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RESEARCH

No differences in inhibitory control or prefrontal activation during a cannabis cue stop-signal task across CUDIT-R-defined CUD-risk groups

Christopher R. Pickering[1] · Valentina Lorenzetti[2,3] · Andrew Jones[4] · Martin Guest[1] · Paul Christiansen[1] · Carl A. Roberts[1]

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Abstract

Rationale Cannabis use continues to increase globally, with a growing proportion of users exhibiting problematic patterns of use. Although neural differences in reward and inhibitory control systems are commonly reported in individuals who use cannabis relative to individuals not reporting cannabis use, findings using behavioural tasks are inconsistent. This is possibly due to lack of differentiation between regular and more problematic patterns of use.

Objectives This study investigated whether inhibitory control performance and associated prefrontal and orbitofrontal activation differed across CUDIT-R-stratified levels of CUD risk.

Methods Participants ($N=81$) were divided into three groups based on CUDIT-R scores: 30 high-CUD-risk individuals who use cannabis (≥ 12), 21 low-CUD-risk individuals who use cannabis (<8), and 30 individuals not reporting cannabis use. Individuals with a current substance use disorder, including cannabis use disorder, were excluded from participation. All completed a cannabis-cue specific Stop-Signal Task (SST) while functional near-infrared spectroscopy (fNIRS) was used to measure oxygenated (oxyHb) and deoxygenated haemoglobin (deoxyHb) concentrations in the prefrontal cortices. Behavioural performance and neural activation were compared across groups.

Results No significant behavioural or neural differences were found between the groups.

Conclusions The study found no evidence of impaired inhibitory control or differential prefrontal activation across CUDIT-R-stratified levels of CUD risk. The results suggest incorporating further diagnostic stratification, multimodal imaging, and ecologically valid methods in future research to better characterise cannabis-related neural adaptations and inform clinical practice.

Keywords cannabis · CUDIT-R · inhibitory control · stop-signal · cannabis use disorder · prefrontal cortex · fNIRS · cannabis cue reactivity · substance use

Introduction

Cannabis is one of the most commonly used psychoactive substances globally, with an estimated 244 million individuals regularly using in 2023 (United Nations Office on Drugs and Crime [UNODC] 2025). Recent evidence indicates that problematic cannabis use, rather than regular use alone, accounts for most of the variance in cannabis-related harms (Lorenzetti et al. 2016; Onaemo et al. 2021). This is supported by studies reporting poorer mental health, higher rates of psychiatric comorbidity, and greater cognitive and functional impairment among those with higher lifetime dose, greater frequency, higher potency, and earlier age of onset (Fergusson and Boden 2008; Figueiredo et al. 2020; Large et al. 2011; Onaemo et al. 2021; Steeger et al. 2021).

✉ Christopher R. Pickering
Christopher.Pickering@liverpool.ac.uk

[1] Department of Psychology, University of Liverpool, Eleanor Rathbone Building, Bedford Street South, Liverpool L69 7ZA, UK

[2] Neuroscience of Addiction and Mental Health Program, Healthy Brain and Mind Research Centre, School of Health and Behavioural Sciences, Faculty of Health Sciences, Australian Catholic University, Fitzroy 3065, Australia

[3] Clinical Pharmacology Unit, Research Department of Clinical, Educational & Health Psychology, University College London, London, UK

[4] School of Psychology, Faculty of Health, Liverpool John Moores University, Byrom Street, Liverpool L3 3AF, UK

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Such use patterns and adverse outcomes are strongly associated with individuals meeting criteria for cannabis use disorder (CUD) or dependence (Connor et al. 2021; Looby and Earleywine 2007; van der Pol et al. 2013). CUD, as defined in the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; American Psychiatric Association [APA]), involves persistent use despite associated harm, and approximately one-fifth of past-year individuals who use cannabis meet diagnostic criteria (Leung et al. 2020). In the present study, the Cannabis Use Disorder Identification Test–Revised (CUDIT-R; Adamson et al. 2010), was used as a validated proxy measure of problematic cannabis use, CUD risk, and dependency.

