had previously been reported in two familial PD cases, one sporadic PD case, and one control individual.⁶

We agree that at this point p.R793M is not a definitively pathogenic mutation, but it may confer risk of neurodegeneration. Our point was that *LRRK2* mutations, which have previously been considered to manifest clinically as parkinsonism, may exhibit cognitive phenotypes.

As other screens for *LRRK2* mutations have been carried out primarily in patients with movement disorders, the assumption that *LRRK2* mutations uniformly exhibit parkinsonian clinical phenotypes may be premature.

A. Chen-Plotkin, W. Yuan, C. Anderson, E. McCarty Wood, H.I. Hurtig, C. Clark, B.L. Miller, V.M.-Y. Lee, J.Q. Trojanowski, M. Grossman, V.M. Van Deerlin, Philadelphia, PA

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CORRECTION

Neurologic consequences of autoimmune polyglandular syndrome type 1

In the article “Neurologic consequences of autoimmune polyglandular syndrome type 1” by Joseph R. Berger, Allison Weaver, and John Greenlee, (*Neurology*® 2008;70:2248–2251), George E. Wahlen was inadvertently listed as an author. Dr. Greenlee is from the George E. Wahlen Veterans Affairs Medical Center and University of Utah. The publisher regrets the error.

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