

COMORBIDITY BETWEEN CANNABIS USE AND PSYCHOSIS: MODELLING SOME POSSIBLE RELATIONSHIPS

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EXECUTIVE SUMMARY

One issue that has raised particular concern over the past few decades is the possibility that cannabis use may be a cause in some sense of psychotic disorders such as schizophrenia. This concern has been raised by clinical research showing high proportions of persons with schizophrenia reporting regular cannabis use and meeting criteria for cannabis use disorders. There has been considerable debate about the reasons for this association. The aim of the present report was therefore to use mathematical modelling to predict what the impact would be of some of the possible relationships on the incidence and prevalence of psychosis, given trends in the prevalence of cannabis use that have occurred over the past several decades.

1. That cannabis use causes psychosis. In this case, cannabis use precipitates schizophrenia in persons who would not otherwise have developed the illness;
2. That cannabis use precipitates schizophrenia only among persons who were vulnerable to developing schizophrenia (e.g. through family history of the illness);
3. That cannabis use by persons with schizophrenia worsens symptoms or prolongs the illness, so that remission of psychotic symptoms is less likely;
4. That those with schizophrenia are more liable to develop heavy or problematic use of cannabis if they begin using it.

Specifically, the study modelled the prevalence of schizophrenia over the lifespan; modelled the prevalence of cannabis use over the life course in eight birth cohorts: 1940-1944, 1945-1949, 1950-1954, 1955-1959, 1960-1964, 1965-1969, 1970-1974, 1975-1979. It also examined trends in the number of cases of schizophrenia that would be observed in these cohorts, given four different hypotheses about the relationship between cannabis use and schizophrenia, namely, (a) that it is causal, (b) that it precipitates the disorder among vulnerable persons, (c) that it exacerbates the disorder, and (d) that persons with schizophrenia are more liable to develop regular cannabis use. These predictions were compared with the published literature on the incidence and prevalence of schizophrenia over the past few decades to evaluate the plausibility of each of the hypotheses.

The prediction of the hypothesis that cannabis causes psychosis was not supported by the data on trends in the incidence of psychosis. There was no evidence that there has been an *increase* in the incidence of psychosis over the past thirty years of the magnitude predicted by the hypothesis. This suggests that cannabis use does *not* cause cases of psychosis that *would not otherwise have occurred*.

All other hypotheses provided a better fit to the available data. However, it is difficult to judge which of these is the best fit, or whether a combination of them is most appropriate. If cannabis use acts as a precipitant of psychosis, we would have seen *small* increases in the number of early onset cases. If cannabis use made relapse to psychotic symptoms, we would have seen *small* increases in the number of chronic cases. Finally, if

persons with psychosis were simply more likely to become regular cannabis users, we would expect to see no differences in the number incident or chronic cases, but a higher prevalence of regular use in this population. Future research needs to examine these possibilities.

This approach has suggested that cannabis use is probably not causally related to psychosis in the strong sense of causing cases that would not otherwise have occurred. Nonetheless, cannabis use may affect persons who are vulnerable to developing psychosis, or who have already developed the disorder. Such persons may be advised of this possible relationship and counselled against using cannabis.

A similar approach to modelling may be useful in empirically assessing the plausibility of different relationships between risk factors and the incidence and prevalence of other mental disorders in the population.