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ORIGINAL PAPER

Influence of recent cannabis use on altered spectral entropy modulation and connectivity strength in patients with psychosis

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Abstract

Cannabis use is highly prevalent in individuals with psychosis, raising concerns about its influence on brain function. Electroencephalography (EEG) studies have identified alterations in brain activity in psychosis, including changes in spectral entropy (SE) modulation and connectivity strength (CS). However, the degree to which cannabis use contributes to these alterations remains unclear. This study investigated the effects of recent cannabis use on specific EEG measures previously found to be altered in psychosis: (i) SE modulation, (ii) pre-stimulus theta and broadband CS, and (iii) baseline CS in the gamma band. We focused specifically on the immediate effects of recent cannabis use, without considering factors like tetrahydrocannabinol content, frequency of use, or age of onset. We included 93 patients with psychosis (32 recent cannabis users, 61 non-users) and 86 age- and sex-matched healthy controls (HC; all non-users). Recent cannabis use was defined as any consumption within the past week, assessed through a clinical interview and confirmed by urinalysis. Patients had diagnosis of schizophrenia or bipolar disorder. EEG data were recorded during a P300 task, and SE modulation and baseline CS were calculated. Both patient groups (cannabis users and non-users) exhibited significantly impaired SE modulation and elevated gamma and broadband CS, compared to HC. Crucially, no significant differences were found between the two patient groups in any of the EEG measures. Recent cannabis use does not appear to be the primary driver of the observed electrophysiological alterations in psychosis. Impaired SE modulation and increased CS are likely core features of psychosis itself, independent of recent cannabis exposure. This suggests that these EEG abnormalities may represent underlying vulnerability markers for psychosis. However, further research is needed to explore the potential long-term and early-onset effects of cannabis use on brain function in individuals with psychosis.

Keywords Cannabis · Psychosis · EEG · P300 · Spectral entropy · Connectivity strength

Introduction

Cannabis is a psychoactive drug with high prevalence of use among individuals with psychosis [16]. Its primary mechanism of action involves binding to cannabinoid receptors but indirectly influences various neurotransmitter systems responsible for modulating brain activity [8, 14, 23]. Consequently, cannabis use may contribute to functional brain alterations reported in psychotic syndromes, such as the electrophysiological abnormalities found in schizophrenia and bipolar disorder. These alterations, detectable through EEG and MEG due to their high temporal resolution, are relevant for understanding the underlying neural mechanisms of these syndromes, particularly the rapidly changing dynamics of neural activity modulation associated with mental functions [3].

In this context, our research group has consistently identified a significant deficit in EEG modulation during a P300 task in individuals with psychosis. This deficit, measured using spectral entropy (SE), has been replicated across multiple studies and samples of patients [1, 19, 20]. Additionally, we observed increased pre-stimulus connectivity strength (CS) in the theta and global bands during the same task, suggesting a hyperactive baseline state [2], which was inversely correlated with SE modulation [9]. These findings

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suggest that a hyperactive baseline state coupled with reduced capacity for neural modulation may play a role in psychoses [5, 15], coherent with excitatory/inhibitory imbalance reported in these conditions [10, 21].

While the influence of cannabis use on these functional alterations remains largely unexplored, evidence suggests a potential link. Cannabinoids significantly affect glutamate transmission, probably affecting (at least) gamma and theta oscillations [24]. Studies have shown that recent cannabis use is associated with increased theta power and decreased beta and gamma power during resting state [28]. This is further supported by research indicating decreased gamma spectral power in healthy cannabis users compared to non-users during an auditory steady-state response (ASSRs) paradigm, without affecting N100 amplitude or inter-trial coherence [25].

This study investigated the possible short-term effects of recent cannabis use on electrophysiological measures previously identified as altered in psychosis. Specifically, and based on previous reports, we examined the impact of cannabis use in the psychotic population on the following measures: (i) SE modulation [1, 19, 20]; (ii) theta and broadband baseline CS [2]; and (iii) gamma baseline CS, given the stablished influence of cannabis on gamma oscillations [25, 28] and their potential role in schizophrenia [6, 10, 17, 26]. To achieve this, we compared these measures between individuals with psychosis who currently use cannabis, those with psychosis who do not, and healthy (non-user) controls. This initial investigation focused solely on the immediate effects of recent cannabis use in currently consumer patients, not addressing at this moment other potentially important issues such as tetrahydrocannabinol content, frequency of use and age of onset, which necessitate specific study design.

