Commentary

Balancing the Bio in a Biopsychosocial Model of Hazardous Drinking and Alcohol Use Disorders

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How alcohol problems are represented, including as 'Alcohol Use Disorder' (AUD), has a broad set of implications for research, policy and practice. A biopsychosocial approach is commonly offered as a means of taking into account the various environmental and individual level factors that may contribute to so called mental and behavioural disorders including AUD. In this reply we argue that the reference article presents a heavy focus on 'bio' factors without sufficiently acknowledging the potential costs of doing so, particularly that a focus on individual level 'bio' factors may undermine the utilization of effective environmental policy levers whilst potentially harming AUD recovery processes. Thus, we call for a more balanced focus on the 'psychosocial' factors related to AUD.

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In their recent primer (*Hazardous drinking and alcohol use disorders. Nat. Rev. Dis. Prim. 2022 81* **8**, 1–25 (2022))[1], MacKillop and colleagues present an extensive account of hazardous drinking and alcohol use disorder (AUD), purporting to take a biopsychosocial evaluation of causes and treatment. Whilst in agreement with much of the evidence presented, on close reading we wish to challenge the heavy focus on *biological* factors, particularly concerning the role of genetics. In response, we wish to highlight several additional concepts and issues concerning the aetiology, prevention and treatment of hazardous drinking and AUD.

Disproportionately emphasising biogenetic factors might lead to the conclusion they are the most important determinants of hazardous drinking and AUD, and as such, warrant interventions narrowly

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focused on modifying individual-level risk. Yet decades of evidence from psychological and behavioural sciences demonstrates that behaviour is significantly shaped by the multiple overlapping environments in which we live – physical, economic, social, and commercial[2]. Specifically, the most effective and cost-effective approaches to reducing hazardous use and preventing AUD are population-level (i.e., public health) strategies including increasing pricing, reducing ease of purchase and modifying social norms, which are significantly under-utilised policy levers[3]. Research supports the concept of an evidence hierarchy, with upstream (i.e., population-level) approaches consistently achieving a larger, more rapid, more equitable, and greater cost-savings relative to downstream individually-targeted interventions[3].

Whilst potentially shifting focus away from actions further up the effectiveness hierarchy, an excessively biogenetic focus may drive placing of responsibility (or 'blame') for AUD on an individuals' essence[4][5]. This strategy is adopted by sections of the alcohol industry to suggest that only a subpopulation of drinkers are at risk for AUD and to push back against effective population-level policies[6]. Further, common perceptions of people with AUD as a distinct outgroup (e.g., genetically different) underpins stigma as a major barrier to AUD recovery[4]. Indeed, emphasising societal over individual causes of addiction can be an important stigma reduction strategy[7] and genetic aetiological perceptions are implicated in a myriad of undesirable social and behavioural outcomes[5][8][9], including for AUD[4]. For example, believing oneself to have a gene associated with 'alcoholism' can engender a sense of reduced control over one's drinking[10]. Conversely, believing oneself not to have an 'alcoholism' gene predicts increased dismissiveness of AUD problems[11].

As MacKillop and colleagues acknowledge, genetic factors may explain only a "relatively small" proportion of AUD risk variance. In this case, we question the level of focus on biomedical factors, when, as the authors state, "the functional significance of genetic variants and polygenic risk scores for AUDs… is largely unclear" and "neuroimaging research has not yet generated clinically informative indicators for improving diagnoses, prognosis or treatment planning" (p.18). In this respect, MacKillop and colleagues' claim to take a biopsychosocial approach is insufficient in accounting for the limitations and potential costs of focusing on 'bio' factors, particularly given the aforementioned significance of sociocultural factors in driving hazardous drinking and AUD prevalence and remission[2][3][6]. Biogenetic attributions can result in individual and cultural beliefs with complex and generally (but not always) harmful consequences for stigma and recovery outcomes[4][5][9][12]. As such, emphasis on biological or individual-

level factors risks undermining both population and individual behaviour change factors conducive to addressing hazardous drinking and AUD.

In one specific example of an overly genetic interpretation, whilst examining environmental risk factors, MacKillop and colleagues state: “Although an earlier age of drinking initiation was initially considered a risk factor for hazardous drinking and AUDs, supporting evidence is inconsistent [citation] and earlier onset drinking may be better understood as a behavioural marker of increased genetic risk”[1] (p.6). Whilst we agree that more evidence is needed to identify what role early onset drinking may play as a risk factor for AUD, we argue that the available evidence is insufficient to suggest early onset drinking may be *better* understood as a behavioural marker of genetic risk (compared to understanding it as a marker of environmental or social risk, for example). To suggest this represents a subtle example of genetic determinism[9]. As MacKillop and colleagues acknowledge, genes appear to interact with environmental factors, yet no clear clinical or policy rationale for understanding early onset alcohol use as a behavioural marker of increased genetic risk is presented. There are also multiple other reasons why early onset alcohol use may be damaging to the developing brain, including harms to brain structure and activity, cognitive functioning, as well as hindering educational achievement[13]. The precautionary principle suggests that claims that have the potential to negatively affect individuals should be held to higher evidential support standards before dissemination[5]. Given the highlighted potential damaging effects of exposure to genetic aetiological claims in general[8], and for AUD specifically[10][11], we argue that such claims should be avoided while robust evidence is absent.

Rather than placing undue emphasis on AUD as a biogenetic disorder, AUD, and hazardous drinking in particular, should be considered *primarily* as a public health issue that reflects the significance of commercial determinants of health in their aetiology and future reduction[3][6]. This does not mean rejecting biomedical approaches to AUD – which would amount to environmental determinism[9] – but that communications such as MacKillop and colleagues’ primer should explicitly and with similar emphasis identify the psychological and sociocultural nature and drivers of hazardous drinking and AUD[14] in their biopsychosocial approach. Other AUD models such as the Addictions Neuroclinical Assessment or Etiologic Theory-based Ontogenetic Hierarchical Framework retain biomedical factors (see[14]) are examples of contemporary models that consider biogenetic, dimensional aetiologic mechanisms of AUD (although have room to incorporate environmental mechanisms) while avoiding essentialism. A continuum model of alcohol use and harm, whereby use, problems, and AUD are emphasised as dimensional (rather than as discrete categories as biomedical models can imply), has also

been proposed as an important public health approach[4]. Further, evidence suggests that continuum beliefs about AUD offer potential benefits to addressing important issues of stigma, low problem recognition, self-change and help-seeking amongst people with AUD[4][15].

We wish to challenge MacKillop and colleagues' conclusion that furthering biobehavioural approaches should be a priority for the field. Despite decades of significant investment in biomedical research and treatments, few significant advances or gains in the prevention or treatment of hazardous drinking have been made. Rather, AUD prevalence and associated harms such as stigma and low treatment engagement have remained persistently high. We call for a prioritisation of AUD models and aetiology that highlight the important sociocultural and commercial drivers that are modifiable through more robust policy action whilst averting the pitfalls of biogenetic determinism and essentialism[5][9]. In offering this critique, we wish to acknowledge our own positionality as, whilst having no conflicts of interest to declare, we acknowledge the backgrounds of the authorship team as primarily rooted in social, clinical, or cognitive psychology and public health.

Competing interests

The authors declare no competing interests.

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Declarations

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