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Corrigendum: Long-term channelrhodopsin-2 (ChR2) expression can induce abnormal axonal morphology and targeting in cerebral cortex

Toshio Miyashita, Yu R. Shao, Jason Chung, Olivia Pourzia and Daniel E. Feldman*

Department of Molecular and Cell Biology, University of California, Berkeley, CA, USA
**Correspondence: dfeldman@berkeley.edu*

Edited by:
Charles F. Stevens, The Salk Institute for Biological Studies, USA

Reviewed by:
Charles F. Stevens, The Salk Institute for Biological Studies, USA

A commentary on

Long-term channelrhodopsin-2 (ChR2) expression can induce abnormal axonal morphology and targeting in cerebral cortex

by Miyashita, T., Shao, Y. R., Chung, J., Pourzia, O., and Feldman, D. E. (2013). Front. Neural Circuits 7:8. doi: 10.3389/fncir.2013.00008

In our recent paper (Miyashita et al., 2013), we showed that long-term, high-level channelrhodopsin-2 (ChR2) expression by *in utero* electroporation (IUE) produces structural abnormalities in the axons of ChR2-expressing pyramidal cells in rat somatosensory cortex. In the Discussion of our paper, we mentioned that such abnormalities were not observed in an earlier study using long-term IUE of ChR2 under the same promoter (Huber et al., 2008). A methodological difference has been brought to our attention that likely explains this difference. Huber et al. expressed wildtype ChR2 (Chop2-315 from Nagel et al., 2003), while we expressed hChR2 that was codon-optimized for higher mammalian expression (Zhang et al., 2006). This suggests that lower ChR2 protein levels in the Huber study may have enabled long-term expression without axonal malformations. This further supports our main conclusion that morphological abnormalities are associated with high-level, long-term expression of ChR2 protein, and that lower level expression is necessary for long-term studies.

REFERENCES

Huber, D., Petreanu, L., Ghitani, N., Ranade, S., Hromádka, T., Mainen, Z., et al. (2008). Sparse optical microstimulation in barrel cortex drives learned behaviour in freely moving mice. *Nature* 451, 61–64.

Miyashita, T., Shao, Y. R., Chung, J., Pourzia, O., and Feldman, D. E. (2013). Long-term channelrhodopsin-2 (ChR2) expression can induce abnormal axonal morphology and targeting in cerebral cortex. *Front. Neural Circuits* 7:8. doi: 10.3389/fncir.2013.00008

Nagel, G., Szellas, T., Huhn, W., Kateriya, S., Adeishvili, N., Berthold, P., et al. (2003). Channelrhodopsin-2, a directly light-gated cation-selective membrane channel. *Proc. Natl. Acad. Sci. U.S.A.* 100, 13940–13945.

Zhang, F., Wang, L. P., Boyden, E. S., and Deisseroth, K. (2006). Channelrhodopsin-2 and optical control of excitable cells. *Nat. Methods* 3, 785–792.

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