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Author for correspondence:
L. De Peri, E-mail: luca.deperi@ti.ch

Commentary on: it might be a wonderful opportunity when patients with a psychotic disorder use cannabis

L. De Peri[1] and A. Vita[2]

[1]Cantonal Psychiatric Clinic, Cantonal Sociopsychiatric Association, Mendrisio, Switzerland and [2]Department of Mental Health, University of Brescia School of Medicine, Spedali Civili Hospital, Brescia, Italy

To the Editor

We read with great interest the editorial by Lieuwe de Haan recently published in your Journal (de Haan, 2022). In the first part of the paper, the Author reviewed the current evidence on the unfavorable impact of cannabis use on the multidimensional outcome of psychotic disorders. Moreover, the editorial put strong emphasis on cannabis as a modifiable risk factor for an unfavorable course of psychosis concluding that interventions to stop or reduce cannabis use are critical. Based on these considerations, the key question posed by de Hann to the readers is why do interventions aimed to reduce or stop cannabis use in psychotic patients fail to make a difference? As suggested by the Author, to date, no answer can be regarded as conclusive.

In our opinion, from a speculative perspective, it could be very interesting to stress the hypothesis that psychotic patients with comorbid cannabis use may be distinguished from those without cannabis use based upon their neurobiological profile. Notably, a limited number of hypothesis-driven studies performed a direct comparison between psychotic patients with and without cannabis use as for biological variables. Genetic, brain structural and functional parameters and also neurocognition have been investigated to some extent, in psychotic patients with and without cannabis use but literature findings on the issue are not univocal. For instance, in a paper recently published by our group, we aimed to determine through a meta-analytic approach whether patients at the onset of schizophrenia with comorbid cannabis use showed a different pattern of brain abnormalities as compared to patients with no comorbid cannabis use. Our results demonstrated that, essentially, there was an overlap of brain abnormalities between the two groups (De Peri, Traber, Bolla, & Vita, 2021) when compared to healthy controls. At variant with neuroanatomical findings, another line of research gave some evidence for better cognitive functioning in first-episode schizophrenic patients with a history of cannabis use with respect to patients without a history of cannabis use (Yücel et al., 2012). Thus, in this case, neurocognitive functioning might be considered a specific target to characterize psychotic patients with cannabis use and build up specific intervention. Finally, genetic association studies between schizophrenia and cannabis use showed that common genetic variants underlying schizophrenia and lifetime cannabis use are partly overlapping evidencing a shared genetic liability (Verweij et al., 2017). Thus, the current global picture deriving both from original studies and quantitative reviews of neurobiological underpinnings in individuals with psychotic disorders and comorbid cannabis use seems to be somewhat unclear and often contradictory.

We agree with the Author that we can not afford the luxury of waiting with starting multidisciplinary interventions to stop or reduce cannabis use in psychotic disorders until all the proof is provided. Nonetheless, we think that future controlled studies aimed to disentangle the neurobiological mechanisms underpinnings psychotic disorders with comorbid cannabis use are warranted right now in order to offer psychotic patients tailored therapies and interventions as soon as possible.

Conflict of interest. None.

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