than in a previous report. 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 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, 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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APPENDIX


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

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