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Psychopathy

Cooke et al (2007) claim that there is no compelling empirical evidence to support the conclusion that antisocial behaviour is a central feature of psychopathy. However, in the same issue of the Journal, Viding et al (2007) report a common genetic component to callous–unemotional traits and antisocial tendencies. Other studies cited by Viding et al report similar results. Moreover, Larsson et al (2007) reported that the same general four factors present in our four-factor model of psychopathy (Vitacco et al, 2003) all loaded onto a single genetic factor. Longitudinal research (not cited by Cooke et al) indicates that antisocial tendencies are significantly linked to the longitudinal stability of psychiatric traits (Frick et al, 2003). Cooke et al refer to the work of Cleckley (1988) to support their position, but in Cleckley's accounts of psychopathy antisocial behaviours play an important role. As Patrick (2006: p. 608) noted, 'There is no question that Cleckley considered persistent antisocial deviance to be characteristic of psychopaths. Without exception, all the individuals represented in his case histories engage in repeated violations of the law – including truancy, vandalism, theft, fraud, forgery, fire-setting, drunkenness and disorderly conduct, assault, reckless driving, drug offences, prostitution, and escape.' As Blackburn (2007: p. 145) recently put it, 'Contra Cooke, . . . antisocial behavior, conceived broadly, is a characteristic feature of psychopathy.'

In our paper based on a very large sample (Vitacco et al, 2005), we demonstrated the conceptual errors and flaws in modelling that went into the development of Cooke's model and provided evidence for the four-factor model. Interestingly, Cooke et al did not cite this large study but rather chose to cite our small preliminary studies, although they are in line with our larger study. We do not view criminality as central to psychopathy. Indeed, the Psychopathy Checklist – Screening Version (PCL–SV) contains two items that refer to antisocial behaviour and that can be scored without evidence of criminality. The PCL–R and PCL–SV are virtually identical psychologically, as noted previously by Cooke et al (1999).

Thus, the results reported by Cooke et al appear to be transparent but in reality no one will be able to unambiguously verify their analyses. When one analyses their published correlation matrix using a non-robust procedure, very different findings result. Also, Cooke et al relied upon a maximum likelihood procedure for estimating model parameters, despite the fact that it is well known that this procedure underestimates model parameters and model fit when used with ordinal data (Everitt & Dunn, 2001) such as the items of the Psychopathy Checklist – Revised. There was no serious discussion on why robust maximum likelihood with polythetic correlations was employed, except that it is recommended in the manual for EQS version 6. None the less, the verisimilitude of this new approach is currently unknown. A program such as Mplus, which employs a robust weighted least-squares procedure for ordinal data is an accepted approach (Neumann et al, 2006). Cooke et al's use of Mplus was limited. Our Mplus analyses of the UK data along with our previously

First, the authors continue to present an over-factored model (i.e. hierarchical three-factor model with testlets), which results in negative variances. This 13-item model actually contains 10 factors: 6 first-order factors/testlets, 3 second-order factors and 1 third-order factor (simply count the number of circles/factors in Fig. 1). Any model can achieve good fit when it is as complex as the data it attempts to summarise. We have shown that this testlet model results in untenable parameters in four separate studies (Neumann et al, 2006). One author of the Cooke et al paper has also suggested that the testlet model is over-factored (Skeem et al, 2003). Cooke does not acknowledge this problem of an over-factored model, even though it is evident in his published work (see Cooke & Michie, 2001, Figs 2 and 3, which contain zero variance terms that the EQS program sets to zero when estimating negative variances). Cooke et al (2007) mention that we have criticised their use of testlets but they do not dispute that it creates a misspecified model with untenable parameters. Our analysis of the testlet model is available upon request.

