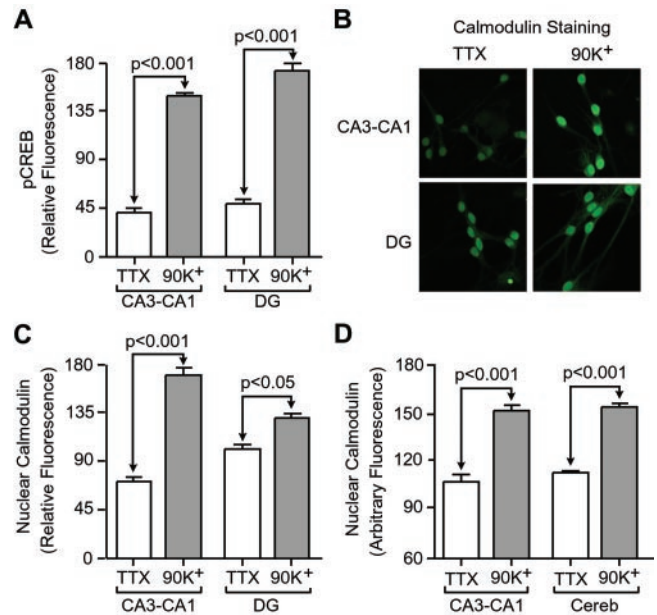


Fig. 3. Qualitative consistency of CaM translocation after depolarization in various neuronal populations. (A) After a 3-min, 90-mM K⁺ stimulation, significant increases in CREB phosphorylation were observed in hippocampal neurons taken from region CA3-CA1 ($P < 0.001$) or dentate gyrus (DG) ($P < 0.001$). (B and C) In granule cells from the dentate gyrus (DG), significant increases in nuclear CaM also were observed ($P < 0.05$), although the changes were less pronounced than in CA3-CA1 cells ($P < 0.001$). (D) Increased nuclear CaM in cerebellar granule cells (Cereb) ($P < 0.001$), similar in magnitude to that observed in CA3-CA1 pyramidal neurons.

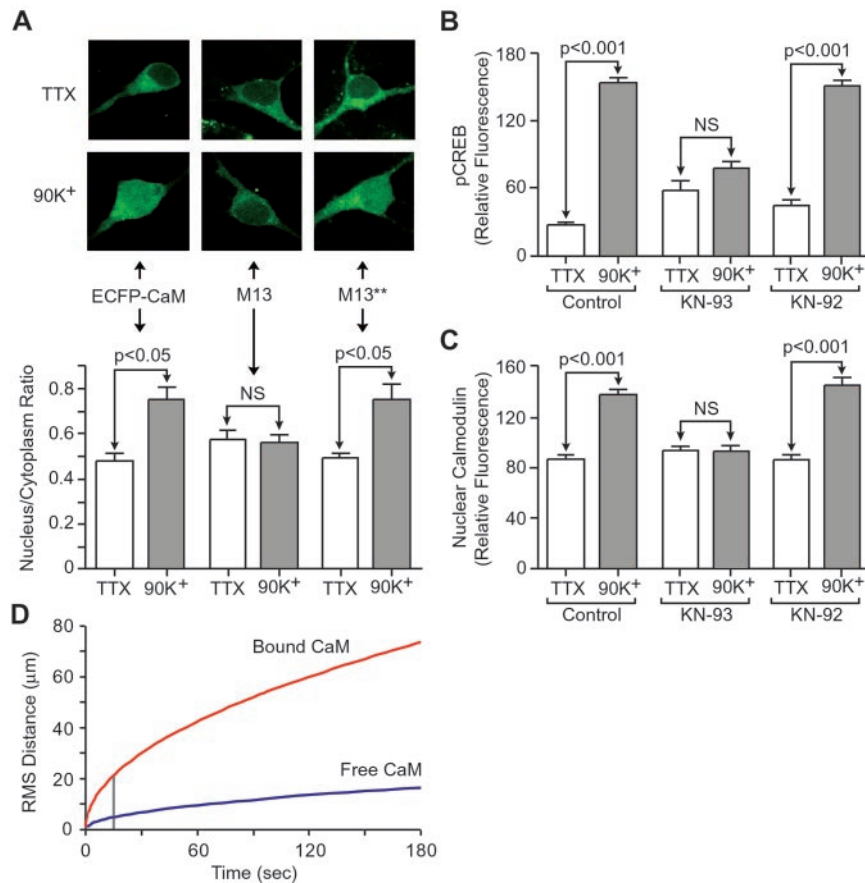


Fig. 4. Translocation of CaM into the nucleus depends on its binding to a target protein in a Ca^{2+} -dependent manner. (A) Hippocampal neurons transfected with cDNA encoding for ECFP-CaM exhibit a depolarization-induced nuclear translocation of fluorophore-labeled CaM, indicated by a significant increase in the ratio of nuclear to cytoplasmic fluorescence ($P < 0.05$). Addition of the Ca^{2+} -CaM binding peptide M13 onto ECFP-CaM prevented translocation. Translocation of ECFP-CaM was restored by mutating the M13 region [W800A, R812A (amino acid residues correspond to the smMLCK sequence)] to prevent Ca^{2+} -CaM binding (34). NS, not significant. (B and C) The CaM kinase inhibitor KN-93 ($1 \mu\text{M}$) blocked both CREB phosphorylation and CaM translocation (10, 45), suggesting that a CaM kinase (e.g., a CaMKK) may be required for CaM translocation. NS, not significant. (D) How interaction of CaM with a kinase could hasten its translocation to the nucleus. Calculated rms diffusion radius in three dimensions, at room temperature, using a diffusion coefficient ($2.5 \times 10^{-9} \text{ cm}^2/\text{s}$) measured for free CaM in smooth muscle cytoplasm (39). Also plotted is the theoretical corresponding rms radius for the diffusion of a typical large soluble protein ($5 \times 10^{-8} \text{ cm}^2/\text{s}$; e.g., kinase-bound CaM) assuming no binding interactions with cytoplasmic structures, showing greater spread over the same time period.