

## Correspondence

Edited by Kiriakos Xenitidis and  
Colin Campbell

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## Depression and dementia

Chen *et al*<sup>1</sup> conclude that a dose–response association exists between severity of depression and the risk of subsequent development of dementia. However, certain methodological issues need to be considered. First, were the Chinese and British cohorts comparable? As per Copeland *et al*,<sup>2</sup> the patients in the Medical Research Council – Ageing in Liverpool Project Health Aspects (MRC–ALPHA) study were drawn from family practitioner lists and included those living in nursing homes, whereas the Chinese cohort was derived wholly from the community. The nature of the British cohort would suggest a predisposition to increased rates of physical and depressive comorbidity even before study entry. Second, are the numbers enough? For instance, the Chinese cohort included only four patients with Level 4 depression out of which three developed dementia. Although the hazard ratio (HR) is 5.05, the confidence interval is quite wide (95% CI 1.56–16.3). The conclusions drawn should be supported by a power analysis as there may be a danger of a type 1 error in concluding that severity of depression is related to subsequent dementia. Third, organic syndromes were significantly more prevalent in those with Level 4 depression compared with patients with Level 3 depression in the MRC–ALPHA study. When these cases are excluded, the CI of the HR for Level 1, 2 and 3 depression overlap (Level 1: 95% CI 0.84–2.21; Level 2: 0.52–1.40; Level 3: 0.53–1.44; Level 4: 1.00–3.57), which would indicate that although differences in the subsequent development of dementia in patients with differing severity of depression are suggestive, these are not significant. The development of subsequent dementia may have been related to the pre-existing and progressive organic insult rather than depression *per se*. Although this study is important and timely, the results and implications thereof are suggestive rather than conclusive.

- 1 Chen R, Hu Z, Wei L, Qin X, McCracken C, Copeland JR. Severity of depression and risk for subsequent dementia: cohort studies in China and the UK. *Br J Psychiatry* 2008; **193**: 373–7.
- 2 Copeland JR, McCracken CF, Dewey ME, Wilson KC, Doran M, Gilmore C, et al. Undifferentiated dementia, Alzheimer's disease and vascular dementia: age- and gender-related incidence in Liverpool. The MRC–ALPHA Study. *Br J Psychiatry* 1999; **175**: 433–8.

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**Authors' reply:** We appreciate Dr Singh's interest in our recent article,<sup>1</sup> but we believe Dr Singh misunderstands our findings. First, in our two cohort studies we did not say that there was an existing dose–response association between the severity of depression and the risk of dementia. On the contrary, our data have suggested that only the most severe syndromes and cases

of depression are a risk factor for developing dementia. Second, we put the two cohort studies from China and the UK in one paper because both of them used the same Geriatric Mental State–Automated Geriatric Examination for Computer Assisted Taxonomy method for the assessment and diagnosis of depression and dementia (in syndromes and cases), which would provide equivalent data between the centres for the proposed analysis. Although there are significant differences in ethnicity, social demographic background and disease patterns between the two populations, the results are consistent, suggesting our findings are robust. Third, it is known that in many Eastern countries elderly people are looked after at home, rather than in nursing homes. Traditionally, the Chinese family would take care of their frail and sick elderly.<sup>2,3</sup> Both studies aimed to enumerate all the cases of dementia wherever they were found. Thus, we believe the composition of the Chinese cohort is similar to that of the UK cohort in terms of its predisposition to physical and depressive comorbidities. Fourth, we have mentioned in the paper that the small number of dementia cases in the Chinese cohort is one of the study limitations. Nevertheless, the UK cohort data support the Chinese findings. Therefore, we believe the findings are quite reliable. Fifth, in our sensitivity analysis excluding all participants with baseline organic syndrome at Level  $\geq 3$ , we did not say that the risk of dementia was associated with depression at Levels 1, 2 and 3 because their HRs are around 1.00 and not significant, but our data have further shown that only the most severe depression (i.e. Level  $\geq 4$ ) is a risk factor for developing dementia.

- 1 Chen R, Hu Z, Wei L, Qin X, McCracken C, Copeland JR. Severity of depression and risk for subsequent dementia: cohort studies in China and the UK. *Br J Psychiatry* 2008; **193**: 373–7.
- 2 Chiu HF, Zhang M. Dementia research in China. *Int J Geriatr Psychiatry* 2000; **15**: 947–53.
- 3 Chen R, Wei L, Hu Z, Qin X, Copeland JR, Hemingway H. Depression in older people in rural China. *Arch Intern Med* 2005; **165**: 2019–25.

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## Alcohol-related brain damage: not a silent epidemic

We read with interest the Editorial by Gupta & Warner,<sup>1</sup> in which they postulated a possible 'silent epidemic of alcohol-related dementia'. We welcome the call for increased awareness, but would make the following points.

First, nosology. The term 'alcohol-related dementia' (alcohol-induced persisting dementia in DSM–IV) has generally been superseded in clinical practice by the (more accurate) term 'alcohol-related brain damage/injury', incorporated nationally in many countries, including Scotland.<sup>2</sup> We were particularly concerned about the authors' exclusion of Korsakoff's syndrome from the aetiology of this entity. The prevailing best synthesis of the evidence on aetiology is the Lishman hypothesis,<sup>3</sup> namely that the alcohol amnesic (Korsakoff's) syndrome exists on a spectrum with other forms of alcohol-related brain injury, thiamine depletion injury interacting with alcohol neurotoxicity, resulting in what the authors conceptualise as alcohol-related dementia.

Second, in Scotland, alcohol-related brain damage has been incorporated into health policy and promotion, with statistics – produced by the Information and Statistics Division (ISD) of