

two of whom had symptom onset at 67 and 68 years of age. One of the two affected siblings carried the pathogenic hexanucleotide expansion; the other did not. All members of this family were heterozygotes for *APOE4* and therefore had a comparatively high risk of Alzheimer's disease.<sup>2</sup>

There are two possible explanations for our data. The first possibility is that the hexanucleotide repeat expansion gives rise to Alzheimer's disease. The second and more likely possibility, which is supported by the autopsy findings, is that these subjects had amnesic frontotemporal dementia that was misdiagnosed as probable Alzheimer's disease. If the second possibility holds true, then it is likely to be a consistent feature across cohorts of patients with Alzheimer's disease, especially since postmortem studies show the diagnostic accuracy of clinically probable Alzheimer's disease to be 83%.<sup>5</sup> However, the prevalence of the repeat expansion in persons with symptoms of Alzheimer's disease and without a family history of the disease is probably less than the prevalence we report here. Regardless, the availability of a test for this genetic mutation may provide an opportunity to correct the misclassification of frontotemporal dementia as Alzheimer's disease in current and future patients.

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#### CORRECTION

A Phase 3 Trial of Bevacizumab in Ovarian Cancer (December 29, 2011;365:2484-96). In the Patients subsection of Results (page 2487), the final sentence should have read, “. . . and 26% had more than 1.0 cm of residual disease after surgical debulking,” rather than “. . . and 26% had 71.0 cm of residual disease.” We regret the error. The article is correct at NEJM.org.