Cooke et al provided a polychoric correlation matrix, ostensibly to give investigators the opportunity to replicate their findings. However, as noted in the EQS program manual, robust procedures can only be conducted with the raw items. Thus, the results reported by Cooke et al appear to be transparent but in reality no one will be able to unambiguously verify their analyses. When one analyses their published correlation matrix using a non-robust procedure, very different findings result. Also, Cooke et al relied upon a maximum likelihood procedure for estimating model parameters, despite the fact that it is well known that this procedure underestimates model parameters and model fit when used with ordinal data (Everitt & Dunn, 2001) such as the items of the Psychopathy Checklist – Revised. There was no serious discussion on why robust maximum likelihood with polythetic correlations was employed, except that it is recommended in the manual for EQS version 6. None the less, the verisimilitude of this new approach is currently unknown. A program such as Mplus, which employs a robust weighted least-squares procedure for ordinal data is an accepted approach (Neumann et al, 2006). Cooke et al's use of Mplus was limited. Our Mplus analyses of the UK data along with our previously

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The article by Cooke et al (2007) contains a number of fundamental modelling errors.
published findings can be found online (http://bjp.rcpsych.org/cgi/eletters/190/49/s39).

Contrary to Cooke et al, the four-factor model clearly fits as well or better than a viable three-factor model. Moreover, our recent research indicates that the four first-order factors are explained by a cohesive superordinate factor (Neumann et al., 2006, 2007).


Involuntary community treatment

Swanson et al (2000) reanalysed the results of the North Carolina trial (Swartz et al, 1999) and their findings are becoming increasingly influential in current debates about mental health legislation in the UK. Our recent systematic review (Churchill et al, 2007), which included these articles, demonstrated that there was no robust evidence to indicate that community treatment orders are associated with either significant benefit or harm. The secondary analyses performed by Swanson et al are, we believe, misleading for two reasons.

First, based on everyone in the trial the intention-to-treat (ITT) effect of randomisation to an involuntary out-patient commitment (OPC) was of a modest and non-significant reduction in violence (risk difference of 4.5%). This overall ITT effect of OPCs is a weighted average of the ITT effects in the two subgroups of participants defined by their post-randomisation management (those who received short-term OPCs and those who eventually received long-term OPCs). These two subgroups would exist in the control arm had they been placed on OPCs. Assuming that there was no benefit in those who received the short-term OPCs (i.e. risk difference 0), the results of Swanson et al suggest that the reduction in violence in those with long-term OPCs would be 12.4%. However, even if considered clinically significant, this finding would still not be statistically significant because the overall ITT effect was not significant (assuming a zero ITT effect in those receiving short-term OPCs implies that a test of the hypothesis concerning those receiving long-term OPCs is equivalent to the test for the overall ITT effect). The only way in which there could have been a beneficial effect in those receiving long-term OPCs is if the effects in those receiving short-term OPCs were actually detrimental (i.e. increased the rate of violence). It is improbable that they would be, and in policy terms it would be unacceptable to impose OPCs in the knowledge that they would cause harm to those in whom they are only applied for a short period.

Second, a post hoc comparison of the outcomes in groups defined by management decisions or patient behaviour following randomisation is potentially subject to selection effects (hidden confounding). That this is in fact the case is illustrated by the results of other subgroup analyses by the same research group (Swartz et al, 1999: Fig. 1). The group destined to be on long-term OPC have a better clinical outcome in the first 1–2 months. In other words there is evidence that the group destined to receive long-term OPCs have a favourable clinical profile before the OPC is renewed. We believe that it is likely that long-term OPCs will only be contemplated under certain circumstances, such as when the short-term OPC has apparently made a difference. Those who have intractable problems or in whom a short-term OPC has failed to make any change might not have their OPC renewed.

The investigators responsible for the North Carolina trial accomplished one of the most extraordinary trials ever performed and as such deserve enormous praise. However, the results described in these and similar secondary analyses are, we believe, flawed and misleading, and should not be taken as evidence for a beneficial effect of OPC. We made a similar point (Szukler & Hotopf, 2001) following the publication of the original trial. The trial data are best interpreted using the main ITT analyses, which show no evidence of benefit or harm.


