Considerable attention has been paid to the hypothesized increase in incidence of psychotic disorders as a result of repeated (adolescent) cannabis use [1,2]. The study by Degenhardt et al. [3] is an excellent example of a prospective longitudinal cohort study, with first assessments in mid-adolescence and follow-ups until age 29 years, that can help to answer the question of whether continued cannabis use in adolescence into adulthood is also associated with some other specific expressions of mental malheur: major depression and/or anxiety disorders in adulthood. Like psychotic disorders, depressive and anxiety disorders can severely hamper adult functioning in multiple ways. In fact, one might consider this particular age, around 30 years, as an important turning point for many individuals, as first career steps are taken, family life is formed and responsibilities as a professional, a partner and, perhaps, as a parent increase. Thus, any mental dysfunction of young adults that could be prevented—already in adolescence—is important, not least for society.

The study by Degenhardt et al. [3], as do many good epidemiological studies, gives us an idea of the strength of the temporal relationship between adolescent cannabis use that is continued into adulthood and adult major depressive episode (MDE) or anxiety disorders (AD). They show an approximately twofold increased risk of AD at age 29 for those who were, at that time, also daily cannabis users. This effect was stronger for individuals who had continued their (at least weekly) cannabis use from adolescence into adulthood. Unfortunately, they did not inform us which anxiety disorders were mainly observed. Surprisingly, no consistent associations were found between (continued) adolescent cannabis use and adult MDE, while we know from the clinical literature that depression and anxiety are comorbid disorders [4]. Others have reported such an effect [5]. Moreover, a recent study suggested that anxiety disorders could precede cannabis abuse in adolescents [6], rather than the other way around. Although Degenhardt et al. [3] controlled for adolescent levels of anxiety, they used a somewhat rough mixed score of combined anxiety and depression. None the less, some anxiety disorders originate typically in early adulthood, and these forms may have occurred more frequently in the persistent cannabis users they described.

The clear downside of such an, albeit impressive, epidemiological study is that it simply describes a relationship, rather than examining its nature or its potential underlying processes. With the data at hand, we can dismiss a few possible explanations for this specific relationship. First, the authors alluded to a ‘social disadvantage’ theory in which cannabis (ab)users have an increased risk for adversities in their social life (e.g. divorce, parental psychiatric illness) which could affect their mental health. If this were true, persistent cannabis use would also increase the odds of MDE by age 30 (e.g. [7]). Secondly, they referred to a lower hypothalamic–pituitary–adrenal (HPA) axis activity that may have played a role. However, an under-aroused HPA axis has been related to risky forms of adolescent cannabis use [8,9], but not to anxiety.

What could help us to understand this finding? In an interesting review, Crippa et al. [10] describes several potential mechanisms by which repeated cannabis use could lead specifically to AD. For example, they suggested that symptoms of cannabis withdrawal include anxiety and, to find relief, cannabis use continues. At the time of their publication, there was still dispute about the actual existence of cannabis withdrawal. Consensus has now been reached, and withdrawal symptoms are part of the proposed DSM-5 criteria for a cannabis use disorder (CUD: http://www.dsm5.org). From this perspective, anxiety symptoms may be part of CUD, embedded in the circular pattern of dependent behaviour, and may therefore be cannabis-induced.

Alternatively, the repeated intake of cannabis may act on the main stress systems, as does nicotine, causing a chronic activation and hypervigilant state [11]. This may help to explain the differential finding on AD (and not on MDE). To differentiate between anxiety and depression, Clark & Watson [12] proposed a tripartite model, redefined slightly by others (e.g. [13]), in which the anhedonic state and unawareness of emotions is typical for depression, while hyperarousal and increased awareness of physiological responses (e.g. increased heart rate) is specific for anxiety. These feelings of arousal in already anxious people may, paradoxically, further enhance cannabis use, as users’ motives commonly include relaxation and tension relief [14]. Interestingly, some preliminary studies have pointed to enhanced stress sensitivity in the adolescent period [15,16] and describe adolescence as an age period in which the stress systems mature further. If repeated cannabis exposure should take place during such a window of stress vulnerability, it may increase further the risk of developing AD.
Some refined modelling of joint developmental trajectories of anxiety and cannabis use, with stress reactivity as predictor, may be helpful to gain an understanding of this potential underlying process. Further, high-risk samples and experimental designs may be needed to test the repeated cannabis use effects on arousal and, conversely, arousal effects on craving and continued cannabis use, in developmentally important periods. We need to understand more of the pathways towards mental health problems—including anxiety—after continued cannabis use, as they can guide us to intervention strategies at early stages. This might prove to be an (cost-) effective approach in many ways and could support healthy adult development.

Declaration of interests

None.

Keywords Adolescence, anxiety, cannabis, hyperarousal.

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