Structural and functional imaging show hippocampal and cerebellar alterations (e.g., Chye et al. 2019; Lorenzetti et al. 2020, 2024), blunted striatal dopamine responses, and reduced OFC/ACC activation in dependent individuals who use cannabis (e.g., Blanco-Hinojo et al. 2017; Pujol et al. 2014; Zehra et al. 2018; Zhou et al. 2018). Taken together, these findings suggest that cannabis-related problems are associated with distinct neural profiles.

These patterns of neuroadaptation align with some traditional models of addiction, whereby repeated substance use results in alterations to brain structure and function, impairing regulatory control (e.g., Everitt and Robbins 2016; Goldstein and Volkow 2011; Noël et al. 2006, 2013; Robinson and Berridge 2008). However, the empirical support for these mechanisms in addiction research, particularly the role of inhibitory control, remains mixed (Fascher et al. 2024).

Inhibitory control is commonly assessed with paradigms such as the Stop-Signal (SST) or Go/No-Go tasks, which require suppression of a pre-potent response (Verbruggen and Logan 2009). Despite evidence of neural differences related to inhibitory control processes in cannabis users, behavioural studies employing the SST have largely reported no significant differences in performance between chronic or 'dependent' individuals who use cannabis and controls (e.g., Filbey and Yezhuvath 2013; Quednow et al. 2007). Data from these studies were included in the mega-analysis by Liu et al. (2019), which found a significant effect for delayed Stop Signal Reaction Time (SSRT) in individuals who use cannabis associated with lifetime cannabis use. However, this was categorised as a binary variable, so it does not provide any context on problematic use, CUD status or dependency.

Findings from neuroimaging studies on a range of cognitive tasks report differences in neural activation between individuals who use cannabis and non-user controls, suggesting potential compensatory neural processes underlying comparable performance to controls (e.g., Morie and Potenza 2021 Tapert et al. 2007; Wrege et al. 2014) though there is considerable heterogeneity in results. Our previous fNIRS study across several cannabis-cue tasks found no significant group differences between regular individuals who use cannabis and controls. This may have reflected heterogeneity within the cannabis-using group, potentially masking effects specific to problematic use (Pickering et al. 2025).

To address this heterogeneity, the present study stratified participants by their CUDIT-R scores (Adamson et al. 2010). This approach enables investigation of behavioural and neural differences across different levels of CUD-risk and problematic use within a non-clinical population, rather than a binary classification of 'user' and 'non-user'.

Previous studies have often collapsed across varying levels of use severity, limiting interpretability and potentially conflating effects specific to problematic use (Fatima et al. 2019). In studies stratifying users by dependence severity or formal CUD diagnosis, more consistent and interpretable neural differences have been reported (e.g., Andriot et al. 2022; Filbey and Yezhuvath 2013; Maij et al. 2017), underscoring the importance of stratification in cannabis research. Furthermore, tasks tend to use neutral, rather than substance-related stimuli. Evidence shows that cannabis cues are associated with deficits in attentional bias task performance (O'Neill et al. 2020) and increased neural activation in dependent individuals who use cannabis and those with CUD (Sehl et al. 2021; Zhou et al. 2019) consistent with incentive-sensitisation accounts of addiction (e.g., Robinson and Berridge 2008; Everitt and Robbins 2016).

The present study therefore compared behavioural and fNIRS responses during a cannabis-cue-specific Stop-Signal Task (SST) across high- and low-CUD-risk groups and non-user controls. The hypotheses for the study were:

1. Relative to controls and the low-CUD-risk group, participants in the high-CUD-risk group were expected to exhibit significantly slower SSRTs.
2. Relative to controls and the low-CUD-risk group, participants in the high-CUD-risk group were expected to exhibit differences in haemodynamic activation in regions associated with both inhibitory control and reward processing. Specifically, differences in prefrontal cortex (PFC), in addition to significantly increased orbitofrontal cortex (OFC) activity.

