weeks following discharge from abstinence-oriented residential treatment.

In common with centres in Britain and Australia, addiction treatment services in Dublin are oriented towards harm reduction. However, there is no conflict between a goal of harm reduction yet continuing to provide patients with the option of an abstinence-based treatment such as that examined in our study. In all medical specialties, doctors are charged with the responsibility of weighing up the advantages and disadvantages of various treatment options. There are many circumstances in which patients will have to choose between a more conservative treatment option and a more aggressive approach with a higher risk but a greater reward.

In the case of opiate dependence, both clinicians and patients in Dublin are fortunate to have the option of both methadone maintenance and abstinence-based treatments. Although there are real risks of accidental overdose associated with the latter, we believe that in a therapeutic relationship that is collaborative and respectful, the patient should be given the choice. Denying them the choice of an abstinence-based treatment would represent a retreat to a paternalistic approach to medicine which was so commonplace a generation ago and which is criticised by patient groups today. At the other end of the spectrum, there are many countries where patients are denied, or have very restricted access to, methadone maintenance treatment (Kakko *et al*, 2003; World Health Organization, 2004). This has occurred when treatment options have been determined by politicians instead of clinicians and decisions have unfortunately being driven by ideology rather than evidence.

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Diagnostic stability and status of acute and transient psychotic disorders

We read with interest the article by Pillmann & Marneros (2005). Acute and transient psychosis is a common clinical presentation in the developing world. We retrieved medical records of all patients with psychotic disorders (F06.0–06.3, F20–29, F30.2, F31.2, F31.5, F32.3, F33.3) who attended our unit from 1 January to 30 September 2003. There were 87 patients (13.9%) with a diagnosis of acute psychosis (ICD–10 F23). The majority were young adults (mean age 29.75 years, s.d.=10.95), male (52%) and without a history of precipitating stress (71%) or similar illness (93%). The mean duration of follow-up was 13.2 months (s.d.=11.7). The diagnosis was revised to affective disorder in 8 patients (9.2%), schizophrenia in 23 (26.4%), and 10 patients (11.5%) presented with recurrent episodes of acute psychosis.

The high drop-out rate has been attributed to a good response to antipsychotic medication, spontaneous remission and/or preference for indigenous treatments (Raguram *et al*, 2002). Most studies of acute psychosis have small samples (Susser *et al*, 1998; Marneros *et al*, 2003; Pillmann & Marneros, 2003; Singh *et al*, 2004) and there are no large long-term follow-up studies of acute psychosis from the developing world.

The introduction of the categories acute and transient psychotic disorders in ICD–10 and brief psychotic disorder in DSM–IV has allowed for coding of patients with a single episode of illness. However, there is also a need to categorise people who present recurrently with such episodes. Future classification should consider such a category.

Acute psychotic presentations can be secondary to organic psychoses and substance dependence. Psychiatrists often subscribe to the Kraepelinian dichotomy and attempt to label all functional psychosis as schizophrenia or affective disorders. However, clinical presentations of acute psychosis challenge such categorisation. Although many patients recover, some relapse with similar acute psychotic presentations, and a significant proportion also develop classic schizophrenia and mood disorders. The difficulty in reaching a diagnosis at the time of the initial presentation is because it is often difficult to recognise the classic syndromes at the onset of the illness. However, these can be identified over time as they become more obvious. Thus, acute psychoses can be a presentation of organic psychoses, substance-induced disorders, schizophrenia, affective illness or may be 'micro-psychotic' episodes that occur in some personality disorders. They can also be separate clinical entities. Clinicians working in the developing world are often aware of this distinction.

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White matter in liars

Yang *et al* (2005) propose a neurodevelopmental theory of pathological lying, finding increased prefrontal white matter and lower prefrontal gray/white ratios in pathological liars compared with antisocial and normal controls. Spence (2005) asks whether these findings represent cause or effect. Since lying is a criterion symptom for childhood conduct disorder, we re-examined a structural magnetic resonance imaging study of early-onset conduct disorders (Kruesi *et al*, 2004 plus unpublished data).

Youths had been classified as liars or not based upon structured interviews and collateral information when documenting criterion symptoms of conduct disorder. Liars (n=6) were compared with individuals with conduct disorder (n=4) and with healthy volunteers (n=10). The mean ages of the three groups (191.5, 195 and 190.8 months) were similar ($F(2,19)=0.015$). In accordance with developmental differences, ratios of prefrontal white volume to total

https://doi.org/10.1192/bjp.188.3.293-a Published online by Cambridge University Press

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).

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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

This study was supported by grants to A.R. from the National Institute of Mental Health (Research Scientist Development Award K02 (MH01114-01, Independent Scientist Award K02 MH01114-01 and 5 RO3 MH50940-02) and from the Wacker Foundation.

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Financial support and conflict of interest

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.

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

https://doi.org/10.1192/bjp.188.3.293-a Published online by Cambridge University Press