

Cannabis and risk of psychosis

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Summary

Legalisation of cannabis use in Switzerland has recently been debated by the Swiss Parliament. Although legalisation has not yet been decided upon, it is still the subject of impassioned public discussion. If cannabis use is legalised, an increase in consumption is to be expected. One of the manifold negative consequences for mental health will probably be an increase in the prevalence of psychoses – not only acute, toxic psychosis but also

chronic psychoses. Schizophrenic psychoses are expected to be triggered at an earlier age and to be negatively influenced in their course. This eventuality could have deleterious consequences not only for many currently healthy individuals predisposed to psychosis, but also for the disability pension.

Key words: cannabis; psychosis; schizophrenia

Introduction

In Switzerland cannabis is a widely used drug due to its psychotropic effects. It enjoys an almost legally accepted status. The National Health Inquiry (Schweizerische Gesundheitsbefragung 2002) showed that the recreational use of cannabinoids increased significantly during the period 1992–2002. In 2002, 225'000 persons in the 15–64 age group consumed cannabinoids, corresponding to 4.7% of the Swiss population. The proportion of consumers increased in the under-40 age group from 12.2% in 1992 to 21.7% in 2002. In 2002 between 36% and 24.4% of men and women respectively of the 15–24 age group reported that they had consumed cannabis at least once [1]. It may be speculated that the prevalence of cannabis consumption would increase further if consumption were legalised in Switzerland, as the Swiss Parliament has debated. Hence the implications for mental health are of importance.

Besides its disturbing effects on psychomotor performance and driving ability [2], development of psychological and physical dependence on cannabis and other drugs [3–5], impairment of cognitive function (memory, attention) [6], changes in personality such as loss of motivation, as described by the term “amotivational syndrome” [7], as well as development of depression and psychosis [5], are reported in the literature. With respect to psychosis, a distinction must be drawn between dose-dependent toxic, so-called drug-induced psychoses and schizophrenic psychoses. The existence of the usually reversible toxic psychoses has been well described [5, 8], but there is still controversy as to whether acute or

chronic cannabis consumption can lead to the development of chronic, especially schizophrenic psychoses.

In the Mannheim ABC Schizophrenia Study the last author of this paper demonstrated that first-admitted patients with schizophrenia showed a twofold higher prevalence of drug abuse (predominantly cannabis) compared with healthy controls [9, 10]. Experience in our Basel FEPSY – (Früherkennung von Psychosen – Early Detection of Psychosis) project show a similar disproportionate use of cannabis: approximately 75% of our newly diagnosed patients with schizophrenia reported regular use of cannabis (at least several times per month). We shall therefore discuss the evidence in the literature for a causal relationship between the use of cannabis and the development of (chronic) schizophrenic psychoses.

We are aware that the kind of relationship between cannabis consumption and development of schizophrenic psychoses is highly controversial [11]: one view is that cannabis use is secondary to psychiatric disorders, or caused by other, concomitantly consumed drugs, or is even due to confounding factors (both cannabis use and psychosis are caused by one or more as yet unknown factors). A different view is that of a causal or modulating effect (in vulnerable patients) of cannabis use for the development of psychoses. And an integrative view, supported by our own earlier data [9], postulates that both kinds of interaction are possible.

In what follows, our criteria for deciding between these views are similar to other authors' [11, 12]:

1) Is there biological evidence of an interaction between the cannabis and the dopaminergic system in cerebral tissue?

2) Is there a statistical association between cannabis use and psychosis and, more specifically,

3) Is there a temporal relationship between antecedent cannabis use and later onset of psychotic symptoms, and, finally

4) Is there a dose-response relationship for cannabis use and development of schizophrenic psychoses?

