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1. Flanagan, J. G. & Vanderhaeghen, P. The ephrins and Eph receptors in neural development. *Annu. Rev. Neurosci.* **21**, 309–345 (1998).
2. Frisen, J., Holmberg, J. & Barbacid, M. Ephrins and their Eph receptors: multitalented directors of embryonic development. *EMBO J.* **18**, 5159–5165 (1999).
3. Henkemeyer, M. *et al.* Nuk controls pathfinding of commissural axons in the mammalian central nervous system. *Cell* **86**, 35–46 (1996).
4. Holland, S. J. *et al.* Bidirectional signalling through the EPH-family receptor Nuk and its transmembrane ligands. *Nature* **383**, 722–725 (1996).
5. Bruckner, K., Pasquale, E. B. & Klein, R. Tyrosine phosphorylation of transmembrane ligands for Eph receptors. *Science* **275**, 1640–1643 (1997).
6. Cowan, C. A. & Henkemeyer, M. The SH2/SH3 domain adaptor Grb4 transduces B-ephrin reverse signals. *Nature* **413**, 174–179 (2001).
7. Eph Nomenclature Committee. Unified nomenclature for Eph family receptors and their ligands, the Ephrins. *Cell* **90**, 403–404 (1997).
8. Gale, N. W. *et al.* Eph receptors and ligands comprise two major specificity subclasses and are reciprocally compartmentalized during embryogenesis. *Neuron* **17**, 9–19 (1996).
9. Henkemeyer, M. *et al.* Immunolocalization of the Nuk receptor tyrosine kinase suggests roles in segmental patterning of the brain and axonogenesis. *Oncogene* **9**, 1001–1014 (1994).
10. Davis, S. *et al.* Ligands for EPH-related receptor tyrosine kinases that require membrane attachment or clustering for activity. *Science* **266**, 816–819 (1994).
11. Himanen, J. P., Henkemeyer, M. & Nikolov, D. B. Crystal structure of the ligand-binding domain of the receptor tyrosine kinase EphB2. *Nature* **396**, 486–491 (1998).
12. Toth, J. *et al.* Crystal structure of an ephrin ectodomain. *Dev. Cell* **1**, 83–92 (2001).
13. Labrador, J. P., Brambilla, R. & Klein, R. The N-terminal globular domain of Eph receptors is sufficient for ligand binding and receptor signaling. *EMBO J.* **16**, 3889–3897 (1997).
14. Lackmann, M. *et al.* Distinct subdomains of the EphA3 receptor mediate ligand binding and receptor dimerization. *J. Biol. Chem.* **273**, 20228–20237 (1998).
15. Hendrickson, W. A. Determination of macromolecular structures from anomalous diffraction of synchrotron radiation. *Science* **254**, 51–58 (1991).
16. Lackmann, M. *et al.* Ligand for EPH-related kinase (LERK) 7 is the preferred high affinity ligand for the HEK receptor. *J. Biol. Chem.* **272**, 16521–16530 (1997).
17. Stein, E. *et al.* Eph receptors discriminate specific ligand oligomers to determine alternative signaling complexes, attachment, and assembly responses. *Genes Dev.* **12**, 667–678 (1998).
18. Bruckner, K. *et al.* EphrinB ligands recruit GRIP family PDZ adaptor proteins into raft membrane microdomains. *Neuron* **22**, 511–524 (1999).
19. Stapleton, D., Balan, I., Pawson, T. & Sicheri, F. The crystal structure of an Eph receptor SAM domain reveals a mechanism for modular dimerization. *Nature Struct. Biol.* **6**, 44–49 (1999).
20. Thanos, C. D., Goodwill, K. E. & Bowie, J. U. Oligomeric structure of the human EphB2 receptor SAM domain. *Science* **283**, 833–836 (1999).
21. Hock, B. *et al.* PDZ-domain-mediated interaction of the Eph-related receptor tyrosine kinase EphB3 and the ras-binding protein AF6 depends on the kinase activity of the receptor. *Proc. Natl Acad. Sci. USA* **95**, 9779–9784 (1998).
22. Torres, R. *et al.* PDZ proteins bind, cluster, and synaptically colocalize with Eph receptors and their ephrin ligands. *Neuron* **21**, 1453–1463 (1998).
23. Buchert, M. *et al.* The junction-associated protein AF-6 interacts and clusters with specific Eph receptor tyrosine kinases at specialized sites of cell–cell contact in the brain. *J. Cell Biol.* **144**, 361–371 (1999).
24. Lin, D., Gish, G. D., Songyang, Z. & Pawson, T. The carboxyl terminus of B class ephrins constitutes a PDZ domain binding motif. *J. Biol. Chem.* **274**, 3726–3733 (1999).
25. Cowan, C. A., Yokoyama, N., Bianchi, L. M., Henkemeyer, M. & Fritsch, B. EphB2 guides axons at the midline and is necessary for normal vestibular function. *Neuron* **26**, 417–430 (2000).
26. Otwinowski, Z. & Minor, W. *Data Collection and Processing* 556–562 (SERC Daresbury Laboratory, Warrington, UK, 1993).
27. Project, C. C. The CCP4 suite: programs for X-ray crystallography. *Acta Crystallogr. D* **50**, 760–763 (1994).
28. Jones, T. A., Zou, J. Y., Cowan, S. W. & Kjeldgaard, I. Improved methods for binding protein models in electron density maps and the location of errors in these models. *Acta Crystallogr. A* **47**, 110–119 (1991).
29. Brünger, A. T. *X-PLOR v. 3.1 Manual* (Yale Univ. Press, New Haven, 1993).

