

Correspondence

Edited by Kiriakos Xenitidis and
Colin Campbell

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Abortion and mental health

The December 2008 issue, with its original papers by Fergusson *et al*,¹ Dingle *et al*² and its commentaries,³ was of great interest to us. Fergusson *et al* have overcome some of the methodological problems of previous studies.⁴ Nevertheless, their latest study has weaknesses: the women's abortion status is not verified objectively, only by self-report. There were 153 abortions in 117 women but insufficient data to distinguish the effects of differing numbers of abortions; it is known that women having more than one abortion may differ in many respects from those having a single abortion.⁴ Also, because of the relatively restrictive law in New Zealand – 'continuance of the pregnancy would result in serious danger . . . to the . . . mental health of the woman' – some selection bias may have been in operation, allowing only women with more traumatic histories to access abortion. We will not discuss the Dingle *et al* paper, as its failure to account for pregnancy intention (wantedness and timing) in those giving birth means that the comparator is inappropriate.⁵

The Royal College of Psychiatrists' Position Statement of 14 March 2008 mentions that a full systematic review is needed. This has now been done.⁵ Only four studies fell into the authors' 'good evidence and low risk of bias' category. All four studies showed a neutral effect of abortion on mental health, indicating no significant differences between the study comparison groups. So Fergusson *et al*'s study can be regarded as the first good-quality study to show a possible negative effect when attempting to answer the question: what is the relative risk of mental health problems for women who chose abortion compared with those who chose to have a live birth and who reported that the pregnancy was unwanted/initially distressing?

As clinicians working in the field of sexual and reproductive health, we favour the approach of Oates *et al*.³ We are supportive of their idea that abortion is not a psychiatric issue and that the Royal College of Psychiatrists should not develop a guideline on abortion. We would never want to go back to the psychiatric referral hurdle-jumping situation before and immediately after the Abortion Act came into force.⁶ The adverse effects of denied abortion must never be forgotten.⁷ Nevertheless, we do value working in partnership with mental health teams for the benefit of certain women requesting abortion who have a history of mental health problems or persistent ambivalence.

Whether abortion causes harm to women's mental health is a question that is not scientifically testable, as women with unwanted pregnancies cannot be randomly assigned to abortion *v.* abortion denied groups.⁷ It seems inappropriate therefore for Casey to talk of potential litigation against abortion providers

for failing to provide information on a possible causal link between abortion and subsequent mental health problems.³ All women should have rights to reproductive health and self-determination, of which safe and dignified access to abortion services is an important part.⁸

- 1 Fergusson DM, Horwood LJ, Boden JM. Abortion and mental health disorders: evidence from a 30-year longitudinal study. *Br J Psychiatry* 2008; **193**: 444–51.
- 2 Dingle K, Alati R, Clavarino A, Najman JM, Williams GM. Pregnancy loss and psychiatric disorders in young women: an Australian birth cohort study. *Br J Psychiatry* 2008; **193**: 455–60.
- 3 Casey P, Oates M, Jones I, Cantwell R. Invited commentaries on . . . Abortion and mental health disorders. *Br J Psychiatry* 2008; **193**: 452–4.
- 4 American Psychological Association, Task Force on Mental Health and Abortion. *Report of the Task Force on Mental Health and Abortion*. APA, 2008 (<http://www.apa.org/pi/wpo/mental-health-abortion-report.pdf>).
- 5 Charles VE, Polis CB, Sridhara SK, Blum RW. Abortion and long-term mental health outcomes: a systematic review of the evidence. *Contraception* 2008; **78**: 436–50.
- 6 Pare CMB, Raven H. Follow-up of patients referred for termination of pregnancy. *Lancet* 1970; **1**: 635–8.
- 7 House of Commons Science and Technology Committee. *Scientific Developments Relating to the Abortion Act 1967: Volume II* (HC 1045-II). TSO (The Stationery Office), 2007 (<http://www.publications.parliament.uk/pa/cm200607/cmselect/cmsctech/1045/1045ii.pdf>).
- 8 Cook RJ, Dickens BM, Fathalla MF. *Reproductive Health and Human Rights*. Oxford University Press, 2003.

