The Composite International Diagnostic Interview in low- and middle-income countries

Steel et al.1 should be commended for using an innovative design to show that the Composite International Diagnostic Interview (CIDI) 2.0 missed a large proportion of diagnoses that could instead be captured by an indigenously based Phan Vietnamese Psychiatric Scale (PVPS) among Vietnamese. Interpretations of the study should also consider the following.

1. Comparison between the self-report PVPS and CIDI included two other methodological issues that have little to do with whether the PVPS was indigenously devised. First, face-to-face structured interviews have long been shown to bias against Asian populations in eliciting psychiatric symptoms. By contrast, Asian populations typically scored as high as Westerners on many self-report scales such as the General Health Questionnaire.2 Second, unlike the 53-item PVPS, the CIDI contains multiple skip-outs from further symptom questioning unless mandatory DSM–IV core symptoms are endorsed. This renders the hierarchically configured CIDI much more prone to false negatives.3

2. The majority of diagnoses captured by the PVPS (72%) were in the somatisation category, but somatoform disorders were not assessed in the CIDI (because of difficulty in operationalising the concept of ‘medically unexplained symptoms’). Recent versions of the CIDI (3.0 and 3.1) contain a section on chronic pains and other physical illnesses, which have been shown to be common and highly comorbid with mental disorders in both high-income and low- and middle-income countries.4

3. The CIDI surely requires improvement regarding downward bias in prevalence estimates in Asian countries. China has used several versions of it (1.0 to 3.1). By adhering strictly to linguistic accuracy, the earlier versions generated unbelievably low prevalence of depression. Prevalence estimates continue to rise with successive versions and the latest survey using CIDI–3.1, by taking careful account of contextual equivalence of stem questions, interviewer training and quality control in the field, has found a prevalence of depression little different from rates in many Western countries. The Chinese CIDI has also provided highly consistent epidemiological data regarding specific disorder distributions, lifetime rates, psychosocial associations, physical/mental comorbidity, treatment-seeking and the opportunity for large-sample cross-national analysis.5 Enhancement of the CIDI may be both challenging and worth reconsidering in Vietnam.


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Authors’ reply: In summary, our report identified lower diagnostic concordance between the CIDI–2.0 and the indigenously derived PVPS among Vietnamese in the Mekong Delta region compared with Vietnamese in Australia. Whereas rates of mental disorder identified by the PVPS were stable across countries, the CIDI-identified mental disorder was three times lower in the Mekong Delta. Of particular importance was that the CIDI failed to detect 75% of Vietnamese with similar levels of disability identified by the PVPS.

Lee et al raise important questions that need to be resolved in order to make sense of the findings of international psychiatric epidemiology. We address some of their concerns in relation to our method. Although technically the PVPS is a questionnaire, it was administered in interview format as is common in the transcultural setting. Moreover, there is some evidence that among Vietnamese, there is a tendency to use a restricted range in reporting symptom severity on questionnaires,6 a factor that would yield conservative rates. Lee et al suggest that the skip rules of the CIDI may lower prevalence rates. We concur that the pre-eminence given to psychological rather than somatic stem symptoms in the hierarchical structure of the CIDI might limit positive endorsements in non-Western countries. However, if this effect was present it differentially had an impact on the Mekong Delta sample, underscoring the importance of culture and “Westernisation” as an influence on psychiatric assessment. We look forward to the publication of the results from the Chinese trials of the CIDI–3.1, which have reformulated the stem questions to be more compatible with somatic idioms of distress.

We do note, however, that removing PVPS cases that only reached threshold on the somatisation scale would have reduced our prevalence rates by 2.8% in Vietnam and 3.0% in Australia. Hence, the PVPS would still have identified a substantial number of cases not yielded by the CIDI. We note too that the Western-derived measure of neurasthenia recorded low rates in all samples, suggesting that somatic measures need to be culture specific.

In summary, there does not seem to be any major disagreement here. Whether we produce indigenous measures ab initio, as we have done, or modify existing measures as undertaken by Lee et al with the CIDI–3.1, the inference we draw remains the same: in order to detect the full range of disabling mental disorders across cultures, we need to have culturally appropriate measurements. We cannot simply apply the same measure with the same wording of items in the same format to all cultures and expect that we can compare the results. The
cost of applying either adapted or culturally developed measures, however, is that it confounds the process of making direct international comparisons of prevalence rates and mental health need. Hence, the real challenge facing world psychiatry is how to combine the strengths of psychiatric epidemiology7 with improvements in culturally valid assessment.8,9 Showing consistent patterns of comorbidity and risk-factor profiles across countries can only partially address this issue.


