

Prospective associations between cannabis use, abuse, and dependence and panic attacks and disorder

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Abstract

The present study prospectively evaluated cannabis use, abuse, and dependence in relation to the development of panic attacks and panic disorder. Participants at the start of the study were adolescents ($n = 1709$) with a mean age of 16.6 years ($SD = 1.2$; time 1) and were re-assessed 1 year later (time 2) and then again as young adults (time 3; mean age = 24.2 years, $SD = 0.6$). Results indicated that cannabis use and dependence were significantly prospectively associated with an increased odds for the development of panic attacks and panic disorder. However, cannabis was not *incrementally* associated with the development of panic after controlling for daily cigarette smoking. The theoretical and clinical implications of these findings are discussed.

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1. Introduction

Cannabis is one of the most commonly used recreational drugs in the United States (US) and beyond (Office of Applied Studies (SAMHSA), 2001; Rey et al., 2002). For example, approximately 4% of adults have used cannabis in the past year and rates of cannabis abuse and dependence are on the rise (Compton et al., 2004). These data are noteworthy given empirical evidence that cannabis use, aside from being linked to negative physical health and social outcomes (Ameri, 1999; Cohen, 1981; Lehman and Simpson, 1992; Lynskey and Hall, 2000), may be related to certain psychological symptoms and conditions (Green and Ritter, 2000; Tunving, 1985).

Panic attacks have historically been linked to cannabis use (Gale and Guenther, 1971). This work was initially stimulated by the observation that cannabis use may acutely promote heightened levels of anxiety symptoms and elicit panic attacks under certain conditions or among certain individuals (Hall et al., 1994; Hollister, 1986; Szuster et al., 1988; Thomas, 1996; Tunving, 1985). Subsequent studies have strengthened confidence that (a) more frequent cannabis use and/or (b) more severe cannabis problems may be related to an increased risk of panic attacks. For example, Hathaway (2003) found that among weekly users of cannabis ($n = 104$), approximately 40% reported having had at least one panic attack related to such use. These prevalence rates are noteworthy in light of lifetime rates of panic attacks among the general population of approximately 5–8% (Katerndahl and Realini, 1993). Although methodological differences in the assessment of panic attacks may hinder comparisons across studies, the

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limited study suggests the possibility of a cannabis-panic linkage. Increasing confidence in the generalizability of these types of associations, a recent study involving a representative sample ($n = 4745$) found that a lifetime history of cannabis dependence, but not use or abuse, was related to an increased risk of panic attacks after covarying the effects of polysubstance use, alcohol abuse, and demographic variables (Zvolensky et al., 2006). This work also is supported by other investigations showing that daily or weekly users of cannabis report greater levels of symptoms of somatic tension and arousal such as feeling dizzy compared to nonusers (Bonn-Miller et al., 2005; Milich et al., 2000; Pickard et al., 2000; Thomas, 1996) and cognitive dyscontrol symptoms (e.g., depersonalization; Dannon et al., 2004; Mathew et al., 1993; Troisi et al., 1998; Zvolensky et al., 2006). The nature of the direction(s) of the putative cannabis-panic association is as of yet unknown from the extant empirical research.

Overall, a growing corpus of evidence collectively suggests that heavier patterns of use or more severe forms of cannabis use problems (e.g., dependence) are related to increased risk of panic attacks. Yet, existing studies on this topic are limited in a number of key respects. Perhaps most notably, the vast majority of investigations evaluating cannabis-panic associations utilized cross-sectional designs. These cross-sectional studies, by definition, cannot explicate temporal order of onset between cannabis and panic attacks, and by extension, it is not possible to differentiate possible risk factor effects from concomitants or consequences. Only prospective studies that test whether putative risk factors predict the subsequent onset of panic attacks can achieve this aim. Unfortunately, of the limited number of prospective tests involving evaluations of cannabis in terms of psychiatric conditions, panic attacks have not been assessed (Block et al., 1991; Fergusson et al., 1996). A second key limitation of past work is that none of the previous studies have tested whether the association between cannabis and panic is not simply attributable to cigarette smoking. This limitation is unfortunate, as there is a strong association between cannabis use and its disorders and cigarette smoking (Degenhardt et al., 2001, 2003). Moreover, daily cigarette smoking is related to an increased risk of panic attacks (Zvolensky and Bernstein, 2005; Zvolensky et al., 2005). A final limitation of past work is that the previously reported association between cannabis dependence and panic attacks may simply be accounted for by a comorbid pattern of more severe non-cannabis drug dependence (Ross et al., 1988). Specifically, because individuals with more severe cannabis use problems (i.e., abuse and dependence) are more likely to have problems with other substances as well as be more prone to psychological problems (Kessler et al., 1997; Regier et al., 1990), it is possible non-cannabis drug dependence could account for the documented association between cannabis dependence and panic attack.

The aim of the present study was to provide a prospective test evaluating cannabis use, abuse, and dependence in

relation to the development of panic attacks and panic disorder. Participants at the start of the study were adolescents ($n = 1709$) with a mean age of 16.6 years ($SD = 1.2$; time 1 [$T1$]) and were re-assessed 1 year later (time 2; [$T2$]) and then again as young adults (time 3 [$T3$], mean age = 24.2 years, $SD = 0.6$; see Method section for details). It was hypothesized that cannabis dependence, but not use or abuse, would prospectively predict an increased risk for panic attacks and panic disorder after controlling for non-cannabis drug dependence and daily cigarette smoking. These hypotheses were collectively driven by past work indicating that more severe or problematic forms of cannabis use are particularly associated with panic symptoms and psychopathology (Zvolensky et al., 2006).

2. Method

2.1. Participants

Participants were a subset of individuals from the Oregon adolescent depression project. Participants were originally randomly selected from nine senior high schools in western Oregon. All participants consented to participate in the investigation. A total of 1709 adolescents (ages 14–18; mean age at initial assessment = 16.6 years, $SD = 1.2$) completed the initial assessment ($T1$), which consisted of an interview and questionnaires, between 1987 and 1989. Approximately 1 year later ($T2$), 1,507 participants (88.2%) participated in a reassessment that used the same interview questions and questionnaires (mean interval between $T1$ and $T2 = 13.8$ months, $SD = 2.3$) (additional details are provided elsewhere; Lewinsohn et al., 1997).

As probands from the Oregon Adolescent Depression Project reached their 24th birthday, participants with a history of MDD at $T2$ ($n = 360$), those with a history of other Axis I disorders at $T2$ ($n = 284$), and a subset of those with no history of mental disorder at $T2$ ($n = 457$) were invited to participate in a $T3$ interview. This sampling strategy was intentional due to the expense of running this longitudinal investigation. The no-disorder comparison group was representative of the entire group of participants with no mental disorder at $T2$ ($n = 863$) in age and gender within age; all racial or ethnic minority participants were invited to participate in the $T3$ assessment.

Of the 1101 young adults selected for $T3$ interview, 941 participated (85.4%). Of those participants, 57.2% were female, 89.0% were white, 34.1% were married, 96.8% had graduated from high school, and 31.4% had a bachelor's degree or a higher educational level. Their average age at $T3$ was 24.2 years ($SD = 0.6$). Women were more likely than men to complete the $T3$ assessments (88.9% versus 81.0%) ($F = 13.55$, $df = 1$, $n = 1101$, $p < .001$). Differences in $T3$ participation as a function of other demographic characteristics or $T2$ diagnostic status were not statistically significant.

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