Childhood Sexual Abuse, Early Cannabis Use, and Psychosis: Testing an Interaction Model Based on the National Comorbidity Survey

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Previous research investigating the etiology of psychosis has identified risk factors such as childhood sexual abuse and cannabis use. This study investigated the multiplicative effect of these variables on clinically assessed diagnoses of psychosis based on a large community sample (the National Comorbidity Survey). Demographic variables (sex, age, urbanicity, ethnicity, education, employment, and living arrangements) and depression were used as predictors in the first block of a binary logistic regression. In the second block, the variables representing early cannabis use, childhood sexual trauma, and the interaction between these variables were entered. There was no significant main effect for early cannabis use or childhood sexual trauma. The interaction was statistically significant (odds ratio [OR] = 6.93, 95% confidence interval [CI] = 1.39–34.63, *P* = .02). The effect for the sexual trauma variable was statistically significant for those who used cannabis under 16 years (OR = 11.96, 95% CI = 2.10–68.22, *P* = .01) but not for those who had not used cannabis under 16 years (OR = 1.80, 95% CI = 0.91–3.57, *P* = .09). Many factors have been shown to be significant in the etiology of psychosis; however, the current research augments previous findings by examining psychosis in terms of an interaction between 2 of these factors.

Key words: trauma/cannabis/psychosis

Introduction

A number of studies using population-based cohorts and prospective measures of cannabis use and adult psychosis have examined the nature of the relationship between these variables.[1–4] For example, in a sample of 45 570 male conscripts in Sweden, a dose-response relationship between cannabis consumption (number of occasions) at conscription when age 18 years and diagnosis of schizophrenia 15 years later was reported. In fact, those who had reported using cannabis more than 50 times were 2.3 times more likely to have had the diagnosis than nonusers after controlling for psychiatric diagnoses at conscription. Only 3% of cannabis users went on to develop schizophrenia, indicating that although cannabis use may be a factor in the etiology of this disorder,[1] it appears that it is only a part of a more broad explanation. In a follow-up of this cohort, participants who were classified as "heavy cannabis users" by the age of 18 years were 6.7 times more likely than nonusers to be diagnosed with schizophrenia 27 years later (*N* = 50 053).[2] This study did control for potential confounds, and although this decreased the risk of diagnosis, it remained statistically significant, leading the authors to suggest that their findings were "consistent with a causal relationship between cannabis use and schizophrenia."

In a general population sample of 4104 males and females, the effect of cannabis use on self-reported psychotic symptoms at baseline, 1 year later, and again 3 years later was examined.[3] Individuals who were using cannabis at baseline (age 16–17 years) were almost 3 times more likely at follow-up to report psychotic symptoms. Again, a dose-response relationship was reported, with the highest risk being for the highest cannabis use, with an odds ratio (OR) of 6.8. The authors contended that cannabis use was an independent risk factor for psychosis in those without psychosis, and those who had an existing vulnerability to psychotic disorders had an increased risk.

Based on a general population cohort from New Zealand[4] (*N* = 1011), the *Diagnostic and statistical Manual for Mental Disorders, Fourth Edition*,[5] criteria for cannabis dependency was used to identify cannabis-dependent participants at age 18 and 21 years. Self-reported rates of psychotic symptoms were significantly higher for those who were dependent compared with those who were not cannabis dependent at age 18 and 21 years. After controlling for a number of confounding factors, including exposure to childhood sexual abuse (CSA) and childhood physical abuse (CPA), a strong

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association remained at age 21 years. With temporal ordering of events clarified by statistically controlling for previous psychotic symptoms, the authors concluded that heavy cannabis use might make a causal contribution to the symptoms of psychosis. A previous study based on a subsample of 759 male and female participants indicated that cannabis users by the age of 15 years and by the age of 18 years had more schizophrenia symptoms than controls at age 26 years. These results remained significant after psychotic symptoms at age 11 years were controlled for. The researchers contended that cannabis use was associated with an increased risk of experiencing schizophrenia symptoms, even after psychotic symptoms preceding the onset of cannabis use were controlled for, indicating that cannabis use is not secondary to a preexisting psychosis.[6]

A recent review of these population-based studies suggested that cannabis use by age 18 years is associated with schizophrenia outcomes in adulthood and indeed that cannabis use most probably precedes schizophrenia.[7] The authors concluded that these studies have provided evidence in terms of direction, by controlling for a wide range of possible confounding variables. It appears that age of first use of cannabis mediates the strength of the cannabis-psychosis relationship. Based on a sample of 3500 Greek 19-year olds,[8] it was reported that using cannabis for the first time before the age of 16 years increased the likelihood of reporting psychotic symptoms compared with those who first used cannabis later. These effects were still significant after controlling for lifetime frequency of use.