Neurobiological background

The importance of dopamine balance in specific brain areas for the development of psychoses has been sufficiently demonstrated, being the basis of the dopamine-antagonistic therapy with neuroleptic drugs. Cannabis may affect this balance by its active psychotropic components, such as tetrahydrocannabinol (THC) and its metabolite 11-OH-THC. It increases dopaminergic activity in relevant areas of the mesolimbic system, possibly by blockade of GABAergic neuronal activity [4, 13]. Cannabis exerts these effects by binding to specific receptors (mainly the CB1-receptor), which interact with local dopamine D₂-receptors [14]. Both receptor genes show a significant homology in regulatory parts [15]. Interactions

between the two receptors were reported for the striatum of rats and monkeys [16]. The most extensive expression of CB1-receptors is found in those brain areas which are also believed to be important for the development of schizophrenia, namely the mesolimbic and mesocortical dopaminergic system [4]. Further, it has been proposed that genetic mutations of the CB1-receptor gene are accompanied by an increased risk of developing cannabis abuse in schizophrenic patients [17]. The finding of increased blood concentrations of anandamide, an endogenous CB1-receptor agonist, in a group of schizophrenic patients [18] corroborates these findings.

Clinical studies

Can cannabis consumption lead to short-lasting, “toxic” psychoses?

In the literature there are many case reports on acute psychiatric symptoms after the consumption of sometimes high doses of cannabis, showing the clinical picture of short, reversible toxic psychosis with organic features such as confusion or disorientation.

In India, 200 patients were hospitalised after consumption of exceptionally high doses of cannabis because of severe psychiatric symptoms (confusion, emotional lability, disorientation, depersonalisation, paranoid symptoms) [19]. The symptoms lasted for some days, but longer in patients with a history of psychiatric disorders.

Can cannabis consumption also lead to non-toxic psychoses/psychotic symptoms?

In a large-scale enquiry in 1000 persons in New Zealand aged between 18 and 35, 38% reported cannabis consumption. 22% of these subjects reported anxiety and panic attacks and 15% psychotic symptoms following cannabis use. Women reported panic attacks significantly more often than men [20]. In Germany, 36,000 US soldiers were questioned about cannabis consumption. 5120 admitted consuming cannabis at least 3 times weekly. Psychiatric symptoms such as panic attacks or toxic psychoses following consumption of a single high dose of cannabis were reported by 720 soldiers [21].

Van Os and co-workers investigated the effect of cannabis in 4045 healthy subjects and 59 psychotic patients [22]. In a 3-year follow-up investigation the consumers showed a 2.8-fold higher risk (95% CI: 1.2–6.5) of developing psychotic symptoms than non-consuming controls. In the group of patients with pre-existing psychotic symptoms, the risk was 24.2-fold (95% CI: 5.4–107.5) It emerged that the severity of psychotic symptoms correlated with the extent of cannabis consumption. In this study more than 50% of psychotic episodes could be related to consumption of cannabis. The authors concluded that cannabis consumption increases the risk of developing psychotic symptoms in healthy subjects and worsens the prognosis in psychotic patients.

The impact of cannabis consumption on the risk of developing psychoses has also been investigated in other studies. Thus, in the National Survey of Mental Health and Well-Being (NS-MHWP) [23], a representative poll (N = 10641) showed that cannabis abuse was accompanied by a significant 2.8-fold (95% CI: 1.4–5.9) increased risk of developing psychosis.

However, these studies did not further investigate the type of psychosis.

Can cannabis consumption cause a schizotypal personality?

Cannabis consumption is associated with a schizophrenia-like personality. Several authors show that young consumers and previous con-

sumers have higher scores on schizotypy, borderline and psychoticism scales even when they do not show other psychiatric symptoms. They also show deficits in attentional inhibition and decreased reaction time compared to never-users [24, 25]. The causal relationship of this association is, however, not yet clear: do these personality traits predispose to cannabis abuse or does cannabis abuse induce these changes of personality?

Can cannabis consumption also trigger or even cause *schizophrenic* psychoses?

A historical cohort study in more than 50,000 Swedish conscripts over an observation period of some 27 years investigated the importance of cannabis consumption for the development of schizophrenia and psychoses [26]: young men who reported previous cannabis consumption on more than 50 occasions at the outset of the observation period showed a 6.7-fold (95% CI: 4.5–10.0) increased risk of later hospitalisation for schizophrenia and other psychoses.

