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White matter in liars

M. J. P. Kruesi and M. F. Casanova

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Contents ■ War and psychological health ■ Post-traumatic stress after non-traumatic events ■ Patient-rated unmet needs and quality of life improvement ■ Abstinence-oriented treatment for opiate addiction ■ Diagnostic stability and status of acute and transient psychotic disorders ■ White matter in liars ■ Financial support and conflict of interest

War and psychological health

The study by Hacker Hughes *et al* (2005) is interesting but I wish to raise a few points. The end of pre-deployment mental health briefing was not the best time for assessment because the soldiers were aware that they would soon be going to war and hence their stress levels must have been high. One month after the return from the war, they must have felt relieved and their stress levels must have been reduced. Since the stress levels were high at the time of initial assessment, lack of increased morbidity at the final evaluation might not mean much. It would have been more appropriate to compare stress levels at the final evaluation with those measured during peacetime.

Although the soldiers were told that the commanders would be informed about only the pooled results, they were told that military mental health practitioners would contact them confidentially if results revealed cause for concern. This means that the answers were not anonymous and hence the soldiers may have hidden their psychopathology for fear of being considered weak and the consequences of being under treatment of the military mental health practitioner. These soldiers were in the war theatre for only 4 months and it has not been mentioned how much experience of combat they had but it is known that Basra was the scene of fewer hostilities than other areas. More combat experience may be associated with a higher prevalence of post-traumatic stress disorder (Hoge *et al*, 2004).

The figures do not add up. It is mentioned that 421 soldiers out of the original sample of 899 completed the questionnaires. Later it is mentioned that 35% ($n=254$) completed both sets of questionnaires. The number 254 is 35% of neither the original sample ($n=899$) nor the sample that completed the questionnaires at follow-up ($n=421$). The follow-up rate is

very low and hence the advantage of the study being longitudinal is minimised. It is also not mentioned how many soldiers did not volunteer for the study before and after deployment although it is mentioned that participation was voluntary.

The conclusion of the study that 'participation in war fighting may sometimes not necessarily be as deleterious to psychological well-being as has previously been thought' is premature. The small sample size compared with studies with positive findings, the high drop-out rate and lack of baseline data do not allow us to draw any conclusions from this study.

Hacker Hughes, J., Cameron, F., Eldridge, R., et al (2005) Going to war does not have to hurt: preliminary findings from the British deployment to Iraq. *British Journal of Psychiatry*, **186**, 536–537.

Hoge, C. W., Castro, C. A., Messer, S. C., et al (2004) Combat duty in Iraq and Afghanistan, mental health problems and barriers to care. *New England Journal of Medicine*, **351**, 13–22.

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Authors' reply: In response to Dr Jhingan's letter we should first point out that the rates of morbidity obtained pre-deployment were entirely compatible with those from other studies (Rona *et al*, 2004). Furthermore, it is illogical to argue that rates of pre-deployment stress must have been high in this group because of anticipatory anxiety. Not only is there no evidence for this assertion in this population but the converse probably applies. Troops in this elite formation would have probably been looking forward to the deployment, confident in the strong belief that they were going to win (Hacker Hughes *et al*, 2006).

The argument that post-deployment stress levels would be low because of relief to be home does not allow for the influence of any adverse events in theatre. In fact, 1 month after return is the earliest time to assess for possible post-traumatic stress using the screening questionnaire (Brewin *et al*, 2002).

For a brigade such as 16 Air Assault Brigade, there is no such thing as true 'peacetime'. This brigade has, to the best of our knowledge, been deployed more often than any other in the British Army since its formation and is constantly training for, or recovering from, deployments when not on operations.

With regard to responses not being anonymised, in fact the converse applies. Soldiers may use the questionnaires as a confidential means of signalling to command, via the mental health chain, that there is a problem. In addition, there are also data from the USA to suggest that when asked questions it is only information on banned activities (such as drug use) that is significantly affected by anonymity, rather than simple distress (Adler & Thomas, 2005).

With regard to the figures, they add up perfectly. There was a population of 899 with 733 initial responses (giving a response rate of 82%); 421 completed the follow-up questionnaires and, in total, 254 of the initial 733 (35%) completed both sets.