However, although these studies have robustly controlled for possible confounds, only one has controlled for exposure to CSA,[4] which has been recently linked to the etiology of psychosis. Research has suggested that the relationship between traumatic events in childhood and psychosis in general, and schizophrenia in particular, may be as strong, or stronger, than the relationship between traumatic events in childhood and other less severe adult disorders.[9] In fact, it has been estimated that psychiatric patients (male and female) were twice as likely to have histories of childhood abuse compared with individuals in the general population.[10,11] Based on a review of the research findings from 1984 to 2003, it was concluded that about two-thirds of female psychiatric patients and 60% of male psychiatric patients had experienced CSA or CPA.[12] Additionally, 77% of children who were admitted to a psychiatric hospital and who also had a history of CSA were diagnosed as psychotic. Only 10% of nonabused children from the same study received a diagnosis of psychosis.[13] Indeed, research examining the prevalence of CSA among patients with different psychiatric diagnoses found that 70% of those with psychosis had CSA histories, while patients with anxiety and mania had CSA rates of 27% and 43%, respectively,[14] and that 83% of patients (mixed gender) diagnosed as schizophrenic had reported histories of CSA, CPA, or emotional neglect.[15]

High rates of CSA were reported (78%) in a sample of 139 female patients diagnosed as schizophrenic.[16] Moreover, among women inpatients diagnosed schizophrenic, 60% had suffered incest,[17] and in an earlier study, 46% of chronically hospitalized psychotic women had suffered incest.[18] Regarding recorded histories of sexual molestation of females, research has shown that rates vary from 12% to 51% between the general population and the psychiatric patient population, respectively, and male histories of sexual molestation at a level of 3% in the general population and 18% among those within psychiatric populations.[19,20]

The research literature has therefore identified both cannabis use and childhood traumas, particularly of a sexual nature, as risk factors for psychosis. This study aimed to test the hypotheses that using cannabis, particularly at an early age, and experiences of childhood sexual trauma would be significant predictors of a diagnosis of psychosis based on a nationally representative sample. In addition, the multiplicative effect, or interaction, between these 2 risk factors was estimated. It was hypothesized that the combined effect of early cannabis use and the experience of a sexual trauma during childhood would significantly increase the likelihood of a diagnosis of psychosis over and beyond the main effects of cannabis use and childhood sexual trauma. Specifically, it was predicted that the interaction would indicate that cannabis use under 16 years and exposure to childhood sexual trauma would produce the highest risk for psychosis.

Method

The National Comorbidity Survey (NCS)[21] was a collaborative epidemiological investigation (1990–1992) based on a stratified, multistage, area probability sample of noninstitutionalized persons aged between 15 and 54 years in the 48 coterminous states of America designed to study the prevalence and correlates of *Diagnostic and Statistical Manual for Mental Disorders, Third Edition Revised* (*DSM-III-R*),[22] disorders. The initial survey employed a household sample of over 8000 respondents, and a subsample of the original respondents completed the additional NCS Part II survey that contained a further detailed risk factor battery and additional diagnoses. All analyses reported in this article were conducted on data from the NCS Part II survey ($N = 5877$). A full description of the NCS is available.[21]

Measurements

Information relating to childhood sexual traumas was derived from the Posttraumatic Stress Disorder module from the modified version of the Composite International Diagnostic Interview (CIDI).[23] During the

administration of this module, participants were provided with a booklet in which each type of abuse was numbered and participants were asked to identify the number of the event rather than naming it, which increases participants' willingness to report such information.[21] Two questions that represented childhood sexual trauma were selected:

1. You were raped (someone had sexual intercourse with you when you did not want to by threatening you or using some degree of force).
2. You were sexually molested (someone touched or felt your genitals when you did not want them to).

These questions were supplemented with information about the respondent's age at first experience of rape or molestation. This information was used to recode the variables to indicate if the respondent was raped or molested under the age of 16 years. The 2 variables were further collapsed into one variable, which indicated if each participant had either (1) experienced neither trauma or (2) experienced one or both traumas.

