Corrections

Correction: Arnold et al., Defective Retinal Vascular Endothelial Cell Development as a Consequence of Impaired Integrin αVβ8-Mediated Activation of Transforming Growth Factor-β

In the article "Defective Retinal Vascular Endothelial Cell Development as a Consequence of Impaired Integrin αVβ8-Mediated Activation of Transforming Growth Factor-β" by Thomas D. Arnold, Gina M. Ferrero, Haiyan Qiu, Isabella T. Phan, Rosemary J. Akhurst, Eric J. Huang, and Louis F. Reichardt, which appeared on pages 1197–1206 of the January 25, 2012 issue, the α and β symbols were missing from Note added in proof. The corrected Note added in proof is listed below:

Note added in proof. A related paper was published recently after the submission of our revised manuscript (Hirota et al., 2011). Both papers used nestin-driven Cre expression to delete *Itgb8* within the retinal precursor cells and observed similar retinal hemorrhagic phenotypes. Harada et al. (2011) report a deficit in tip cell filopodia density not observed in our study. The authors did not include a description of the abnormalities in vascular branch point density, coverage, or stability that were observed in our study. Both papers provide data that strongly supports a role of this integrin in development of the secondary (deep) vascular plexus. Hirota et al. (2011) did not examine the role in the outer versus inner deep vascular plexus. Retinal anti-TGFβ injection by Hirota et al. (2011) provided data complementary to ours on the role of TGFβ as a downstream effector of this integrin. The most important disagreement between our papers concerns the role of αVβ8 in retinal astrocytes versus Müller glia. Hirota et al. (2011) concluded that retinal astrocyte-expressed αVβ8 is required for normal retinal angiogenesis because they observed expression of this integrin in cultured retinal astrocytes. Our data indicates that nestin-Cre promotes efficient gene deletion in Müller glia, but not retinal astrocytes *in vivo*. We also observed expression of the β8 subunit in Müller glia, but not retinal astrocytes *in vivo*. Following astrocyte-specific, GFAP-Cre-mediated *Itgb8* deletion *in vivo*, we did not detect retinal hemorrhage or significant abnormalities in vascular branching, coverage, or stability. It seems likely that the cultures examined by Hirota et al. (2011) contained Müller glia that mediated TGFβ activation.

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