Subjects and methods

Study sample

The study included 93 patients with psychosis (32 current cannabis users, 61 non-users; see Table 1) and 86 age- and sex-matched healthy controls (HC; all non-users). Recent cannabis use was defined as any consumption within the past week, assessed through a clinical interview and confirmed by urinalysis. The patient group comprised 75 individuals with schizophrenia (34 first episodes) and 18 individuals

Table 1 Participant sociodemographic, clinical and electrophysiological characteristics, and group comparisons

	Patients (n = 93)		HC (n = 86)
	CNN(+) (n = 32)	CNN(-) (n = 61)	
Age (years)	28.34 (7.02)	26.30 (5.05)	26.23 (5.72)
Sex (Male: Female)	25:7	37:24	47:39
Education (years)	12.11 (3.27)**	14.27 (3.79)**	16.13 (2.35)
Parental Education (years)	10.38 (3.92)**	12.16 (3.98)*	14.14 (3.91)
Diagnosis (SZ:FE:BP)	29:10:3	46:24:15	n.a
CPZ equivalents (mg)	321.90 (192.98)	355.77 (238.61)	n.a
Illness duration (months)	40.84 (53.71)	56.61 (77.64)	n.a
PANSS–Positive symptoms	13.55 (4.68)	11.70 (4.79)	n.a
BNSS–Total	32.48 (17.56)	20.89 (18.74)	n.a
WAIS-Total IQ	86.39 (11.68)**	87.57 (14.89)**	113.55 (12.28)
BACS-Verbal learning	40,39 (7.71)**	40.30 (10.61)**	53.44 (8.83)
BACS-Working memory	17.65 (3.55)**	16.79 (4.69)**	22.39 (3.14)
BACS-Motor speed	62.65 (18.14)**	61.59 (16.22)**	73.67 (17.55)
BACS-Verbal fluency	20.35 (4.85)**	19.60 (5.07)**	27.51 (4.94)
BACS-Performance speed	45.13 (9.47)**	48.30 (13.23)**	69.96 (11.65)
BACS-Problem solving	17.23 (3.27)*	16.29 (3.58)**	18.30 (2.30)
WCST-Perseverative errors (%)	18.52 (12.21)**	14.85 (13.18)**	8.58 (3.71)
SE modulation	0.062 (0.649)**	0.185 (0.679)**	−0.492 (1.216)
Pre-stimulus CS – Theta band	0.372 (0.052)*	0.366 (0.054)	0.355 (0.031)
Pre-stimulus CS – Gamma band	0.286 (0.053)*	0.280 (0.065)#	0.264 (0.040)
Pre-stimulus CS – Broadband	0.324 (0.042)*	0.317 (0.056)	0.305 (0.034)

In the patient group columns, symbols indicate significant differences compared to healthy controls (HC) using independent samples t-tests: **p <.005, * p <.05. The # symbol indicates a trend towards significance at p=.07. Additionally, the two patient groups differed significantly in years of education (p =.013) and BNSS negative symptomatology scores (p =.016)

with bipolar disorder, all with normal hearing. Diagnoses were established by an expert psychiatrist (VM or OM) involved in the patients' clinical care, according to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5). Among the 61 recent cannabis non-users, 38 had used this drug to a variable amount during recent years, although data on frequency of consumption, age of onset, tetra-hydro-cannabinol concentration and duration of use were not available.

Exclusion criteria were: (1) neurological disease; (2) history of head trauma with loss of consciousness; (3) current substance abuse (excluding nicotine and caffeine); (4) Intelligence Quotient (IQ) below 70; (5) any psychiatric treatment (for controls); and (6) current psychiatric diagnosis other than schizophrenia or bipolar disorder (for patients). Sociodemographic, behavioral, cognitive, and clinical data are presented in Table 1. All participants provided written informed consent after receiving comprehensive study information. This study was approved by the ethical committees of all participating hospitals.