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Authors’ reply: Hotopf et al make essentially the same point that we stated in the article ‘... the study found no significant difference in the prospective rate of violence between the two randomly assigned groups: 32.3% in the OPC group v. 36.8% in the control group (Fisher’s exact test, one-tailed: P = 0.292; two-tailed: P = 0.567)’ (Swanson et al, 2000).

Critics of OPC policy might wish we had left it at that, but straightforward analysis of randomised controlled trials does not tell the whole story. In this case it excluded people with a documented history of serious violence (n = 64), since the court did not permit us to randomise these to the control group. However, variability in the real-world application of OPC allowed us to examine whether longer periods of court-ordered treatment were associated with lower rates of violence over the study year. They were.

Hotopf et al are rightly concerned about the possibility of favourable selection bias, but we think this is an unlikely explanation for our findings. Indeed, people with a history of treatment non-attendance were more than twice as likely to receive an extended period of OPC (40.0 v. 18.75%). If anything, this should have stacked the deck against finding an effect for long-term OPC.
Hotopf et al recalculated the post-randomisation effect for longer-term OPC in what they refer to as our ITT sample, rather than the sample we actually used. They say the effect is not significant but their calculation excludes the historically violent subgroup.

For hospital outcomes, unlike violence, we obtained follow-up information on the entire ITT sample through admission records. Here we found a statistically significant experimental result. For any month during the study year, the randomly assigned OPC group had a lower risk of readmission than the control group (OR = 0.64, P < 0.01). Hotopf et al do not mention this finding.

About one-third of the OPC group had their court orders expire very early in the study – during the first or second month – and more of these individuals were rehospitalised than those remaining on OPC, which explains the early separation of the lines in the figures from Swartz et al (1999).


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Psychosocial interventions for self-harm

Crawford et al (2007) conclude that the results of their meta-analysis ‘do not provide evidence that additional psychosocial interventions following self-harm have a marked effect on the likelihood of subsequent suicide’. This conclusion is far too bold considering the weaknesses inherent in the analytical approach employed. In my opinion Crawford et al have not allowed adequate weight for several methodological problems, the most prominent being the rationale for including studies in the analysis. They acknowledge the ‘lack of statistical power’ in the meta-analysis but offer a definitive and sweeping conclusion.

The lack of statistical power is only one reason not to conduct the meta-analysis. The central rationale for clustering the included studies is seriously flawed. Not only have they mixed simple interventions and treatments, the target populations range from latency-age children (some as young as 12 years) to older adults (> 50 years), intervention methods and theoretical orientations vary considerably (employing individual, group, case-management and home-based care), samples include those making suicide attempts as well as those engaging in self-harm (non-suicidal) behaviour, and they have also included studies that employed questionable intervention or treatment protocols for suicidality. A review of the intervention and treatment protocols of the studies included reveals wide variability in the nature, oversight and fidelity of the services being offered. I have serious concerns about at least 8 of the 19 study protocols. Some of the interventions cannot realistically be described as appropriate for suicidality, at least from the perspective that they have a serious chance of reducing subsequent pathology of suicide attempts, much less actual deaths. For example, Harrington et al (1998) employed four home visits by a social worker. Similarly, Guthrie et al (2001) included four sessions delivered in the patient’s home. Cedereke et al (2002) explored the utility of random telephone interventions and Clarke et al (2002) included ‘management enhanced by nurse-led case management’. As these examples illustrate, not all psychosocial interventions are the same, something Crawford et al (2007) failed to clarify in their article.

Why would we expect that a meta-analysis of randomised trials of interventions or treatments that are this broadly disparate (with samples equally disparate) would actually provide evidence of effective reduction of subsequent suicides?

