the rest regarding neuropathologic markers. The presence of comorbid depression in Alzheimer disease (AD) has been demonstrated to correspond to increases in AD-related neuropathologic changes beyond age, sex, level of education, and cognitive status. Moreover, lifetime history of depression in patients with AD has been associated with higher levels of plaque and tangle formation in the hippocampus.

The authors did not provide information on the use of psychotropic agents such as antidepressants or lithium. Antidepressants or mood stabilizers may not only counteract depression, but also exert neuroprotective and neurotrophic actions extending beyond mood effects.

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Author Response: Robert S. Wilson, Chicago:

The authors thank Kunte et al. for their comments. We agree that the levels of depressive symptoms in the cohort were mostly mild (i.e., 54.6% with none, 30.7% with 1 or 2, and 14.7% with 3 or more). It is surprising that the variability within this relatively narrow range of depression severity accounted for nearly 5% of the variance in rates of cognitive decline (after adjustment for demographic and postmortem pathologic variables). If the cohort contained a higher proportion of participants with severe depression (defined psychometrically or diagnostically), the association of depression with cognitive decline would likely have been stronger. We agree that our findings do not rule out the possibility that depression only influences the accumulation of dementia-related pathologies when it is severe. We plan to investigate this possibility. We agree that antidepressants may have a neuroprotective effect, but we have not yet seen evidence suggestive of this.

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CORRECTION

MS disease activity in RESTORE: A randomized 24-week natalizumab treatment interruption study

In the article “MS disease activity in RESTORE: A randomized 24-week natalizumab treatment interruption study” by R.J. Fox et al. (Neurology® 2014;82:1491–1498), the supplemental file listing all study coinvestigators and sites was omitted. It is now available as supplemental data with the article. The authors regret the error.

Author disclosures are available upon request (journal@neurology.org).
MS disease activity in RESTORE: A randomized 24-week natalizumab treatment interruption study

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