- The integrity of the scientific mission is a collective responsibility
- Content and data must be original and accurate
- Priority of data and ideas must be respected
- Authorship should reflect a significant intellectual contribution
- Every author shares responsibility
- Conflict of interest must be declared
- Original content and data should only be published once
- Pre-published material is confidential
- Research using animals and human subjects must be conducted ethically

The integrity of the scientific mission is a collective responsibility

- Society
- Politics
- Institutions
- Academia
- Scientific Societies
- Scientific Communities
- Authors
- Readers

Content and data must be original and accurate

It is essential that readers be able to trust the validity of published material. That trust permits readers to build on prior observations and thus facilitates the progress of knowledge. It also allows individuals to form opinions and make policies based on those observations.

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Falsification (altered in any way)
Misrepresenting(minimize the possibility of misinterpretation)

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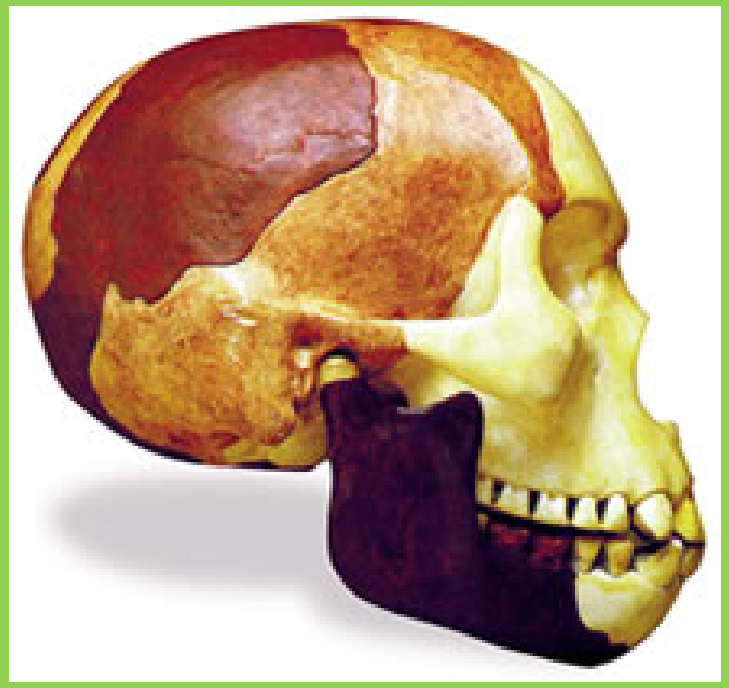