Meta-analyses have become increasingly popular and increasingly misleading in their findings. Prior to inclusion in a meta-analysis of intervention or treatment outcome, I would suggest a thorough review of the intervention/treatment approach and related fidelity. Only those studies meeting strict and predefined criteria should be included. When considering strategies for including and clustering treatment studies for meta-analysis, it is particularly important to consider the targeted problem or disorder. Many, if not most problems targeted by psychosocial interventions and treatments are recurrent, persistent and potentially chronic in nature. Hence, the need for careful scrutiny of studies included.

Compounding the problems noted above, the follow-up periods for all of the studies included by Crawford et al ranged from 6 to 12 months. The efficacy of treatment or interventions for suicide will only be known after 5, 10 or 20 years. In shorter-term studies even if the results did show a reduction in subsequent suicides, we would not know whether the interventions or treatments were ‘delaying’ suicide or actually preventing it without longitudinal data.

There are many other factors that need to be scrutinised prior to inclusion of studies in a meta-analysis (e.g. sample size, categorisation of attempt status and suicide intent, fidelity/oversight of intervention or treatment) but space does not allow a full discussion. The point is that identifying appropriate inclusion criteria for such a study is a complex process which is far more complicated than simply taking all randomised controlled trials.

The definitive nature of the conclusion offered by Crawford et al belies the current state of the science in this area. In an age when legislators and funding agencies rely on science for direction, studies like this one generate ill-informed conclusions on what interventions, treatments and approaches to suicide prevention offer the most promise. Many readers will sadly and mistakenly carry away the message that psychosocial interventions offer no promise to reduce suicide rates.


Author’s reply: Professor Rudd raises important questions about whether it was appropriate to undertake this meta-analysis given the nature of interventions studied and the length of follow-up periods used. We believe that it can be appropriate to synthesis data from randomised trials to examine clinically important rare outcomes that individual studies are unlikely to be able to detect. For instance, psychosocial interventions for alcohol misuse are effective in reducing alcohol consumption but a range of factors, including clinical inertia, mean that they are not widely delivered. By synthesising data from trials conducted in a range of different settings, Cuijpers et al (2004) demonstrated that they are associated with a 30% reduction in subsequent mortality, a finding which may help to overcome some of the barriers to their delivery.

Although none of the studies we examined set out specifically to try to reduce suicide, it seems logical that interventions that are designed to reduce the incidence of suicidal behaviour should have an impact on the likelihood of fatal as well as non-fatal self-harm. Although several studies we included involved only brief interventions, such interventions have been shown to reduce the rate of suicide in other contexts, for instance in the period following discharge from in-patient psychiatric care (Motto & Bostrom, 2001).

Most of the studies we included followed people for between 6 and 12 months after the initial episode of self-harm. Although this is a relatively short period it is also the period during which suicide is most likely to occur (Owens et al, 2002). By focusing on the period immediately following an episode of self-harm we maximised the likelihood of being able to demonstrate an impact on the rate of suicide.

However, we fully endorse Professor Rudd’s comment that the results of our meta-analysis need to be interpreted with caution. Lack of data on suicide deaths in several of the trials that we identified meant that study power was limited. This resulted in wide confidence intervals around the pooled difference in suicide rates and it is therefore possible that additional psychosocial interventions do lead to reductions in subsequent suicide.


Psychiatric disorder and looked after status

Ford et al (2007) investigated the possible explanations for the increased prevalence of psychiatric disorder in children looked after by local authorities and linked looked after status with higher levels of psycho-pathology, educational difficulties and neurodevelopmental disorders. They suggested that services should bear in mind that a change of environment might be appropriate in providing help, at least in some cases.

After carefully reading the article, I think that Ford et al have missed an important aetiological factor: the influence of genetics. Studies (e.g. Howard et al, 2001) have shown that children of parents with mental disorder are likely to be looked after by another person or organisation. Biological factors which caused mental illness in the parents of children currently looked after by services might operate to cause the increased prevalence of psychiatric disorder in these children. Hence by neglecting the biological component of the bio-psychosocial model of mental illnesses, Ford et al have failed to provide a comprehensive assessment of causative factors in these children.

