Psychological Medicine, **38** (2008)
doi:10.1017/S0033291707002267

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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Title:
Letter to the editor: Drawing conclusions about cannabis psychosis

Date:
2008

Citation:
Castle, D. (2008). Letter to the editor: Drawing conclusions about cannabis psychosis. Psychological Medicine, 38(3), 459-460.

Publication Status:
Published

Persistent Link:
http://hdl.handle.net/11343/33022