

Biochemistry. In the article “Characterization of recombinant phytochrome from the cyanobacterium *Synechocystis*” by Tilman Lamparter, Franz Mittmann, Wolfgang Gärtner, Thomas Börner, Elmar Hartmann, and Jon Hughes, which appeared in number 22, October 28, 1997, of *Proc. Natl. Acad. Sci. USA* (**94**, 11792–11797), a name appeared incorrectly in the acknowledgments on page 11797 due to a printer’s error. Prof. Silvia Braslavski (Max-Planck-Institut für Strahlenchemie, Mülheim) should be listed as Prof. Silvia Braslavsky.

Neurobiology. In the article “The synthesis of ATP by glycolytic enzymes in the postsynaptic density and the effect of endogenously generated nitric oxide” Kuo Wu, Chiye Aoki, Alice Elste, Adrienne A. Rogalski-Wilk, and Philip Siekevitz,

Neurobiology. In the article “ β subunits influence the biophysical and pharmacological differences between P- and Q-type calcium currents expressed in a mammalian cell line” by Herman Moreno, Bernardo Rudy, and Rodolfo Llinás, which appeared in number 25, December 9, 1997, of *Proc. Natl. Acad. Sci. USA* (**94**, 14042–14047), the following correction should be noted. Due to an editorial change at PNAS, the meaning of the last sentence on page 14046 was altered. The sentence originally read as follows: On the other hand, this structure does not reproduce the pharmacological properties of either P or Q channel exactly, as the ID_{50} to sFTX and ω -Aga IVA for P-type channels is lower than for the $\alpha 1A$, $\alpha 2\delta$, $\beta 1b$ channels in HEK cells.

which appeared in number 24, November 25, 1997, of *Proc. Natl. Acad. Sci. USA* (**94**, 13273–13278), the quality of the reproduction of Fig. 2A was poor. The figure and its legend are shown below:

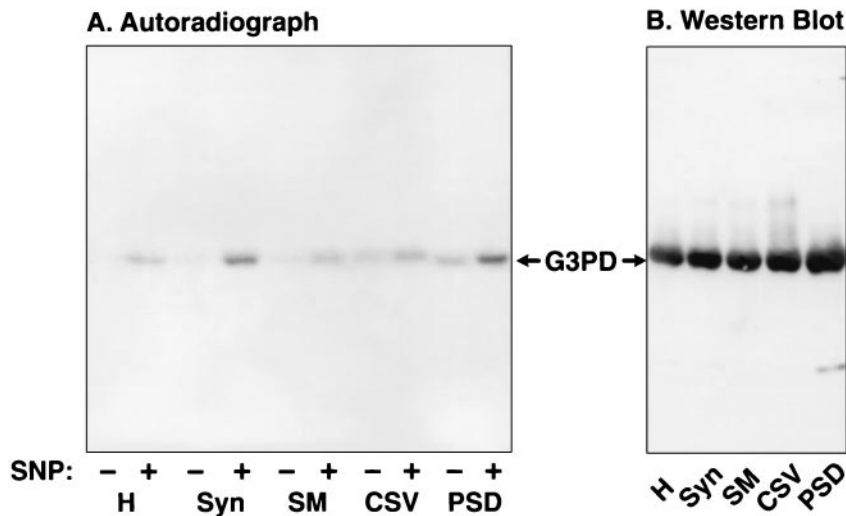


FIG. 2. (A) NO-stimulated [adenylate-³²P]NAD incorporation into G3PD in subcellular fractions isolated from adult porcine cerebral cortex. Assays are described in *Materials and Methods*. PSD (50 μ g) and 100 μ g each of the other fractions, in 100 μ l final volume, including whole homogenate (H), synaptosomes (Syn), synaptic plasma membranes (SPM), and crude synaptic vesicles (CSV), were incubated at 37°C for 15 min. NAD incorporation was performed in the absence (–) or presence (+) of SNP as exogenous source of NO. The mixtures were subjected to SDS/PAGE and then autoradiography. (B) Western blot analysis of the G3PD in the subcellular fractions. To confirm that the radioactive protein in the subcellular fractions was indeed G3PD, Western blot analysis was performed by using specific anti-G3PD antibodies as described.

Biochemistry. In the article “KSR stimulates Raf-1 activity in a kinase-independent manner” by Neil R. Michaud, Marc Therrien, Angela Cacace, Lisa C. Edsall, Sarah Spiegel, Gerald M. Rubin, and Deborah K. Morrison, which appeared in number 24, November 25, 1997, of *Proc. Natl. Acad. Sci. USA* (**94**, 12792–12796), the following correction should be noted.