The authors could have included psychiatric disorder in the parents as a variable and divided the looked after group into children of parents with or without mental disorder. Ford et al have identified that neurodevelopmental disorders and learning difficulties are associated with increased prevalence of psychiatric disorder. Both are also associated with the future development of mental illnesses such as schizophrenia (Done et al, 1994; Lawrie et al, 2001) in which genetic factors play an important aetiological role (Cardno et al, 1999).


Authors’ reply We totally agree with Dr Sekar’s point that biological factors make an important aetiologial contribution to the development of psychiatric disorder in children. We certainly did not intend to suggest that biological factors are any less important than psychological or social factors. Many childhood disorders are known to have a high level of heritability (Rutter et al, 2006). However, we should not forget that both our and previous studies suggest that similar risk factors operate in looked after children as in children living in private households, but that looked after children tend to have been exposed to more of them, sometimes at greater intensity (Stein et al, 1996; Ford et al, 2007). In our opinion, this includes biological as well as psychological and social factors.

Many studies have shown that parental psychiatric disorder is correlated with childhood psychiatric disorder (Rutter,
2002). Although parental psychiatric disorder might increase the chance that children become12 after, this is not inevitable and there are many other reasons why children may enter the care system. In fact, only 6% of the children participating in the survey on which our analysis was based were accommodated primarily as a result of any type of parental illness. As the survey involved no contact with the biological parents of participants and historical information about children who are looked after is notoriously scarce, we had no way of accurately assessing the mental health of the biological parents. Our paper refers to our frustration at the extremely limited amount of information available to the survey and in clinical practice, and we explicitly state that our analysis cannot be seen as covering all potential risk and resilience factors.

Even if we had access to data on the mental health of the biological parents, an excess of children with psychiatric disorder among parents with psychiatric disorder would not necessarily indicate a biological or genetic basis for this finding. The mean age that children participating in this survey entered the care system was between 7 and 8 years, and we know that mental illness can have an impact on parenting practices. Do the children of parents with mental illness have raised rates of psychiatric difficulties as a result of genetic vulnerability and/or exposure to maladaptive parenting, or perhaps both processes occur at the same time and/or moderate each other? The literature suggests that parenting is an important mediating variable, although other genetic and environmental factors also play a part in the familial aggregation of psychopathology (Ramachandani & Stein, 2003). Cross-sectional surveys are not able to disentangle such complex questions, as data about exposures and outcomes are gathered at the same time. Longitudinal designs would be needed to explore Dr Sekar’s theory.


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Lithium for prevention of Alzheimer’s disease

Nunes et al (2007) reported that the prevalence of Alzheimer’s disease in a group of elderly patients with bipolar disorder who were on continuous lithium treatment was significantly less than in a similar group without recent lithium therapy. After controlling for age, lithium use remained associated with a smaller risk of Alzheimer’s disease (age-adjusted OR = 0.079, 95% CI 0.020–0.321). Conversely, Dunn et al (2005) showed that patients who received lithium had a significantly higher risk of dementia than those who did not (age-adjusted OR = 1.8, 95% CI 1.1–2.8).

Nunes et al (2007) found no differences between the lithium and the comparison group in neuropsychological performance after excluding patients with Alzheimer’s disease. This is in accordance with our study using Mini-Mental State Examination (MMSE) scores (Terao et al, 2006). Our study, however, showed that patients with present and/or past history of lithium treatment had significantly better MMSE scores than patients without any history of lithium treatment (Terao et al, 2006). It is important to further investigate lithium in the prevention of Alzheimer’s dementia with a large number of patients in prospective studies.