On this basis, it is totally reasonable to have stated that, for highly trained professional soldiers involved in brief, focused operations with positive outcomes, participation in active war fighting may not be necessarily bad for mental health, at least in the short term.

Declaration of interest

J.G.H.H., F.C., R.E., M.D. and N.G. are or were employed by Defence Medical Services. S.W. is honorary Civilian Adviser in Psychiatry (unpaid) to the British Army Medical Services.

Adler, A. & Thomas, J. L. (2005) Measuring up: comparing self reports with unit records for assessing soldier performance. *Military Psychology*, **17**, 3–24.

Brewin, C. R., Rose, S., Andrews, B., et al (2002) Brief screening instrument for post-traumatic stress disorder. *British Journal of Psychiatry*, **181**, 158–162.

Hacker Hughes, J. G. H., Campion, B., Cameron, F., et al (2006) Psychological morbidity in soldiers following an emergency operational deployment. *Military Psychology*, in press.

Rona, R. J., Jones, M., French, C., et al (2004)

Screening for physical and psychological illness in the British Armed Forces. III: The value of a questionnaire to assist a Medical Officer to decide who needs help. *Journal of Medical Screening*, **11**, 148–153.

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Post-traumatic stress after non-traumatic events

Authors' reply: We thank Ben-Ezra & Aluf (2005) for their letter, in which they broadly support our findings (Mol *et al*, 2005) that life events may cause as many symptoms of post-traumatic stress disorder (PTSD) as traumatic events classified according to the A1 criterion of the DSM-IV. However, they also have some criticisms. Ben-Ezra & Aluf argue that 'serious illness (self)' – classified as a life event in our study – can be considered a traumatic event. We decided against this classification as many respondents had experienced an illness that was chronic but not life-threatening in the short term. However, when we re-analysed the data with 'serious illness (self)' as a traumatic event the PTSD scores of the traumatic and life events groups still did not differ (total log PTSD score 0.68 in both groups).

As suggested by Ben-Ezra & Aluf we have also excluded accidents and sudden deaths from the trauma events group, since this might be a heterogeneous group regarding the magnitude of the event. This resulted in a mean total log PTSD score of 0.76 (*v.* 0.71), which is not an essential change compared with the original difference.

Ben-Ezra & Aluf argue that the magnitude (severity) of an event is related to the likelihood of developing PTSD, and that we should have allotted events to either of our two groups on the basis of their severity. We agree that symptoms are related to severity but we found a striking overlap in PTSD symptomatology after life events and traumatic events (Tables 2 and 4) and similar mean symptom levels (Table 3).

The severity of an event can be assessed objectively and subjectively. Ben-Ezra & Aluf allude to the objective assessment but the subjective appraisal of an experience also plays an important role (McNally *et al*,

2003). It is likely that objective and subjective severity are associated with PTSD symptoms after both traumatic and life events.

Declaration of interest

The Achmea Foundation for Victim Support in Society paid the salary of S.S.L.M. but had no influence on the methodology or analyses of the study.

Ben-Ezra, M. & Aluf, D. (2005) Traumatic events *v.* life events: does it really matter? *British Journal of Psychiatry*, **188**, 83–84.

McNally, R. J., Bryant, R. A. & Ehlers, A. (2003) Does early psychological intervention promote recovery from posttraumatic stress? *Psychological Science in the Public Interest*, **4**, 45–79.

Mol, S. S. L., Arntz, A., Metzmakers, J. F. M., et al (2005) Symptoms of post-traumatic stress disorder after non-traumatic events: evidence from an open population study. *British Journal of Psychiatry*, **186**, 494–499.

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Patient-rated unmet needs and quality of life improvement

Slade *et al* (2005) have published a potentially important study of the relationship between patient-rated unmet needs, quality of life and the effect of meeting those needs. They draw the conclusion that 'meeting patient-rated unmet needs should be the starting point for mental healthcare'. Although much psychiatric care is indeed directed towards reducing unmet need, we believe that this research shows (over the time scale of the study) that reducing unmet need is actually largely ineffective. A longer study might confirm continuing incremental improvement but this would need to be demonstrated.