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Authors' reply: We would like to thank Rowlands & Guthrie for their positive comments about our paper.¹ We do not agree that the Dingle *et al*² paper should be dismissed on the grounds that it uses an inappropriate comparison. Although this dismissal is consistent with the opinions stated in the review your correspondents cite,³ it reflects a common misunderstanding. There are, in fact, two closely related causal questions that one can ask about abortion and mental health. The first concerns whether or not abortion is an adverse life event that increases risks of mental health problems. Answering this question is important for understanding the extent to which women having abortions are an at-risk population for subsequent mental health problems. The second question concerns whether any mental health risks of abortion are greater or less than the mental health risks of unwanted pregnancies that come to term. Answering this question is important for understanding the extent to which abortion may mitigate or exacerbate any mental health problems associated with unwanted pregnancy. Dingle and colleagues² address the first question by showing that women having abortions are an at-risk population for mental health problems, and that these responses seem similar to those of women who experience pregnancy loss.

Rowlands & Guthrie suggest that our paper has a number of limitations relating to the assessment of abortion, the number of abortions and the social context of the research. However, these problems have different implications for interpreting our research. Any under-ascertainment of abortion is likely to have the effects of biasing estimates of relative risk downwards (providing that under-ascertainment is statistically independent of mental health outcomes); failure to show the effects of multiple abortions does not threaten the validity of our conclusions, but may call the precision of our conclusions into question; and the sociolegal context within which the research was conducted implies that it

is important to take pre-abortion mental health into account (which we did).

Rowlands & Guthrie also suggest that an adequate review of this issue has been conducted by Charles *et al.*³ We do not agree. This review has been criticised on the grounds of investigator bias⁴ and these concerns are supported by the somewhat anomalous conclusions the authors draw. For example, the study by Schmiege & Russo⁵ using the National Longitudinal Study of Youth (NLSY) data is ranked as one of the four 'good' studies despite the facts that only 40% of women having an abortion were included, and mental health was measured using a single scale of depression, with this outcome being assessed up to 13 years after the abortion. All of these features will bias results towards the null hypothesis. In addition, Charles *et al.*³ failed to distinguish between the different causal questions that may be asked about abortion, and misleadingly dismissed a number of studies showing links between abortion and mental health on the grounds that they did not include an appropriate comparison group.

Finally, Rowlands & Guthrie claim that conclusions cannot be drawn about the causal effects of abortion on mental health in the absence of randomised controlled trials. We believe that this argument overstates the case and there is now growing evidence for two major conclusions about abortion and mental health. First, exposure to abortion is an adverse life event which is associated with a modest increase in risks of mental health problems. Second, the mental health risks associated with abortion may be larger, and certainly are not smaller, than the mental health risks associated with unwanted pregnancies that come to term.

- 1 Fergusson DM, Horwood LJ, Boden JM. Abortion and mental health disorders: Evidence from a 30 year longitudinal study. *Br J Psychiatry* 2008; **193**: 444–51.
- 2 Dingle K, Alati R, Clavarino A, Najman JM, Williams GM. Pregnancy loss and psychiatric disorders in young women: an Australian birth cohort study. *Br J Psychiatry* 2008; **193**: 455–60.
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- 4 Ertelt S. *Researcher: Johns Hopkins paper biased on abortion-mental health issues*. LifeNews.com, 2008; 4 December (<http://www.lifeneews.com/nat4629.html>).
- 5 Schmiege S, Russo NF. Depression and unwanted first pregnancy: longitudinal cohort study. *BMJ* 2005; **331**: 1303.

