Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

Contents

- Letter to the editor about ‘Adolescent cannabis use, baseline prodromal symptoms and the risk of psychosis’
- Author’s reply

Letter to the editor about ‘Adolescent cannabis use, baseline prodromal symptoms and the risk of psychosis’

We read, with great interest, the article by Antti Mustonen et al entitled ‘Adolescent cannabis use, baseline prodromal symptoms and the risk of psychosis’, it is a tremendous study and is well conceptualised.1 We would like to make certain comments.

In the Method section, under the subheading of Psychosis diagnoses, ICD diagnoses have been mentioned as (F22-F29, F302, F312… whereas the fourth character in ICD-10 codes is always a ‘dot’.2 Second, the codes F302 and F312 are also mentioned under the category of psychosis. If these codes refer to F30.2 and F31.2, respectively then these fall in the category of affective disorder without psychosis. These errors in reporting make the article quite difficult to follow.

There is also a mismatch between the figures and the text in the article. In the Results section, Figure 2, explaining the cumulative incidences of psychosis in four groups with and without cannabis use and prodromal symptoms in the Northern Finland Cohort 1986 is not self-explanatory as the figures (n = 13/134, 5/134…) has not been explained in the text and I found it difficult to understand the origin and meaning of these figures.

Similarly, while mentioning the association between adolescent cannabis use and subsequent psychosis (on page 230 in the first paragraph of the Associations between adolescent cannabis use and subsequent psychosis subsection) the authors state that 18 out of 375 (4.8%) cannabis users received a diagnosis of psychosis during the 15-year follow-up (4 narrow-defined schizophrenia, 4 schizophrenia spectrum disorder, 0 bipolar disorder with psychotic features, 7 major depression with psychotic features, 3 other psychosis). As 7 out of 18 participants had major depression with psychotic features, which is a mood disorder, inclusion of it in the criteria would lead to inaccurate results. If these 7 participants are excluded, there would be 11 participants who had psychosis. There should have been more participants for a better exploration of the hypothesis formulated for the study. It would be of great help if the authors could clarify these points. Thank you.

Author’s reply

In our recent paper on adolescent cannabis use and risk of psychosis we found out that heavy cannabis use was associated with increased risk of any psychotic disorder or condition during 15 years of follow-up. Colleagues Savya Saluja and Hitesh Khurana made some important comments on this paper and we will try our best to clarify the concerns they raise.

The major argument in their letter was that affective psychoses should not be included with other psychoses, and they raise concern that this would not provide accurate results in general. In our study the objective was to study the associations between cannabis use and any psychotic condition and not to focus only on non-affective psychoses. We disagree that F30.2 (mania with psychotic features), F31.2 (bipolar disorder with psychotic features) and F32.3 (major depressive episode with psychotic features) should be considered solely as mood disorders; indeed ICD-10 also categorises them as severe psychotic states.3 The risk factors for psychotic disorders are likely to be pluripotent and further increase the risk across all psychotic disorders.2 Furthermore, the broad definition of psychosis has also been used in other studies as a primary or secondary outcome.3-5 It should also be noted that many of the previous studies on adolescent cannabis use and psychosis outcomes have focused on psychotic symptoms instead of clinical diagnoses, which is the major strength in our article.

We reported in the paper also that when the psychosis diagnoses were analysed separately, both schizophrenia spectrum disorder (HR = 11.18, 95% CI 3.16–39.62) and psychotic depression (HR = 9.74, 95% CI 3.83–24.73) are associated with cannabis use, whereas similar associations were not found for schizophrenia, bipolar disorder with psychotic episodes and other psychosis. We agree that the number of individuals with psychosis was small in the subgroup analyses, as we noted in the limitations of the study. Whether substance use contributes to an increased risk across different psychosis categories differentially is a matter requiring replication, and we think that in future it would be worthwhile studying affective psychoses under their own category if sample size permits.

There were also some minor comments. In the Method section they pointed out correctly that ‘dots’ in ICD-10 psychosis diagnosis codes are missing, which is unfortunately true, and should be there, for example F30.2 instead of F302. They also point out some discrepancies between the figures and the main text and raise concern that the figures are not self-explanatory. The key point in these figures is that participants with both prodromal symptoms (cut-off ≥3 items in PROD-screen) and cannabis use are more likely to develop psychosis during the 15-year follow-up than participants with just prodromal symptoms or cannabis use alone, suggesting that cannabis use might be more harmful for those with preceding psychotic experiences. We hope that this answers the questions.


Navya Saluja, Junior Resident, Department of Psychiatry, Pt. B.D. Sharma PGIMS Rohtak, India; Hitesh Khurana, Senior Professor, Department of Psychiatry, Pt. B.D. Sharma PGIMS Rohtak, India. Email: navyasaluja@gmail.com

doi:10.1192/bjp.2020.27

Antti Mustonen, MD, PhD, Center for Life Course Health Research, University of Oulu; and Medical Research Center Oulu, University of Oulu Hospital, Finland; Solja Niemelä, MD, PhD, Department of Psychiatry, Research Unit of Clinical Neuroscience, University of Oulu; and Department of Psychiatry, Lapidus Hospital District, Finland; Tanja Nordström, PhD, Center for Life Course Health Research, University of Oulu; and Medical Research Center Oulu, Oulu University Hospital, Finland; Graham K. Murray, MRCPSych, Department of Psychiatry, University of Cambridge, UK; Erika Jääskeläinen, MD, PhD, Center for Life Course Health Research, University of Oulu; and Medical Research Center Oulu, Oulu University Hospital Finland; Jouko Miettunen, PhD, Center for Life Course Health Research, University of Oulu; and Medical Research Center Oulu, Oulu University Hospital Finland. Email: antti.mustonen@oulu.fi

doi:10.1192/bjp.2020.28