monkeys did have relatively smaller frontal cortices.

As Semendeferi and colleagues point out, a more informative analysis would look at the relative sizes of the subdivisions of the frontal cortex, and in particular the prefrontal cortex proper. However, identifying these areas from sulcal anatomy is very difficult, if not impossible, and a detailed cytoarchitectonic study — the only way to analyse these subdivisions definitively — would be extremely costly and very difficult. As a first step, the authors compared the sizes of the frontal cortices when the precentral gyri were excluded, on the grounds that the remaining cortex contains all of the prefrontal cortex and only a few other small cortical areas. Once again, they found no significant difference between the relative sizes of this section of cortex in humans and great apes.

How can we reconcile these results with previous studies that claimed to find large differences in the relative sizes of human and ape frontal cortex? One explanation might be sample size. Previous studies used much smaller groups of subjects and looked at fewer species, whereas Semendeferi and colleagues included several examples of every extant species of great ape. Differences in how the frontal cortex is defined could also have contributed.

Of course, size isn't everything. It is likely that the human frontal cortex differs in other ways from those of apes and monkeys — for example, in being more densely interconnected (as supported by data showing that more white matter underlies the frontal cortices in humans than in apes). In addition, specific areas of the frontal cortex might have evolved to be relatively larger at the expense of other subdivisions, without altering the overall size. But one thing is certain: we will have to consider more than just size if we are to figure out what makes humans so different.

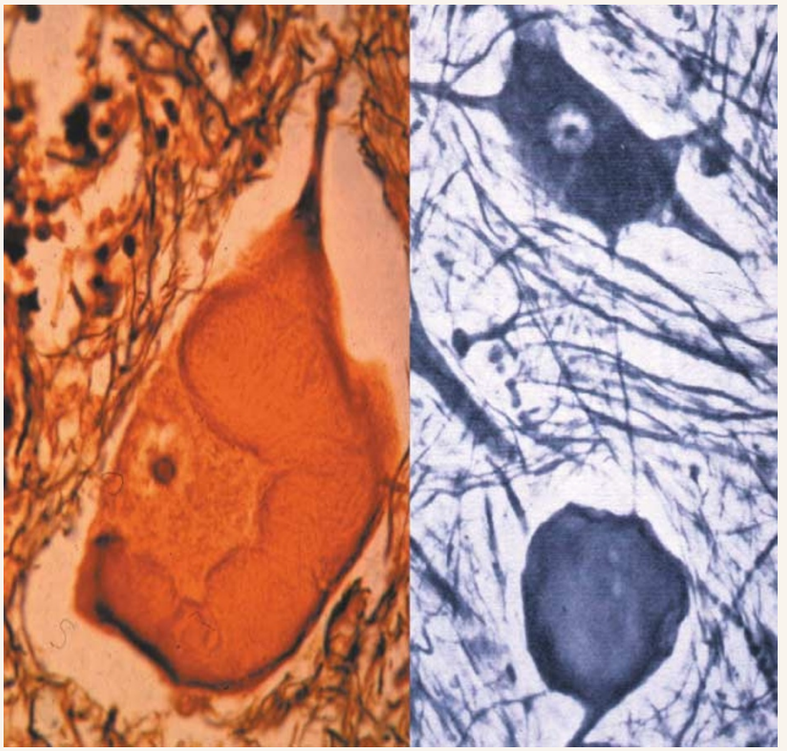
Rachel Jones

References and links

ORIGINAL RESEARCH PAPER Semendeferi, K. *et al*. Humans and great apes share a large frontal cortex. *Nature Neurosci*. 19 February 2002 (10.1038/nn814)

WEB SITES

Semendeferi's laboratory:
http://anthro.ucsd.edu/anthfac/semendeferi.html



Neurofilament aggregates distend a motor neuron in the spinal cord of a patient with ALS (left). A neurofilament-containing spheroidal swelling in the axon of a motor neuron from a patient with ALS (right).

IN BRIEF

DEVELOPMENT

Tangential migration in neocortical development.

Jiménez, D. *et al. Dev. Biol.* 25 February 2002 (10.1006/dbio.2002.0586)

Ventricle-directed migration in the developing cerebral cortex.

Nadarajah, B. *et al. Nature Neurosci.* **5**, 218–224 (2002)

It is known that some cortical neurons migrate tangentially from the basal telencephalon during development, but their precise site of origin has not been clear. Jiménez *et al*. have now shown that distinct populations of cortical neurons are derived from two regions — the medial and lateral ganglionic eminence. Nadarajah *et al*. have addressed a different but related question; namely, how do tangentially migrating cells know where to go once they have reached the cortex? Their data indicate that the cells initially migrate towards the cortical ventricular zone, where they acquire the information that determines their final position in the cortex.

AGEING

Under-recruitment and nonselective recruitment: dissociable neural mechanisms associated with aging.

Logan, J. M. *et al*. *Neuron* **33**, 827–840 (2002)

Using functional magnetic resonance imaging, Logan *et al*. found that older adults showed less recruitment of frontal regions during the self-initiated memory encoding of words than did younger adults. This under-recruitment could be reversed if the memory encoding was supported, for example, by requiring semantic elaboration. A second difference between younger and older adults — nonselective activation of multiple frontal regions for both words and faces — was not reversed by this strategy. The results might have implications for understanding and ameliorating age-related cognitive decline.

ION CHANNELS

Models of the extracellular domain of the nicotinic receptors and of agonist- and Ca^{2+}-binding sites.

Le Novère, N. *et al*. *Proc. Natl Acad. Sci. USA* **99**, 3210–3215 (2002)

Experimentally based model of a complex between a snake toxin and the α7 nicotinic receptor.

Fruchart-Gaillard, C. *et al*. *Proc. Natl Acad. Sci. USA* **99**, 3216–3221 (2002)

These two papers constitute significant progress in the elucidation of the extracellular domain of nicotinic acetylcholine receptors (nAChRs). The authors took advantage of the crystal structure of a molluscan acetylcholine-binding protein (AChBP), which shows substantial homology to nAChRs, and constructed three-dimensional models of the receptor. They identified key differences between AChBP and nAChRs in the binding pocket, and provided a structural basis for previous mutagenesis experiments. In the second paper, the authors model the α7 nAChR subunit in association with a toxin antagonist, identifying the interaction sites and paving the way to the design of new receptor blockers.