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Longitudinal course of cognition in schizophrenia

In their meta-analysis,¹ Szöke *et al* found significant improvements in most neuropsychological variables, along with well-known potential practice effects, and that semantic verbal fluency holds promise as a suitable cognitive endophenotype in schizophrenia. We acknowledge that this review is a step forward, attempting to merge and quantify the evidence from both naturalistic observational studies² and clinical trials.³

We agree with the authors that the current literature is limited by the virtual absence of healthy control groups. Since only 9 out of 53 studies reported longitudinal neurocognitive data for controls, it is difficult to disentangle whether patients' cognitive changes are true improvements or confounded by the non-specific effects of practice-related learning.⁴ The use of healthy individuals from test–retest studies ('external controls') is also problematic

and the parallel assessment of controls would rule out the possibility that patients' apparent cognitive stability is not deterioration in disguise, as the authors accurately suggest.

In addition, we would like to highlight other critical issues that may limit the conclusions. First, the authors' choice to lower the minimum study duration to 1 month led to the median test–retest interval being only 4 months, which is shorter than the recommended trial duration to evaluate cognitive changes (e.g. at least 6 months).⁵ This also may have biased the review towards short-term clinical trials intended to improve cognitive deficits, especially with second-generation antipsychotics. Had a more stringent and clinically relevant 1-year follow-up cut-off² been used, only 24 out of 53 studies would have been reviewed. Second, approximately half of the 20 clinical trials previously reviewed³ are open, and these are mostly clozapine trials of patients who are treatment-resistant. Significant associations between cognitive change and change in negative symptoms are more likely to occur in these studies than in naturalistic follow-up studies.⁵ Third, we feel the authors missed an opportunity to include the distinction between patients with first-episode or chronic schizophrenia and geriatric patients as a potential moderator variable. This could have contributed to a better understanding of the probably complex cognitive pathways during the lifespan.

Despite the number of longitudinal neurocognitive reports, less is known than was originally supposed about the course of cognition in schizophrenia. Only a small subset ($n = 4$) of longitudinal reports have compared these neurocognitive pathways with those of healthy controls over at least 1 year. When reviews² are not focused on the neurocognitive effects of antipsychotics, stable long-term performances and, in some cases, cognitive gains could be expected, thus offering a rather pessimistic picture for cognitive enhancement. This approach seems more useful for understanding the long-term natural history of cognition in schizophrenia. Conversely, this meta-analysis¹ relies on short-term data, mostly from clinical trials,³ and therefore more likely overestimates the potential for cognitive improvement.

In summary, it would be misleading if the conclusions were regarded as a major leap forward instead of as tentative hypotheses awaiting further investigation. Since the possibility remains that the current findings are more valuable to researchers, a new meta-analysis that takes into consideration these and other limitations might be more helpful for clinicians, patients and caregivers.

- 1 Szöke A, Trandafir A, Dupont ME, Méary A, Schürhoff F, Leboyer M. Longitudinal studies of cognition in schizophrenia: meta-analysis. *Br J Psychiatry* 2008; **192**: 248–57.
- 2 Rund BR. A review of longitudinal studies of cognitive functions in schizophrenia patients. *Schizophr Bull* 1998; **24**: 425–35.
- 3 Woodward ND, Purdon SE, Meltzer HY, Zald DH. A meta-analysis of neuropsychological change to clozapine, olanzapine, quetiapine, and risperidone in schizophrenia. *Int J Neuropsychopharmacol* 2005; **8**: 457–72.
- 4 Goldberg TE, Goldman RS, Burdick KE, Malhotra AK, Lencz T, Patel RC, et al. Cognitive improvement after treatment with second generation antipsychotic medications in first-episode schizophrenia: is it a practice effect? *Arch Gen Psychiatry* 2007; **64**: 1115–22.
- 5 Buchanan RW, Davis M, Goff D, Green MF, Keefe RS, Leon AC, et al. A summary of the FDA-NIMH-MATRICS workshop on clinical trial design for neurocognitive drugs for schizophrenia. *Schizophr Bull* 2005; **31**: 5–19.

