CORRECTION

Structural and functional brain connectivity in presymptomatic familial frontotemporal dementia

In the article "Structural and functional brain connectivity in presymptomatic familial frontotemporal dementia" by E.G.P. Dopper et al. (*Neurology*® 2013;80:814–823), there is an error in the DNA results in 6 subjects. An Editorial Expression of Concern was published (*Neurology* 2014;82:735) and the authors have reanalyzed the data. Reanalysis of data concluded that there was significantly reduced resting-state functional connectivity within the salience network and reduced structural connectivity in the uncinate fasciculus in presymptomatic carriers, compared with controls, in the absence of gray matter atrophy. These findings support the conclusion of the original article; see authors' full correction below. The authors regret the error.

In our article published in the February 26, 2013, issue of *Neurology*, we reported alterations in structural and functional connectivity in carriers of *progranulin* (*GRN*) and *microtubule-associated protein tau* (*MAPT*) mutations before the onset of symptoms of frontotemporal dementia (FTD).[1] In the follow-up study of our cohort of 75 at-risk individuals, we discovered an erroneous interchange of DNA results in 4 subjects and incorrect laboratory results in 2 additional subjects in our baseline article. Therefore, we reanalyzed our baseline data to investigate to what extent this error affected our results.

Similar to the reported results, reanalysis shows no differences in neuropsychological results between carriers and controls, except for significantly worse performances on the Letter Digit Substitution Test in carriers compared with controls. Reported age correlations with Stroop III and Happé cartoon performances in carriers, but not controls, are still present. In addition, there is a significant correlation between Rivermead Behavioral Memory Test performances with higher age in carriers, whereas the correlation for Ekman faces is no longer significant.

As reported, carriers and controls show no statistical differences in gray matter volume, except for a small cluster of higher volume in the right precentral gyrus in carriers that emerged in the reanalysis.

The reported differences in fractional anisotropy and radial diffusivity in frontotemporal white matter tracts are no longer significant, but using the uncinate fasciculus and forceps minor, tracts most consistently affected in FTD,[2–6] as regions of interest,[7] we find significantly reduced fractional anisotropy in the right uncinate fasciculus in carriers compared with controls.

The main reported resting-state fMRI findings were (1) a correlation between reduced frontoinsula (FI) connectivity with the anterior midcingulate cortex (aMCC) and age in carriers but not controls, and (2) reduced aMCC connectivity with parietal regions in carriers compared with controls. Both findings are still present in our reanalysis. In addition, we now find significantly decreased aMCC connectivity with FI and surrounding structures in carriers compared with controls, which strengthens our conclusion that salience network functional connectivity deteriorates prior to symptom onset. We also found reduced FI connectivity with several temporal and occipital regions.

To conclude, reanalysis of our data after error correction showed significantly reduced resting-state functional connectivity within the salience network and reduced structural connectivity in the uncinate fasciculus in presymptomatic carriers compared with controls, in the absence of gray matter atrophy. These findings support the conclusion of our original article that resting-state fMRI and diffusion tensor imaging changes in FTD develop before symptom onset.

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Author disclosures are available upon request (journal@neurology.org).

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