In the descriptive part of the study the authors show that low quality of life is associated with high unmet need. Figure 1 shows a clear gradient which can be estimated to be -0.2 by inspection (no summary statistics are given). By contrast, in the second part of the study, which looks at the effect of reducing unmet needs, Fig. 2 shows almost no relationship between change in unmet need and change in quality

of life (summary statistics: $B = -0.04$, *s.d.* = 1). Although B indicates high statistical significance it seems to be clinically irrelevant: one would have to meet 25 unmet needs to improve quality of life by one point; B is very small compared with the standard deviation and importantly is only one-fifth of the gradient in Fig. 1.

Thus quality of life and unmet need are associated (gradient = -0.2) but meeting unmet needs has a negligible effect (gradient $B = -0.04$) on quality of life. This suggests that unmet needs do not cause low quality of life and that the relationship between the two may be mediated by some third factor, such as psychiatric illness, that causes both. If this were the case, treating psychiatric illness should be the starting point for mental healthcare and not 'meeting patient-rated unmet needs'.

Furthermore, if the justification for meeting unmet needs of psychiatric patients is to improve quality of life *per se*, then this research shows that in terms of size of effect (and over the period of the study), reducing unmet need is largely ineffective, and is therefore a questionable use of resources.

Slade, M., Leese, M., Cahill, S., et al (2005) Patient-rated mental health needs and quality of life improvement. *British Journal of Psychiatry*, **187**, 256–261.

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Authors' reply: We are grateful to Drs McQueen & St John-Smith for their response, which highlights that our study raises the question of the purpose of mental healthcare.

We agree that the effect we showed is small but we believe it is more meaningful than that shown by other study designs. Our data comprised repeated measures at monthly intervals over 7 months, and we demonstrated temporal precedence in the relationship between patient-rated unmet need and quality of life – reduction in the former precedes improvement in the latter. Cross-sectional studies more easily demonstrate apparent associations, which prove on further investigation to be spurious.

The analysis controlled for baseline symptomatology (assessed using the Brief Psychiatric Rating Scale) and diagnosis, and found no evidence of a mediating role

for psychiatric illness. Furthermore, our use of random-effects regression models controlled for further unmeasured individual characteristics that are stable over time. Our finding of a modest but robust effect is meaningful and therefore clinically important, especially when combined with other small effects. Further research into determinants of quality of life will provide other levers of change for improvement, which are unlikely to be staff-rated symptomatology (Lasalvia *et al*, 2002).

We agree that interventions to improve mental health will have an impact on patient-rated unmet need, which in turn (as we demonstrate) will improve quality of life. However, the advantage of identifying a modest but robust causal relationship is that it highlights the importance of a more comprehensive approach to meeting needs. Mental healthcare that focuses exclusively on treating psychiatric illness can risk neglecting the importance of other consequences of mental ill health, such as discrimination in travel (Driver and Vehicle Licensing Agency, 2005), insurance (Association of British Insurers, 2003) and debt (Meltzer *et al*, 2002). Mental health services that also address a wide range of health and social needs (as, for example, assessed in our study by the Camberwell Assessment of Need) are more likely to improve quality of life.

Declaration of interest

The Health Services Research Department, where this study was based, receives royalties from sales of the *Camberwell Assessment of Need* published by Gaskell.

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Meltzer, H., Singleton, N., Lee, A., et al (2002) *The Social and Economic Consequences of Adults with Mental Disorders*. London: Stationery Office.

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Abstinence-oriented treatment for opiate addiction

Smyth *et al* (2005) reported outcomes of abstinence-oriented in-patient treatment for opiate users at 2–3 years and found that 23% of patients were abstinent for the preceding 30 days according to self-report without methadone maintenance. At the start of the treatment 49% had injected heroin. There was, however, a group of patients who were truly abstinent: those who had died.

Of the 109 patients who had been located out of the original 149, 5 had died. The total expected number of deaths from the original sample would therefore be closer to 7, but would perhaps be even higher if we assume that those lost to follow-up led more 'chaotic' lifestyles. The authors rightly note that abstinence-oriented treatment is associated with accidental overdose (Strang *et al*, 2003).

