Corrections

NEUROBIOLOGY. For the article "Remodeling of α-amino-3-hydroxy-5-methyl-4-isoxazole-propionic acid receptor subunit composition in hippocampal neurons after global ischemia" by Thoralf Optiz, Sonja Y. Grooms, Michael V. L. Bennett, and R. Suzanne Zukin, which appeared in number 24, November 21, 2000, of *Proc. Natl. Acad. Sci. USA* (**97,** 13360–13365), the authors note the following correction. The name of the first author was misspelled. The correct spelling is Thoralf Opitz. The online version has been corrected.

Correction published online before print: *Proc. Natl. Acad. Sci. USA*, 10.1073/pnas.041602198. Text and publication date are at www.pnas.org/cgi/doi/10.1073/pnas.041602198

NEUROBIOLOGY. For the article "Sparing of neuronal function postseizure with gene therapy" by John McLaughlin, Benno Roozendaal, Theodore Dumas, Anurag Gupta, Olusola Ajilore, Joseph Hsieh, Dora Ho, Matthew Lawrence, James L. McGaugh, and Robert Sapolsky, which appeared in number 23, November 7, 2000, of *Proc. Natl. Acad. Sci. USA* (**97,** 12804–12809), the authors note the following correction. The words necrotic and apoptotic that appeared at the top of Fig. 2 should be omitted. The corrected figure and its legend are reprinted below.

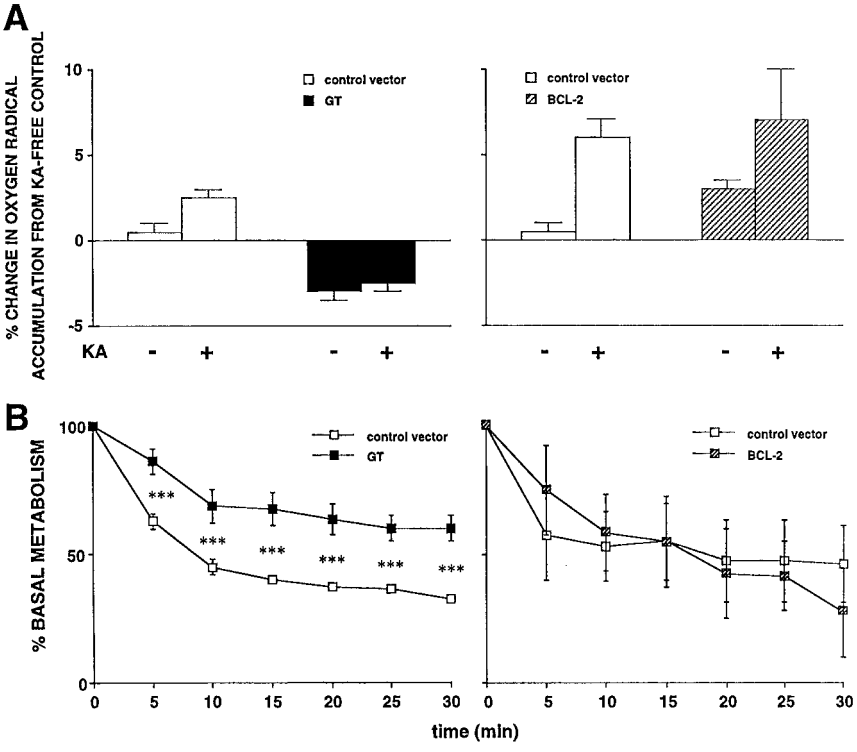

Fig. 2. (*A*) Effects of GT (*Left*) or Bcl-2 (*Right*) on ROS accumulation post-KA. Values are expressed as percentage above that in mock-infected control cultures without KA. In control vector-treated wells, KA significantly increased ROS accumulation ($P < 0.05$ and 0.01 in GT and Bcl-2 studies, respectively, post hoc test after two-way ANOVA). In GT-infected wells, KA did not significantly increase accumulation, and in either condition (±KA), there was significantly less accumulation than in cognate control wells ($P < 0.01$ for both). In contrast, KA significantly increased accumulation in Bcl-2-treated wells ($P < 0.05$), and values did not differ significantly from the cognate control wells. (*B*) Effects of GT or Bcl-2 on metabolism in primary hippocampal cultures under hypoglycemic conditions as assessed by proton efflux rates, measured by microphysiometry. vIE1GT (*Left*) attenuated the drop of metabolism posthypoglycemia (***, $P < 0.001$ by post hoc test after two-way ANOVA, comparing experimental vector versus control vector at the same time point), whereas vα22βgalα4bcl-2 (*Right*) had no effect. Data on the *Left* previously published (7), making use of a related HSV vector expressing either GT (vIE1GT) or β-Gal as a reporter gene (vIE1βGal).