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Editorial

# Addressing alcohol-related dementia should involve better detection, not watchful waiting

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**Summary**

Alcohol-related dementia represents an underrecognised mental disorder with both clinical and public mental health aspects. There is considerable scope for improving its assessment within both mainstream and specialist mental health services, but ongoing challenges remain in ensuring its timely detection so that appropriate preventative and rehabilitative interventions can be applied.

**Declaration of interest**

None.



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Fifty years ago, the underrepresentation of older people presenting to alcohol information centres was observed as being partly attributable to the 'diminished life expectancy of the alcoholic'.[1] This observation has now been superseded by one that reflects the cohort of 'baby boomers' born between 1946 and 1964, all of whom are currently over the age of 50 and presenting to alcohol services in larger numbers than previous generations. This has been reflected in larger rises in alcohol misuse (including alcohol-related admissions and deaths) for this population compared with younger people over the past 20 years.[2]

The neuropathology and pathophysiology of Wernicke–Korsakoff (amnestic) syndrome (WKS) as a result of thiamine deficiency has been clearly defined for many years. However, the relationship between heavy alcohol consumption and dementia has been less clear for reasons that include the apparent lack of a specific neuropathology, the role of comorbidities such as cerebrovascular disease and traumatic brain injury, and the difficulties in distinguishing the effects of alcohol from degenerative dementias such as Alzheimer's disease and frontotemporal dementia.[3,4] In spite of this, it is becoming clearer that alcohol-related dementia is emerging as a distinct mental disorder.

## Brain damage or dementia?

The nosological status of alcohol-related dementia as a distinct mental disorder remains hotly contested when interpreting the findings from studies of alcohol-related neuropathology and pathophysiology. Alcohol-induced brain injury is a consequence of direct neurotoxicity, oxidative stress, excitotoxicity from alcohol withdrawal, apoptosis, disruption of neurogenesis and mitochondrial damage. It is also further compounded by neuropathological changes typical of WKS, that can also accompany alcohol-related dementia. The direct effects of alcohol toxicity result in a continuum of cognitive dysfunction and behavioural change. These changes vary from less severe uncomplicated alcohol-induced brain injuries to those that are more severe and are complicated by nutritional deficiencies.[5] Neuroimaging and neuropathology studies indicate that the cardinal feature of alcohol-induced brain injury is a reduction in white matter within the frontal cortex, which is responsible for the disruption of the fronto-cerebellar circuit.[5]

The neuropsychological profile of alcohol-related dementia has not been as well studied, but there is evidence that in comparison with Alzheimer's disease, language impairment is unlikely; with better performance on semantic tasks and on verbal memory recognition, but worse performance on visuospatial tasks.[4] However, as many also have features of WKS, there is often profound anterograde amnesia, impaired recall of past events and impaired executive functioning.[4]

A UK cohort study of people with a mean age of 56 found that drinking above 32 UK units of alcohol per week was associated with greater global impairment in cognitive function, as well as in memory and executive function 10 years later.[6] These global changes in cognitive function go beyond the neuropsychological profile of amnestic syndrome, which does not characteristically affect visuospatial function. There is also evidence for some degree of improvement in both white matter changes and neuropsychological impairment following abstinence in people with alcohol-related dementia.[7]

The clinical presentation of alcohol-related dementia brings us closer to defining a mental disorder with a better defined neuropsychological profile. Although Oslin *et al* refined the diagnostic criteria to improve specificity by including measures of duration and severity of alcohol consumption, excluding characteristic features of Alzheimer's disease and vascular dementia and linking dementia with a minimum abstinence time before onset,[8] these criteria have yet to be validated in methodologically sound prospective studies.[4]

## Challenges and solutions

There remains a need to further explore the extent to which alcohol misuse can exacerbate primary degenerative and vascular dementias, traumatic brain injury and alcohol-related brain damage from other mechanisms such as other substance misuse and other physical disorders affecting brain function. There is also a dearth of evidence around the role of drug treatment in alcohol-related dementia, including the treatment of behavioural and psychological symptoms.



