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Re: Caution urged in interpreting a negative study of cannabis use and schizophrenia: A response to Dr. Christine Miller

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On behalf of my co-authors, I thank Dr. Miller for her thoughtful comments about our study (Proal et al., 2014). However, her title is quite misleading in that it implies that our study was “negative” and that we were simply reporting that cannabis does not “cause” schizophrenia. This is not so; unfortunately one sentence in the conclusion was taken out of context and as such was misleading. Our study was a family study of people who did and did not abuse cannabis as adolescents. There were 4 groups: non-psychotic individuals who did and did not abuse cannabis and people with schizophrenia who did and did not abuse cannabis prior to their illness. What the main study analysis showed was that those individuals who developed schizophrenia after cannabis use in adolescents had a significantly greater family history of schizophrenia than those who used cannabis and did not develop schizophrenia and an elevated family history no different than those with schizophrenia who never used cannabis. These results suggest that the key element for determining whether someone develops schizophrenia after cannabis use is an elevated genetic risk.

We did not test in this study design whether cannabis use without a family history of psychosis could cause schizophrenia. In order to test that hypothesis one would need to perform a longitudinal study examining people with and without cannabis abuse who have no family history of schizophrenia and determine whether over time there would be a significant excess of schizophrenia in those that used cannabis compared with those who did not. The converse of this longitudinal design, which we proposed as the next step to our study, would be to examine people at high genetic risk for schizophrenia and compare those who did and did not abuse cannabis for significant differences in later development of schizophrenia. The latter would show an interaction of genes with environment to cause illness. The former might show that cannabis alone could “cause” schizophrenia. We believe the issue is not so much that the sample size was underpowered to detect differences related

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Conflict of interest

None

Contributors:

None

to cannabis use, but rather that longitudinal studies are the necessary way forward to understand the interaction of cannabis with genetic risk factors to lead to schizophrenia.

Population studies of cannabis use are complicated by a lack of ability to know the composition of the substance being purchased as a street drug by individuals in the study, and knowing that this composition varies considerably depending on where and when the cannabis is purchased. Finally, the word “cause” is not a scientific term, and can be interpreted in many ways. “Googling” the word, leads to the following definition “something...that produces an effect, result, or condition: something...that makes something happen or exist”. Can cannabis alone “cause” schizophrenia. We suggest by our study that it cannot, but rather that elevated genetic risk factors are the key variable. However, large longitudinal studies are needed as stated above to further investigate this important issue. Meanwhile, we are in agreement with Dr. Miller, that cannabis is not a benign substance and our study should in no way be misinterpreted to imply that it is safe to use cannabis and one will not develop adverse effects.

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References

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