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BDNF Val66Met polymorphism and the affective component

I read the paper by Lencz et al1 with concern for the future of psychosis genetics. The authors claim that their candidate gene study of BDNF is ‘the first to demonstrate association with schizoaffective disorder but not schizophrenia’ and therefore that ‘BDNF variation is associated with psychiatric disorders with a primary affective component’. To reach this conclusion they argue on the basis of a sample size of 596 individuals against two meta-analyses and two cohort studies with sample sizes between 6 and 26 times larger (Table 1). Each of these studies examined the Val66Met polymorphism (the subject of Lencz et al’s report) and reached the conclusion that BDNF genotype does not exert an influence on the development of affective illness whether or not associated with psychosis.

A literature survey indicates that between 2004 and 2009 these authors between them published 25 papers relating to associations of 19 genes with aspects of psychiatric disease. Concerning one gene (FEZ1) they drew negative conclusions, but concerning each of the other 18 they claim a relationship was established. Such a rate of gene discovery would be a remarkable achievement. My review of the linkage literature,4 as represented by the four largest (each > 300 sibpairs) studies, suggests that none of Lencz et al’s candidate genes were replicated in these systematic searches, and the association study of Sanders et al5 that investigated six of them (DISCI, DAOA, HTTLPQ, DTPBP, COMT, DRD2) in 1870 individuals with schizophrenia or schizoaffective disorder and 2002 controls concluded these genes were unrelated to psychosis.

When large numbers of variables are examined, simultaneously alluring relationships can often be discerned that evaporate in the wider context of large and systematic studies. It appears that by ignoring this context Lencz et al are operating an algorithm for generating positive associations in selected data-sets.


Table 1 Main findings of two recent studies of the Val66Met variation in BDNF in relation to psychiatric diagnosis compared with Lencz et al1

<table>
<thead>
<tr>
<th></th>
<th>Controls, n</th>
<th>Schizophrenia, n</th>
<th>Schizoaffective disorder, n</th>
<th>Bipolar disorder, n</th>
<th>Depression, n</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kanazawa et al2</td>
<td>Meta-analysis</td>
<td>4035</td>
<td>2955</td>
<td>0.944</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Meta-analysis</td>
<td>6347</td>
<td>3143</td>
<td>0.161</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chen et al2</td>
<td>BMHHS</td>
<td>2367</td>
<td>553</td>
<td>0.360</td>
<td>596</td>
<td>0.834</td>
</tr>
<tr>
<td></td>
<td>ALS PAC</td>
<td>6042</td>
<td>596</td>
<td>0.834</td>
<td>596</td>
<td>0.834</td>
</tr>
<tr>
<td></td>
<td>Meta-analysis</td>
<td>11040</td>
<td>3879</td>
<td>0.537</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lencz et al3</td>
<td>HC V. S2</td>
<td>222</td>
<td>211</td>
<td>NS</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HC v. (S2A+B2p+MDD)</td>
<td>222</td>
<td>61</td>
<td>77</td>
<td>29</td>
<td>0.015</td>
</tr>
<tr>
<td></td>
<td>S2 v. (S2A+B2p+MDD)</td>
<td>211</td>
<td>61</td>
<td>77</td>
<td>29</td>
<td>0.008</td>
</tr>
</tbody>
</table>

ALSPAC, Avon Longitudinal Study of Parents and Children; BMHHS, British Women’s Heart and Health Study; HC, healthy controls; MDD, major depressive disorder; NS, not Significant; S2, schizophrenia; S2A, schizoaffective disorder.

Authors’ reply: Dr Crow is concerned that the publication of our recent study on BDNF endangers the field of psychiatric genetics. We would suggest that this concern may be overstated for the following reasons.

First, Dr Crow claims that the two meta-analyses and two cohort studies invalidate our results. We find this conclusion to be puzzling, given that none of these studies assessed the phenotype of schizoaffective disorder. Notably, the cohort studies relied on a single self-report item as the primary assessment of psychiatric diagnosis.
psychopathology. We addressed limitations of the meta-analyses in our original paper. We suggest that careful and comprehensive examination of the diverse phenotypes associated with neuropsychiatric illness may be a more fruitful approach.

Second, Dr Crow cites his own review of the linkage literature to suggest that most of the candidate genes reported by our group, and many others, are not supported by linkage studies and thus should be discounted. This reasoning is based on a flawed understanding of the role of linkage in complex disorders and is inconsistent with a large body of recent empirical evidence in complex genetics. In other complex disorders, a majority of susceptibility loci that have been unambiguously replicated in association studies fall outside of previously identified areas of even suggestive linkage (e.g. Barrett et al). Therefore, an argument utilising non-significant linkage data to invalidate a subsequent candidate gene association is erroneous.