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In 1990, alcohol ranked as the sixteenth leading cause of morbidity in England for the 50–69 age group. In 2013, it became the sixth leading cause.[9] At a public health level, there are mixed reports about the role of alcohol consumption and the development of dementia. An overview of systematic reviews of epidemiological studies predominantly involving participants aged 60 years and over concluded that light-to-moderate alcohol consumption might reduce the risk of Alzheimer's disease whereas heavy or excessive consumption did not affect the risk.[10] However, a recent prospective study of people with a mean age of 43 at baseline followed up for 30 years found evidence of cognitive decline and hippocampal atrophy even with moderate alcohol consumption.[11]

The potential for reversibility has considerable implications for early intervention within addiction and old age psychiatry services. An increasing focus in the community on the prevention of dementia might provide another incentive for the early detection of alcohol-related dementia. Encouraging engagement with addiction services enables a smooth passage to the provision of cognitive rehabilitation interventions.[12] With alcohol-related dementia displaying an identifiable neuropsychological profile, there is a need to incorporate wider neuropsychological testing and explore problems that distinguish frontal-lobe-mediated personality change from other features of alcohol dependence such as withdrawal and intoxication.

Given the presence of such cognitive impairment at younger ages, routine cognitive testing within addiction services would help to guide the primary and secondary prevention of alcohol-related dementia. This should also be mirrored by the provision of screening, brief intervention and referral for treatment in mainstream mental health services.

We need to address service provision and establish parity of esteem for alcohol-related dementia with alcoholic-related physical disorders such as alcohol-related liver disease; however, this cannot happen unless we tackle the broader issue of help-seeking, overcoming stigma and improving access to services. Against the background of a growing population of older people with alcohol misuse, no time can be lost to watchful waiting.

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## References

1 Edwards G, Fisher MK, Hawker A, Hensman C. Clients of alcoholism information centres. *BMJ* 1967; **4**: 346.

2 Rao R, Crome I. Alcohol misuse in older people. *BJPsych Adv* 2016; **22**: 118–26.

3 Lishman WA. Alcoholic dementia: a hypothesis. *Lancet* 1986; **327**: 1184–6.

4 Ridley NJ, Draper B, Withall A. Alcohol-related dementia: an update of the evidence. *Alzheimers Res Ther* 2013; **5**: 3.

5 Savage L. Alcohol-related brain damage and neuropathology. In *Alcohol and the Adult Brain* (eds J Svanberg, A Withall, B Draper, S Bowden): 108–25. Psychology Press, 2015.

6 Sabia S, Elbaz A, Britton A, Bell S, Dugravot A, Shipley M, et al. Alcohol consumption and cognitive decline in early old age. *Neurology* 2014; **28**: 332–9.

7 Bartsch AJ, Homola G, Biller A, Smith SM, Weijers HG, Wiesbeck GA, et al. Manifestations of early brain recovery associated with abstinence from alcoholism. *Brain* 2007; **130**: 36–47.

8 Oslin D, Atkinson RM, Smith DM, Hendrie H. Alcohol related dementia: proposed clinical criteria. *Int J Geriatr Psychiatry* 1998; **13**: 203–12.

9 Ilomaki J, Jokanovic N, Tan EC, Lonnroos E. Alcohol consumption, dementia and cognitive decline. *Curr Clin Pharmacol* 2015; **10**: 204–12.

10 Public Health England. *Health Profile for England: Trends in Morbidity and Behavioural Risk Factors*. Public Health England, 2017. (https://www.gov.uk/government/publications/health-profile-for-england/chapter-3-trends-in-morbidity-and-behavioural-risk-factors).

11 Topiwala A, Allan CL, Valkanova V, Zsoldos E, Filippini N, Sexton C, et al. Moderate alcohol consumption as a risk factor for adverse brain outcomes and cognitive decline: longitudinal cohort study. *BMJ* 2017; **357**: j2353.

12 Wilson K, Halsey A, Macpherson H, Billington J, Hill S, Johnson G, et al. The psycho-social rehabilitation of patients with alcohol-related brain damage in the community. *Alcohol Alcohol* 2012; **47**: 304–11.



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