

# The Journal of Neuroscience

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**Cover legend:** Cerebrovascular amyloid angiopathy (CAA)-affected vessel in the Tg2576 mouse model of amyloid deposition. Representative example of CAA in a vessel segment of a living Tg2576 mouse imaged with multiphoton microscopy. Vascular and parenchymal A $\beta$  deposits are identified by fluorescence from systemically administered methoxy-X04 (red pseudocolor). Fluorescent angiograms with Texas Red dextran were performed to identify fiduciary markers (blue pseudocolor). The serial imaging of individual segments of CAA-laden leptomeningeal vessels allowed detection of clearance of CAA after treatment with anti-A $\beta$  antibody. For more information, see the article by Prada et al. in the February 21, 2007 issue (pages 1973–1980).

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**Correction:** In the article “Extracellular Signal-Regulated Kinase-Regulated Microglia–Neuron Signaling by Prostaglandin E2 Contributes to Pain after Spinal Cord Injury” by Peng Zhao, Stephen G. Waxman, and Bryan C. Hains, which appeared on pages 2357–2368 of the February 28, 2007 issue, the sentence in the abstract “Selective targeting of dorsal horn microglia with the Mac-1–synapse-associated protein (SAP) immunotoxin resulted in reduced microglia staining, reduction in PGE2 levels, and reversed pain-related behaviors” should read as “Selective targeting of dorsal horn microglia with the Mac-1-SAP immunotoxin, a chemical conjugate of mouse monoclonal antibody to CD11b and the ribosome-inactivating protein saporin, resulted in reduced microglia staining, reduction in PGE2 levels, and reversed pain-related behaviors.”

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