Piltdown man

Gregor Mendel

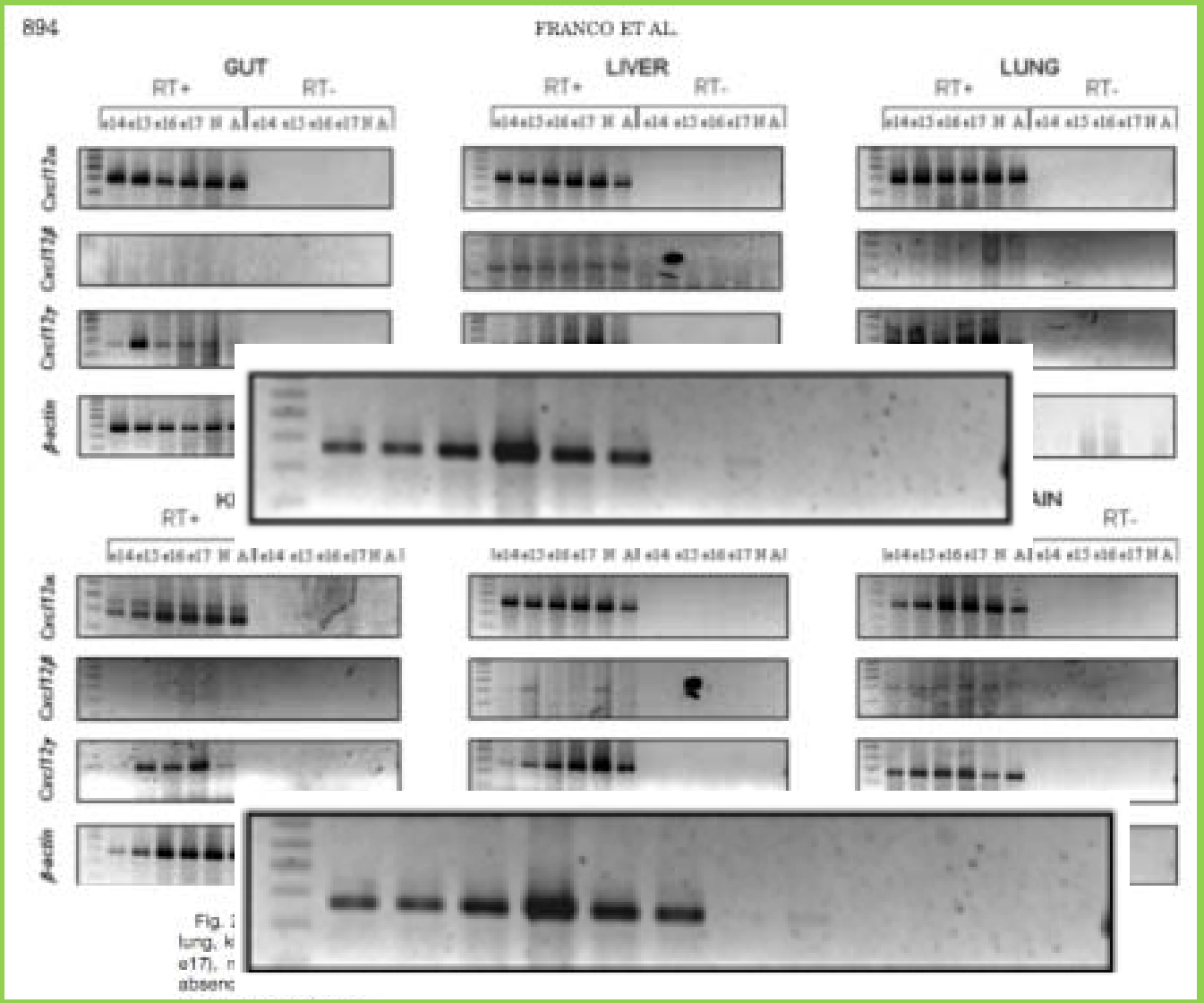

Fig. 1
lung, k
e17), r
absenc

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In the neuropil of the temporal cortex, TLN immunoreactivity appeared first at 2 postnatal months and was rapidly increased by 5 months. At first, labeling was weak and restricted to the cytoplasm of pyramidal neurons from 35 to 39 GW, but thereafter became diffuse and intense in the cortical layers, The staining was most intense in the molecular (Ist) layer, moderate in the external (IIIrd), and internal pyramidal (Vth) layers, and least intense in the external (IInd) and internal granular (IVth) layers. Immunoreactivities for synaptophysin and microtubule-associated protein (MAP) 2 appeared earlier than that for TLN, being detectable in the 25 GW cerebrum. In the hippocampus and temporal cortex, the neuronal perikarya was positively stained for synaptophysin at 25 GW, and their dendrites at 32–36 GW. The peak level of expression was reached at 37–40 GW. MAP2 staining was noted in the neuronal perikarya and dendrites at 25 GW. The intensity was increased by 32–36 GW, and decreased thereafter (Arii et al., 1999).

Western Blotting

Pieces of the cerebral cortex were taken at autopsy from the frontal lobe of six patients, aged from 21 GW to 14 years, and were kept frozen at -80°C until use. The tissues were thawed, stripped of meninges, and homogenized in an ice-cold buffer comprising 20 mM Tris-HCl, pH 7.6, 4 mM EDTA, 1 mM phenylmethylsulfonyl fluoride, and 1% Triton X-100. The homogenates were cleared by centrifugation at 12,000*g* for 30 minutes, and their protein contents were determined by Bradford's method. Proteins were then subjected to sodium dodecyl sulfate/polyacrylamide gel electrophoresis on a 7.5% gel with 30μg of protein loaded onto each lane, and were transferred to an Immobilon membrane (Millipore, Bedford, MA). After blocking with 8% skim milk in PBS, they were reacted with the TLN antibody at 4°C overnight, followed by biotinylated horse anti-mouse IgG (diluted 1:500) and alkaline phosphatase-conjugated avidin-biotin complex (both from Vector, Burlingame, CA) for 1 hour each, at room temperature. After each step, the membrane was washed extensively with PBS containing 0.1% Tween 20. The immunoproducts were visualized with a Vector Black substrate kit (Vector).

of a 25-GW fetus, there was no
vity. The hippocampus was the first
positivity at 29 GW. At 29–33 GW,
noted in the neuropil of the cornu
r and pyramidal layers) and area
layer) (Fig. 1A). At 35 GW, the
tivity was comparable to that of an
adult brain (Fig. 1C and E). In the molecular layer of the cornu ammonis, apical dendrites of the pyramidal neurons were intensely stained. Thereafter, the zone showing maximal staining moved from the middle to the upper third of the layer.

In the neuropil of the temporal cortex, TLN immunoreactivity appeared first at 2 postnatal months and was rapidly increased by 5 months. At first, labeling was weak and restricted to the cytoplasm of pyramidal neurons from 35 to 39 GW (Fig. 2), but thereafter became diffuse and intense in the cortical layers, The staining was most intense in the molecular (Ist) layer, moderate in the external (IIIrd), and internal pyramidal (Vth) layers, and least intense in the external (IInd) and internal granular (IVth) layers (Fig. 1B,D,F).