In Glasgow, before the advent of supervised consumption, rates of methadone-related overdose were around 2.5 per 100 treatment-years. This rate fell to less than 0.5 per 100 treatment-years (Advisory Committee on the Misuse of Drugs, 2000) after the supervised consumption of methadone was introduced. Supervised methadone consumption is known to be effective in reducing the risk of overdose and there is a dose-related effect in reducing mortality, with doses over 75 mg being more effective than doses below 55 mg (van Ameijden *et al*, 1999). Methadone also reduces the risk of injecting; this in turn reduces viral transmission, which is the other significant risk of increased mortality among drug users (Dolan *et al*, 1998).

However, the attitude of treatment agencies towards extended maintenance is changing in the direction of delineated treatment episodes (National Treatment Agency for Substance Misuse, 2005). In these days of crack cocaine, the belief that methadone treatment works (Gossop *et al*, 2003) and saves money (Godfrey *et al*, 2004) has diminished. This is despite evidence for interventions such as contingency management and cognitive-behavioural therapy using substitute prescribing (Rowan-Szal *et al*, 2004).

Of course, abstinence should be a potential goal of drug treatment. Deciding those patients for whom abstinence-oriented treatment is appropriate, and the risk of such treatment, is more difficult. There is no reliable evidence for matching

patients to optimal treatments in addiction. However, those who inject, isolated users and alcohol/benzodiazepine co-users are all over-represented in the morgue (Warner-Smith *et al*, 2001). Risk awareness might well be a reasonable first step and for many abstinence might be more dangerous than desirable.

Advisory Committee on the Misuse of Drugs (2000) *Reducing Drug Related Deaths*. London: Stationery Office.

Dolan, K., Wodak, A. D. & Hall, W. D. (1998) Methadone maintenance treatment reduces heroin injection in New South Wales prisons. *Drug and Alcohol Review*, **17**, 153–158.

Gossop, M., Marsden, J., Stewart, D., et al (2003) The National Treatment Outcome Research Study (NTORS): 4–5 year follow-up results. *Addiction*, **98**, 291–303.

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Smyth, B. P., Barry, J., Lane, A., et al (2005) In-patient treatment of opiate dependence: medium-term follow-up outcomes. *British Journal of Psychiatry*, **187**, 360–365.

Strang, J., McCambridge, J., Best, D., et al (2003) Loss of tolerance and overdose mortality after inpatient opiate detoxification: follow up study. *BMJ*, **326**, 959–960.

van Ameijden, E. J., Langendam, M. W. & Coutinho, R. A. (1999) Dose-effect relationship between overdose mortality and prescribed methadone dosage in low-threshold maintenance programs. *Addictive Behaviors*, **24**, 559–563.

Warner-Smith, M., Darke, S., Lynskey, M., et al (2001) Heroin overdose: causes and consequences. *Addiction*, **96**, 1113–1125.

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Author's reply: I agree with Critchlow & Nadeem that abstinence-based treatment may only be appropriate for a minority of opiate-dependent patients and that risk awareness is an essential first step for both patient and treatment provider. There is an increased risk of accidental overdose in the

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 been driven by ideology rather than evidence.

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World Health Organization (2004) *The Practice and Context of Pharmacotherapy of Opioid Dependence in Central and Eastern Europe*. Geneva: WHO.

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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.

Marneros, A., Pillmann, F., Haring, A., et al (2003) What is schizophrenic in acute and transient psychotic disorder? *Schizophrenia Bulletin*, **29**, 311–323.

Pillmann, F. & Marneros, A. (2003) Brief and acute psychoses: the development of concepts. *History of Psychiatry*, **14**, 161–177.

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Singh, S. P., Burns, T., Amin, S., et al (2004) Acute and transient psychotic disorders: precursors, epidemiology, course and outcome. *British Journal of Psychiatry*, **185**, 452–459.

Susser, E., Varma, V. K., Mattoo, S. K., et al (1998) Long-term course of acute brief psychosis in a developing country setting. *British Journal of Psychiatry*, **173**, 226–230.

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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

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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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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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.

