

Neurite growth inhibitors restrict plasticity and functional recovery following corticospinal tract lesions

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Nature Neurosci. 1, 124–131 (1998).

We reported in the June 1998 issue of *Nature Neuroscience* that the monoclonal antibody IN-1 promotes collateral sprouting in the rat spinal cord, red nucleus and pons following a lesion to the corticospinal tract. This sprouting was accompanied by functional recovery. We also published a paper in the 15 June 1998 issue of *Journal of Neuroscience* (Vol. 18: 4744–4757), which described experiments that were performed in parallel in our laboratory and reached similar conclusions; specifically, whereas the *Nature Neurosci.* paper concentrated on spinal effects, the *J. Neurosci.* paper employed identical lesions and antibody treatments, and described in detail sprouting in red nucleus and pons as well as functional recovery. Although the datasets are largely unique to each paper, the experimental design and results were very similar. Moreover, some of the data presented in the two papers are also identical (Fig. 5b of the *Nature Neurosci.* paper and Fig. 4a of the *J. Neurosci.* paper), while other data represent different time points from the same animals (Fig. 6b of the *Nature Neurosci.* paper and Fig. 6a of the *J. Neurosci.* paper).

We greatly regret that neither of these papers cites the other, and that we failed to inform the editors of either *Nature Neuroscience* or *Journal of Neuroscience* of the existence of another closely related paper that was under consideration elsewhere. We also regret that an important result presented in the *J. Neurosci.* paper (the effect of a second lesion rostral to the first) was mistakenly described in the *Nature Neurosci.* paper as “W.J.Z., in preparation” while it was already in press at *Journal of Neuroscience*.

We apologize to the editors, referees and readers of both *Nature Neuroscience* and *Journal of Neuroscience* for these errors, and for any confusion which we may have caused.

A similar correction has been submitted to *Journal of Neuroscience*.

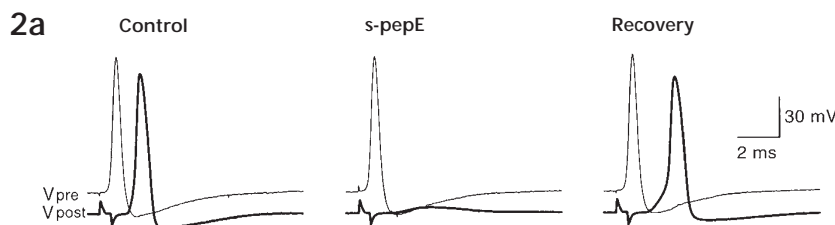
Martin E. Schwab, Michaela Thallmair, Werner Z'Graggen and Gerlinde Metz.

Two sites of action for synapsin domain E in regulating neurotransmitter release

Sabine Hilfiker, Felix E. Schweizer, Hung-Teh Kao, Andrew J. Czernik, Paul Greengard and George J. Augustine

Nature Neurosci. 1, 29–35 (1998).

In preparing Fig. 2a, the same presynaptic traces were inadvertently used for all three panels. The correct version, which is virtually identical to our published Fig. 2a, is shown here. The authors regret the error.



Also, because of an editorial error, the x-axes in Figs 2b, 2c, 2d, 3a, 3b, 3e and 5c were mislabeled as ‘ms.’ The labels should read ‘min.’

Cortisol levels during human aging predict hippocampal atrophy and memory deficits

Sonia J. Lupien, Mony de Leon, Susan de Santi, Antonio Convit, Chaim Tarshish, N.P.V. Nair, Mira Thakur, Bruce S. McEwen, Richard L. Hauger and Michael J. Meaney

Nature Neurosci. 1, 69–73

One of the references in this manuscript is incorrect. Reference 18 should read:

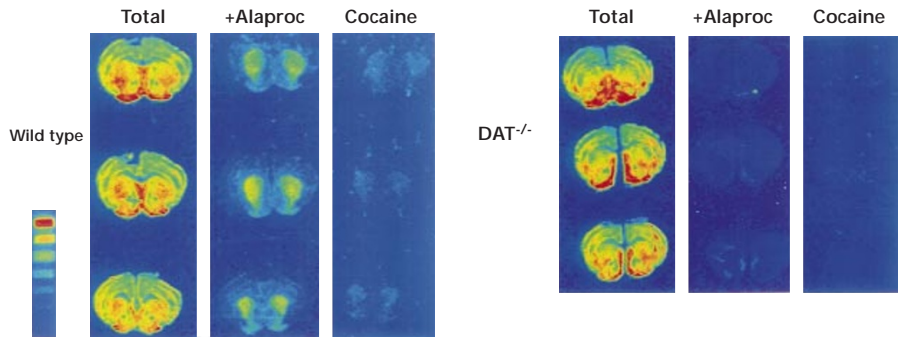
18. Convit, A. *et al* Specific hippocampal volume reductions in individuals at risk for Alzheimer’s disease. *Neurobiol. Aging* 18, 131–138 (1997).

