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Correspondence

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Western depression is not a universal condition

Thornicroft *et al*[1] assume that 'mental disorder' is an entity essentially lying outside situation, society and culture, which is identifiable anywhere using a common (Western) methodology such as the Composite International Diagnostic Interview (CIDI). Biologically triumphalist studies like this simply have to be challenged, because once something – in this case, depression as a unitary pathological entity arising naturalistically anywhere in the world – is declared real, it becomes real in its consequences.

The authors cite at the outset the World Health Organization (WHO) claim that depression is the first or second most burdensome disease, disability-wise, in the world. To me this is perhaps the most bizarre statement to come out of a major medical institution in the modern era: more burdensome than AIDS or tuberculosis, which each take around 1.5 million lives per year, and with millions more disabled over the years? The disability-adjusted life-years metric (DALY) on which the WHO claim rests is epistemologically lamentable when applied in this way.

The CIDI is described by the WHO as a survey instrument produced for standard use across cultures. This does not mean it is valid. The authors concede that 'no attempt was made to go beyond DSM-IV criteria to assess depression-equivalents that might be unique to specific countries', and that 'the reliability and validity of diagnoses made with the WMH CIDI may vary across countries'. This doesn't appear to deter the authors, yet it renders their conclusions risible.

Western psychiatric templates simply cannot generate a universally valid knowledge base, since they fail the core test of validity, which relates to the 'nature of reality' of subjects under study. Invalid approaches cannot be redeemed by 'reliability' – using a standard, reproducible method – since the very ground they stand on is unsound.[2] This is hardly surprising since, organic categories aside, diagnoses are merely descriptive constructions, conceptual devices, and are drawn up by us, not by nature. Ironically there is a WHO study, reported by Sir David Goldberg and colleagues, which showed that in 15 cities around the world those people recognised as depressed by doctors did no better (indeed they did slightly worse) than comparable others who were not so recognised.[3]

Depression has no exact equivalent in non-Western cultures, not least because these do not share a Western ethnopsychology that defines 'emotion' as internal, often biological, unintentioned, distinct from cognition, and a feature of individuals rather than situations.[4] Here we see the Western psychological discourse setting out abroad to instruct, regulate and modernise, presenting contemporary Western mentality and ways of being a person as definitive anywhere. Why should this imperialism suit the rest of the world?[5]

Half the countries surveyed here were low-income ones. What is 'mental health' in the poverty-haunted, near-broken parts of the world? Thinking of my own country, Zimbabwe, how would invalid approaches distinguish between depression and situational distress? Does Africa need the category of Western depression at all, and does it need the marketing of antidepressants which will ride in on the back of papers like this in international psychiatric journals? I think not.

1 Thornicroft G, Chatterji S, Evans-Lacko S, Gruber M, Sampson N, Aguilar-Gaxiola S, et al. Undertreatment of people with major depressive disorder in 21 countries. *Br J Psychiatry* 2017; **210**: 119–24.

2 Summerfield D. How scientifically valid is the knowledge base of global mental health? *BMJ* 2008; **336**: 992–4.

3 Goldberg D, Privett M, Ustun B, Simon G, Linden M. The effects of detection and treatment on the outcome of major depression in primary care: a naturalistic study in 15 cities. *Br J Gen Pract* 1998; **48**: 1840–4.

4 Lutz C. Depression and the translation of emotional worlds. In *Culture and Depression. Studies in the Anthropology and Cross-Cultural Psychiatry of Affect and Disorder* (eds A Kleinman, B Good): 63–110. University of California Press, 1985.

5 Summerfield D. Afterword: Against global mental health. *Transcult Psych* 2012; **49**: 519–30.

Derek A. Summerfield, consultant psychiatrist, South London and Maudsley NHS Foundation Trust. Email: derek.summerfield@slam.nhs.uk

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Does prescribing psychiatric medication really make it less likely that alcohol is involved in a self-poisoning?

Making causal assertions from complex cross-sectional data is risky and may lead to erroneous clinical advice. Although a negative association has been revealed between alcohol ingestion in self-poisoning and taking psychiatric medications (particularly a tricyclic or a typical antipsychotic),[1] individuals who are prescribed these medications may be different from those who are not, even after adjusting in covariate analysis for a generic category of 'psychiatric diagnosis'. This association even led Chitty *et al* to speculate that D2 antagonists might reduce the use of alcohol. However, there is evidence to the contrary: flupenthixol led to more drinking when tested in randomised controlled trials (RCTS),[2] and olanzapine caused a similar trend.[3] In the remaining 10 of 13 RCTs found in a systematic review, antipsychotics did not reduce drinking.[4]

Clearly, there are various interpretations of the association that was found. For example, perhaps people who have access to highly sedating and potentially lethal drugs such as tricyclics and antipsychotics can self-poison seriously without recourse to added alcohol.

While Chitty *et al* raise some interesting questions, we are concerned that those reading the abstract alone might misperceive a role for antipsychotics in drinkers. Suicide rates in people who drink heavily might be best prevented by improving treatment and access to treatment for alcohol use disorders.

1 Chitty KM, Dobbins T, Dawson AH, Isbister GK, Buckley NA. Relationship between prescribed psychotropic medications and co-ingested alcohol in intentional self-poisonings. *Br J Psychiatry* 2017; **210**: 203–8.

2 Wiesbeck GA, Weijers HG, Lesch OM, Glaser T, Toennes PJ, Boening J. Flupenthixol decanoate and relapse prevention in alcoholics: results from a placebo-controlled study. *Alcohol Alcohol* 2001; **36**: 329–34.

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4 Kishi T, Sevy S, Chekuri R, Correll CU. Antipsychotics for primary alcohol dependence: a systematic review and meta-analysis of placebo-controlled trials. *J Clin Psychiatry* 2013; **74**: 642–54.