Clinical and cognitive assessments were conducted as previously described [1, 2, 19, 20]. The Positive and Negative Syndrome Scale (PANSS) [11] and the Brief Negative Symptom Scale (BNSS) [13] were used to assess positive and negative symptoms, respectively. Cognitive function was evaluated using the Brief Assessment in Cognition in Schizophrenia Scale (BACS) [12] and the Wisconsin Card Sorting Test (WCST), specifically the percentage of perseverative errors.

EEG data acquisition and measures

Electrophysiological procedures for measuring SE and CS have been detailed in previous reports [1, 2, 19, 20].

Briefly, SE quantifies the degree of uncertainty or randomness in the EEG signal during task performance [7]. High SE values indicate a more uniform distribution of spectral content (a highly random signal), while low SE values reflect concentrated power within a narrower frequency range (a more regular signal). Thus, SE serves as a global index of EEG activity, capturing changes from the pre-stimulus to the active task windows (*i.e.,* SE modulation measure). Differences in EEG modulation between groups during the task can be assessed by comparing SE values between the windows immediately prior and posterior to a target stimulus. In this study, SE was calculated for each sensor during a P300 task (pre-stimulus and response windows), and data were summarized using principal components analysis, yielding a single factor that captured most of the variance. In HC, SE modulation values are typically negative, indicating a more regular –less entropic– signal during the response compared to the pre-stimulus window.

Connectivity strength (CS) quantifies global functional connectivity within a brain network, with higher values indicating greater synchrony. This parameter, derived from the concept of density in binary networks and represents the average edge values of all node's connections. These connections are typically quantified using phase-locking values (PLV), which, in the context of EEG, reflect the degree of synchronization between sensors. Higher synchronization is associated with greater PLV. Thus, CS provides a single, quantitative index summarizing the overall synchrony among neural assemblies within the network. For this study, CS values were calculated during the P300 task, focusing specially on the pre-stimulus window, as this previously demonstrated significantly higher CS in patients with psychosis [2].

Statistical analysis

Between-group differences in sociodemographic, clinical and cognitive measures were assessed by t-test or chi-square as appropriate. The effects of current cannabis use on the mentioned EEG parameters (SE modulation and baseline CS in the theta, gamma and global bands), were analyzed using a one-way analysis of variance (ANOVA). This analysis compared three groups: patients with psychosis who currently use cannabis, patients with psychosis who do not use cannabis, and HC. Post-hoc pairwise comparisons (t-test for independent samples) were conducted to identify specific group differences (or trends) where appropriate.

Results

There were no significant differences in age or sex between the three groups (cannabis-using patients, non-using patients and HC). Similarly, no significant differences were found between the two patient groups in terms of cognitive scores, family education level, or chlorpromazine (CPZ) equivalent doses. However, both patient groups had significantly lower cognitive scores and education levels compared to HC. Cannabis-using patients had lower education level ($t(91) = 1.619$, $p = 0.013$) and higher scores on the BNSS ($t(91) = 2.479$, $p = 0.016$) than non-using patients (Table 1).

The one-way ANOVA revealed a significant main effect of group for SE modulation ($F(2, 176) = 9.704$, $p < 0.001$), with a trend toward significance for baseline CS in the gamma band ($F(2, 176) = 2.698$, $p = 0.070$) and the broadband ($F(2, 176) = 2.606$, $p = 0.077$). There was no significant group effect on theta CS ($F(2, 176) = 2.379$, $p = 0.096$).