Cocaine self-administration in dopamine-transporter knockout mice

Beatriz A. Rocha, Fabio Fumagalli, Raul R. Gainetdinov, Sara R. Jones, Robert Ator, Bruno Giros, Gary W. Miller and Marc G. Caron

Nature Neurosci. 1, 132–137 (1998).

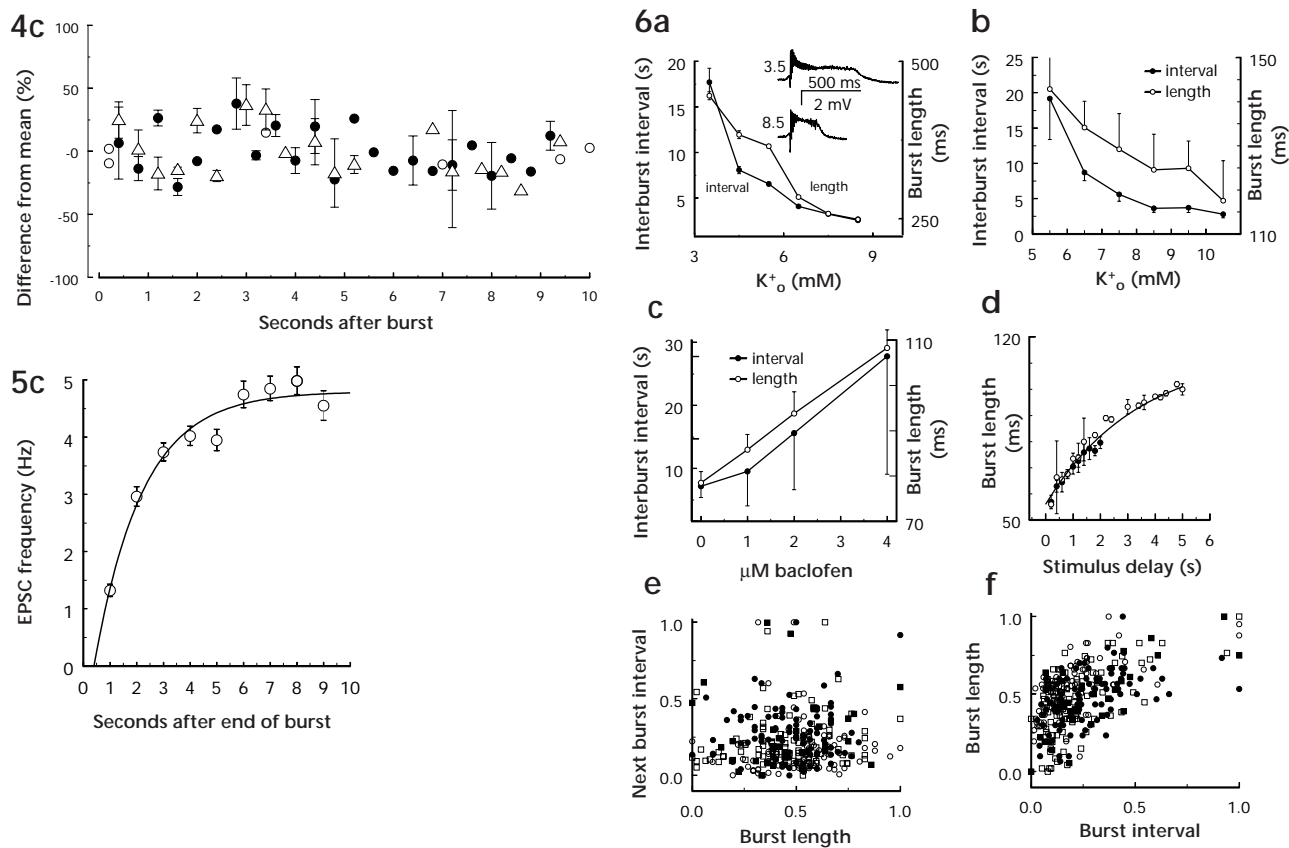
On page 134, Fig. 4 was inadvertently printed in black and white instead of color. The corrected version is printed below.



Presynaptic modulation of CA3 network activity

Kevin J. Staley, Mark Longacher, Jaideep S. Bains and Audrey Yee
Nature Neurosci. 1, 201–209 (1998).

Because of a printing error, Figs 4c, 5c and 6 were not properly reproduced. The corrected versions are printed below.



In addition, the penultimate sentence in the legend to Fig. 4b was inadvertently omitted. The corrected legend should read:

Fig. 4. (b) The experiment shown in (a) was repeated in a slice in which CA3 bursting was induced by tetanic stimulation of the CA3 pyramidal cell layer. The average interburst interval in this preparation was 13 seconds, which permitted longer delays between the end of a burst and glutamate application. As in (a), glutamate application at delays closer to the interburst interval triggered a burst more rapidly, but the initial response to glutamate was unchanged (inset). In this cell, bursts triggered calcium escape spikes. Glutamate was applied after every fourth burst in (b) and (c).