Third, Dr Crow notes the productivity of our lab over the past several years as a source of concern for him. In so doing he mischaracterises our papers. First, he is simply incorrect in stating that only one paper reports strictly negative results (see Fubke et al and Hodgkinson et al). Moreover, many of our papers report complex relationships that are not so simplistically reduced to ‘positive’ v. ‘negative’. More importantly, Dr Crow fails to mention that most of our papers are not simply analyses of association to schizophrenia diagnosis, but instead examine alternative phenotypes. For example, our study of DRD2 assessed the relationship between a functional promoter region polymorphism and clinical response to olanzapine and risperidone in the context of a randomised controlled clinical trial in first-episode schizophrenia.4 Therefore, it is not surprising that our DRD2 results were not ‘replicated’ in either linkage studies or the association study of Sanders et al,5 as these papers were restricted to mere association to diagnosis.

Although Dr Crow is entitled to his opinions, the field of psychiatric genetics may be better served by more constructive discussion leading towards a better understanding of the complexities of these devastating disorders.


Outcome of group psychoeducation for stabilised bipolar disorders

The article by Colom et al further enhanced our understanding about the role of psychoeducation in the management of bipolar disorders. The study draws its strengths from the fact that it included an active control group and individuals with bipolar disorder and Axis II comorbidity, follow-up rates were excellent and the authors assessed the outcome in the form of the number and type of recurrences, time to recurrence, time spent ill and number of hospitalisations at 5 years. However, some of the issues require further clarification.

When one looks at the article reporting 2-year follow-up of the same cohort,2 the authors report that individuals with Axis I comorbidity were excluded, but at 5-year follow-up the authors report that only those with severe Axis I diagnosis were excluded. Further, the authors do not define ‘severe’. Individuals with bipolar disorder can have a high rate of comorbidity, hence clarification of this fact is very important from the perspective of generalisability of the study findings. In addition, Colom et al do not provide details of status and/or type of Axis I/II comorbidities and whether the drop-out rate and the number of completers made any difference with regard to clinical and demographic features.

Another important aspect is the way the authors defined recurrence based on rating scale scores. This type of definition in the true sense does not include the subsyndromal symptoms and can influence almost all the outcome measures such as time spent ill, time to recurrence and the number of recurrences, especially when the cohort is being followed up at a frequency of every 2 weeks. Similarly, although the study included the number and duration of hospitalisations as an outcome measure, the authors have not discussed the criteria for hospitalisation.

Another important aspect which needs clarification is the analysis of data. In many places Colom et al have used parametric tests to compare the numerical variables, although the standard deviation is more than the mean. Similarly, mean values are given for the number of recurrences without standard deviations, and comparison statistics are given as F-values. In Table 1, again the authors compare the mean values using Fisher F statistics and demonstrate that there was a significant difference in the number of days spent in each episode for all types of episodes. However, when one looks at the data, it is difficult to understand this contention. In the same table when one adds the mean number of days spent in each episode for the control group, the data regarding each episode and the total duration do tally, but the same is not the case for the psychoeducation group.


Authors’ reply: We would like to provide some clarifications in response to Gaur & Grover’s queries.

First, only those patients with ‘severe’ Axis I comorbidity diagnoses were excluded. This means that patients were excluded if
Abortion and mental health: established facts reconsidered

Tyrrer’s ‘From the Editor’s desk’ lyrically asserted that in relation to the paper by Fergusson et al and other studies, ‘In the parched desert of ignorance and prejudice every established fact becomes an oasis. By “established fact” I mean one that defines the field, the one that all the related and restless inchoate facts gather round and say “I belong here”, and then fall into line behind it.’

Fergusson et al conclude that there is evidence that abortion may be associated with a small increase in risk of mental disorders and in comparison, other pregnancy outcomes were not associated with increased risk. Although we acknowledge that aspects of their analytic design are strong and carefully implemented, we believe that the analyses have not maximised the potential of the data-set and that therefore, your editor’s rhetorical confidence is not yet justified. We advance the following reasons.

First, Fergusson et al dichotomised each pregnancy exposure. Of 534 women in the Christchurch cohort, 284 had had pregnancies. Women making decisions about terminating pregnancies may have prior pregnancy events and potentially cumulative losses will have different mental health impacts compared with termination as the outcome of a first pregnancy. Pregnancy variables are not independent and mutual adjustment in models for other outcomes will not account for the interactions between pregnancy outcomes. A more useful analysis would have been with a composite variable with never having had a pregnancy event as the reference category.

