1. Chaturvedi S, Zivin J, Amerenco P, et al. Effect of atorvastatin in elderly patients with a recent stroke or transient ischemic attack. *Neurology* 2009;72:688–694.
2. The Stroke Prevention by Aggressive Reduction in Cholesterol Levels (SPARCL) investigators. High-dose atorvastatin after stroke or transient ischemic attack. *N Engl J Med* 2006;355:549–559.
3. Pfeffer MA, Jarcho JA. The charisma of subgroups and the subgroups of CHARISMA. *N Engl J Med* 2006;354:1744–1746.
4. Rothwell PM. Treating individuals 2: subgroup analysis in randomised controlled trials: importance, indications, and interpretation. *Lancet* 2005;365:176–186.

NEURODEGENERATION ASSOCIATED WITH GENETIC DEFECTS IN PHOSPHOLIPASE A₂

To the Editor: In their article, Gregory et al.¹ describe 2 main pathologic features of infantile neuroaxonal dystrophy and idiopathic neurodegeneration with brain iron accumulation: axonal spheroids and iron deposits. The authors describe these 2 elements as unrelated and induced by the defective phospholipase A₂.

However, I believe that the accumulated iron plays a role in spheroid formation. As the authors note, the association of spheroids and iron accumulation appears in another disease, pantothenate kinase-associated neurodegeneration (formerly Hallervorden-Spatz syndrome).² Iron-positive macrophages were described in another rare disease, hereditary diffuse leukoencephalopathy with spheroids.³

Moreover, the combination of axonal spheroids and iron deposits is seen in non-metabolic disorders, such as superficial siderosis of the CNS. In this disorder, chronic or intermittent spilling of blood into the subarachnoid space and spread of heme by the CSF causes infiltration of iron and ferritin in the cortex

associated with abundant axonal spheroids.⁴ Spheroids were described in the vicinity of hemosiderin-laden macrophages surrounding arteriovenous malformations, and even in hemorrhagic infarctions.⁵ Thus, accumulation of iron alone may induce spheroid formation.

The spheroids of the hereditary diseases may differ from those of superficial siderosis of the CNS. However, the strong association of spheroids and iron in hereditary diseases and acquired disorders suggests a causative relation, and perhaps the toxic effect of iron has a role in spheroid formation.

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Editor's Note: The authors of the article were offered the opportunity to respond but declined.

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1. Gregory A, Westaway SK, Holm IE, et al. Neurodegeneration associated with genetic defects in phospholipase A₂. *Neurology* 2008;71:1402–1409.
2. Hayflick SJ. Pantothenate kinase-associated neurodegeneration (formerly Hallervorden-Spatz syndrome). *J Neurol Sci* 2003;207:106–107.
3. Marotti JD, Tobias S, Fratkin JD, Powers JM, Rhodes CH. Adult onset leukodystrophy with neuroaxonal spheroids and pigmented glia: report of a family, historical perspective, and review of the literature. *Acta Neuropathol* 2004;107:481–488.
4. Koeppen AH, Barron KD. Superficial siderosis of the central nervous system: a histological, histochemical and chemical study *J Neuropathol Exp Neurol* 1971;30:448–469.
5. Sadeh M, Sandbank U. Neuroaxonal dystrophy and hemosiderin in the central nervous system. *Ann Neurol* 1980;7:286–287.

CORRECTION

Association between late-life body mass index and dementia: The Kame Project

In the article "Association between late-life body mass index and dementia: The Kame Project" by T.F. Hughes et al. (*Neurology*® 2009;72:1741–1746), reference 6 (Atti et al.) should not be referenced in the Introduction, page 1741, paragraph 1, sentence 2. All other uses of this reference are correct. The authors regret the error.

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Association between late-life body mass index and dementia: The Kame Project

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