Genetic dissection of Alzheimer's disease and related dementias: amyloid and its relationship to tau

John Hardy, Karen Duff, Katrina Gwinn Hardy, Jordi Perez-Tur and Mike Hutton Nature Neurosci 1, 355–358 (1998).

In preparing Table 1, an old version with incomplete data was inadvertantly used. The correct version is shown here. The authors regret the error.

Table 1: Tau mutations relative to FTDP17 pathology					
Mutation type	Mutations	Soluble tau	Tau inclusions	Tau filaments	Glial inclusions?
Missense NOT exon 10	G272V, V337M, R406W	Normal ratio of 4 to 3 repeat	All six isoforms	AD-like PHF***	No
Missense in exon 10	P301L	Normal ratio of 4 to 3 repeat**	4 repeat predominates	LPF*** ('Twisted ribbons')	Yes
Exon 10 5' splice site	+13* +14* +16* +3*	Increased 4 repeat	4 repeat predominates	LPF*** ('Twisted ribbons')	Yes

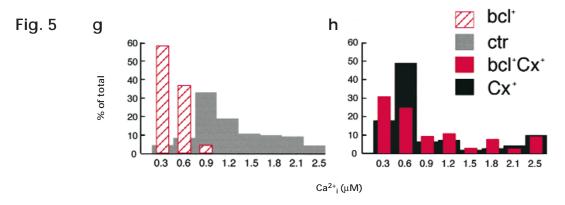
^{*} Exon 10 5'-splice-site mutations numbered from 3' end of exon 10

Gap-junction-mediated propagation and amplification of cell injury

Jane H.-C. Lin, Helga Weigel, Maria Luisa Cotrina, Shujun Liu, Earl Bueno, Anker J. Hansen, Thomas W. Hansen, Steven Goldman and Maiken Nedergaard

Nature Neurosci. 1, 494-500 (1998).

In preparing Fig. 5g and h, the bars were inadvertantly colored incorrectly. The correct version is shown here. The authors regret the error.



errata

Functional anatomy of saccadic adaptation in humans

M. Desmurget, D. Pélisson, C. Urquizar, C. Prablanc, G.E. Alexander and S.T. Grafton *Nature Neurosci.* 1, 524–528 (1998).

Because of an editorial error, the last line of the Discussion section was misprinted. The sentence should read as follows:

From a theoretical point of view, the congruence between our data and observations emphasizing the role of the medial cerebellum for saccadic adaptation in monkeys supports the hypothesis that saccadic adaptation involves similar neural circuits in humans and animals.

^{**}Inferred from unchanged ratio of exon 10+/- RNA

^{***}LPF, long periodicity filaments; PHF, paired helical filaments

Proposed relationship between site of mutation in *tau* gene and type of pathogenic *tau* mutation. Proposal based on data taken from refs 50–54 and unpublished data from the authors' laboratories.