Second, the combining of therapeutic abortion for fetal malformation with abortion by choice is inappropriate. Most abortions are first trimester. There is an argument for separating termination of pregnancy by gestational age, so that the mental health impact of those in the second or third trimester are visible and separate. It is possible that terminating a wanted pregnancy because of fetal abnormality would be more distressing than an early unwanted pregnancy.

Third, many authors (including Fergusson et al) have found strong relationships between intimate partner violence and poor mental health, and between intimate partner violence and increased association with reporting terminations. Despite the potential to include the much more rigorous measure from their previous study of partner violence among this cohort, the authors have excluded their strongest measures of partner violence in this analysis, leaving a major covariate poorly measured.

Fergusson et al conclude that the evidence for abortion impact is small but clear – even causal. Yet there is no evidence that the risks associated with other pregnancy outcomes, particularly loss, are different from those estimated for abortion (see Charles et al), nor that mental health disorders are incident after an abortion. This could have been statistically tested using logistic regression among the range of statistical tests already carried out.

It is a pity that such a good cohort study has not been better analysed. With the above adjustments, the authors would be better placed to more clearly identify the vulnerable groups they are wisely seeking to identify.


Authors’ reply: Taft & Watson claim that we measured pregnancy history using dichotomous measures and that this fails to represent the complexities of pregnancy history. This claim misrepresents our analysis.
Correspondence

What we did was create four dichotomous measures of pregnancy history corresponding to: abortion ($X_1$); pregnancy loss ($X_2$); unwanted pregnancy coming to term ($X_3$); and other pregnancy ($X_4$). These dichotomous response variables were assessed cumulatively at four time periods (15–18, 18–21, 21–25 and 25–30 years). The consequence of this method of scoring is that the pregnancy history of the cohort was represented by four cumulative distributions assessed at four times. In our main analysis, the properties of these distributions were represented by the model:

$$G(Y_t) = B_0 + B_1X_{1it} + B_2X_{2it} + B_3X_{3it} + B_4X_{4it} + u_i + e_{it}$$

where $Y$ was the mental health outcome of interest assessed at time $t$ (15–18, 18–21, 21–25 and 25–30 years). This analysis takes into account the accumulative pregnancy history of our cohort and provides an effective method for representing the properties of multiple non-independent events assessed at multiple times.

Second, our definition of abortion could include fetal malformation. Although the reasons for abortion were not recorded in our study, available population figures show that in New Zealand, 0.6% of elective terminations are performed because of fetal malformation. ¹

Third, Taft & Watson claim that we did not use the strongest measure of domestic violence that we had available. This is not so, since measurements of domestic violence were not available at ages 15–18. The measure of sexual or physical violence victimisation used was based on repeated life event reports that included all physical or sexual assaults occurring at each time period. This covariate was significant in eight of the twelve regressions reported in Table 2.

Finally, the argument that our analysis does not establish that the mental health risks of abortion were greater than the risks of other pregnancy outcomes is not correct. What we showed was that: (a) abortion was associated with a small significant increase in mental health risks; and (b) other pregnancy outcomes were not associated with significantly increased risks. Recently, we have extended these findings to conduct a Bayesian analysis of the probability that the increase in risk associated with abortion (RR = 1.37) was greater than any increase in risk associated with unwanted pregnancy (RR = 1.11). This analysis used Markov chain Monte Carlo methods to model the distribution of $P(B_1 > B_2)$ adjusted for covariates, given the observed data and a non-informative prior distribution. This analysis showed that there was a greater than 90% probability that the small adverse effects associated with abortion were greater than the smaller adverse effects associated with unwanted pregnancy. This approach provides a more sensitive assessment of the equality of small effects than the logistic model proposed by Taft & Watson. The evidence from our study is consistent with the view that the adverse effects of abortion on mental health were greater than the adverse effects of unwanted pregnancy.


Corrections

Strengths and Difficulties Questionnaire Added Value Scores: evaluating effectiveness in child mental health interventions. *BJP*, 194, 552–558. The first equation on p. 554 should read:

Raw SDQ Added Value Score (in SDQ points)  
= 2.3 + 0.8 x baseline total difficulties score  
+ 0.2 x baseline impact score – 0.3  
x baseline emotional difficulties subscale score  
– follow-up total difficulties score

The online version of the paper has been corrected post-publication, in deviation from print and in accordance with this notice.

Prevalence of autism-spectrum conditions: UK school-based population study. *BJP*, 194, 500–509. The following should be included under ‘Funding’ (p. 508): This study was conducted in association with the NIHR CLAHRC for Cambridgeshire and Peterborough. Also, Patrick Bolton’s affiliation is now MRC SGDP Centre, Institute of Psychiatry, London.

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Authors' reply
David M. Fergusson, L. John Horwood and Joseph M. Boden
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