($\epsilon 4- = 7$, $\epsilon 4+ = 6$). 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.

Suzanne Craft, PhD, Seattle, WA

Disclosure on article to which this Correspondence refers: W.D. is a founding member of Kurve Technology, the maker and patent holder of the electronic atomizer used in this study. W.D. holds equity interest in excess of \$10,000 in Kurve Technology. The remaining authors have nothing to disclose.

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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 ϵ L78P (c.233C>T) was for the analysis of mutation AChR ϵ S278del (c.833_835delCTC). It is mutation ϵ S278del that causes a slow channel syndrome, not ϵ L78P. The study of the clinical features within the kinship harboring mutation ϵ S278del finds that the slow channel syndrome phenotype is revealed through the presence of a low expression mutation, ϵ R217L, 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/entrez/viewer.fcgi?val=NM_000080) by two rare missense polymorphisms, ϵ V108A and ϵ T117S, that were present in the original clone isolated from a lambda₁₀ 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.

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