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Effects of Cannabis Use and Subclinical ADHD Symptomology on Attention Based Tasks in Adolescents and Young Adults

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Abstract

Objective: Research has demonstrated comorbidity between Attention-Deficit Hyperactivity Disorder (ADHD) and cannabis use, and some have proposed that subclinical ADHD symptoms may explain attentional deficits in cannabis users. Here we investigated whether subclinical ADHD symptoms and cannabis use independently or interactively predict performance on attention tasks in adolescents and young adults.

Method: Seventy-two participants (cannabis users (MJ) = 34, Controls = 38) completed neuropsychological tasks of inhibition and attention. Parent report on the Child Behaviors Checklist reflected current ADHD symptoms. Multiple regression analyses examined whether ADHD symptoms and cannabis use independently or interactively predicted cognitive outcomes.

Results: Cannabis use was significantly associated with slower CPT hit rate response. Subclinical ADHD symptoms did not independently predict or moderate cannabis effects.

Conclusions: Cannabis users demonstrated slower response rate during an attentional task. Subclinical ADHD symptoms did not predict any deficits. As such, attention deficits seen in cannabis users are more related to substance use than ADHD symptomatology.

Keywords: ADHD; Attention; Drug and alcohol abuse

Cannabis use remains the most common illicit drug used by adolescents and emerging adults, with approximately 38% of young adults reported using cannabis in the past 12 months (Johnston, O'Malley, Bachman, Schulenberg, & Miech, 2016). Cannabis use in youth has previously shown poorer performance on tasks that require attention functioning, including cognitive control, sustained attention and working memory (Lisdahl, Wright, Kirchner-Medina, Maple, & Shollenbarger, 2014). Specifically, studies have shown that cannabis use is associated with decreased attention, verbal working memory, and decreased executive functioning (Hanson et al., 2010; Lisdahl & Price, 2012). However, one criticism of this literature is that perhaps subclinical symptoms of attentional problems, as seen in Attention-Deficit Hyperactivity Disorder (ADHD), may predate cannabis exposure.

ADHD can be characterized as a persistent pattern of inattentive, hyperactive, and impulsive behaviors. Childhood ADHD symptoms, specifically in areas of sustained and focused attention, may persist well into adulthood (Balint et al., 2009). Studies show that there are increased rates of comorbid substance use in individuals diagnosed with ADHD, including cannabis. Specifically, children with ADHD are about 1.5 times more likely to develop cannabis abuse or cannabis dependence than their non-ADHD counterparts (Lee, Humphreys, Flory, Liu, & Glass, 2011). Calls to examine the impairment within individuals with subclinical ADHD have become more prevalent (Kobor, Takacs, Urban, & Csepe, 2012). Recent research has even begun to suggest that late onset ADHD, that otherwise appears subclinical in adolescence, may play a significant role in these neurocognitive deficits that are otherwise attributed to substance users (Faraone et al., 2007). It can be reasoned, then, that even subclinical ADHD symptoms may contribute to the neurocognitive outcomes seen in cannabis users.

Indeed, we see similar patterns of attention and cognitive control deficits in youth with ADHD or regular cannabis use, including deficits if attention, working verbal memory, and executive control (Willcutt, Sonuga-Barke, Nigg, & Sergeant,

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2008), though this research often fails to factor cannabis use into investigations of the impact ADHD symptoms have on attention and inhibition performance. Recent research in young adults with childhood ADHD have found that ADHD predicted poor executive function performance, even after controlling for cannabis use (Tamm et al., 2013), although individuals with ADHD who had early onset cannabis use (before age 16) demonstrated worsened attention and executive functioning. In contrast, Lisdahl and colleagues (2016) found that after controlling for ADHD, cannabis use was linked with abnormal frontal and parietal brain structure. Thus, more information about the impact of ADHD disorder or symptomology and cannabis use on neurocognition is needed.

In the current study, we examined whether cannabis use and ADHD subclinical symptoms independently and interactively predicted attention and cognitive control performance in adolescents and emerging adults. We expect cannabis use and parent-reported ADHD symptoms to independently predict decreased performance on these neuropsychological tasks. Further, we predict that cannabis users with higher ADHD symptoms will demonstrate more robust attentional deficits.