Information relating to cannabis use was derived from the Medication and Drugs module. Respondents were asked "Have you ever used either marijuana or hashish, even once?" This question was supplemented with "How old were you the first time (you used marijuana or hashish)?" This information was used to recode the variable to indicate if the respondent (1) did not try marijuana or hashish or tried aged 16 years or over or (2) first used marijuana or hashish under the age of 16 years.

The CIDI was used to assess the lifetime prevalence of nonaffective psychosis (a summary category made up of schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, and atypical psychosis). The diagnosis of psychosis was based on clinical reinterviews administered by experienced clinicians using an adapted version of the Structured Clinical Interview for the *DSM-III-R*.[22,24]

Analysis

Initial analysis was conducted using an additive hierarchical binary logistic regression model in SPSS 11.0. The dependent variable was the diagnosis of psychosis. The following background variables, or covariates, were used as predictors in the first block: sex, age, depression (lifetime prevalence), urbanicity (a binary variable representing current urban or nonurban location), ethnicity (white or nonwhite), years in education (0–11, 12, 13–15, 16+ years), employment status (working, not working), and living arrangements (lives alone, does not live alone). In the second block, the variables representing cannabis use and sexual trauma and the cannabis use and sexual trauma interaction were entered.

Table 1. Incidences of Cannabis Use and Sexual Traumas

Cannabis Use and Sexual Traumas	Count (%)
First used cannabis under 16 y	643 (10.9)
Raped + cannabis under 16 y	143 (2.4)
Molested + cannabis under 16 y	469 (8.0)
Any sexual trauma under 16 y	543 (9.2)

Results

Data on cannabis use and sexual trauma experiences were available for 5877 participants who had a mean age of 32.02 years (SD = 10.59), and males comprised 48.1% of the sample. The majority of participants were white (76.3%), with others classified as black (11.3%), Hispanic (9.1%), or other (3.2%). The frequency of cannabis use and sexual traumas before the age of 16 years are presented in table 1, and the cross tabulated frequencies are presented in table 2.

The ORs from the logistic regression analyses are presented in table 3.

The first block of the regression model was significant (χ^2 = 88.27, df = 8, P = .00) and indicated that being nonwhite, having a lifetime prevalence of depression, not working, and living alone were all significantly related to a diagnosis of psychosis. The variables in the second block resulted in a significant model (χ^2 = 100.43, df = 11, P = .00), which was a significant improvement over the previous model ($\Delta\chi^2$ = 12.16, Δdf = 3, P = .01). The main effects for cannabis and trauma were not significant, but the interaction was statistically significant. The additive scale interaction was also estimated using the BINREG procedure in STATA by fitting a generalized linear model estimating risk differences (RDs). The results were similar in that the main effects were not significant, but the interaction was significant (RD = 0.025, 95% confidence interval [CI] = 0.021–0.030, P = .00).

To interpret the interaction, the cannabis and the interaction variables were removed from the second block and the analysis was rerun separately for those who used cannabis under 16 years and those who did not use cannabis under 16 years. The effect for the sexual trauma variable was statistically significant for those who used cannabis under 16 years (OR = 11.96, 95% CI = 2.10–68.22, P = .01) but not for those who had not used cannabis under 16 years (OR = 1.80, 95% CI = 0.91–3.57, P = .09).

Discussion

This article assessed the impact of cannabis use and CSA on cases of psychosis in a nationally representative sample. The analysis indicated no main effect for cannabis use on a diagnosis of psychosis. This is inconsistent with previous

Table 2. Psychosis Frequency by Early Cannabis Use and Sexual Trauma

| Cannabis Use | Sexual Trauma | Psychosis | | |
		Absent	Present	Total
No cannabis use <16 y	No sexual trauma <16 y	4828 (99.5%)	26 (0.5%)	4855 (100.0%)
	Sexual trauma <16 y	375 (97.8%)	8 (2.2%)	383 (100.0%)
First cannabis use <16 y	No sexual trauma <16 y	601 (99.4%)	4 (0.6%)	605 (100.0%)
	Sexual trauma <16 y	28 (88.0%)	4 (12.0%)	32 (100.0%)

research that has found significant associations between these variables.[1–4] However, this study controlled for many more potential confounding factors, which have been previously identified as risk factors for psychosis than other studies. Furthermore, a recent review of these studies suggested that although there may be an association between cannabis use and psychosis, there is not enough evidence to suggest that cannabis is a necessary cause for psychosis, given that within the studies reviewed not all adults with schizophrenia used cannabis in adolescence. Moreover, the authors suggested that cannabis is not a sufficient cause for psychosis, given that the majority of adolescent cannabis users do not go on to develop schizophrenia in adulthood.[7]

