CORRIGENDUM

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For the article Shelley & Colquhoun (2005), we regret to say that, after sequencing from end to end all the DNAs that have been used in experiments with nicotinic receptors, we have found that the clone used in this paper did not contain the ε L78P. mutation. This has been confirmed with Dr D. Beeson

(Neurosciences Group Weatherall Institute of Molecular Medicine), who supplied the DNA.

The actual mutation used in the study was ε S278del (c.833-835delCTC). It is mutation ε S278del that causes a slow channel syndrome, not ε L78P. The ε S278del mutation occurred in a sequence that was not quite the same as that of the wild-type human muscle type nicotinic acetylcholine receptor, epsilon subunit cDNA as deposited in GenBank (reference sequence (http://www.ncbi.nlm.nih.gov/entrez/viewer. fcgi?val=NM_000080). The clone supplied to us contained two rare missense polymorphisms, eV108A and eT117S, that were present in the original clone isolated in Beeson's lab from a lambdagt10 human muscle cDNA library.

Reference

Shelley C & Colquhoun D (2005). A human congenital myasthenia-causing-mutation (εL78P) of the muscle nicotinic acetylcholine receptor with unusual single channel properties. J Physiol 564, 377–396.