(e4− = 7, e4+ = 6). These cell sizes are too small to allow exploration of a differential APOE treatment response, but our ongoing larger clinical trial is powered to determine such an effect.[3] We hope to soon have data to address this important question.

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VOLUNTARY PARTIAL RETRACTION

Voluntary partial retraction of: Recessive inheritance and variable penetrance of slow-channel congenital myasthenic syndromes

The authors state that they inadvertently published incorrect data in the analysis of one of the mutations in this manuscript. The data presented for the analysis of mutation AChR ϵL78P (c.233C>T) was for the analysis of mutation AChR ϵS278del (c.833_835delCTC). It is mutation ϵS278del that causes a slow channel syndrome, not ϵL78P. The study of the clinical features within the kinship harboring mutation ϵS278del finds that the slow channel syndrome phenotype is revealed through the presence of a low expression mutation, ϵR217L, on the second allele so the observational hypothesis in the original manuscript is valid. In the experiments described, the core AChR ϵ-subunit cDNA varies from the Genbank reference sequence (http://www.ncbi.nlm.nih.gov/entrz/viewer.fcgi?val=NM_000080) by two rare missense polymorphisms, ϵV108A and ϵT117S, that were present in the original clone isolated from a lambdagt10 human muscle cDNA library.

Croxen R, Hatton C, Shelley C, Brydson M, Chauplannaz G, Oosterhuis H, Vincent A, Newsom-Davis J, Colquhoun D, Beeson D. Recessive inheritance and variable penetrance of slow-channel congenital myasthenic syndromes. Neurology 2002;59:162–168.