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Commentary on Connor: Strong epidemiological evidence that alcohol causes cancer but how might the public interpret this information?

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Statement of competing interest

Carol Emslie is a member of the Alcohol Research UK Grants Advisory Panel and has received research funding from Scottish Health Action on Alcohol Problems. Sara Macdonald has no financial or other relevant links to companies with an interest in the topic of this article.

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Concise Statement

Strong epidemiological evidence about the causal role of alcohol consumption in cancer may not fit easily with complex lay understandings of the causes of ill health

Commentary

Connor (1) argues there is strong epidemiological evidence that alcohol causes cancer but highlights uncertainty about how this message may be understood by the 'wider public'. We agree there is public confusion and scepticism about public health advice on drinking, and that alcohol industry actors will seek to exploit this by 'framing' the debate in ways which further their commercial interests (2, 3). However, there is also a pressing need to take into account the wider social context in which any new scientific evidence is introduced (4).

People have multifactorial understandings of the causes of chronic disease which encompass family history, environmental factors, stress and luck, as well as behavioural factors such as smoking and drinking; these factors are perceived as interconnecting rather than independent and may become more or less salient at particular points in the lifecourse (5, 6). Davison and colleagues (7, 8) argue that, in an iterative process similar to formal epidemiology, 'lay' epidemiologists extract information from mainstream health messages and observe patterns of illness and death among family, friends, acquaintances and those in public life to generate and reformulate hypotheses about 'candidates' for particular health problems. Gender, age, socio-economic status are also relevant here (9, 10). Crucially, uncertainty is a key feature of lay epidemiology; some 'candidates' are observed to 'do all the wrong things' but live long lives ('unwarranted survivals': "the fat 'Uncle Norman' figure who has survived into a healthy old age, despite extremely heavy smoking and drinking" (5) p682) while other people lead apparently 'healthy' lives but still suffer premature illness or death ('anomalous deaths'). The 'anomalous death' of a relative is particularly important in undermining epidemiological evidence about risk factors for major diseases (11)

There are a number of reasons why the message 'alcohol causes cancer' may not fit easily with lay epidemiology. First, candidacy only indicates increased risk – not an inevitable outcome – so a message which emphasises certainty rather than probability may be disregarded. Research investigating the acceptability of cancer warning messages among Australian drinkers (12) suggests that statements about alcohol leading to an 'increased risk of cancer' performed better than those which stated that alcohol 'can cause cancer'.

Secondly, lay epidemiology encompasses an understanding of the different meaning of risk factors at the population and individual level: "most people have notions about what renders a person 'a candidate' for a specific disease (incorporating 'expert' epidemiological understandings of risk at the population level) whilst simultaneously understanding that life, health and death defy prediction at an individual level" (11)(p445). Thus, simple health messages which focus exclusively on behavioural risk factors at an individual level draw attention to 'unwarranted survivals' and particularly to 'anomalous deaths' in people's social networks. The observation that behavioural change does not guarantee a reduction of risk at an individual level (mirroring epidemiological concerns about the 'prevention paradox' (11, 13)) may therefore result in public scepticism.

Thirdly, while smokers are currently viewed as ‘candidates’ for cancer, it appears that drinkers are not; lay people struggle to find an explanation for non-smokers who get cancer and instead emphasise the unpredictability and randomness of the disease (14). Finally, it is important to explore how terms such ‘drinkers’ and ‘drinking’ might be interpreted by the wider public. While ‘smoking causes cancer’ draws on the commonly understood binary opposition between smokers and non-smokers, ‘drinking causes cancer’ may well be understood as comparing ‘heavy’ drinkers with ‘light’ or ‘moderate’ drinkers, rather than contrasting drinkers with non-drinkers. Qualitative research in the UK demonstrates that drinking is perceived as a routine activity associated with sociability, pleasure, and relaxation and that heavy weekend drinking and drinking to intoxication are normalised; thus, drinking alone or choosing NOT to drink alcohol are behaviours which require explanation, not excessive drinking in general (13, 15-17). In addition, people construct themselves as responsible, moderate drinkers and position other groups as the ‘problematic’ drinkers. For example, respondents in midlife portray themselves as ‘experienced’ drinkers and younger people as irresponsible ‘problem’ drinkers (15), younger adults position older people, especially older women drinking heavily in public, as problematic (18) while younger middle class women position working class women as vulgar and excessive drinkers (19). This suggests that ‘drinking causes cancer’ may be interpreted as *‘other people’s excessive drinking causes cancer’*.

Connor demonstrates the strength of the evidence for alcohol consumption as a cause of cancer. Further research on how lay people conceptualise drinkers and drinking when assessing candidacy is necessary before this message can be communicated effectively to the public.

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