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Authors' reply: Balanzá-Martinez *et al* made a detailed review of our article¹ and also provided several suggestions for future research.

Although we agree with the authors that the conclusions of our article are by no means definitive, we disagree with them on the following important issues.

First, Balanzá-Martinez *et al* suggest that our meta-analysis 'more likely overestimates the potential for cognitive improvement' as compared with other reviews (the authors cite as an example the review by Rund²). On the contrary, we think that the comparison of results in individuals with schizophrenia with controls, which is an original feature of our review, has a sobering effect as it points out that 'practice [is] more likely than cognitive remediation to account for most of the improvements observed'. On the other hand, reviews (such as the one cited earlier) in which performances in people with schizophrenia are not compared with those of controls could mistake improvement in results for improvement in cognitive abilities.

Second, commenting on the methods we used in our meta-analysis, Balanzá-Martinez *et al* criticise three of our options: (a) the inclusion of studies with a test–retest duration under 1 year; (b) the inclusion of open trials (especially clozapine trials); and (c) the fact that we did not differentiate between 'patients with first-episode or chronic schizophrenia and geriatric patients'. They suggest therefore that it would be better to limit the analyses to a subset of the available data assuming, without formally testing, that some of the studies' characteristics significantly influence results.

At the time we made our analysis, there were only 11 studies with more than a year test–retest interval and only 4 reported data for a control group. It was not possible to limit our analysis only to this subset of studies, let alone further exclude studies or separately analyse subgroups of studies.

Instead, we chose to include all methodologically sound studies and test the role of potentially confounding variables (including test–retest interval). By using this method, we limited the loss of important available information. It is our belief that we could not gain more insight from fewer data.

As we report in our article, only 2 out of the 17 variables tested showed a larger improvement in studies with shorter test–retest intervals. Thus, for the vast majority of variables, excluding studies with shorter test–retest intervals would lead to an unjustified loss of information.

Third, Balanzá-Martinez *et al* consider that their suggestions may lead to results 'more helpful for clinicians, patients and caregivers'. We think that limiting the analysis to studies with large test–retest intervals (which usually have high attrition rates) or excluding the more naturalistic, open trials would achieve just the contrary.

In conclusion, although we agree with Balanzá-Martinez *et al* that the conclusions of our meta-analysis are not definitive, we also consider that to improve our knowledge on the subject we need new data, not new analyses, in subsets of (the same) data.

1 Szöke A, Trandafir A, Dupont ME, Méary A, Schürhoff F, Leboyer M. Longitudinal studies of cognition in schizophrenia: meta-analysis. *Br J Psychiatry* 2008; **192**: 248–57.

2 Rund BR. A review of longitudinal studies of cognitive functions in schizophrenia patients. *Schizophr Bull* 1989; **24**: 425–35.

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Neural correlates of formal thought disorder

Horn *et al*¹ investigate a very important, somewhat underexplored area of neural correlates of schizophrenic speech disturbance. Given the probability of underlying deficits in contextual integration and theory of mind, formal thought disorder yields a fertile ground for structural and functional connectivity analysis in schizophrenia. Although the use of hitherto unused techniques such as resting perfusion scan to study formal thought disorder must be lauded, the results of this preliminary study must be treated with caution for various reasons.

The composite score of the Scale for the Assessment of Thought, Language and Communication (TLC) has been used as a measure of severity of formal thought disorder. The authors have administered the scale 45 min before the scanning procedure for each participant. It is widely perceived that uncontrolled generation of thought is required to reliably measure formal thought disorder in schizophrenia. The TLC itself lacks a standardised practical method of eliciting such thought flow in contrast to some recently developed instruments.² The cross-sectional use of the TLC to measure formal thought disorder severity must be treated with prudence.