Method

Participants

Seventy-two participants (cannabis users (MJ) = 34, Controls = 38) were recruited through flyers and advertisements in the local community and college campuses. Participants were between the ages of 16 and 26 ($M = 21.3$, $SD = 2.6$), were gender balanced (52.8% male), and predominately Caucasian (66.7%). Participants were included if they were right handed, spoke English, and were willing to abstain from substance use over a 3-week period. Exclusion criteria included having an independent DSM-IV Axis I (attention, mood, anxiety, or psychotic) disorder in the past year, major medical or neurological disorders, prenatal medical issues or premature birth (gestation <35 weeks), reported prenatal alcohol/illicit drug exposure, or excessive other drug use (>20 times of lifetime use for each drug category). Cannabis users were categorized as weekly users who used cannabis at least 45 times in the last year or had at least 100 lifetime uses. Controls used cannabis up to 5 times in the past year and less than 20 times lifetime.

Procedures

Data was used from a larger parent study examining the neurocognitive effects of cannabis use in youth (R01 DA030354; PI: Lisdahl); all aspects of the protocol were IRB-approved. Potential participants who expressed interest in the parent study were screened through an initial semi-structured interview for independent past-year Axis I Disorders other than substance use disorder (SUD) over the phone. If determined eligible, study staff obtained written consent from participants (aged 18 or older). All minors below 18 years of age provided written assent after parent consent was acquired. Participants who were eligible for the study came in for five study sessions over the course of three weeks. Data from the baseline session (Day 1) and fourth session (Day 20 of 21) are assessed here. Participants completed a series of psychological questionnaires, drug use interview, neuropsychological battery, and an MRI scan over the course of three weeks. During that period, participants were required to remain abstinent from alcohol, cannabis, and other drug use, which was confirmed through breath, urine and sweat toxicology screening. Parents of participants were consented for a parent-administered phone interview that screened for medical, psychiatric and prenatal history. Parents were also administered the Child Behavior Checklist (CBCL) to fill out in relation to their participating child's current behavior over the past 6 months (Achenbach & Rescorla, 2001).

Measures

Detailed phone screen. Lifetime substance use—To determine lifetime patterns of drug and alcohol use, youth participants were given the Customary Drinking and Drug Use Record (CDDR) (Brown et al., 1998) at baseline to measure frequency of alcohol, nicotine, cannabis, and other drug use, SUD symptoms, and the age of onset for first time and regular (weekly) use.

Mini Psychiatric Interview—Participants and parents of minors were interviewed using the Mini International Psychiatric Interview (MINI) (Sheehan et al., 1998) or MINI-Kid (Sheehan et al., 2010) to screen out for psychiatric comorbidities.

Inattention symptoms—Parents completed the Child Behavior Checklist (CBCL) (Achenbach & Edelbrock, 1991) to provide a measure of current (past 6 month) symptoms of psychopathology, including inattention. Continuous ADHD symptom scores were computed based on DSM-IV syndrome scores proposed by the ASEBA manual (including combined inattentive and hyperactive-impulsive symptoms).

Study session. Substance use—Timeline Follow-Back interviews were conducted by trained RAs to measure substance use patterns on a weekly basis for the past year while providing memory cues such as holidays and personal events (Sobell & Sobell, 1995). Substances were measured by standard units [alcohol (standard drinks), nicotine (number of cigarettes and hits of chew/snuff/pipe/cigar/hookah), cannabis (all methods converted to joints or mg in concentrates), ecstasy (number of tablets), sedatives (number of pills or hits of GHB), stimulants (cocaine and methamphetamine use converted to mg and number of amphetamine pills), hallucinogens (number of hits or occasions of ketamine/salvia/shrooms/other hallucinogens), opioids (number of hits of heroin/opium), and inhalants (number of hits)].

Neuropsychological battery—Following 3 weeks of monitored abstinence, a neuropsychological battery was administered; tasks of interest are listed below.