If lithium has a preventive effect for Alzheimer’s disease, there may be two possible mechanisms. First, it might indirectly prevent dementia via its prophylactic effects on mood disorders, because the rate of dementia increased 13% with every episode leading to admission for patients with depressive disorder and 6% for patients with bipolar disorder, when adjusted for differences in age and gender (Kessing & Andersen, 2004). Second, lithium might directly prevent dementia via its inhibition of glycogen synthase kinase 3 (GSK-3) alpha (Phipel et al, 2003) and GSK-3 beta (Phipel & Klein, 2001). Although Nunes et al (2007) found no significant differences in the number of previous depressive and manic episodes between the lithium and comparison groups, at present both possibilities should be borne in mind.


Authors’ reply: Dunn et al (2005) identified from the General Practice Research Database in the UK all cases of dementia between 1992 and 2002 (n=9954) and compared the number of prescriptions of lithium for individuals with this diagnosis with a control group without dementia (n=9374). They found that more patients with dementia (n=47, 0.47%) than controls (n=40, 0.43%) were exposed to lithium. We feel that this finding does not allow conclusions to be drawn as to whether lithium protects against or confers a risk for dementia, because it has been shown that patients with dementia have an increased risk of developing mania and depression (Nilsson et al, 2002) and are thus more likely to receive treatment, including lithium. Conversely, affective disorders themselves seem to increase the risk for dementia.

In a series of studies based on data from the Danish psychiatric case register, Kessing et al found that 14% of elderly patients with bipolar disorder and 16% with

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Dr Terao mentions the possible effects of lithium on GSK-3 beta. We recently investigated the effects of lithium on the transcriptional regulation of GSK-3 beta and found a significant reduction of its expression in primary cultures of rat hippocampal neurons as well as a reduction in regional intracerebral expression in lithium-treated adult rats and in leukocytes of elderly patients undergoing chronic lithium therapy for bipolar disorder (details available from the authors). These observations suggest a mechanism for GSK-3 beta inhibition by lithium, which may influence the formation of both amyloid plaques and neurofibrillary tangles, the two neuropathological hallmarks of Alzheimer’s disease.

We think that it is important to investigate further the potential protective effect of lithium in Alzheimer’s disease, as this could represent a low-cost universally available strategy to reduce the prevalence.

**Mortality and electroconvulsive therapy**

Munk-Olsen et al (2007) reported that the mortality rate from natural causes was lower for patients undergoing electroconvulsive therapy (ECT) than for other psychiatric in-patients. The lower relative risk was particularly significant for mortality linked to respiratory disease (RR=0.67, 95% CI 0.55–0.95) and a trend was found for cardiovascular disease (RR=0.85, 95% CI 0.70–1.03). The authors concluded that this decreased risk of mortality from natural causes is unlikely to be the result of a selection bias. They based this statement on: (a) the absence of absolute contraindications to ECT in patients with medical illness, and are more likely to ask for the opinion of a colleague in such a case (e.g. anaesthetist, cardiologist) (Benbow & Shah, 2002). Thus, patients with severe medical illness could be less likely to be treated by ECT. Furthermore, identification of cardiovascular diseases or pulmonary disorders, as well as physical examination and standard laboratory tests are part of a systematic screening procedure before ECT. This practice improves the diagnosis and the treatment of medical comorbidities. Indeed, the absence of such preliminary medical examination led to a high level of cardiac complications after ECT in the past (Gerring & Shields, 1982).

Accordingly current guidelines emphasise the importance of identifying and carefully managing patients with risk factors before, during and after ECT, as well as assessing the risks associated with anaesthesia (National Institute for Clinical Excellence, 2003). Patients receiving ECT are therefore not representative of all psychiatric in-patients. The careful assessment and treatment of their physical comorbidities contrasts with the increased rate of untreated physical illness in psychiatric patients, mostly because of inadequate somatic care in psychiatric units (Rasanen et al, 2006). Therefore, the observed diminution of mortality from natural causes in patients with ECT is more likely to be related to appropriate medical assessment and treatment than to a direct effect of ECT on physical health.