Immunoreactivities for synaptophysin and MAP2 appeared earlier than that for TLN (Tables 2 and 3), being detectable in the 25 GW cerebrum. In the hippocampus and temporal cortex, the neuronal perikarya was positively stained for synaptophysin at 25 GW, and their dendrites at 32–36 GW. The peak level of expression was reached at 37–40 GW. MAP2 staining was noted in the neuronal perikarya and dendrites at 25 GW. The intensity was increased by 32–36 GW, and decreased thereafter.

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2) drafting the article or revising it critically for important intellectual content;
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Fezf2 directs the differentiation of corticofugal neurons from striatal progenitors *in vivo*

Caroline Rouaux[1,2] & Paola Arlotta[1–4]

AUTHOR CONTRIBUTIONS
P.A. and C.R. conceived the experiments and wrote the manuscript. P.A. supervised the project and C.R. executed all the experiments.

A crosstalk between β1 and β3 integrins controls glycine receptor and gephyrin trafficking at synapses

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AUTHOR CONTRIBUTIONS
C.C. designed, performed and analyzed the experiments except for the *in vitro* phosphorylation assays and mass spectrometry and wrote the manuscript with help from the other authors. P.M. performed the *in vitro* phosphorylation assays. R.Y.T.-C. and D.R. performed mass spectrometry and analyzed data. I.M.M. supervised mass spectrometry and phosphorylation analyses. A.T. supervised the project.

The MAP kinase phosphatase MKP-1 regulates BDNF-induced axon branching

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AUTHOR CONTRIBUTIONS
F.J. and K.D. designed, performed and analyzed experiments. F.J., K.D. and M.V.C. wrote the manuscript. G.M. helped with the *in utero* electroporations. G.M. and A.M.B. provided mice and reagents.

Categorical speech representation in human superior temporal gyrus

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AUTHOR CONTRIBUTIONS
E.F.C. designed the experiments, collected the data and wrote the manuscript. E.F.C. and J.W.R. analyzed the data, evaluated results and edited the manuscript. J.W.R., N.M.B. and M.S.B. helped with data collection. K.J. and R.T.K. reviewed the manuscript.

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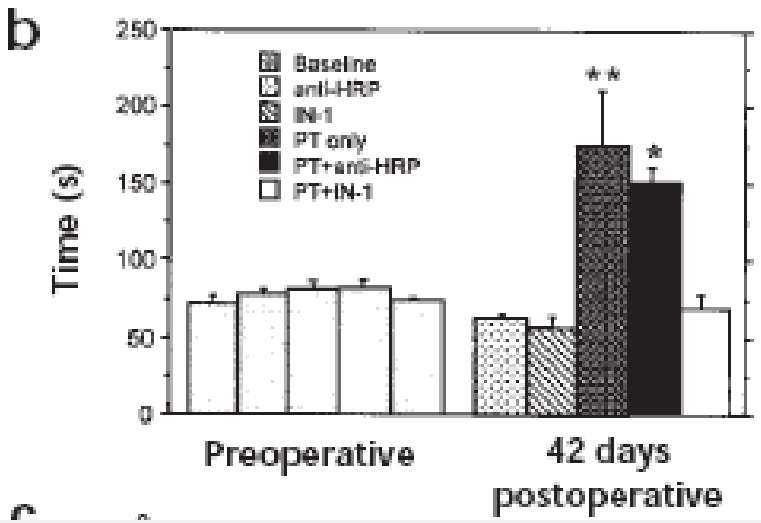

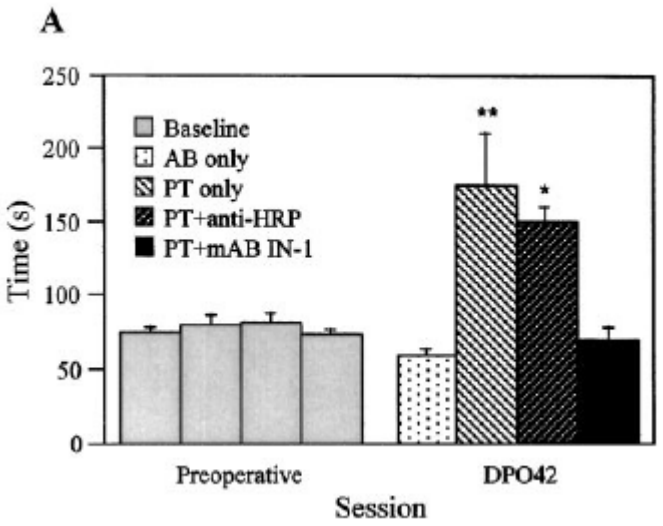

Neurite growth inhibitors restrict plasticity and functional recovery following corticospinal tract lesions

Michaela Thallmair, Gerlinde A.S. Metz, Werner J. Z'Graggen, Olivier Raineteau, Gwendolyn L. Kartje and Martin E. Schwab

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.