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Correspondence

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Contents

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,[1] 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[2] 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

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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 epidemiology[3] with improvements in culturally valid assessment.[4,5] 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 al*[1] 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 al*[5] that investigated six of them (*DISC1*, *DAOA*, *HTTLPR*, *DTNBP1*, *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.

1 Lencz T, Lipsky RH, DeRosse P, Burdick KE, Kane JM, Malhotra AK. Molecular differentiation of schizoaffective disorder from schizophrenia using *BDNF* haplotypes. *Br J Psychiatry* 2009; **194**: 313–8.

2 Kanazawa T, Glatt SJ, Kia-Keating B, Yoneda H, Tsuang MT. Meta-analysis reveals no association of the Val66Met polymorphism of brain-derived neurotrophic factor with either schizophrenia or bipolar disorder. *Psychiatric Genet* 2007; **17**: 165–70.

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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

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

	Controls, *n*	Schizophrenia, *n*	Schizoaffective disorder, *n*	Bipolar disorder, *n*	Depression, *n*	*P*
Kanazawa *et al*[2]						
Meta-analysis	4035	2955				0.944
Meta-analysis	6347			3143		0.161
Chen *et al*[3]						
BWHHS	2367				553	0.360
ALSPAC	6242				596	0.834
Meta-analysis	11 040				3879	0.537
Lencz *et al*[1]						
HC *v.* Sz	222	211				NS
HC *v.* (SzAf+Bip+MDD)	222		61	77	29	0.015
Sz *v.* (SzAf+Bip+MDD)		211	61	77	29	0.008

ALSPAC, Avon Longitudinal Study of Parents and Children; BWHHS, British Women's Heart and Health Study; HC, healthy controls; MDD, major depressive disorder; NS, not significant; Sz, schizophrenia; SzAf, schizoaffective disorder.

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