The authors extract components from the Positive and Negative Syndrome Scale (PANSS) using factor analysis and demonstrate that none of these components correlate with formal thought disorder severity as measured by the TLC total score. The validity of factor analysis in such a small sample is questionable and not in synchrony with available factorial structures of PANSS.³ As a result, all principal components extracted were from negative symptoms in PANSS (except the conceptual disorganisation item, which was rightly excluded from further analysis). Consequently, the results only show a lack of correlation between severity of formal thought disorder and negative symptoms as measured by PANSS. Findings from the magnetic resonance imaging may still be explained by positive symptoms alone and not by formal thought disorder. Lastly, the pervasive issue of sample size in neuroimaging studies becomes more prominent when correlation analyses are attempted in whole brain analyses.

1 Horn H, Federspiel A, Wirth M, Müller TJ, Wiest R, Wang J-J, et al. Structural and metabolic changes in language areas linked to formal thought disorder. *Br J Psychiatry* 2009; **194**: 130–8.

2 Liddle PF, Ngan ETC, Caissie SL, Anderson CM, Bates AT, Quedest DJ, et al. Thought and Language Index: an instrument for assessing thought and language in schizophrenia. *Br J Psychiatry* 2002; **181**: 326–30.

3 Fitzgerald PB, de Castella AR, Brewer K, Folia K, Collins J, Davey P, et al. A confirmatory factor analytic evaluation of the pentagonal PANSS model. *Schizophr Res* 2003; **61**: 97–104.

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Authors' reply: Palaniyappan suggests using the Thought and Language Index (TLI) instead of the TLC in order to quantify formal thought disorder. The TLI, he argues, would have the advantage over the TLC of a standardised method of eliciting thought flow. Unfortunately, the TLI was established after the start of our study,¹ whereas the TLC was an established instrument that has been successfully used in numerous studies as a reliable instrument to quantify formal thought disorder.² We agree that future studies might benefit from the application of the more standardised TLI. However, the distribution of the severity of formal thought disorder in the patient group should not change

substantially just by changing from the TLC to the TLI. Therefore, we regard our results as reliable.

Furthermore, Palaniyappan criticises the factor analysis of the PANSS in the small patient samples used in neuroimaging studies. He is right that a factor analytic approach in such small samples is critical if the patients group is randomly selected. In order to be able to study formal thought disorder with neuroimaging techniques, we recruited a specifically selected patient sample, which mainly differs in formal thought disorder severity and barely in other psychopathological categories. To obtain such a selected sample, the recruitment took years. Patients were matched as closely as possible for all other psychopathology traits in addition to the items of formal thought disorder of PANSS. The factor analysis and the correlation of the factors with the severity of formal thought disorder were only used to document this special patient selection. Therefore, it is no surprise that the factors of our factor analysis do not match the factors of unselected samples of patients with schizophrenia.³ To study specific psychopathological phenomena like formal thought disorder by means of neuroimaging, such a specific selection of patients is necessary to extract reliable results. In our patient group we did not observe any significant correlation between formal thought disorder and (a) positive symptoms (without PANSS item P2): $r=0.39$, $P=0.19$; and (b) negative symptoms (without PANSS item N5): $r=0.04$, $P=0.90$. We therefore can conclude that our results are due to positive or negative symptoms in general.

Concerning the issue of the sample size in neuroimaging studies, our results were corrected for multiple comparisons. This approach is a common and accepted way to handle this problem in neuroimaging studies in general.

- 1 Liddle PF, Ngan ETC, Caissie SL, Anderson CM, Bates AT, Queded DJ, et al. Thought and Language Index: an instrument for assessing thought and language in schizophrenia. *Br J Psychiatry* 2002; **181**: 326–30.
- 2 Andreasen NC, Grove W. Thought, language and communication in schizophrenia: diagnosis and prognosis. *Schizophr Bull* 1986; **12**: 348–59.
- 3 Fitzgerald PB, de Castella AR, Brewer K, Folia K, Collins J, Davey P, et al. A confirmatory factor analytic evaluation of the pentagonal PANSS model. *Schizophr Res* 2003; **61**: 97–104.