The latter author also reported very interesting results in the ABC study (Age, Beginning and Course of Schizophrenia): this study investigated 276 first-time hospitalised schizophrenic patients (232 with first episode) [27, 28]. Twice the number of patients reported a lifetime history of substance abuse than healthy controls – 14.2% versus 7% [9, 29]. Of these patients 88% consumed cannabis, in approx. 60% of whom cannabis consumption preceded even the first still very unspecific symptoms of schizophrenia [29], on average by about 4.5 years [9]. In 35% of cases the first unspecific symptoms were reported to have occurred in the same month as the start of drug consumption. Cannabis-consuming patients were significantly younger than non-consuming patients. The authors therefore concluded that cannabis consumption may have precipitated (triggered) the onset of schizophrenia in predisposed (vulnerable) patients and aggravated the symptoms [29, 30]. However, there may also be a subgroup of patients who have started to use cannabis to attenuate the first (pre-)psychotic symptoms as a form of self-therapy [9, 31, 32].

In a recent reanalysis of five large (N = 1011 to 50,053 patients) studies [22, 26, 33–35] Smit et al. pointed out that in these studies there was a clear temporal relationship of antecedent cannabis use before first psychiatric symptoms were observed [11]. Effects of other concomitantly used drugs were excluded and the effects of other confounders were taken into account in four of these studies. In two of them [22, 26] a dose-dependent increase in psychosis risk was observed. As the authors pointed out, this does not imply that psychotic patients “do not use cannabis as a form of ‘self-medication’, but these results do imply that cannabis use increases the risk of later schizophrenia even when self-medication can be ruled out as an explanation” [11]. Verdoux et al. [36] found that young undergraduate students with high vulnerability to develop-

ment of psychosis reported more unusual perceptions and feelings of thought influence after cannabis consumption than subjects with low vulnerability. Interestingly, they could not find evidence of increased use of cannabis following the occurrence of psychotic experiences. This contradicts the self-medication model in the psychotic stage but not necessarily in the still unspecific prodromal stage.

Young age appears to increase the risk of developing psychotic symptoms following cannabis use. In a prospective longitudinal study Arsenault et al. [34] investigated whether adolescent cannabis use is a risk factor for adult schizophreniform disorders. The study was performed in 1037 adolescents from New Zealand and showed that cannabis consumption at age 15 years significantly increased the risk of developing schizophrenia (4.5-fold, 95% CI: 1.1–18.2) up to age 26. However, cannabis consumption at age 18 increased the risk only 1.7-fold (95% CI: 0.7–4.2). These results were corroborated by a birth cohort in 1265 children [35] studied to age 21. The authors showed that subjects fulfilling DSM-IV criteria for cannabis dependence had elevated rates of psychotic symptoms as compared to individuals without cannabis dependence – at age 18 the increase was 3.7-fold (95% CI: 2.8–5.0) and at age 21 2.3-fold (95% CI: 1.7–3.2). This significant increase was still present when data were adjusted for pre-existing symptoms or other background factors.

Despite this close association of cannabis use with schizophrenia, the causal relationship between cannabis consumption and schizophrenic psychosis is still controversial. On the one hand, many studies show an increased risk of developing schizophrenia in patients consuming cannabis; on the other hand, in a simulation Degenhardt [37] showed that a causal relationship would have led to a significant increase in the incidence of schizophrenia in Australia, which has, however, not been observed [8].

A possible explanation for the lack of evidence of an increase in schizophrenia rates in epidemiological studies may be a reporting bias: in clinical studies, which usually find this association, exposed patients have been systematically studied for psychiatric disturbances in one or more follow-up investigations. This has led to detection of mild psychotic symptoms, which outside clinical studies may not have been detected (underreporting). Also, the proportion of cannabis-induced schizophrenia may be small and, therefore, an increase in schizophrenia rate attributed to cannabis use may not be easily detectable. On the other hand, Boydell et al. [38] recently reported a continuous and statistically significant increase in the incidence of schizophrenia from the London area for the period 1965–1997. Interestingly, this increase was most marked in people under 35 years of age and was not gender-specific.

