Correspondence on Curran et al. (2018): ‘Which biological and self-report measures of cannabis use predict cannabis dependency and acute psychotic-like effects’

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To the Editor:

The interactions between cannabis use, use disorder and psychosis is an important topic that is worthy of systematic investigation. Therefore, we read with great interest our colleagues’ article ‘Which biological and self-report measures of cannabis use predict cannabis dependency and acute psychotic-like effects’, recently published in Psychological Medicine (Curran et al., 2018). Within the framework of this otherwise well-conducted study, we respectfully suggest that accounting for the participants’ predisposition to psychosis in the study methodology would have strengthened the findings.

The investigators employed a series of clinical and biological measures in the context of participants’ (verified) naturalistic use and nonuse of cannabis, and found that: (1) cannabis intoxication increased the level of psychotic-like symptoms overall, and (2) evidence of greater recent delta-9-tetrahydrocannabinol (Δ9-THC) exposure was associated with decreased psychotic-like effects of, but greater dependency, on cannabis. Essentially, while cannabis may have acute psychotic-like effects, more experience with cannabis is associated with lower psychotic-like effects but a greater likelihood of cannabis dependence. While this is a reasonable and well-supported conclusion, and the study had many methodological strengths, the investigators neglected to address a potential major factor in the acute psychotic-like effects of cannabis – namely, the predisposition to psychosis.

A substantial literature, employing a variety of methodologies and populations, has examined the psychotic-like sequelae of cannabis in the broad psychosis spectrum. Naturalistic/clinical studies have demonstrated that cannabis use is concurrently and prospectively associated with increases in psychotic-like experiences/symptoms in such individuals (e.g. Verdoux et al., 2003; Henquet et al., 2004; Hides et al., 2006; Corcoran et al., 2008; Henquet et al., 2010). Further, placebo-controlled drug administration studies have shown, compared to healthy cannabis users, evidence consistent with a stronger psychotic-like effect of (or psychosis-associated neurochemical response to) active cannabis/Δ9-THC in individuals at genetic (Henquet et al., 2006), familial (Kuepper et al., 2013), clinical (Vadhan et al., 2017), or realized (D’Souza et al., 2005; Henquet et al., 2006; Kuepper et al., 2013) risk for a psychotic disorder. Finally, naturalistic studies have converged on these findings of differential effects (Henquet et al., 2010; Spriggens and Hides, 2015) and prospective cohort observation studies have provided further clinical significance (McHugh et al., 2017).

Although it could be argued that such populations were outside the scope of the Curran et al. (2018) study, individuals who have psychotic-like experiences are overrepresented in the cannabis-using population (van Os et al., 2008), and about 20% of individuals from our extensive database of nontreatment-seeking cannabis users endorsed having CHR-level psychotic-like experiences (unpublished data) on a screening questionnaire (Miller et al., 2004). This suggests the presence of a subpopulation of, at minimum, psychosis-prone individuals within the larger population of nontreatment-seeking cannabis users. If it is possible for the investigators to identify these individuals within their study (perhaps by the baseline Brief Psychiatric Rating Scale measures), a series of interesting analyses could be accomplished. For example, the notion that cannabis’ psychotic-like effects are stronger in psychosis-predisposed individuals could be tested, and the similarities and differences in the interrelationships between previous cannabis exposure, dependency and psychotic-like effects, between these cannabis user subtypes could be assessed.

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References


