WHO's Global Clinical Practice Network for mental health

The core constitutional responsibilities of the WHO include the promotion of global cooperation, acting as a directing authority for international initiatives that contribute to the advancement of health. The Global Clinical Practice Network (GCPN), created by WHO's Department of Mental Health and Substance Abuse, holds promise for promoting collaborative initiatives that enhance training, research, and clinical capacity for mental health worldwide. Eventually, these initiatives can change the way that mental health care is practised globally.

The GCPN was established as a multidisciplinary and multilingual vehicle for the investigation of proposed diagnostic guidelines for mental and behavioural disorders in the ICD-11, which is being prepared to present to the World Health Assembly in 2017. The first GCPN members were international psychiatrists and psychologists participating in formative field studies to inform initial revision efforts and the overall architecture of the mental disorders classification.[1–3] Since then, the GCPN has expanded substantially to include more than 11 700 mental health and primary care professionals from 139 countries. GCPN members are contributing their time and expertise to essential research initiatives that support WHO's goal to reduce the global disease burden of mental disorders through developing a more clinically useful and globally applicable diagnostic manual.

38% of GCPN members live in low-income and middle-income countries, which shows its feasibility as a platform for research collaboration in these settings. GCPN registration is available in nine languages, and 57% of participants have registered in languages other than English. GCPN members are participating in multilingual, systematic case-controlled field studies[4] of proposed ICD-11 diagnostic guidelines to test their reliability, clinical utility, and global applicability. Several studies have been successfully implemented, providing a novel and valuable set of methods for rigorous and efficient worldwide field studies.

Beyond the ICD-11, the GCPN holds great potential as a laboratory for advancing research, training, and clinical initiatives to improve the quality and coverage of mental health care worldwide through the advancement of the objectives outlined in WHO's Mental Health Action Plan 2013–2020.[5] Specifically: (1) the GCPN is a powerful research platform that can provide direct point-of-service information about how health professionals encounter and treat people with mental health needs; (2) the GCPN can provide the necessary structure and governance to enable participation and collaboration by clinicians in critical international mental health research initiatives; (3) the GCPN can serve as a vehicle for training and professional development to enhance the capacity of clinicians to provide the best, evidence-based clinical care; and (4) the GCPN can help with the development, implementation, and assessment of innovative mental health services and strategies for promotion and prevention in mental health.

In view of available technology and the GCPN's wide-ranging platform and ability to communicate in many languages in real time, the GCPN represents an important part of WHO's effort to make isolated research silos a thing of the past and bring evidence-based mental health care to those who need it most, greatly shortening the gap between research and clinical implementation.

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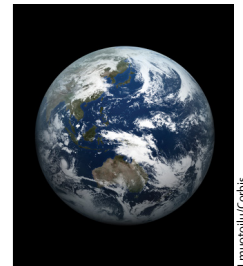
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Mental health and primary care professionals are invited to join the GCPN by registering at www.globalclinicalpractice.net

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Cannabis and psychosis

Marta Di Forti and colleagues[1] report that the use of herbal cannabis (skunk), but not resin (hash), is associated with increased risk of psychosis. They make the reasonable assumption that cannabis type is a proxy for strain potency and predominant cannabinoid present. Their findings are broadly consistent with recent experimental studies showing a psychomimetic effect of tetrahydrocannabinol (THC), and a potential antipsychotic effect of cannabidiol.[2]

At first glance, these findings suggest skunk use is hazardous to mental health whereas hash use is relatively safe in this context. This finding would have great implications for public health. However, it is important to consider whether this association is likely to be causal. Although there is largely consistent evidence that cannabis use is associated with psychotic symptoms, the strongest evidence for an effect on risk of clinical psychosis derives from populations in which most cannabis use would have been either resin cannabis or relatively low-potency herbal strains.[3] Moreover, findings from previous studies generally suggested a dose-response relation, with the strongest association being noted in the heaviest users, and little or no association noted in those who use infrequently.[3] Di Forti and colleagues[1] found this pattern in skunk users but not hash users. These earlier findings of a dose-dependent relation

have been used in support of claims that cannabis use is causally associated with psychosis risk.[4]

This discrepancy with previous studies suggests an alternative interpretation of the data. Specifically, it is possible that the association reported by Di Forti and colleagues[1] does not represent a biological effect, but rather some other predisposing risk factor for psychosis that could also lead people to select the most potent drug available to use. When associations depend on where an exposure lies in a distribution (rather than its absolute level), this pattern suggests confounding rather than a causal relation. For example, low concentrations of cholesterol predict negative non-vascular health outcomes in both European and east Asian populations, despite absolute cholesterol concentrations at the low end of the distribution in European populations being in the middle of the distribution in east Asian populations.[5] The similarity in the shape of risk curve between the two populations indicates that the observed association does not indicate a biological effect of low cholesterol on increased mortality, but rather that various confounding factors (including illness-induced reductions in cholesterol concentrations) give rise to the association.

We took the figures from the study by Di Forti and colleagues and combined the hash and skunk groups. The resulting effect size (OR 1·92, 95% CI 1·35–2·73) is similar to that reported in the meta-analysis by Moore and colleagues[3] (ever use 1·41, 1·20–1·65, heavy use OR 2·09, 1·54–2·84). In other words, the basic association between cannabis use and risk of psychosis was noted in populations where skunk use was uncommon. When skunk became available, a subgroup already at high risk of psychosis might have selected to use this form of cannabis. Therefore, although it is certainly plausible that use of skunk could be causally associated with psychosis, it is important to consider alternative explanations for the associations

noted. Moreover, any message that hash is comparatively safer than other forms of cannabis might have negative public health consequences, given its potential effect on respiratory health.[6]

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Marta Di Forti and colleagues' retrospective case–control study[1] distinguished between self-reported use of high-potency (skunk) versus lower potency (hash) cannabis. Scaremongering headlines in the media predictably followed. The *Daily Mail* screamed "Scientists show cannabis TRIPLES psychosis risk: Groundbreaking research blames 'skunk' for 1 in 4 of all new serious mental disorders." Undoubtedly, politicians and policy makers who have already made up their minds about regulation of cannabis will seize on the study as support for their views.

The authors must share some of the blame for misinterpretations of their results. They were pre-committed to causal language and failed to acknowledge important limitations