brain volume were lower in our three groups of youths (0.039, 0.026 and 0.034 for liars, antisocial controls and healthy volunteers respectively) than in the corresponding groups of adults reported by Yang *et al* (0.069, 0.054 and 0.054). However, prefrontal white to whole brain ratio, prefrontal white volume, or prefrontal grey/white ratios did not differ between our youth groups ($F(2,19)=1.105$, 0.973 and 0.337 respectively).

We also examined the corpus callosum morphometrically using the method of Casanova *et al* (1990). Since Raine *et al*'s (2003) strongest effect size was seen for corpus callosum volume and limited data were available, we calculated the ratio of corpus callosum area to whole brain volume as a proxy for corpus callosum volume. A trend for ratio differences between the three groups was seen ($F(2,19)=2.748$, $P=0.092$), with the smallest ratios in the liars (0.080), followed by antisocial controls (0.086) and healthy controls (0.091).

Thus, we did not find prefrontal differences in lying youths but did find suggestion of corpus callosum differences. Our results are consistent with the notion that prefrontal findings are not causal, although they may be linked to the maintenance of the symptom of lying and consistent with myelination proceeding rostrally and from the inside (longer connections) outward (short association fibres and arcuate fibres).

Casanova, M. F., Sanders, R. D., Goldberg, T. E., *et al* (1990) Morphometry of the corpus callosum in monozygotic twins discordant for schizophrenia: a magnetic resonance imaging study. *Journal of Neurology, Neurosurgery and Psychiatry*, **53**, 416–421.

Kruesi, M. J. P., Casanova, M. F., Mannheim, G., *et al* (2004) Reduced temporal lobe volume in early onset conduct disorder. *Psychiatry Research: Neuroimaging*, **132**, 1–11.

Raine, A. Lencz, T., Taylor, K., *et al* (2003) Corpus callosum abnormalities in psychopathic antisocial individuals. *Archives of General Psychiatry*, **60**, 1134–1142.

Spence, S. A. (2005) Prefrontal white matter – the tissue of lies? Invited commentary on Prefrontal white matter in pathological liars. *British Journal of Psychiatry*, **187**, 326–327.

Yang, Y., Raine, A., Lencz, T., *et al* (2005) Prefrontal white matter in pathological liars. *British Journal of Psychiatry*, **187**, 320–325.

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Authors' reply: The findings reported by Kruesi *et al* are intriguing. We showed that adult pathological liars had 22% more prefrontal white matter than normal controls and 26% more than antisocial controls. Based on mean values reported by Kruesi *et al*, they too found higher prefrontal white matter/whole brain volumes in adolescent liars compared with both normal controls (14.7%) and antisocial controls (50%). Their sample of adolescent liars was small ($n=4$) and therefore underpowered for the detection of a true increase in prefrontal white matter. We therefore believe that the results of Kruesi *et al* support our findings rather than refute them. With a larger sample size they may well have found a statistically significant increase in prefrontal white matter in liars. An important difference between the two studies is that the mean age of our adult pathological liars (36.5 years) was more than twice that of the adolescent liars (15.9 years). Since prefrontal white matter may not be fully developed until 30 years of age (Paus *et al*, 2001), there may be insufficient development of prefrontal white matter in adolescents to facilitate pathological lying. Taken together the findings suggest a neurodevelopmental hypothesis whereby individual differences in white matter predispose more to lying in adulthood when neurodevelopment is complete.

A further difference between the studies is that although our pathological liars were matched with controls for IQ, the control group of Kruesi *et al* had a 31 point higher IQ than the liars, which may affect their findings. A further important difference is that we assessed pathological lying in adults, whereas Kruesi *et al* appear to be assessing excessive lying in adolescents. There may be a continuum of lying from normative lying (controls) to excessive lying (the adolescents of Kruesi *et al*) to pathological lying (our adults). Whether prefrontal white matter (or any other brain structure) is related in a neurodevelopmental context to this lying continuum remains to be determined.

Declaration of interest

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Paus, T., Collins, D. L., Evans, G., *et al* (2001) Maturation of white matter in the human brain: A review of magnetic resonance studies. *Brain Research Bulletin*, **54**, 255–266.

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The *Journal* apologises, as does Dr Calton (British Journal of Psychiatry, 2005), for giving the impression that the views expressed by authors were influenced by their occasional support from pharmaceutical companies. Your column (Tyrer, 2005) comments that assuming that such support necessarily creates a conflict of interest is 'sometimes' unwarranted. I am sure that it would be of great interest to readers to know how you judge when such an assumption is warranted. Does it depend on how often you receive support? Or on the financial value of such support? Or on some multiplication of both? Or on the obviousness of the relationship between the support and the views expressed? We must be told.

Declaration of interest

A.J.D.M. received direct support for attending conferences and meetings until 2001 from Pfizer UK and from other companies. He cannot recall ever attending a major academic meeting that was not heavily sponsored by industry. He works with user and carer charities which also receive such support. He attends lunchtime meetings at which food is never available from any other source, and uses a USB memory stick provided by Eli Lilly UK.

British Journal of Psychiatry (2005) Apology. *British Journal of Psychiatry*, **187**, 390.

Tyrer, P. (2005) From the Editor's Desk. *British Journal of Psychiatry*, **187**, 394.

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Editor's reply: The declaration of interest attached to Professor Mcdonald's letter is

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