Correspondence

INTELLECTUAL ENRICHMENT LESSENS THE EFFECT OF BRAIN ATROPHY ON LEARNING AND MEMORY IN MULTIPLE SCLEROSIS

To the Editor: Sumowski et al. showed that greater intellectual enrichment lessened the negative effect of brain atrophy on cognitive performance in multiple sclerosis (MS). Patients with MS with high intellectual enrichment performed similarly in memory tasks regardless of brain atrophy values on MRI while brain atrophy was associated with lower performances in patients with low intellectual enrichment.

We previously showed that educational background protects cognitive efficiency in patients with newly diagnosed relapsing-remitting MS (RRMS). Low-educated (LE) patients with RRMS, but not high-educated (HE) patients, had lower performances than matched healthy controls on most neuropsychological tests including the memory test used by Sumowski et al. However, cognitive scores between LE and HE controls showed no difference. LE and HE patients with RRMS did not differ regarding age, gender, disability, or MRI measures, so the effect of education on cognition was not attributable to a different distribution by chance.

Sumowski et al. only observed a correlation between brain atrophy and cognition in patients with lower reserve, but not patients with higher reserve. In contrast, Bonnet et al. found only one correlation between MRI measures and cognition in patients with lower reserve, but several correlations among patients with higher reserve.

The cognitive reserve hypothesis states that patients with higher reserve can withstand more severe neurologic disease before suffering cognitive decline. The negative impact of neuropathology on cognition is weaker among higher reserve patients, as we have shown in MS and others have shown in aging and Alzheimer disease. In this context, the findings of Bonnet et al. conflict with the cognitive reserve literature, and therefore prompted this review of their study.

Bonnet et al. defined low reserve as educational attainment less than 12 years. However, the LE MS group had much lower verbal intelligence than the LE control group. Considering that verbal intellectual decline is very rare in MS, the MS group probably had lower intelligence before disease onset. The observed cognitive differences between the LE MS and control groups likely represent premorbid differences rather than disease-induced cognitive decline, especially given the short disease duration of the MS group.

Further studies are needed to understand the compensatory effect of intellectual background on cognitive efficiency preservation in patients with MS. The ceiling effect due to the progression of the disease should also be elucidated.

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Disclosure: Dr. Bonnet has received speaker honoraria from Bayer Schering Pharma. Dr. Brochet serves on scientific advisory boards for Bayer Schering Pharma, Novartis, and Merck Serono; received funding for travel or speaker honoraria from Bayer Schering Pharma, Merck Serono, Biogen Idec, Novartis, and Teva Pharmaceutical Industries Ltd./Sanofi-Aventis; serves as LEN editor for SEP et Neurosciences; and has received institutional research support from Biogen Idec, Novartis, Roche, Sanofi-Aventis, Bayer Schering Pharma, Teva Pharmaceutical Industries Ltd., Peptimmune, Eli Lilly and Company, and AB Science.

Reply from the Authors: We reported negative correlations between brain atrophy and cognition in patients with MS with lower reserve, but not patients with higher reserve. In contrast, Bonnet et al. found only one correlation between MRI measures and cognition in patients with lower reserve, but several correlations among patients with higher reserve.

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Because lower cognition among patients with MS was likely developmental rather than disease-induced, a correlation between MRI measures and cognition cannot be expected. Among HE patients with MS, Bonnet et al. reported correlations between MRI parameters and performance on 8 cognitive tasks. However, patients with MS only differed from controls on one task, suggesting that most of the correlations were due to an uncontrolled nondisease variable, such as age, which likely correlates with both cognition and MRI measures. If so, correlations should also be evident among controls, but these analyses were not performed. Finally, differential statistical power is another explanation for fewer correlations among LE patients (n = 19) relative to HE patients (n = 24), especially given the high risk of type I error (5 MRI parameters \times 15 cognitive scores = 75 correlations performed for each group, with an uncorrected \( \alpha \) of 0.05).

The cognitive reserve literature supports the notion that neuropathology is more likely related to cognition among patients with lesser reserve,1,4–7 and the exception posed by Bonnet and colleagues2 appears confounded.

James F. Sumowski, West Orange, NJ

Disclosure: See original article for full disclosure list.


**CORRECTION**

An unusual cause of isolated vomiting

In the NeuroImage “An unusual cause of isolated vomiting” by R. Schlaeger and M. Sollberger (Neurology® 2010;75:1303), the small ischemic lesion shown in the figure is not in the left area postrema, but likely a few millimeters above. The authors are still convinced that the depicted lesion is related to therapy-refractory vomiting, either by affecting parts of a network of interacting neuronal structures related to vomiting or by irritation of the area postrema, for instance by ischemia-induced, transitory bleeding on the lower surface of the fourth ventricle. The authors regret this error.
An unusual cause of isolated vomiting

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