With regard to CSA and its association with psychosis, research has consistently found high rates of sexual abuse in childhood within psychotic populations.[13–20] In fact, a number of studies have provided important evidence in support of a causal relationship between child abuse and psychosis[25–27]; however, no main effect was found within this sample. One explanation of this may be that incidents of CSA have been underreported because, although general population data was used in this study, evidence suggests that psychiatric patients underreport

Table 3. Hierarchical Logistic Regression Estimates With Background Variables, Childhood Cannabis Use, and Childhood Sexual Trauma Predicting Psychosis

Predictors	P-value	Odds Ratios	95.0% Confidence Interval
Block 1			
Sex	0.16	0.68	0.40–1.16
Age	0.21	1.01	0.99–1.04
Urbanicity	0.38	0.78	0.45–1.36
Ethnicity	0.03	0.55	0.33–0.94
Depression	0.00	6.31	3.68–10.84
Education	0.25	0.86	0.66–1.11
Employment	0.00	0.30	0.17–0.51
Living arrangements	0.04	1.87	1.02–3.44
Block 2			
Cannabis under 16 y	0.43	0.66	0.23–1.86
Sexual trauma under 16 y	0.09	1.80	0.91–3.57
Cannabis by sexual trauma interaction	0.02	6.93	1.39–34.63

rather than overreport abuse.[28,29] The underreporting of CSA poses a serious concern for epidemiological research, and the issue regarding the stability and consistency of reports of this nature are addressed elsewhere.[30–32] A possible limitation of the present research therefore is the underestimation of abuse. Previous research has shown that asking about ''abuse'' as opposed to asking specific questions regarding abuse can lead to lower rates of acknowledgment by around 50%.[30] Using more specific and indeed comprehensive definitions of traumatic sexual experiences may increase the likelihood of obtaining a more accurate history of an individual. These findings do however support results from one prospective study that found no association between CSA and schizophrenic disorders in adulthood. The authors of this study did, however, note a number of methodological limitations that may have decreased the probability of finding an association between CSA and mental disorders.[33]

An additional purpose of this article was to assess the impact of the multiplicative effect of early cannabis use and CSA on a diagnosis of psychosis. Results indicated that the impact of sexual trauma was statistically significant for those individuals who used cannabis under 16 years but not for those who had not used cannabis under 16 years. In fact, those who used cannabis under the age of 16 years and had also been sexually abused were almost 12 times more likely to receive the diagnosis of psychosis. The mediating role of cannabis suggests that early cannabis use may increase the strength of the proposed trauma-psychosis relationship. This may be explained by both cannabis use and trauma acting indirectly on the dopaminergic system. Previous rat-based research has indicated that the active ingredient of cannabis (Δ9-tetrahydrocannabinol) mediates dopamine transmission[34] and that pubertal cannabinoid administration, compared with administration in adulthood, was associated with enhanced behavioral disturbances.[35] Sexual traumas are unquestionably stressful events, which will produce elevated cortisol levels, which have been demonstrated to be associated with dopamine activity.[36]

In terms of a vulnerability-stress model, the results of the current analysis raise the question as to which of these factors constitutes vulnerability and which constitutes stress. It may be possible in light of recent

evidence that childhood abuse enhances an individual's susceptibility/vulnerability to the experience of psychosis symptoms[9] and that cannabis use acts as an exacerbating (stressful) agent heightening psychotic experience in those who have suffered abuse. Alternatively, cannabis use could be considered the vulnerability factor and CSA the environmental stressor that increases the likelihood of psychosis. This is particularly important given the high rates of cannabis use in the general and clinical population.[37]

Research findings based on reports from psychiatric patients have indicated that assessments of traumatic experiences, particularly childhood traumas, are not routinely taken.[38] The findings of this study highlight the importance of evaluating drug use history and interpersonal victimization experiences during clinical assessment to ensure comprehensive formulation of the patient's difficulties and appropriate treatment planning.[39]

Further research is necessary in order to demonstrate the robustness of this finding. It should be noted that only 4 individuals were exposed to early sexual trauma and early cannabis use and were identified as psychotic. Findings based on such a small cell size require replication and do not represent unequivocal support of the hypothesis. In addition, it would be valuable to establish if a different temporal ordering of cannabis use and trauma was associated with different risks of psychosis. For example, it may be that exposure to a trauma followed by cannabis use (self-medication) and cannabis use followed by trauma produce significantly different likelihoods of psychosis.

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