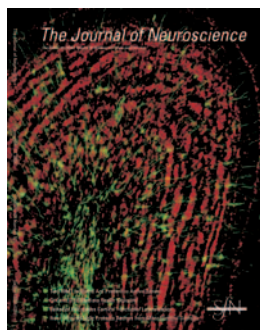


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Cover legend: Coronal section of the olfactory bulb of an adult wild-type mouse that received a homotopic graft of subventricular zone cells from a GFP⁺/TrkB^{-/-} mouse and was killed 15 d later. Graft-derived neurons (green) migrate to the olfactory bulb, disperse radially into several olfactory bulb layers (granular layer shown in picture; red, DAPI nuclear dye), and extend dendrites even in the absence of the neurotrophin receptor TrkB. For more information, see the article by Galvão et al. in this issue (pages 13368–13383).

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Correction: In the article “NMDA Receptor Activation by HIV-Tat Protein Is Clade Dependent” by Wenxue Li, Yan Huang, Rollie Reid, Joseph Steiner, Tanya Malpica-Llanos, Thomas A. Darden, Susarla K. Shankar, Anita Mahadevan, Parthasarathy Satishchandra, and Avindra Nath, which appeared on pages 12190–12198 of the November 19, 2008 issue, some key references were accidentally omitted. HIV-Tat protein from clade C virus when compared with that of clade B virus has been previously shown to have impaired chemotactic activity and decreased neurotoxicity, and *in vivo* studies in a rodent model have shown decreased cognitive abnormalities associated with decreased macrophage infiltration. The Cys31Ser mutation in Tat has been implicated in each of these effects.

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