In an era that has seen ECT being opposed for political not clinical reasons, it was heartening to see an article on ECT addressing the very important issue of mortality. The study of Munk-Olsen et al (2007) is based on the Danish registry system which is acclaimed for its reliability, but certain issues need further clarification. It would have been relevant to know the total number of patients who received ECT and the total number of ECT treatments received by patients over the study period. Furthermore, the results could be better understood if information regarding physical comorbidity and the age of patients at the time of ECT had been provided. These variables can have a strong influence on mortality rates. In addition, as the study included only in-patients it is likely that the sample included patients who were severely ill. Also, the results show that inclusion of ‘days since last ECT treatment’ in the analysis causes the relative risk of mortality from natural causes of patients ‘discharged within the past 8–30 days’ to rise.

The relative risk of mortality from natural causes is also highest within 7 days of last ECT (RR=2.11), which is similar to the trend seen in deaths due to unnatural causes, especially suicide. Both these figures go against the conclusion of the authors that the mortality from natural causes is lower with ECT. It must also be noted that the relative risk of dying by suicide after...
ECT is 1.20, which is not significant but which the authors refer to as 'a marginally significant trend', and 'significantly increased suicide rate'. The finding that the risk from suicide is highest in the first 7 days after discharge and ECT is based on a small sample (n=6). Although the authors concede that admission status and time since discharge are important confounders in the analysis of suicide in patients with affective disorders, the statistical analysis does not consider these factors when calculating the relative risk of suicide after ECT. The authors discuss in some length the lack of a selection bias of patients with poor physical health. However, it is likely that patients with very poor physical health are not given ECT and this introduces a selection bias. Also, given the bias that occurs as patients at high risk for suicide are given ECT preferentially, this calls into question the validity of the conclusions. Further, it would have been very useful if the authors could have compared the death rates with those in the general population. This study provides several good research questions which need to be pursued further.


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Authors’ reply: Both Le Strat & Gorwood and Bharadwaj & Grover comment on the finding of a decrease in mortality in ECT-treated patients. In Denmark, all psychiatric patients are given a thorough medical assessment prior to any somatic treatment. This is partly because of the well-known cardiac contraindications for the use of tricyclic antidepressants which were widely used during the study period from 1976 to 2000, as the selective serotonin reuptake inhibitors (SSRIs) were only available in the latter part of the period described. Furthermore, SSRIs were generally considered less effective than tricyclic antidepressants or ECT in patients with severe depression. Accordingly, ECT was often used in patients with contraindications for tricyclic antidepressants. We are aware that this notion is at variance with several British guidelines (e.g. National Institute for Clinical Excellence, 2003) but it is in accordance with Danish and American Psychiatric Association guidelines, which state that the only contraindications to ECT are cerebral and other aneurysms. In Denmark, a preponderance of patients with medical illness is thus found among ECT-treated patients compared with those treated with tricyclic antidepressants and we therefore maintain our conclusion.

Drs Bharadwaj and Grover point out that admission status and time since discharge are important confounders. We fully agree and have hence adjusted for these variables in the analysis. The variables in Table 3 on risk of suicide in ECT recipients were mutually adjusted but this was not mentioned specifically in the footnote.

The number of patients dying by suicide in the first week after ECT discontinuation was small, and therefore our results should be interpreted with caution, as we mention in the discussion. Electroconvulsive therapy is often administered to patients who are assessed to be suicidal and we acknowledge that this could introduce selection bias (confounding by indication), which we also mention in our paper. These are the reasons why we concluded that: ‘the increased suicide rate among ECT patients shortly after treatment is probably a result of bias’ and we therefore disagree that the validity of the study is questionable regarding suicide rates after ECT.

A more in-depth description of the ECT patients can be found in a paper based on the same data (Munk-Olsen et al, 2006).