- The Ruff 2&7 is a neuropsychological task that is designed to measure sustained and selective attention within individuals (Ruff & Allen, 1996). The total number of correct responses to target stimuli as well as speed of completion was recorded and assessed as total accuracy and total speed per participant.
- The CPT-II measures selective and sustained attention as well as levels of impulsivity (Conners & Staff, 2000). Participants are required to respond to target stimuli and inhibit their response during distractor stimuli on a computer for a period of 14 min. The total number of commission errors and participant average response time was utilized to measure performance on the CPT-II.
- The WAIS-III LNS subtest measures working memory, selective attention and mental control and requires participants to remember a series of number and letters that gradually increase by a unit of one digit or letter (Wechsler, Coalson, & Raiford, 1997). The number of trials that were correctly completed by participants was used to tabulate the total score.
- D-KEFS Color-Word Interference measures inhibition and cognitive control (Delis, Kaplan, & Kramer, 2001). Participants were required to complete an interference trial where they must correctly identify the ink colors of words while inhibiting their response to read the word written as a contrasting color.

Psychological questionnaires—Participants also completed the Beck's Depression Inventory (BDI) and the State Trait Anxiety Inventory (STAI) to establish if subclinical psychiatric factors such as depression and anxiety differ between groups and therefore may mediate the relationship between cannabis use and cognition.

Statistical Analysis

A series of regressions were run with cannabis group, ADHD total symptoms, and cannabis group-by-ADHD symptoms as the independent variables of interest; covariates included age and past year alcohol use (total standard drinks). Cannabis group-by-ADHD symptoms was examined by creating an interaction term and multiplying the ADHD total symptom count by cannabis use classification. Before the interaction term was created, both predictor variables were centered to eliminate nonessential multicollinearity. Decisions about statistical significance were made if $p \leq .05$.

Results

Demographics

Controls and cannabis users were not significantly different in race ($\chi^2 = 5.84$, $p = .44$), ethnicity ($\chi^2 = 1.15$, $p = .56$), gender ($\chi^2 = 3.58$, $p = .06$), or years of education ($t = 0.93$, $p = .59$). There was a significant difference between cannabis users and controls in age ($t = 2.00$, $p = .05$), with the average age of the cannabis user group being 21.91 ($SD = 2.33$) compared to 20.71 ($SD = 2.7$) in the control group; therefore, age was included in all regressions. See Table 1.

Table 1. Demographics

	Age (*M*,*SD*)	Race (% Caucasian)	Ethnicity (% Not Hispanic)	Gender (% Male)	Years of Education (*M*,*SD*)
MJ Users	21.9 (2.3)	64.7%	85.3%	64.7%	14.4 (2.0)
Controls	20.7 (2.7)	68.4%	86.8%	42.1%	14.1 (2.3)

Substance Use

Controls were limited to smoking cannabis less than 5 times in the past year ($M = 0.28$, $SD = 0.89$, Min = 0.00, Max = 4.80) and less than 20 in their lifetime ($M = 2.38$, $SD = 5.05$, Min = 0, Max = 20.00). Cannabis users had an average of 289 joints in the past year ($M = 289.18$, $SD = 301.96$, Min = .05, Max = 1,394) and over 900 joints in their lifetime ($M = 966.59$, $SD = 1{,}060.93$, Min = 100, Max = 6,000); as expected, cannabis users had significantly higher cannabis use in the last year ($t(70) = -5.90$, $p < .001$) and in their lifetime ($t(70) = -5.61$, $p < .001$) compared to controls. Cannabis users had significantly higher alcohol use in the past year ($M = 290.56$, $SD = 255.97$, Min = 0, Max = 897.00) compared to controls ($M = 111.31$, $SD = 175.13$, Min = 0, Max = 598.50), $t(70) = -3.50$, $p = .001$ (alcohol was a covariate in regressions). On average, cannabis users last used cannabis 32.53 days prior to testing ($SD = 32.08$, Min = 16, Max = 201).

ADHD Subclinical Symptoms

There were no significant difference in ADHD symptoms ($t(70) = -1.57$, $p = .12$) between the cannabis ($M = 2.94$, $SD = 3.40$, Min = 0, Max = 13.00) and control ($M = 1.74$, $SD = 2.37$, Min = 0, Max = 8.00) groups.