Furthermore, it is conceivable that cannabis is not causal in a narrow sense, but merely triggers

the outbreak of schizophrenic psychoses in individuals with a specific (genetic) vulnerability for this disease. Cannabis use would then simply result in earlier manifestation of schizophrenia in these vulnerable patients rather than an increased incidence. Evidence for this has just been published [39]. This alone would also be relevant, since it has important implications for the mental and educational development of these patients. If the onset of first symptoms is earlier, these patients have not usually completed their professional education and have not developed a sufficient social network. Earlier onset due to a “cannabis trigger” may therefore be associated with a worse prognosis of schizophrenia, especially regarding its psychosocial course.

Can cannabis consumption worsen the progression of schizophrenia?

The productive symptoms of psychosis are amplified by concomitant consumption of cannabis. Compliance with antipsychotic treatment and utilisation of rehabilitation programmes are impaired [29]. Schizophrenia patients with cannabis use suffer from more frequent and earlier relapse episodes [40, 41]. Because of the dopaminergic effects of cannabis it can be speculated that cannabis-consuming patients suffering from schizophrenia may need higher doses of antipsychotic (anti-dopaminergic) medication. Psychotic patients with cannabis consumption are usually younger, predominantly male and show more criminal behaviour [42].

Conclusions

On the basis of our criteria, the following conclusions can be drawn:

- Cannabis consumption modulates dopamine concentrations in certain brain areas, and can thus induce or modulate the development of psychotic symptoms and psychosis.
- Cannabis in high doses may induce acute, reversible (toxic) psychoses.
- Cannabis may also induce the manifestation of schizophrenic psychoses in vulnerable patients (“dopaminergic stress”) or at least trigger an earlier onset in this population.
- Young age is an additional risk factor for the development of psychoses.
- Concomitant cannabis consumption may affect the progression of schizophrenic psychoses and worsen the prognosis.
- Cannabis consumption is associated with a schizotypal personality, the causal relationship of which is not yet clear.
- Cannabis consumption can also lead to other psychiatric disorders, including depression and may result in a severe loss of energy and cognitive disturbances.

To obtain more definite answers to the questions of causality, dose-effect relationship, the severity and time course of these effects, the importance of other confounding factors, and, finally, the size of the impending burden for the individual and the society, we urgently need additional prospective longitudinal studies.

Nevertheless, at this stage of research we can and should, depending on our view, already sound a note of warning, especially as the potency of the substances used and the prevalence of abuse and dependence are apparently growing [43]. Increased cannabis consumption in our society could have deleterious consequences for many so far healthy individuals, not only because of the negative influences on education and work performance due to impairment of cognitive function and

loss of energy, but also due to the “psychotogenic” properties of cannabis. Healthy individuals with a currently “hidden” predisposition to psychosis could develop full blown psychosis. In individuals vulnerable to schizophrenia the outbreak of this disorder could be triggered at an earlier age and negatively influenced in its course. This would not only have severe psychosocial consequences for the individual and his family. As schizophrenia is already one of the most expensive diseases, this could also have negative consequences for the national economy – *inter alia* due to the high level of disability pensions at a very young age.

What conclusions can be drawn? Politicians, health professionals and teachers should more intensively pinpoint the potential health risks of cannabis use, in particular for young adolescents. Information campaigns should be launched in the media. Although complete prohibition of cannabis may be neither enforceable nor successful in our society, legalisation of cannabis could – on the other hand – send the wrong message concerning the potential harm done by its use.

It goes without saying that people who are already dependent need our help and should not be criminalised; but methods of controlled use for dependent persons have also been found for other substances and drugs. Our main focus should in any case be on the prevention of new cases of cannabis dependence.

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