| Section A: CPH to complete | |
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| **Address:** | Chair, Liverpool Health Partners, Foundation Building, University of Liverpool, |
| **Guidance title:** | Disability, dementia and frailty in later life - mid-life approaches to prevention |
| **Committee:** | PHAC D |
| **Subject of expert testimony:** | Alcohol |
| **Evidence gaps or uncertainties:** | |
| Relationship of alcohol consumption and the development and onset of dementia, disability and frailty | |
| Effectiveness of interventions to reduce alcohol consumption on the development and onset of dementia, disability and frailty in the general population | |
| How the mid-life population may differ from the general adult population | |
| | |
| **Section B: Expert to complete** | |
| **Summary testimony**: | [Please use the space below to summarise your testimony in 250 – 1000 words – continue over page if necessary ] |

There is strong evidence on the impact of alcohol on disability and death. The WHO acknowledge alcohol as the biggest single risk factor globally for disability adjusted life years lost before the age of 60. This is because of the peak age of death / disability of alcohol-related deaths being 40-60, for example from violent deaths or cirrhosis. There are possible cardiovascular benefits of low-level consumption but these benefits accrue from 1-2 drinks a week, apply only to older people and are negated by 'binge drinking' at least once a month. There is no good evidence of differential effects of different drinks but spirits are more likely to be associated with heavier consumption. Patterns of drinking are important as well as total amount and binge drinking more strongly associated with violence and sudden death. There is a strong association between death and disability form alcohol and other measures of health inequality. There is increasing evidence and awareness about the link between alcohol consumption and many common cancers, such as breast and colon cancer.

The levers to reduce this disability in a population have been well characterised and the strongest are price, availability and marketing. Evidence on education and information alone changing behaviour is weak. Price appears the most effective measure and a minimum price per unit of alcohol (MUP) has the benefit of targeting the cheapest alcohol, favoured by the heaviest and youngest drinkers, and that purchased in off-licenses and supermarkets. Cheap supermarket alcohol has changed the UK into a predominately home-drinking culture. In marketing, there are

many examples of a targeted advertising campaign changing youth culture and consumption patterns. There is evidence that young people exposed to alcohol marketing are more likely to start drinking younger and drink more when they do.

There is some evidence on a protective effect of low-level alcohol consumption for dementia but it is difficult to separate this from the clustering of low level drinking with dietary and many other healthy lifestyle factors. There is strong evidence of alcohol related brain damage (ARBD) producing a significant burden on NHS resources and there is NICE guidance on its management underway. Much of the damage is related to thiamine deficiency. There is also increasing evidence for brain development continuing into the third decade of life (20's) and so concern of the long-term impact of heavy teenage drinking on later cognitive function.

**References (if applicable):**

Ridley NJ et al. Alcohol-related dementia: an update of the evidence. Alzheimer's Research & Therapy 2013, 5:3 http://alzres.com/content/5/1/3