Due to a printer’s error, background was incorrectly added to Fig. 2A and B on page 12793, Fig. 3B on page 12794, and Fig. 4A–C on page 12795 so that multiple panels from different gels or Western blots appear as one continuous panel. Correct versions of Figs. 2, 3, and 4 and each figure legend are reproduced here and on the opposite page.

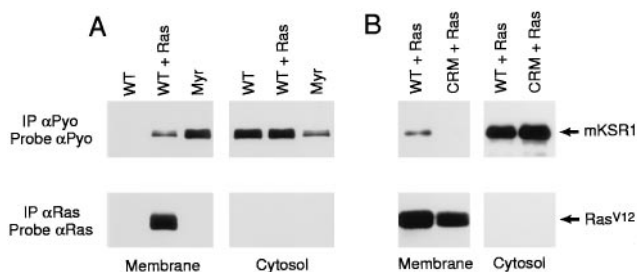


FIG. 2. The mKSR1 CA3 domain is required for the Ras-dependent membrane localization of mKSR1. (A) 293 cells transiently expressing WT and Myr mKSR1 or coexpressing WT and Ras^{V12} were fractionated into membrane and cytosolic fractions. The mKSR1 proteins were immunoprecipitated and examined by immunoblot analysis using α Pyo antibody. (B) 293 cells coexpressing Ras^{V12} and either WT or CRM mKSR1 proteins were analyzed as in A.

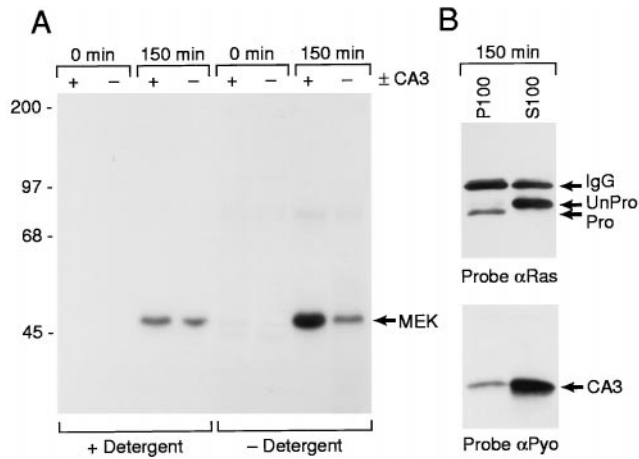


FIG. 3. The mKSR1 CA3 domain augments Raf-1 activity in a detergent-sensitive manner. *Xenopus* oocytes expressing Raf-1 alone (-) or coexpressing Raf-1 and the mKSR1 CA3 domain (+) were injected with Ras^{V12} RNA. Immediately after (0 min) or 150 min after Ras^{V12} injection, oocytes were lysed in hypotonic buffer and membranes were isolated. (A) Raf-1 proteins were immunoprecipitated from membrane fractions resuspended in RIPA buffer (+ detergent) or phosphate-buffered saline (- detergent), and *in vitro* kinase assays were performed using kinase-inactive MEK as a substrate (1). Phosphorylation of MEK1 on Ser-218 and Ser-222 was determined by tryptic peptide mapping analysis. (B) Ras^{V12} and CA3 proteins were immunoprecipitated from membrane (P100) and cytosolic fractions (S100) isolated at 150 min after injection with Ras^{V12} RNA and were examined by immunoblot analysis using Ras and Pyo antibody, respectively. The migration of processed (Pro) and unprocessed (UnPro) Ras proteins is indicated.