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 Macdonald's letter is

a clear answer to his question. It is almost impossible to exist as a medical practitioner without receiving support from one organisation or another at some point in your duties, but most of the time this is quite irrelevant to a piece of published information. The declaration of interest may need to be defined more specifically in our instructions but at present we are inclined towards the views of the *BMJ* in identifying those interests which are competing as those which should be declared. The *BMJ* defines a competing interest as one that

‘exists when professional judgment concerning a primary interest (such as patients’ welfare or the validity of research) may be influenced by a secondary interest (such as financial gain or personal rivalry)’ (<http://www.bmj.com>). This properly identifies the element that might, wittingly or unwittingly, create a bias in the written material that is submitted. In most instances the interest declared will be a financial one, but I would welcome more of the personal rivalry interests that are highly relevant in academic circles.

Although not expecting ‘I am a visceral opponent of Dr X’s work and cannot bear to be in the same room as him/her’, I think ‘I have a general bias against Dr X because I do not think he/she has the clinical experience to pronounce on these matters, whereas I have’, might not be out of place in an open and honest exchange of views.

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One hundred years ago

Fragment of the History of a Case of Hysteria. [Bruchstück einer Hysterie-Analyse]. (Monatsschr. f. Psychiatrie u. Neurolog., Bd. xviii, Heft. 4, 1905.) Freud, S.

The volume of *Studies of Hysteria* which Professor Freud published in conjunction with Dr. Breuer some ten years ago aroused much controversy, but even many of those who were by no means prepared to accept its teaching at every point could not fail to recognise that it was an epoch-marking book in the history of hysteria. In method it introduced a refined and penetrating psychic analysis which had never before been known, and in theory it brought back in a more acceptable form the conception of the large part played in hysteria by the sexual emotions, which, under the influence of Charcot, had been too absolutely rejected.

While Freud’s method and theory remain substantially the same, he has very considerably developed the technique of his analytical process. He has abandoned the use of hypnosis as a method of investigation, and attaches still more importance than before to what may be called “symbolic manifestations” of the psychic

condition. He seeks to obtain a complete and sympathetic knowledge of the patient’s outer and inner life, and to interpret the data thus obtained by means of clues which often seem of the slightest character. It is obvious that such a method must be carried out in an extremely elaborate manner to be in any degree convincing. Even the present fragment of a history, which might easily be dismissed as a quite ordinary case of hysteria, covers nearly a hundred pages, and though it really reveals itself as an exceedingly complex and many-sided history, which, under the investigator’s hands, slowly falls into order, there is still much that a cautious and critical reader is inclined to view with suspicion, notably as regards the interpretation of dreams (a subject to which of recent years Freud has devoted special study); even here, however, the clues often prove such excellent guides that one hesitates to condemn them on account of their extreme tenuity. It should be remarked that Freud now attaches very great importance to dreams in the interpretation, not only of hysteria, but of all allied psycho-neurotic conditions; without a study of dream-life, indeed, he believes we can make very little progress in this field.

It is necessary, however, to pay close attention to all the automatic and involuntary manifestations of the psychic and physical organism. “He who has eyes to see and ears to hear becomes convinced that no mortal can hide his secret. He whose lips are silent chatters with his finger-tips and betrays himself through all his pores. That is how it is that the task of bringing even the most hidden regions of the soul to consciousness becomes quite possible.”

It is impossible to analyse this analysis, but by many readers its study will be found highly fascinating and profitable. There are other readers for whom it will seem unsatisfactory, trivial, and unwholesome. Of this type of mind was the little girl who criticised the operations of the Divine mind with the remark that it “must be fiddling work making flies.” People of this mental type cannot, however, be advised to study hysteria.

HAVELOCK ELLIS.

REFERENCE

Journal of Mental Science, April 1906, 406–407.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey

Corrigenda

Specialised care for early psychosis: symptoms, social functioning and patient satisfaction. Randomised controlled trial. *BJP*, 188, 37–45. The seventh author’s name is Jason Read. The online version of

this article has been corrected post-publication in deviation from print and in accordance with this correction.

Going to war always hurts (letter). *BJP*, 188, 83. The signatories to the ‘Authors’ response’ should include N. Greenberg, King’s Centre for Military Health Research, London, UK.