Editors' Note: In WriteClick this week, Kumfor et al. comment on the differences between brain reserve and cognitive reserve in reference to "Cognitive reserve in frontotemporal degeneration: Neuroanatomic and neuropsychological evidence." Authors Placek et al. agree that more research is required to better characterize both mechanisms. Malhotra et al. correctly comment that in the NeuroImage "Tuberculous optochiasmatic arachnoiditis and vision loss," T1-weighted postcontrast coronal images are shown rather than T2-weighted images, as labeled in the legend. A correction appears on page 1591.

—Megan Alcauskas, MD, and Robert C. Griggs, MD

LETTER RE: COGNITIVE RESERVE IN FRONTOTEMPORAL DEGENERATION: NEUROANATOMIC AND NEUROPSYCHOLOGICAL EVIDENCE

Fiona Kumfor, Sydney, Australia; Cristian E. Leyton, Boston, MA; Olivier Piguet, Sydney, Australia: In the article by Placek et al.¹, letter fluency was used as a proxy of executive function and as one of the tasks to assess cognitive reserve (CR). Letter fluency relies on multiple cognitive processes and, in such a diverse clinical sample, is unlikely to reflect a single neurocognitive process. These findings are therefore difficult to interpret.

Further, the results seem interpreted by conflating 2 distinct, but related, mechanisms: brain reserve (i.e., passive process where larger brains are less affected by damage than smaller brains) and CR (i.e., active use of compensatory processes to maintain function despite advancing neuropathology).² Accordingly, larger brain volume appears supportive of greater brain reserve, whereas better letter fluency may reflect greater CR. These 2 mechanisms may help reconcile apparently contradictory findings in dementia, whereby higher CR is associated with shorter disease duration in Alzheimer disease (AD),³ but longer duration in frontotemporal dementia.⁴ It is plausible that, in AD, compensatory mechanisms can delay symptomatology, whereas, in frontotemporal dementia, brain reserve may be more important. Longitudinal studies that combine behavioral and neuroimaging data are likely to shed light on this important issue.⁵

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AUTHOR RESPONSE: COGNITIVE RESERVE IN FRONTOTEMPORAL DEGENERATION: NEUROANATOMIC AND NEUROPSYCHOLOGICAL EVIDENCE

Katerina Placek, Lauren Massimo, Murray Grossman, Corey T. McMillan, Philadelphia: Letter fluency is a verbally mediated measure of executive control and we agree that it is a complex task. In our study, we demonstrated in patients with frontotemporal degeneration (FTD) that higher cognitive reserve (CR) and higher frontal gray matter density are selectively associated with superior performance on letter fluency and not on the Boston Naming Test.¹ Prior work demonstrated that inferior frontal gyrus and frontal lobe white matter support letter fluency task performance.^{2,3} Our observations converge with these prior demonstrations.

We agree that additional longitudinal studies are necessary to distinguish between active compensatory and passive brain reserve mechanisms. Our work,⁴ associating higher CR with early frontal atrophy and prolonged survival in autopsy-proven FTD, contrasts with observations in AD that show shorter survival in patients with higher CR and later frontal atrophy.⁵ While our imaging methods account for interindividual differences in intracranial volume,⁶ and control for differences in disease duration and age, we cannot rule out the possibility that individuals with higher CR are able to sustain cognitive function due to greater neuronal richness or frontal brain connectivity. Nonetheless, these 2 studies provided a valuable start to disentangling the mechanisms underlying CR.

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LETTER RE: TUBERCULOUS OPTOCHIASMATIC ARACHNOIDITIS AND VISION LOSS

Hardeep S. Malhotra, Neeraj Kumar, Ravindra Kumar Garg, Lucknow, India: The NeuroImage by Drs. Chee and Dinkin included T1-weighted gadolinium contrast coronal images instead of T2-weighted sequences. The legend should be revised to include more details, such as having performed a shunt procedure, as evident from the image.¹

1. Chee RI, Dinkin MJ. Tuberculous optochiasmatic arachnoiditis and vision loss. *Neurology* 2016;87:1845.

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AUTHOR RESPONSE: TUBERCULOUS OPTOCHIASMATIC ARACHNOIDITIS AND VISION LOSS

Marc J. Dinkin, Ru-Ilk Chee, New York: We thank Malhotra et al. for pointing out that our NeuroImage was of T1 postcontrast coronal images, not T2, and apologize for the error.¹

Malhotra et al. are also correct in observing a right-frontal ventriculoperitoneal shunt, which had been placed to treat hydrocephalus associated with tuberculous meningitis. Hydrocephalus is a common feature of this disease, occurring in up to 26% of adults and 90% of children,^{2,3} due to either an adhesive leptomeningitis in the basal cisterns (communicating) or obstruction of the aqueduct or fourth ventricle (noncommunicating). Shunting for tuberculous hydrocephalus demonstrated substantial clinical improvement in 7 children, 3 of whom had near complete recovery of vision after weeks of blindness, attributed to reduced compression on the ophthalmic arteries and optic apparatus by exudate and dilated ventricles.⁴

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CORRECTION

Tuberculous optochiasmatic arachnoiditis and vision loss

In the NeuroImage “Tuberculous optochiasmatic arachnoiditis and vision loss” by R.I. Chee and M.J. Dinkin,¹ the image should have been labeled “T1 postcontrast coronal MRI” rather than “T2 coronal MRI.” The authors regret the error.

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Tuberculous optochiasmatic arachnoiditis and vision loss

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