Psychological questionnaires. There were no significant differences in scores on the STAI for State Anxiety (S-STAI) nor Trait Anxiety (T-STAI) between cannabis users (S-STAI: $M = 26.97$, $SD = 6.11$; SDT-STAI: $M = 32.39$, $SD = 7.18$) and controls (S-STAI: $M = 26.92$, $SD = 6.44$, $t(70) = -.033$, $p = .97$; T-STAI: $M = 29.50$, $SD = 5.19$, ($t(70) = -1.40$, $p = .17$). However, there was a significant difference in depression symptoms on the BDI between cannabis users ($M = 5.08$, $SD = 4.10$, Min = 0, Max = 15.00) and controls ($M = 2.84$, $SD = 3.14$, Min = 0, Max = 10.00, $t(70) = -2.63$, $p = .01$), with cannabis users reporting higher levels of depressive symptoms. Due to these differences, Total BDI scores were incorporated into the regressions.

Neuropsychological Outcomes

See Table 2 for means and standard deviations for the neuropsychological outcomes according to group.

Primary Findings

Cannabis use status significantly predicted CPT-II slowed hit response time ($t = 2.02$, $p = .05$). Increased age predicted decreased commission errors ($t = -2.02$, $p = .05$). ADHD symptoms, cannabis-by-ADHD symptoms interaction, BDI symptoms, and alcohol use did not predict any of the other cognitive variables. See Table 3 for regression table including beta coefficients and *t*-values.

Post-Hoc Analyses

Although gender was not significantly different between groups, there was a general trend of more males in the MJ group compared to the control group ($p = .06$). A separate regression was run taking into account gender as a covariate. By including gender, cannabis use status still significantly predicted CPT-II slowed hit response time ($t = 2.07$, $p = .04$), but significant effects between age and commission errors became statistically insignificant. Gender did significant predict performance on LNS, with males performing significantly better than females ($t = -3.08$, $p = .003$).

Table 2. Neuropsychological outcomes

	Ruff TA	Ruff Speed	CPT Commission Errors	CPT RT	LNS	DKEFS I	DKEFS I/S
MJ Users	95.3 (16.1)	113.7 (24.4)	12.3 (6.2)	408.7 (56.9)	13.3 (3.3)	38.6 (7.7)	47.8 (8.5)
Controls	96.1 (14.6)	117.7 (24.0)	17.4 (8.9)	378.8 (58.9)	13.7 (2.5)	37.1 (9.0)	47.8 (12.1)

Note: Standard deviations are in parentheses. Ruff TA = Ruff Total Accuracy scores; CPT = Continuous Performance Task; CPT RT = Continuous Performance Task Reaction Time; LNS = Letter Number Sequencing; DKEFS I = Delis-Kaplan Executive Function System Color Word Interference Trial; DKEFS I/S = Delis-Kaplan Executive Function System Color Word Interference/Switching Trial.

Table 3. Multiple regression table

	CPT Commission Errors	CPT RT	LNS	Ruff TA	Ruff Speed	DKEFS I	DKEFS I/S
Constant	35.49 (4.19)	328.57 (4.95)	14.45 (4.51)	80.12 (4.78)	100.90 (3.73)	23.35 (2.47)	50.31 (4.21)
ADHD Symptoms	−0.63 (−1.84)	−1.06 (−0.4)	0.05 (.38)	−1.00 (−1.49)	−0.46 (−.42)	0.23 (.60)	−0.12 (−.24)
MJ Group	−2.73 (−1.31)	33.11* (2.02)	−0.52 (−.66)	−2.05 (−.50)	−2.37 (−.36)	0.83 (.36)	1.10 (.37)
Past Year Alc Use	−0.003 (−.58)	−0.03 (−0.90)	0.01 (1.64)	0.01 (.47)	−0.01 (−.80)	−0.01 (−1.24)	−0.01 (−1.34)
Age	−0.80* (−2.02)	2.65 (0.85)	−0.03 (.86)	0.80 (.31)	0.90 (.71)	0.63 (1.43)	−0.11 (.84)
BDI Symptoms	−0.13 (−.50)	0.09 (.05)	−0.19 (−1.90)	0.20 (.39)	−0.25 (−.30)	0.35 (1.17)	0.30 (.43)
MJ-ADHD Interaction	0.82 (1.28)	1.84 (.37)	−0.13 (−.54)	−0.61 (−4.8)	3.54 (1.73)	−0.13 (−.18)	0.42 (.47)

Note: Results from the multiple regressions examining cannabis group, ADHD symptoms, and their interaction on predicting neuropsychological task performance. Values represent standardized regression coefficients followed by *t* statistics in parentheses.
*$p < .05$.