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Public attitudes towards mental illness

It is tempting to accept the conclusion from the paper by Mehta *et al*¹ that the Scottish anti-stigma campaign ‘see me’ has successfully influenced public attitudes north of the border. However, it is not clear that this is an appropriate conclusion from the data they present.

They describe random sampling techniques whereby 2000 adults representative of the UK population were surveyed. One presumes that this would give rise to cohorts in England which were roughly ten times larger than those in Scotland. Comparing the year 2000 against 2003, they observed a deterioration for 17/25 stigma questionnaire items in England against only 4/25 in Scotland, and concluded that Scotland’s dubious distinction of having done less badly suggested that ‘see me’ had been effective. Can they assure us that this difference did not arise simply because the much larger samples in England would be more likely to show a statistically significant difference than smaller Scottish samples?

- 1 Mehta N, Kassam A, Leese M, Butler G, Thornicroft G. Public attitudes towards people with mental illness in England and Scotland, 1994–2003. *Br J Psychiatry* 2009; **194**: 278–84.

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Authors’ reply: Professor Eagles writes to ask whether our conclusion from the paper is that the Scottish ‘see me’ anti-stigma campaign has positively influenced public attitudes about mental illness in Scotland. He suggests that this conclusion may be inappropriate because the populations in Scotland and England produce different sample sizes, given that the population of England is roughly ten times that of Scotland. We are very grateful to Professor Eagles for his comments as they allow us to provide some more information on these surveys than we could include in the original paper.¹

As he rightly says, direct evidence of the position in Scotland *vis-à-vis* England is not provided by comparison of only those significant changes within the two sites. We had hoped to pursue this question further with analysis of future surveys, which would have given us power to make the comparisons between Scotland and England adequately, but unfortunately the wording of the Scottish survey has been changed so this will not be possible. The existing data-sets do, however, show limited evidence in favour of Scotland when comparing their respective mean changes from immediately pre- to post-campaign periods (2000 *v.* 2003). Of the 25 items, 6 differed between the sites at a nominal 0.1 significance level. One item (26: fear of downgrading residential areas) favoured England at $P=0.05$. The others favoured Scotland: items 7–9 at $P=0.1$ (to do with tolerance), and items 10 and 13 (the need to spend money and care for people with mental illness) at $P=0.05$. As we stated in our paper, the evidence may be consistent with an early positive effect of ‘see me’, but this possible association requires further investigation, although we accept that it is far from conclusive and needs further verification.

- 1 Mehta N, Kassam A, Leese M, Butler G, Thornicroft G. Public attitudes towards people with mental illness in England and Scotland, 1994–2003. *Br J Psychiatry* 2009; **194**: 278–84.

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Is there core diffusion tensor imaging pathology in schizophrenia?

Kanaan *et al*¹ reported widespread abnormalities in white matter in 76 patients with schizophrenia compared with 76 healthy controls. A secondary analysis of 45 patients showed mean extracted fractional anisotropy scores to be unrelated to illness duration and duration of antipsychotic treatment. We wish to make two comments.

First, their main hypothesis that they would reconcile inconsistencies in the literature is a worthy, but elusive, goal. The problem of nosological heterogeneity ‘afflicts’ not only the definition of schizophrenia, but also the interpretation of fractional anisotropy localisation. Fractional anisotropy score localities are commonly cited in terms of grey or white matter terminology. Given such heterogeneity, it would suffice to adopt

the lesser goal of showing core pathology (in other words, the Venn diagram intersection). Coreness of pathology allows for diversity, without having to reconcile everything.

Second, we note that the secondary analysis was performed to dissect out the effects of the disorder from that of illness duration and treatment. The authors achieved this by using fractional anisotropy scores extracted from the principal analysis, which were then used to compare chronically with briefly medicated patients. Perhaps newly diagnosed, antipsychotic-naïve patients help most to partition out these effects² but they are not essential. An alternative approach is voxel-based ANOVA covarying for illness duration and atypical antipsychotic duration/dosage since this can help maximise anatomical coverage (particularly in the striatum where antipsychotic effects are detectable at even 2–3 weeks of treatment^{3,4}).

