

## 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

the Scottish Health Service (available since 1997) – showing increased rates of general hospital admissions over the past 8 years (155 patients identified under age 65 and 99 patients over 65 in 1996–1997 compared with 287 and 185 respectively in 2004–2005) (ISD, personal communication, 2006). There is acknowledgement in Scotland of the effects on both those under and over 65 years, with provision of specific services, distinct from dementia services, recognising the differing needs of this group.

Third, although we accept there is no conclusive evidence on effects of hazardous alcohol use over the life-course in terms of the emergence of a dementia syndrome in old age, we would urge caution in claiming a ‘silent epidemic’. To date there has been no definitive neuropathological evidence of alcohol being a primary aetiological factor in a dementia syndrome of old age. We would suggest, from clinical experience, that the effects of significant alcohol use in the elderly are better conceptualised as short-term contributory effects on more significant causes of cognitive impairment (e.g. Alzheimer’s disease, vascular dementia), in keeping with current thinking.<sup>3</sup> This would be seen as a separate process from the spectrum of primary alcohol-related brain damage.

Fourth, the quoted epidemiological studies do not provide clear evidence to suggest that ‘alcohol-related dementia’ will increase at a population level, other than in cohorts with sustained alcohol dependence and poor nutrition. These findings, for those without dependency, are equivocal at best, and do not demonstrate population effects of hazardous drinking on increasing dementia rates. These results illustrate the heterogeneity of alcohol’s effects on differing populations, with there being equivalent evidence for moderate drinking having an association with better cognitive function.<sup>4</sup>

In conclusion, our view would be that the effects of alcohol excess on cognition are heterogeneous in terms of clinical syndrome, and multifactorial in terms of aetiology (including individual susceptibility factors, yet to be determined). The more relevant concept is that of alcohol-related brain damage, where undoubtedly presentations have increased in recent decades, in parallel with rates of alcohol dependency in the UK.

1 Gupta S, Warner J. Alcohol-related dementia; a 21st-century silent epidemic? *Br J Psychiatry* 2008; **193**: 351–3.

2 Dementia Services Development Trust, University of Stirling. *A Fuller Life. Report of the Expert Group on Alcohol Related Brain Damage*. Scottish Executive, 2004. (<http://www.alcoholinformation.isdscotland.org/>)

[alcohol\\_misuse/files/ARBD\\_afullerlife.pdf](#).

- 3 Lishman WA. *Organic Psychiatry: The Psychological Consequences of Cerebral Disorder* (3rd edn). Blackwell Science, 1998.
- 4 Rodgers B, Windsor TD, Anstey KJ, Dear KBG, Jorm AF, Christensen H. Non-linear relationships between cognitive function and alcohol consumption in young, middle-aged and older adults: the PATH Through Life Project. *Addiction* 2005; **100**: 1280–90.

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**Author’s reply:** We thank Drs Jauhar and Smith for their comments which serve to highlight the lack of consistent approach to alcohol-related dementia.

Among clinicians there is generally good awareness of Korsakoff’s syndrome as a subacute sequel to prolonged heavy drinking and nutritional deficiency (among other causes). We agree there may be a continuum between pure Korsakoff’s and dementia. However, our article was intended to raise awareness of the less well-recognised, broader dementia category at the other end of this spectrum.

Getting tied up in nosological arguments (alcohol-related brain damage or alcohol-related dementia) is unlikely to help get across the health message. We believe ignoring the word ‘dementia’ may reduce the impact of the message and conflate several neurological sequelae of alcohol misuse. The increase in general hospital admissions in Scotland reported by Jauhar & Smith serve to reinforce our message.

We agree there is no definitive neuropathological link between alcohol consumption and dementia, although epidemiological studies do suggest an association. There is simply insufficient research on this point. To conjecture that absence of evidence equates to evidence of absence is hazardous.

We are overwhelmed by the level of positive national and international media and scientific interest in our article. Hopefully, this will result in our twin aims: increasing awareness and stimulating research in this area.

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## Correction

Look into my eyes: psychiatry in the movies. *BJP*, **194**, 116. Sentences six to nine should read: The crossover with psychiatry was established early: *Dr Mabuse the Gambler* (1922, plus six remakes) is credited as cinema’s first psychoanalyst and *Testament of Dr Mabuse* (1932) was spiked with quotes from *Mein Kampf*. *Cabinet of Dr Caligari* (Germany, 1919, pictured) was at once horror show and Nazi metaphor: the Nazis’ detestation of both this film (banned as ‘degenerate Art’) and psychiatry are documented elsewhere. Hypnotists steal and defraud (*The Magician* 1926), but ‘taking advantage of the ladies’ was a central activity, beginning with *Under the Hypnotist’s Influence* (1897). In the decade ending 1939, 75% of screen hypnotists were evil or mad; mostly both.

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