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Letter to the Editor

Should we advise patients with serious mental illness to stop using cannabis?

Advising patients with serious mental illness to stop their cannabis intake tends to be unpopular with patients, and also with some psychiatrists. Patients enjoy using cannabis, and can perceive it as being therapeutically beneficial. Thus, their reluctance to stop their cannabis intake is understandable. The argument of psychiatrists and other mental health-care professionals against giving such advice is that since the effect of cannabis on psychosis is nothing or negligible, psychiatry must not portray itself as a 'killjoy' profession, because this could lead to fewer mentally ill patients trusting psychiatrists to help them. On the other hand, Macleod (2007), in his excellent commentary on Degenhardt *et al*. (2007), considers that trying to prevent or reduce cannabis use amongst the mentally ill patients is justifiable, even when one considers its apparently small effect on psychosis. He supports this opinion by underlining that cannabis is not only deleterious on psychosis, but it is also a contributing factor to physical health problems. I agree with the reasons he gave but I also believe that this approach is totally justifiable also on the basis of the effect of cannabis on psychosis only. As clinicians, we should provide the best possible holistic treatment to our patients. Since schizophrenia is a 'dreadful illness that is substantially unexplained and ineffectively treated' (Macleod, 2007), it poses unique challenges. Thus, any measure that can contribute towards relieving suffering, however negligible is its effect, should be considered seriously. Also, one must not forget that such measures do not influence patients uniformly, and a negligible measure for one patient can be a significant one for another patient. How can we convince more psychiatrists and more mental health-care professionals to advise patients to stop their cannabis intake? An obvious need is to conduct research into the efficacy of different ways of persuading patients with serious mental illness to refrain from using cannabis.

Declaration of Interest

None.

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Letter to the Editor

Drawing conclusions about cannabis and psychosis

The renewed interest in the association between cannabis and mental illness is well reflected in three articles and a commentary in volume 37, number 7, of *Psychological Medicine*. In some ways it is surprising that there still seems so much diffidence regarding the drawing of definite conclusions from what is an increasingly converging literature. One reason for this may be the confusion regarding predisposing, precipitating, and perpetuating effects of delta-9-tetrahydrocannabinol (THC) on psychosis, as well as a reliance on positive symptoms of psychosis as the hallmark of schizophrenia. In sum:

(1) There is no doubt that THC can precipitate psychosis: indeed, it does so reliably in a dose–response way, and arguably anyone could manifest positive psychotic symptoms given a large enough dose (see Castle & Solowij, 2004).
(2) The individual liability to the manifestation of positive symptoms on exposure to THC depends upon individual 'psychosis proneness', as shown nicely in a non-clinical sample by Verdoux and colleagues (Verdoux, 2004). Clearly people with schizophrenia are very 'psychosis prone' and they would be expected to manifest positive symptoms at even low dose, similar to someone with diabetes eating sugar (see Linszen *et al*. 2004). The study of Degenhardt *et al*. (2007) that found only a modest

exacerbation of positive psychotic symptoms in their schizophrenia cohort may be a product, *inter alia*, of: selection bias; the fact that the cohort already had positive symptoms (mean BPRS score at baseline 43.2): this assertion is supported by the fact that prior-month BPRS score was a much stronger predictor of increased BPRS score at each assessment point; the fact that few were using high quantities of cannabis (only a fifth using more than 3 g a week); and that they were a treated sample, so most would have been receiving dopamine blocking medication.

(3) The symptoms that drive cannabis use in people with schizophrenia are very much the same as those that drive its use in people without schizophrenia: what we (Spencer *et al*. 2002) have called 'negative affect': so, the self-medication hypothesis is true, but self-medication is for negative rather than positive symptoms (see also Macleod, 2007).

(4) Some individuals have a predisposition to schizophrenia but do not quite manifest positive symptoms until they are exposed to a stressor such as THC. In this small group, THC is the 'straw that breaks the camel's back' and acts as a cumulative causal factor for schizophrenia (see Arseneault *et al*. 2004): using this model, very few 'cases' of schizophrenia (estimated population attributable fraction around 8%) would actually be prevented with the global abolition of cannabis.

So, the facts appear clear, and the message must be that anyone with high psychosis proneness should avoid cannabis: the tough part is helping people with negative affect (of which those with schizophrenia have a surfeit) to find alternative ways of ameliorating those symptoms.

Declaration of Interest

None.

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Letter to the Editor

The coherence of the evidence linking cannabis with psychosis

Similar to the link between smoking and lung cancer, it is the level of *coherence* between observational, biological and experimental studies on the link between cannabis and psychosis that will finally inform the community about the validity and causality of any association. Macleod (Macleod, 2007) cogently argues that observational studies in psychiatry may be rich sources of bias and confounding. Therefore, no matter how many studies are conducted, one can always think of more or less plausible sources of bias and confounding. Whilst this is true, it is also true that any discussion of observational evidence is incomplete and selective if other sources are ignored. Furthermore, a discussion of observational evidence is biased if it fails to take into account important findings. For example, while Macleod agrees that the acute effects of cannabis include psychotic symptoms, he does not discuss the Danish Psychiatric Central Register follow-up of such acute intoxications, showing that the great majority were later re-diagnosed with schizophrenia (Arendt *et al*. 2005).

Macleod is selective with regard to the scope of the evidence assessing links between cannabis and psychosis. He ignores randomized experimental studies and does not discuss studies showing that the effect