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.[3] 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.[4]

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.

Sameer Jauhar, Kershaw Unit, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 OXH, UK. Email: sameerjauhar@gmail.com; **Iain D. Smith**, Kershaw Unit, Gartnavel Royal Hospital, Glasgow, UK

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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.

James Warner, Central North West London NHS Foundation Trust, London, UK. Email: j.warner@ic.ac.uk; Susham Gupta, St Charles Hospital, London, UK

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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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