- 1 Kanaan R, Barker G, Brammer M, Giampietro V, Shergill S, Woolley J, et al. White matter microstructure in schizophrenia: effects of disorder, duration and medication. *Br J Psychiatry* 2009; **194**: 236–42.
- 2 Cheung V, Cheung C, McAlonan GM, Deng Y, Wong JG, Yip L, et al. A diffusion tensor imaging study of structural dysconnectivity in never-medicated, first-episode schizophrenia. *Psychol Med* 2008; **38**: 877–85.
- 3 Szeszko PR, Ardekani BA, Ashtari M, Kumra S, Robinson DG, Sevy S, et al. White matter abnormalities in first-episode schizophrenia or schizoaffective disorder: a diffusion tensor imaging study. *Am J Psychiatry* 2005; **162**: 602–5.
- 4 Chua SE, Deng Y, Chen EY, Law CW, Chiu CP, Cheung C, et al. Early striatal hypertrophy in first-episode psychosis within 3 weeks of initiating antipsychotic drug treatment. *Psychol Med* 2009; **39**: 793–800.

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Authors' reply: We agree that the process of establishing a definitive extent of white matter disruption in schizophrenia, and its relationship with illness duration and antipsychotic medication, is likely to be a lengthy one – larger studies such as ours notwithstanding. But we would like to clarify our reasoning

with respect to Chua & McAlonan's comments and the methodological alternatives they suggest.

First, we suspect that a 'core pathology' for white matter abnormalities in schizophrenia may be rather more elusive than the reconciliation we attempted. Although the recent meta-analysis by Ellison-Wright & Bullmore¹ has found areas of most common difference in the 15 studies they examined, it should be noted that only a fraction of the studies they looked at shared these differences – and the history of diffusion tensor imaging in schizophrenia is full of such conflicts.² Although there may indeed be areas of greater difference, the evidence is against any difference that is common to all.

Second, with regard to distinguishing the effects of duration of illness and antipsychotic medication, drug-naïve cohorts clearly offer enormous potential as the authors acknowledge. Such cohorts are difficult to obtain in high-income countries however, and the alternative approach they suggest – of ANOVA with covariation – has similar difficulties, since duration of treatment and illness will be so strongly correlated in most samples. We also note that the studies they cite as demonstrating the effectiveness of this approach either did not covary for medication exposure³ or did not use diffusion tensor imaging.⁴

- 1 Ellison-Wright I, Bullmore E. Meta-analysis of diffusion tensor imaging studies in schizophrenia. *Schizophr Res* 2009; **108**: 3–10.
- 2 Kanaan RA, Kim JS, Kaufmann WE, Pearlson GD, Barker GJ, McGuire PK. Diffusion tensor imaging in schizophrenia. *Biol Psychiatry* 2005; **58**: 921–9.
- 3 Szeszko PR, Ardekani BA, Ashtari M, Kumra S, Robinson DG, Sevy S, et al. White matter abnormalities in first-episode schizophrenia or schizoaffective disorder: a diffusion tensor imaging study. *Am J Psychiatry* 2005; **162**: 602–5.
- 4 Chua SE, Deng Y, Chen EY, Law CW, Chiu CP, Cheung C, et al. Early striatal hypertrophy in first-episode psychosis within 3 weeks of initiating antipsychotic drug treatment. *Psychol Med* 2009; **39**: 793–800.

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Corrections

Differential efficacy of escitalopram and nortriptyline on dimensional measures of depression. *BJP*, **194**, 252–259. The two last-named authors, Dr K.J. Aitchison and Professor P. McGuffin, are the GENDEP Principal Investigators and contributed equally to the work. In addition, Dr Aitchison's qualifications include MRCPsych.

Caption for cover picture, April 2009. *BJP*, **194**, A14. The text and image were submitted by Wojtek Wojcik; edited by Allan Beveridge.

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