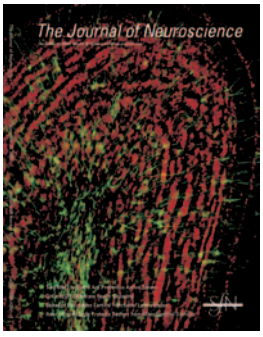
The Journal of Neuroscience

December 10, 2008 • Volume 28 Number 50 www.jneurosci.org



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).

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, Parthasarthy 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.

References

Mishra M, Vetrivel S, Siddappa NB, Ranga U, Seth P (2008) Clade-specific differences in neurotoxicity of human immunodeficiency virus-1 B and C Tat of human neurons: significance of dicysteine C30C31 motif. Ann Neurol 63:366–376.

Ranga U, Shankarappa R, Siddappa NB, Ramakrishna L, Nagendran R, Mahalingam M, Mahadevan A, Jayasuryan N, Satishchandra P, Shankar SK, Prasad VR (2004) Tat protein of human immunodeficiency virus type 1 subtype C strains is a defective chemokine. J Virol 78:2586–2590.

Rao VR, Sas AR, Eugenin EA, Siddappa NB, Bimonte-Nelson H, Berman JW, Ranga U, Tyor WR, Prasad VR (2008) HIV-1 clade-specific differences in the induction of neuropathogenesis. J Neurosci 28:10010–10016.

Persons interested in becoming members of the Society for Neuroscience should contact the Membership Department, Society for Neuroscience, 1121 14th St., NW, Suite 1010, Washington, DC 20005, phone 202-962-4000.

Instructions for Authors are available at http://www.jneurosci.org/misc/itoa.shtml. Authors should refer to these Instructions online for recent changes that are made periodically.

Brief Communications Instructions for Authors are available via Internet (http://www.jneurosci.org/misc/ifa_bc.shtml).

Submissions should be submitted online using the following url: http://sfn.manuscriptcentral.com. Please contact the Central Office, via phone, fax, or e-mail with any questions. Our contact information is as follows: phone, 202-962-4000; fax, 202-962-4945; e-mail, jn@sfn.org.