Jonathan Chick, Medical Director, Castle Craig Hospital, and Visiting Professor, School of Health and Social Care, Napier University, Edinburgh. Email: jonathan.chick@gmail.com; **Colin Drummond**, Professor of Addiction Psychiatry; **Julia Sinclair**, Senior Lecturer

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Co-consumption of alcohol and psychotropic medications in episodes of non-fatal self-poisoning attended by ambulance services in Victoria, Australia: evidence of potential modification by medical severity

Chitty and colleagues' recent investigation[1] into the association between psychotropic medication use and alcohol consumption during emergency department presentations for self-poisoning raises an interesting perspective on the putative role of psychopharmacology in reducing risky alcohol use among those at risk of self-harm and suicide.

Episodes of attempted suicide resulting in hospital presentation may underestimate the true extent of psychotropic medication and alcohol co-ingestion across the community, given recent findings suggesting that just over half of patients treated by ambulance paramedics following an episode of self-harm and/or attempted suicide are transported to hospital.[2]

Using data from our ongoing study of psychiatric presentations to ambulance services,[3] we extracted information on all episodes of non-fatal self-poisoning in the state of Victoria, Australia, from January 2012 to December 2016 ($N = 24\,726$). In contrast to Chitty and colleagues, we found that, overall, use of psychotropic medications was associated with an increased, not decreased, risk of alcohol co-consumption in the self-poisoning episode (odds ratio (OR) = 1.35, 95% CI 1.28–1.42).

While anticonvulsants (OR = 0.74, 95% CI 0.65–0.84), antipsychotics (OR = 0.81, 95% CI 0.75–0.86) and psychostimulants (OR = 0.52, 95% CI 0.32–0.85) were associated with a decreased risk of alcohol co-consumption, in contrast to Chitty and colleagues, we found that benzodiazepines (OR = 1.60, 95% CI 1.52–1.69) were associated with an increased risk of alcohol co-consumption. Additionally, we found no significant association between antidepressant use and risk of alcohol co-consumption for these presentations (OR = 1.04, 95% CI 0.97–1.11).

Importantly, however, we found that medical severity may modify these associations. Specifically, most associations were reduced to non-significance when considering those not requiring hospital treatment following the self-poisoning episode: all psychotropic medication classes (OR = 1.12, 95% CI 0.76–1.65), anticonvulsants (OR = 0.39, 95% CI 0.09–1.80), antidepressants (OR = 1.05, 95% CI 0.63–1.77), antipsychotics (OR = 0.81, 95% CI 0.48–1.36), benzodiazepines (OR = 1.40, 95% CI 0.94–2.07) and psychostimulants (OR = 0.44, 95% CI 0.02–9.21).

This highlights the importance of considering the breadth of services that people who engage in self-harm come into contact with, so as to provide a fuller picture of the treatment needs of this population and how these may vary as a consequence of medical severity.

1 Chitty KM, Dobbins T, Dawson AH, Isbister GK, Buckley A. Relationship between prescribed psychotropic medications and co-ingested alcohol in intentional self-poisonings. *Br J Psychiatry* 2017; **210**: 203–8.

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Katrina Witt, Post-Doctoral Research Fellow, Turning Point, Eastern Health Clinical School, Monash University, Australia. Email: katrina.witt@monash.edu; **Dan Lubman**, Professor of Addiction Studies and Services; **Belinda Lloyd**, Associate Professor of Addiction Studies and Services; **Karen Smith**, Manager, Research and Evaluation and Adjunct Professor, Monash University, Australia

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Authors' reply: We thank Chick *et al*, and Witt *et al*, for their welcome responses to our article.[1] We agree that both improving access and facilitating referral to alcohol services are essential strategies with regard to reducing deliberate self-poisoning that may be a product of harmful use of alcohol.

We share the concerns of Chick and colleagues – it is dangerous to make causal assertions from cross-sectional data, especially if preliminary analyses and author interpretations are introduced into clinical practice from the abstract alone. We agree that people prescribed tricyclic antidepressants and typical antipsychotics are different from those on other drugs – that they are less likely to co-ingest alcohol during intentional self-poisoning is one such example. As highlighted by Chick *et al*, the underlying nature of this relationship (whether it is causal or correlated because of shared factors) has many possibilities, for which we presented three interpretative and non-mutually exclusive speculations. We agree with the further interpretation put forth in their letter – individuals with increased access to higher-toxicity medications may negate any perceived role of alcohol in the poisoning. Of course, this is only relevant in cases when alcohol is used as a tool to facilitate the self-harm (i.e. to 'numb fears') as opposed to the person being intoxicated before the desire to self-harm arises. It is noteworthy that a recent study found that over 70% of people interviewed after a suicide attempt that involved acute alcohol use reported they did not use alcohol to facilitate the action.[2] However, we recognise that the methods of suicide attempts in this aforementioned small sample size study were heterogeneous and that self-poisoning is more likely to involve alcohol as a substance perceived to increase the toxicity of the poison or mask the taste of the co-ingested substances. We are currently conducting a study to investigate patient self-reported reasons for use of alcohol before and during deliberate self-poisoning, which will further shed light on this.

We are pleased that our analysis prompted Witt and colleagues to investigate a similar line of enquiry within their own cohort. The similarities between the data analysis conducted by Witt *et al* and our findings are notable – those prescribed antipsychotics, anticonvulsants and stimulants were less likely to co-ingest alcohol during a non-fatal self-poisoning.

Compared with the Japanese study cited by Witt *et al*, in which nearly half of individuals are not transported to hospital after suicide attempts or episodes of self-harm, our experience specifically for deliberate self-poisoning (via toxicology services and Poison Information Centres) tells us this is not the case in