Prefrontal white matter – the tissue of lies?

Invited commentary on . . . Prefrontal white matter in pathological liars†

SEAN A. SPENCE

Yang *et al* (2005, this issue) report what is probably the first structural neuroimaging study of lying. Adults were recruited from temporary employment agencies in Los Angeles. This will have been a complex and demanding study to perform; it has already yielded significant insights into the neural correlates of antisocial personalities drawn from that environment, indicating reduced prefrontal grey matter and diminished autonomic responsiveness (Raine *et al*, 2000). The current findings derive from a re-analysis of these data, themselves obtained from reliable blinded measurement of prefrontal white matter by magnetic resonance imaging. The groups were imperfectly matched on some variables (e.g. age), nevertheless, this did not detract significantly from the authors' findings: namely, greater prefrontal white matter volume among those identified as 'liars' (relative to 'antisocial' and 'normal' controls).

This study raises a number of intriguing questions. Why should prefrontal structure affect deceptive behaviour? What is the contribution of white matter? How specific (and replicable) are the findings likely to be? What do we mean by 'pathological' lying?

WHITE MATTER

The adult human brain weighs approximately 1400 g and 40–50% of its cerebral volume consists of white matter. White matter comprises axons (invested with myelin), millions of which provide the substrate for neuronal connectivity. Grey and white matters exhibit distinct developmental trajectories: nerve cells (grey matter) developing early in gestation, white matter in the second trimester. The brain grows rapidly in the first 2 years of postnatal life but myelination continues well after this (perhaps until the sixth decade), advancing rostrally: the brain stem and cerebellum myelinating early, the frontal regions late. While the volumes of white and grey matters reach approximate equivalence in middle age, in old age there is a disproportionate reduction of the former (possibly reflecting vascular change). Disorders that diffusely affect white matter may disproportionately affect visuospatial (cf. language) function, thereby appearing to preserve verbal IQ (of possible relevance, given the findings of Yang *et al*; for review see Filley, 2001). White matter projections are especially abundant in the frontal lobes, consistent with the prefrontal executive role in modulating emergent behaviour via subordinate brain structures (Spence *et al*, 2002). Hence, white matter is pivotal to the connectivity and cognitive function of the human brain, and abnormal prefrontal white matter might affect complex behaviours such as deception.

Examples of reduction in prefrontal white matter in neuropsychiatric disease are relatively easy to find. A literature search performed in January 2005, entirely confined to prefrontal white matter volume, revealed approximately 40 records describing volume reductions in schizophrenia (variably replicated, possibly related to chronicity or medication), alcohol dependence (probably confined to clinical samples and older age groups) and attention-deficit hyperactivity disorder. Neither bipolar disorder nor the neuroses have been consistently associated with such changes to date. However, increased prefrontal white matter is rarely reported, neurofibromatosis type 1 providing an exception (Cutting *et al*, 2002). To my knowledge, this condition is not associated with deception *per se*, although of course it may have severe consequences for cognitive development. Thus, Yang *et al*'s finding of increased prefrontal white matter volume (in community 'liars') is uncommon among clinical samples.

THE LYING BRAIN

Deception itself resembles an executive task. It elicits greater activation of the prefrontal regions (compared with telling the truth) and also incurs a processing cost, manifest in longer response times (for review see Spence *et al*, 2004). That humans utilise greater cognitive resource while lying (cf. telling the truth) also seems congruent with emerging primate studies suggesting that non-human primate species exhibiting more frequent tactical deception possess larger neocortices (Byrne, 2003). Hence, deception may constitute a weapon in the evolutionary biological arms race, supported by 'higher' brain regions.

This prompts a question: should a prefrontal white matter abnormality result in more or less, or perhaps 'better or worse', deception? If deception relies upon cognitive resource, then cognitive deficit should impair deception (as in the case of autism; Sodian & Frith, 1992), while the cognitively advantaged should be 'good' at deceiving. So, is increased white matter likely to impede or facilitate information processing (and, thereby, deception)? Yang *et al*'s findings imply that excess prefrontal white matter confers some 'capacity' for deception, akin to an advantage. King & Ford (1988) reached similar conclusions when they found that those with pseudologia fantastica might exhibit superior verbal abilities (despite increased prevalence of neurological abnormality). Such conclusions evoke the stereotypic image of the liar as one who is 'smooth-tongued' and Machiavellian.

Thus, a straightforward interpretation of Yang *et al*'s study would conclude that increased prefrontal white matter confers a 'predisposition' to lying, which itself confers a competitive edge in day-to-day life. But there are caveats.

First, we do not know whether the findings reflect cause or effect (whether anatomy drives deception or is driven by its practice). We cannot form a firm conclusion on the basis of cross-sectional data.

Second, the meaning of pathological requires elucidation. While the authors rightly use the term in a very specific way, their criteria differ from those of certain other studies. What is pathological lying? It is not lying *per se*, as 'normal' people tell lies regularly, often to their nearest and dearest (Vrij, 2001). Lying may, on occasion, be considered altruistic. Not so the lying attributed to those in the study of

†See pp. 320–325, this issue.

Yang *et al*; this is predominantly antisocial lying (e.g. 'conning' and benefit fraud). It seems inherently instrumental (and, hence, akin to malingering). Yet, it differs from the pathological lying described by Ford (1995) and in the Munchhausen's literature, where emphasis is placed upon self-defeating, impulsive or compulsive lying, not associated with tangible personal gain. Hence there may be different types of pathological lying, with that described by Yang *et al* pertaining to the more antisocial variant.

Third, how accomplished were those described by Yang *et al* at telling lies? They attended temporary employment agencies, consented to being studied and admitted to lying. This is not very Machiavellian! We might contrast them with those successful social predators who lie and cheat and yet retain enormous influence in the world (the doubting reader might reflect upon Smith's (2002) account of these powerful people). Yang *et al*'s findings may be specific to an underprivileged milieu: where a subgroup of unemployed antisocial people resort to deception for instrumental gain but are not necessarily very good at lying.

The study of Yang *et al* has opened up a new area in the use of neuroimaging technologies to examine aspects of human behaviour, yielding findings that may have profound consequences for the way we view immoral and forensic activity, responsibility and mitigation. Philosophically, they point towards behaviour (and, by extension, morality) that is constrained by biology. It might be remarked that although the science in this area is relatively recent the assumption has been implicit in much forensic psychiatric practice for some time. The very seriousness of this proposition underscores the need for further careful work in this area.

SEAN A. SPENCE, MD, MRCPsych, Professor of Psychiatry, Department of Academic Clinical Psychiatry, Division of Genomic Medicine, University of Sheffield, The Longley Centre, Norwood Grange Drive, Sheffield S5 7JT, UK. Tel: +44 (0)114 22 61519; fax: +44(0)114 22 61522; e-mail: S.A.Spence@Sheffield.ac.uk

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DECLARATION OF INTEREST

None.

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