

Research report

Cannabis use and expression of mania in the general population

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Received 23 March 2006; received in revised form 3 May 2006; accepted 5 May 2006

Available online 21 June 2006

Abstract

Background: Cannabis use is common in patients with bipolar disorder, however little is known about cannabis as a risk factor for mania. In order to investigate the association between exposure to cannabis and subsequent development of manic symptoms whilst controlling for psychotic symptoms, a longitudinal population-based study was carried out.

Methods: 4815 individuals aged 18 to 64 years were interviewed using the Composite International Diagnostic Interview at baseline, 1 year follow up and 3 year follow up, including assessment of substance use, manic symptoms and psychotic symptoms.

Results: Use of cannabis at baseline increased the risk for manic symptoms during follow-up (adjusted OR 2.70, 95% CI: 1.54, 4.75), adjusted for age, sex, educational level, ethnicity, single marital status, neuroticism, use of other drugs, use of alcohol, depressive symptoms and manic symptoms at baseline. The association between cannabis use and mania was independent of the prevalence and the incidence of psychotic symptoms. There was no evidence for reverse causality, as manic symptoms at baseline did not predict the onset of cannabis use during follow-up (OR=0.35, 95% CI: 0.03, 3.49).

Limitations: As 3 years is a relative short period of follow-up, long-term effects of cannabis use on mania outcomes could not be detected.

Conclusion: The results suggest that cannabis use may affect population expression of manic symptoms (and subsequent risk to develop bipolar disorder [Regeer, E.J., Krabbendam, L., R, DE Graaf, Ten Have, M., Nolen, W.A., Van Os, J., 2006. A prospective study of the transition rates of subthreshold (hypo)mania and depression in the general population. *Psychol Med*, 1-9.]). These findings may not be due to the emergence of psychotic symptoms or the effects of self-medication.

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Keywords: Tetrahydrocannabinol; Bipolar disorder; Psychosis; General population; Cohort study

1. Introduction

Manic symptoms are common in patients diagnosed with schizophrenia and, conversely, psychotic symptoms often occur in those with bipolar disorder. The two comorbid but separable symptom dimensions of mania and psychosis (McGorry et al., 1998; Peralta and Cuesta, 1999) also display a degree of overlap in genetic and non-genetic aetiological influences (Murray et al., 2004; Walker et al.,

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2002). Increased risk for schizophrenia has been reported in relatives of patients with bipolar illness characterised by a high familial loading (Valles et al., 2000) and twin studies suggest overlap in the genes contributing to schizophrenia, schizo-affective and mania syndromes (Cardno et al., 2002). High rates of mental illness among minority groups are not specific to schizophrenia and have been described in mania as well (van Os et al., 1996). Furthermore, neuroticism has been associated with the development of both schizophrenia (van Os and Jones, 2001)/psychotic symptoms (Krabbendam et al., 2002) and bipolar disorder (Angst et al., 2003a,b). Other risk factors however, such as obstetric complications (Browne et al., 2000) and urbanicity (Mortensen et al., 2003) have been associated with schizophrenia/psychotic symptoms, but not with bipolar disorder.

Although evidence is accumulating that cannabis is a risk factor for schizophrenia/psychotic symptoms (Andreasson et al., 1987; van Os et al., 2002), little is known about cannabis as a shared risk factor for both mania and psychosis. In studies of psychotic outcomes, there is evidence that exposure to cannabis plays a role not only in the expression of psychotic disorder, but also in the emergence of psychotic experiences at lower levels of severity in non-clinical samples (Henquet et al., 2005; van Os et al., 2002; Verdoux et al., 2003). Results from population-based studies furthermore suggest that cannabis use interacts synergistically with pre-existing liability to psychosis, indicating that the risk-enhancing effect of cannabis is much stronger in individuals with prior evidence of psychosis diathesis (Caspi et al., 2005; Henquet et al., 2005; van Os et al., 2002). Patients with bipolar disorder have elevated levels of substance use (Regier et al., 1990; Strakowski and DelBello, 2000), including cannabis (Sherwood Brown et al., 2001). There is also evidence that substance use in these patients is associated with poor treatment response and poorer clinical outcome (Sonne et al., 1994; Tohen et al., 1990). There are no data shedding light on whether associations between cannabis use and mania may be causal (Strakowski and DelBello, 2000). Clinical data, however, suggest that in many patients the use of substances precedes the onset of bipolar disorder (Strakowski et al., 1998). Several case studies, on the other hand, report that patients may start using cannabis to moderate their manic symptoms (Grinspoon and Bakalar, 1998; Khantzian, 1997; Strakowski and DelBello, 2000). Prospective studies, however, have not provided evidence to support the self-medication hypothesis, as patients with prior histories of substance use often did not resume their substance use after onset of the disease (Strakowski and DelBello, 2000; Strakowski et al., 1998). Only few population-based

studies have actually investigated the temporal sequence of substance use and bipolar disorder (Escamilla et al., 2002). To our knowledge, no prospective study to date has investigated the hypothesis of cannabis as a risk factor for mania outcomes, disentangling co-morbidity with psychotic symptoms, potential confounding variables (such as use of other drugs), and reverse causality (i.e. the self-medication hypothesis).

The aims of the current study, therefore, were to investigate prospectively (i) if baseline cannabis use increases the risk for development of manic symptoms, (ii) if the association between cannabis and mania is independent of the emergence of psychotic symptoms, and (iii) if baseline mania predicts cannabis use at follow-up (the self-medication hypothesis).

2. Methods

2.1. Sample

The Netherlands Mental Health Survey and Incidence Study (NEMESIS), is a prospective study with three measurement points over a period of 3 years (Bijl et al., 1998a,b). A multistage, stratified, random sampling procedure was used to first select 90 municipalities, then a sample of private households, and finally a Dutch-speaking individual aged 18–64 years within each household. Selected households were sent an introductory letter by the Minister of Health, inviting them to participate. A total of 7076 individuals provided informed consent and was interviewed at baseline, representing a response rate of 69.7%. At T₁, 5618 subjects participated at the first follow-up and at T₂, 4848 subjects participated at the second follow-up; 4815 individuals had completed the mania section of the CIDI at both follow ups. The sample was found to be representative of the Dutch population in terms of gender, marital status and level of urbanisation (Bijl et al., 1998b), with the exception of a slight underrepresentation of individuals in the age group 18–24 years.

2.2. Measures

Subjects were interviewed at home using the Composite International Diagnostic Interview (CIDI) version 1.1 (Smeets and Dingemans, 1993). The CIDI was designed for trained interviewers who are not clinicians and has been found to have high inter-rater reliability (Cottler et al., 1991; Wittchen et al., 1991) and high test–retest reliability (Wittchen, 1994). Ninety interviewers experienced in systematic data collection collected the data, having received a 3-day training course in recruiting and interviewing, followed by a 4-day course at the

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