Discussion

We aimed to investigate whether subclinical ADHD symptoms and cannabis use independently or interactively predicted attention, working memory, and cognitive control functioning in adolescents and young adults. Cannabis users demonstrated slower response on a continuous performance attention task. ADHD symptomology did not independently predict or moderate the impact of cannabis on cognitive outcomes, nor did other potential psychological moderators such as subclinical symptoms of depression. Further, these effects remained significant when factoring in the role of gender in post-hoc analyses.

Prior research shows that cannabis users experience significant attentional deficits compared to their non-using counterparts (Hanson et al., 2010; Lisdahl & Price, 2012). Our findings showed that while our cannabis users did not significantly differ from controls across every attentional task, there were significant slowed reaction time on CPT-II performance. This slowed response is indicative of general inattentiveness (Conners & Staff, 2000) within cannabis users. Further, cannabis users did see general trends of poorer cognitive performance; even if these deficits did not reach statistical significance from their control counterparts (see Table 2). Previously we also found impaired working memory, inhibitory control and sustained attention on the Ruff 2 & 7 task in cannabis users; although our previous studies had shorter average length of abstinence and heavier recent cannabis use (Lisdahl & Price, 2012).

These subtle attentional differences in regular cannabis users were not explained by subclinical ADHD symptoms often observed in adolescents and young adults. These findings are in contradiction with previous work examining the effects of ADHD and cannabis use on attentional differences. Tamm and colleagues (2013) found that ADHD diagnosis, not cannabis use, was significantly associated with attentional deficits. However, as noted in the introduction, this research examined the impact of childhood-diagnosed ADHD. Here, we examined *current* ADHD symptoms in cannabis users without a substance-independent ADHD diagnosis. Thus, it remains possible that premorbid childhood ADHD diagnosis is associated with more significant attention deficits, compared to subclinical current ADHD symptoms.

ADHD symptoms and the interaction between cannabis use did not have significant effects on attention. Previous work has highlighted the impact of ADHD diagnosis on attentional performance, so it is surprising that ADHD symptoms had no significant differences on neurocognitive performance. It is possible that subclinical ADHD symptoms in youth are not significantly associated with laboratory tests of attention. Alternatively, previous research finding cognitive impairments in ADHD may actually be due to the impact of substance use (Balint et al., 2009; Lisdahl et al., 2016); thus, our findings highlight the need to include measures of cannabis use when looking at even subclinical symptoms of inattention.

There are limitations to the current study. Due to the cross-sectional nature of our study, we are unable to determine the timing and causality of the ADHD symptomology, cannabis use, and cognitive outcomes. Reports on ADHD symptoms were reported by parents and evaluated for current symptoms and independent, childhood ADHD diagnosis that occurred prior to cannabis use initiation was exclusionary. Thus, these findings do not generalize to samples with childhood onset ADHD. Further, youth-rating may yield differential findings. Secondly, our study incorporated a three-week abstinence of cannabis use before testing; making it possible that some participants began to experience recovery of neurocognitive functioning during abstinence (Hanson et al., 2010), indicating that these findings may not generalize to cannabis users with heavier use or shorter length of abstinence. Prospectively, longitudinal data is needed to tease apart the causation, such as the Adolescent Brain Cognitive Development (ABCD) study (https://abcdstudy.org/).

We found that cannabis users had abnormal sustained attention compared to controls, and even after controlling for parental-report of current ADHD subclinical symptoms. Research investigating subclinical ADHD symptomology should consider the impact cannabis use might play in cognitive performance. Simultaneously, more work should be done to tease apart

the subtle effects subclinical ADHD symptoms may play in cannabis users cognitive functioning. Prospectively, longitudinal data is needed to determine timing and causality of cannabis exposure, ADHD symptoms and cognitive outcomes.

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Conflict of interest

None declared.

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