Post-hoc comparisons showed that both patient groups exhibited significantly more positive SE modulation (*i.e.,* a smaller change in SE from pre- to post-stimulus) compared

to HC. This effect was observed for both cannabis users ($t(116) = 3.180$, $p = 0.002$) and non-users ($t(145) = 3.937$, $p < 0.001$) (Table 1; Fig. 1). Regarding the pre-stimulus CS parameters, post-hoc pairwise analyses found significant differences between cannabis user patients and HC for the theta band ($t(116) = 2.301$, $p = 0.023$), gamma band ($t(116) = 2.320$, $p = 0.022$) and the broadband ($t(116) = 2.483$, $p = 0.014$); and a trend between cannabis non-users and HC for the gamma band ($t(145) = 1.835$, $p = 0.069$). We found no significant differences between both groups of patients after any pairwise analysis (Table 1; Fig. 2). Thus, patients recently using or not cannabis did not differ in SE modulation or CS values.

Discussion

Our results suggest that recent cannabis use does unlikely account for the previous findings supporting a hyperactive baseline and a deficit in task-related cortical modulation in psychosis. Specifically, we found no significant differences in SE modulation or pre-stimulus CS between patients with psychosis who used cannabis recently and those who did not. Moreover, both patient groups exhibited significantly impaired SE modulation and similarly elevated CS values compared to HC, irrespective of cannabis use. This suggests that these electrophysiological alterations are likely core features of psychosis, rather than being primarily driven by recent cannabis exposure.

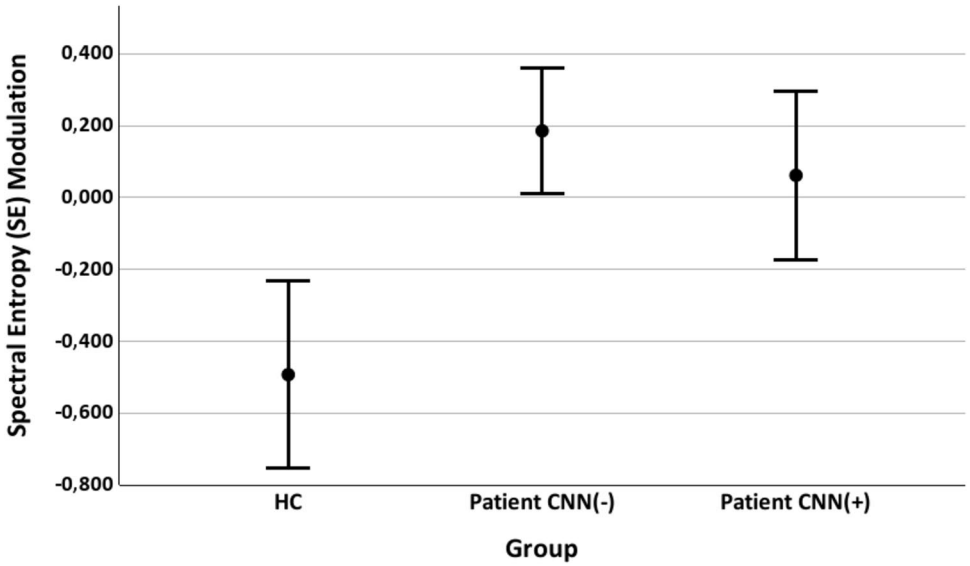

Fig. 1 Spectral entropy (SE) Modulation values (mean and 95% confident intervals, CI) in cannabis user patients (CNN(–)), non-user patients (CNN(+)) and healthy controls (HC). Significant differences were found between each group of patients and controls, but not between both groups of patients

