than in a previous report.[4] This formally excludes an ascertainment bias in the previous study. Given the low frequency of G2019S carriers in healthy controls (~1%), the relative risk for a carrier to develop PD is ~41 in this population. Intriguingly, we found a healthy control who was homozygous for the G2019S mutation. This case and also the large number of homozygous mutation carriers in North Africa[5] are likely due to the high rate of consanguinity in this population. However, this asymptomatic 41-year-old individual was 14 years younger than the average age at onset of affected carriers (~55 years).

Our study also suggested that G2019S mutation carriers might be more likely to develop levodopa-related dyskinesias than patients without this mutation. However, although several genes have been suggested to confer a genetic predisposition to levodopa-induced motor complications,[6,7] a similar role for the *LRRK2* G2019S mutation, suggested by our study, needs further confirmation.

**Members of the French Parkinson's Disease Genetics Study Group (FPDGSG) are listed in the appendix.*

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Supported by the Agence Nationale de la Recherche (ANR-05-NEUR-019).

Disclosure: The authors report no disclosures.

Received March 11, 2008. Accepted in final form June 16, 2008.

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ACKNOWLEDGMENT

The authors thank the patients and their families. They thank Merle Ruberg for critical reading of the manuscript, Jean-Christophe Corvol for help with the statistical analyses, and the DNA and Cell Bank of the IFR 070 for sample preparation.

APPENDIX

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CORRECTION

CNS aquaporin-4 autoimmunity in children

In the article "CNS aquaporin-4 autoimmunity in children" by A. McKeon et al. (*Neurology*® 2008;71:93–100), the unit of measurement for AQP-IgG detected in serum by immunoprecipitation assay should read nmol/L (not pmol/L).

CNS aquaporin-4 autoimmunity in children

Neurology 2008;71;1552
DOI 10.1212/01.wnl.0000338461.66925.93