Corrections

NEUROSCIENCE. For the article "Sex and estrogenic effects on coexpression of mRNAs in single ventromedial hypothalamic neurons," by N. Devidze, J. A. Mong, A. M. Jasnow, L.-M. Kow, and D. W. Pfaff, which appeared in issue 40, October 4, 2005, of Proc. Natl. Acad. Sci. USA (102, 14446-14451; first published September 26, 2005; 10.1073/pnas.0507144102), the authors note that "%" was added after several numbers, due to a printer's error. On page 14449, right column, the second sentence under Estrogen Treatment Increases Number of ERα, ERβ, OTR, and PKCs in Female Rats, "In estrogen-treated animals, these numbers were higher than in the oil group, ER α (66% vs. 20%), ERβ (39% vs. 14%), OTR (56% vs. 15%), PKCδ (28% vs. 8%), PKC ε (30% vs. 8%), and PKC η (44% vs. 12%) (\dot{P} < 0.05 in all cases) (Tables 3 and 4)," should read: "In estrogen-treated animals, these numbers were higher than in the oil group, ER α (66 vs. 20), ERβ (39 vs. 14), OTR (56 vs. 15), PKCδ (28 vs. 8), PKC ε (30 vs. 8), and PKC η (44 vs. 12) (P < 0.05 in all cases) (Tables 3 and 4)." This error does not change the conclusions of the article.

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GENETICS. For the article "Promoters of the murine embryonic β -like globin genes Ey and $\beta h1$ do not compete for interaction with the β -globin locus control region," by Xiao Hu, Michael Bulger, Julia N. Roach, Susan K. Eszterhas, Emmanuel Olivier, Eric E. Bouhassira, Mark T. Groudine, and Steven Fiering, which appeared in issue 3, February 4, 2003, of Proc. Natl. Acad. Sci. USA (100, 1111-1115; first published January 13, 2003; 10.1073/pnas.0337404100), the authors note the following. It was incorrectly reported that β -major and β -minor do not change transcription level in primitive erythroid cells when either Ey or $\beta h \hat{1}$ is deleted. This error is attributable to sample misidentification. Subsequent studies have shown that deletion of either Ey or $\beta h1$ mediates a 3-fold increase of β -major and β-minor mRNA expression in primitive cells. This revised conclusion supports the hypothesis that expression of the embryonic β -like globin genes influences expression of the fetal/adult β -like globin genes in primitive erythroid cells.

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