Supplementary information accompanies the paper on Nature's website (<http://www.nature.com>).

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correction

Neurogenesis in the adult is involved in the formation of trace memories

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The original Fig. 2e–j is in error because it was based on incorrectly sorted brain tissue. A corrected figure is shown below and demonstrates that the anti-mitotic agent MAM does not affect the gross morphology or the volume of either the hippocampus or the cerebellum. The revised figure is based on a new set of tissue obtained from brains in experiment 3 that had been exposed to MAM ($n = 3$) or saline ($n = 3$) for 14 days and processed as described. Sections from each of these brains are presented. This error (and its correction) does not alter the main findings we reported, namely that treatment with MAM depletes newly generated neurons in the hippocampus and impedes learning of trace eyeblink conditioning in the adult rat. □

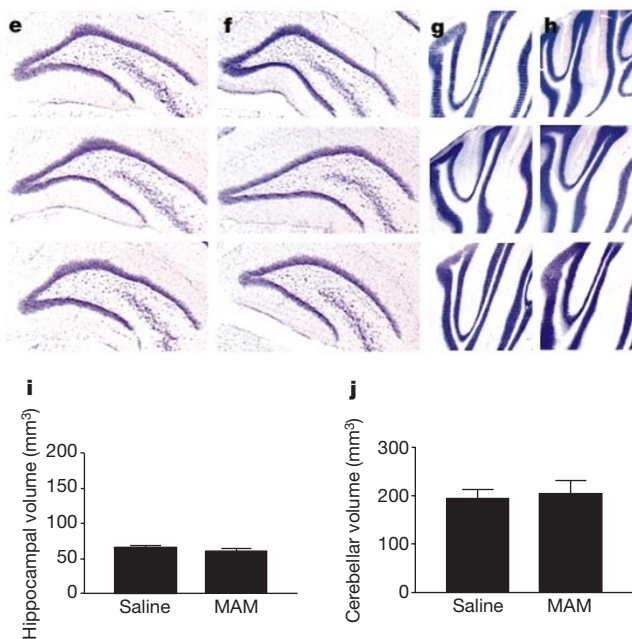


Figure 2e–j MAM treatment does not alter the gross morphology of the hippocampus or cerebellum. **e, f**, Cresyl violet stained sections of the dentate gyrus from three saline-treated (**e**) and three MAM-treated (**f**) adult rats. **g, h**, Cresyl violet stained sections of the cerebellar cortex from three saline-treated (**g**) and three MAM-treated (**h**) adult rats. **i, j**, Hippocampal (**i**) and cerebellar (**j**) volume (mean + s.e.m.).