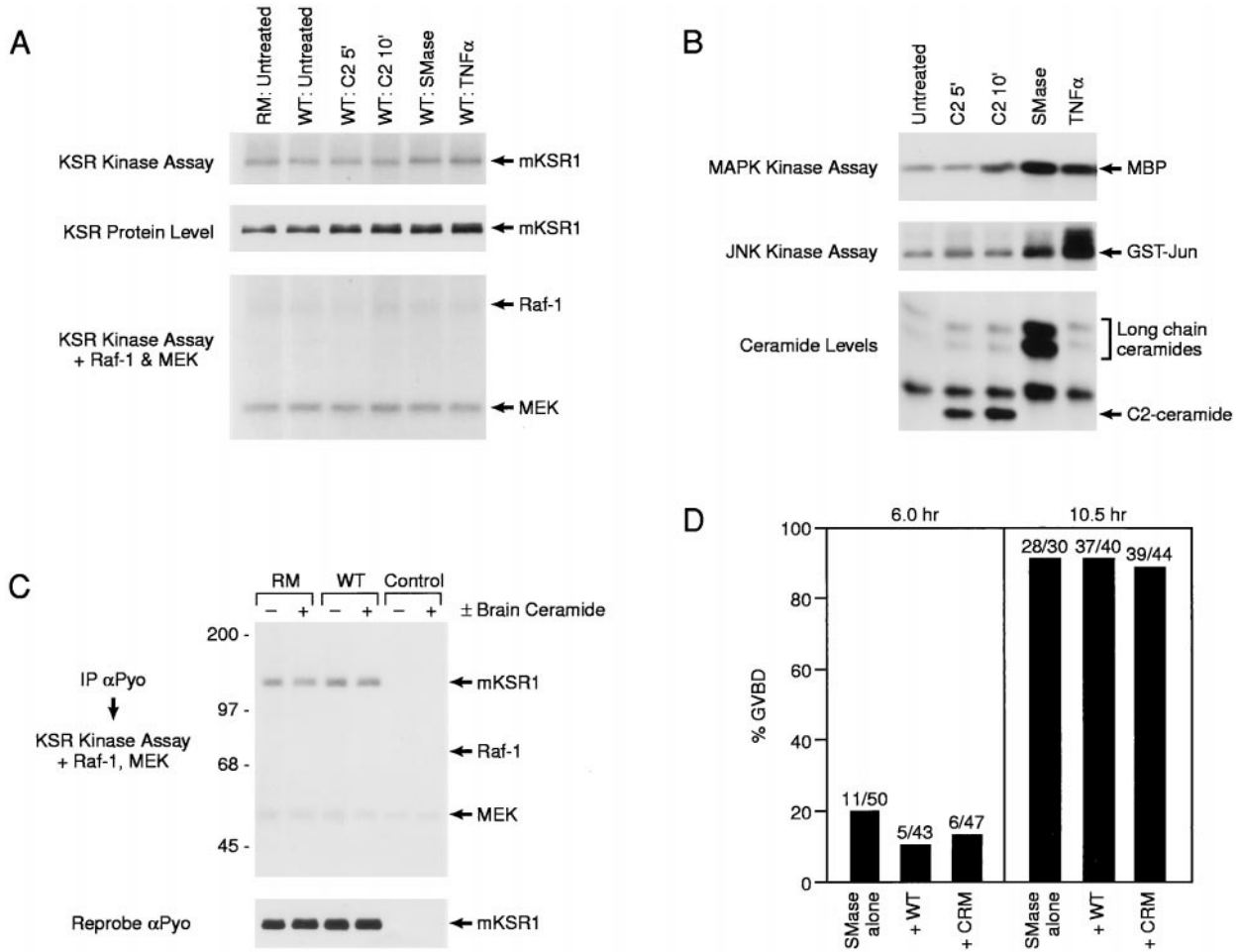


FIG. 4. Augmentation of Ras signaling by mKSR1 does not involve ceramide. (A) Cos cells were transiently transfected with constructs encoding wild-type (WT) or kinase-inactive (RM) mKSR1. At 60 hr posttransfection, serum starved cells were left untreated or were stimulated with 20 μ M C2 ceramide for 5 or 10 min, 100 milliunits/ml sphingomyelinase (SMase) for 20 min or 10 nM tumor necrosis factor α (TNF α) for 20 min. KSR proteins were immunoprecipitated using α Pyo antibody, and mKSR1 immune complex kinase assays were performed *in vitro* as described by Zhang *et al.* (ref. 19; *Top*). Immunoprecipitated mKSR1 was detected by immunoblot analysis (*Middle*). To observe phosphorylation of Raf-1 or modulation of Raf-1 activity, purified activated Raf-1, coexpressed in Sf9 cells in the presence of Ras^{V12} and v-src, and kinase-inactive MEK1 were added to the mKSR1 immune complex kinase assays previously described (ref. 19; *Bottom*). (B) Cos cells were treated as in A and endogenous ceramide levels, JNK activity, and MAPK activity were determined. Ceramide levels were normalized to the untreated control. C2-ceramide levels were elevated 2.9- and 3.7-fold at 5 and 10 min, respectively, and long-chain ceramide levels were elevated 12-fold by SMase and 1.9-fold by TNF α . (C) Purified brain ceramide (100 nM) (+) or diluent (-) was added *in vitro* to mKSR proteins immunoprecipitated from transfected Cos cells and immune complex kinase assays performed in the presence of activated Raf-1 and kinase-inactive MEK1 as previously described (ref. 19; *Top*). Immunoprecipitated mKSR1 was detected by immunoblot analysis (*Bottom*). (D) Oocytes preinjected with buffer or RNA encoding WT and CRM mKSR1 constructs were treated with 250 milliunits sphingomyelinase. GVBD was then scored 6 and 10.5 hr after treatment.