Measuring stigma

King et al (2007) frequently state that their stigma scale is measuring ‘the stigma of mental illness’ but, when closely scrutinised, it measures nothing other than stigmatisation perceived by users in outpatient, in-patient and crisis settings. There is no evidence that this is an objective assessment of stigmatisation. Users’ perception of stigma is affected by their mental state, depression, persecutory delusions or hallucinations. These symptoms can help to exaggerate the estimate of social stigmatisation (including rejection and discrimination) and hence the assessment is by no means an accurate measure. Measurements of more objective perceptions of stigmatisation can only be obtained from users in remission.

The reported negative correlation between self-esteem and perceived stigma can be confounded by high rates of both low self-esteem (e.g. Axford & Jerrom, 1986; Barrowclough et al, 2003; Blairly et al, 2004) and persecutory ideation and depressive cognition, including ‘self-stigmatisation’ in people with mental illness. Indeed, low self-esteem is a common symptom in psychiatric conditions such as depressive disorders, in which people can perceive more rejection and discrimination than warranted. Overemphasis on this correlation can divert attention from the fact that the correlation has to do more with people’s mental state than objective level of social stigmatisation.

An instrument can only be called ‘standardised’ if it is shown to be both reliable and valid. This instrument is not validated and so cannot be called standardised, on the basis of mere test–retest reliability. The correlation between the stigma scale and self-esteem scale is not an indication of validity of the instrument and although King et al admit this, they end up referring to their instrument as ‘standardised’ and to the correlation as ‘concurrent validity’.

A wide range of people with diverging diagnoses and mental states were recruited by King et al but there was no randomisation and no exclusion criteria. Even the ‘perceived stigmatisation’ cannot be attributed to a particular category of patients with a given diagnosis, or at least to psychiatric users in general, owing to lack of randomisation and inclusion of arbitrary proportions of participants with different diagnoses. This is likely to cause problems
Authors’ reply We were puzzled by Dr Haghighat’s criticism of our development of a stigma scale and would like to respond to his points. First, ours is a self-report measure of perceived stigma and we do not claim otherwise. Perceived stigma is a valuable construct that may have a greater impact on mental and social well-being (including relationships and occupation) than so-called objective acts of discrimination. This is also true of social support. Second, we agree that the relationship between perceived stigma and low self-esteem is potentially confounded by low mood. However, our sample contained a heterogeneous group of participants from a range of settings and thus it is unlikely that a sizeable proportion were depressed at the time of the study. In addition, Dr Haghighat overlooks the complexity of any putative association between stigma and depressive symptoms. Perceived stigma may cause or maintain depressive episodes.

Finally, participants in our earlier qualitative study (Dinos et al., 2004) emphasised that positive outcomes may arise from experiencing mental illness and thus such items were included in our scale. We reversed their scores to indicate that stigma might be greater when such positive aspects were lacking. This is not the same thing as assuming mental illness has only negative aspects. In parallel fashion the opposite of risk is not protection, it is lack of risk.

**Metabolic syndrome and intellectual disability**

Mackin et al. (2007) highlight the importance of screening and management of metabolic syndrome in patients with severe mental illness. This is particularly important in patients with intellectual disability as they have high rates of both physical and psychiatric comorbidities compared with the general population (Welsh Office, 1996). In addition, considerable evidence points to a disparity between the health of people with learning disability and the general population, and this was also highlighted in two Mencap reports (Mencap, 2004, 2007).

Suggested causes for this disparity include specific patterns of complex health needs associated with the aetiology of their intellectual disability, sensory and communication difficulties, reliance on carers to communicate their health needs, and barriers to healthcare accessibility due to poor professional knowledge and attitudes.

The Government White Paper **Valuing People** (Department of Health, 2001) acknowledges this disparity and identifies the improved healthcare of people with intellectual disability as a key outcome. However, the document is a little unclear on how these aims will be achieved.

As Mackin et al point out few studies specifically examine the impact of different models of care on physical well-being and comorbidities in people with severe mental illness, and this is also the case for people with intellectual disability. There is a pressing need for evidence-based integrated models of care for delivering high standards of care for this patient group.
Metabolic syndrome and intellectual disability
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