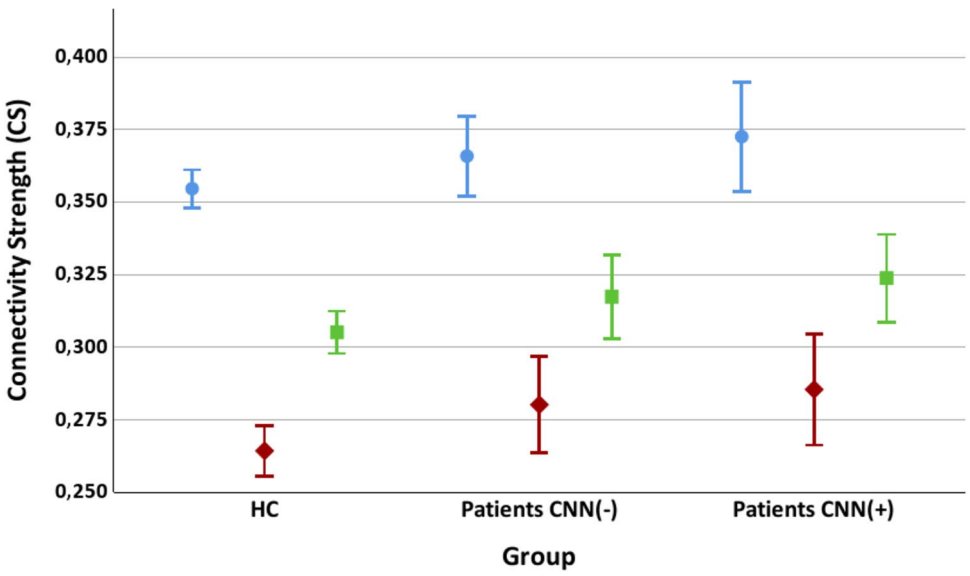

Fig. 2 Connectivity strength (CS) values (mean and 95% confident intervals, CI) in the theta band (black), gamma band (green) and broadband (red). Significant differences or trend towards significance were found between each group of patients and controls, but not between both groups of patients

Interestingly, while previous research in healthy individuals has shown that cannabis use decreases gamma activity [25, 28], our findings indicate increased gamma CS in patients with psychosis who use cannabis. This suggests that the relationship between cannabis use and gamma activity may differ in the context of psychosis, and alterations in this frequency band are unlikely explained by this substance. Furthermore, the similar pattern of elevated pre-stimulus CS in the gamma band observed in both cannabis users and non-users among our patients reinforces the idea that this alteration is likely inherent to psychosis, rather than being solely attributable to cannabis.

Our data therefore support the notion that task-related EEG modulation deficits are independent of cannabis use, at least from recent use. This aligns with our previous work, which demonstrated that these deficits are unrelated to CPZ equivalent doses and independent of treatment with antipsychotics, antidepressants, benzodiazepines and lithium, as a major effect of treatment and chronicity on these alterations [20]. Therefore, SE modulation deficit may be more closely linked or primarily related to the neurobiological underpinnings of psychosis, as suggested by its significant association with cognitive deficits [19] and baseline hyperactivity [9].

It is worth noting that some pre-existing brain structural differences, as thinner left precentral and right inferior parietal gyri and lower right caudate volume, have been reported in cannabis users before they initiate use [18]. This raises the possibility that certain brain vulnerabilities, usually related to cannabis use, may predate and potentially contribute to the onset of psychosis, rather than merely follow cannabis use. However, we cannot rule out that cannabis consumption may aggravate such brain activity alterations.

Such an (at least) relative independence between cannabis use and brain dysfunction may be relevant, since the observed pattern of baseline hyperactivity and task-related hypomodulation aligns with the concept of a relative inhibitory deficit in psychosis, which is supported by neurobiological [10] and neuroimaging research [21] in schizophrenia. Thus, while cannabis has a known effect on glutamate transmission [24], our data suggests that acute cannabis use is not a primary driver of this possible excitatory/inhibitory imbalance in psychoses.

However, while our findings suggest that acute cannabis use is not the primary factor behind SE modulation deficits or hyperactive baseline CS in psychosis, we cannot rule out the impact of long-term cannabis use or early use during adolescence, a period when cannabis use is known risk factor for schizophrenia [27]. Long-term cannabis use has been reported to influence P300 amplitude in schizophrenia patients, although both users and non-users exhibit reduced amplitude compared to HC [22].

Finally, our reliance on clinical interviews and urinalysis to assess cannabis use has limitations. These methods do not provide an objective measure of cannabis use beyond the past week, nor can they differentiate the potential influence of varying THC concentrations. Future research should address these limitations, particularly given –for example– the reported geographical variations in schizophrenia incidence linked to cannabis potency [4].

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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Ethical standards All participants gave their informed consent prior to their inclusion in the study. The study received the corresponding Institutional Review Board approval and was in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. We herein acknowledge that each author has reviewed and approved the manuscript and no substantial portion of the study has been published or is under consideration for publication elsewhere.

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