Method

Participants

Participants ($n = 81$) were recruited via advertisements posted on campus notice boards and the Psychology undergraduate experimental research participation scheme (EPR).

Recruitment channels were identical for all participants. Student status was not recorded as a demographic variable. Participants were allocated to three groups: non-user controls ($n=30$), low-CUD-risk individuals who use cannabis (CUDIT-$R<8$; $n=21$), and high-CUD-risk individuals who use cannabis (CUDIT-$R\geq12$; $n=30$). All participants were aged 18 years or older. Individuals with a history of neurological or psychiatric illness or a current diagnosis of a substance use disorder (including CUD) were excluded. Exclusion was based on self-report of a current clinical diagnosis; no structured diagnostic interview was administered. Current individuals who use cannabis were defined as using cannabis at least once per month as per Sutherland et al. (2021). The study was approved by the University of Liverpool Central University Research Ethics Committee D (Ref. 14067).

Further details regarding recruitment procedures are provided in Appendix A.

Design

The study used a between-subjects design comparing three groups: high-CUD-risk individuals who use cannabis, low-CUD-risk individuals who use cannabis, and non-user controls. Differences between groups were assessed behaviourally using the SST, and neurophysiologically via average concentrations of oxyHb and deoxyHb in the PFC and OFC relative to baseline during task performance, recorded using fNIRS. In addition, CUDIT-R scores, total lifetime dose, and age of onset were used as predictors in regression models examining both inhibitory control (SSRT) and neurophysiological responses (oxyHb and deoxyHb). Additional exploratory analyses were conducted and are summarised in the results section.

Materials

Questionnaires

Background drug use questionnaire (Montgomery et al. 2005)

A background drug use questionnaire was used to examine current and historic patterns of drug use amongst participants. Following the approach of Montgomery et al. (2005), lifetime dose was estimated using a structured retrospective method combining recent use and representative months from prior years (See Appendix A for full description).

Mood adjective checklist (MAC) (Fisk and Warr 1996)

State Anxiety, Arousal, and Hedonic Tone were measured using the 18-item mood adjective checklist (MAC) developed by Fisk and Warr (1996). Participants rated their feelings at the time of testing on a 5-point Likert scale from 1 (not at all) to 5 (extremely) on 6 items related to each subscale. A high score on each subscale indicates increased anxiety, arousal, and hedonic tone. Internal consistency was acceptable for all subscales: Anxiety ($\omega_t = 0.82$), Arousal ($\omega_t = 0.78$), and Hedonic Tone ($\omega_t = 0.78$).

NASA task load index (NASA-TLX) (Hart and Staveland 1988)

Included here as a complementary subjective measure, the NASA-TLX is a multi-dimensional measure of subjective workload, consisting of six distinct dimensions: mental demand, physical demand, temporal demand, effort, frustration, and perceived performance. Each dimension is assessed with a single item rated from 0 (very low) to 100 (very high) in increments of 5. The dimensions were analysed separately, consistent with recommendations that treat each as conceptually distinct (Hart 2006; Rubio et al. 2004). As this study did not involve physical exertion, physical demand was not included in analyses.

Raven's progressive matrices (RPM) (Raven 1936)

Participants completed sets D & E (12 items in each) of Raven's Progressive Matrices (Raven 1936). RPM is a non-verbal measure of fluid intelligence in which participants are required to select - from 8 options - the missing element from a pattern (presented in 3×3 matrices). Higher scores indicate higher levels of fluid intelligence.

AUDIT-C (Bush et al. 1998)

AUDIT-C (the standard 3-item version of the Alcohol use disorders identification test [AUDIT]), measures frequency of consumption, typical units on a drinking occasion, and frequency of binge drinking (Bush et al. 1998) Sum total of the responses to these three items results in a score from 12. A total of 5 or more is a positive screen for potential harmful drinking, with higher scores representing an ascending risk of alcohol use disorder. Internal consistency indices were not calculated for this three-item scale, as reliability coefficients are not considered meaningful for very short measures (Cortina 1993; McNeish 2018).

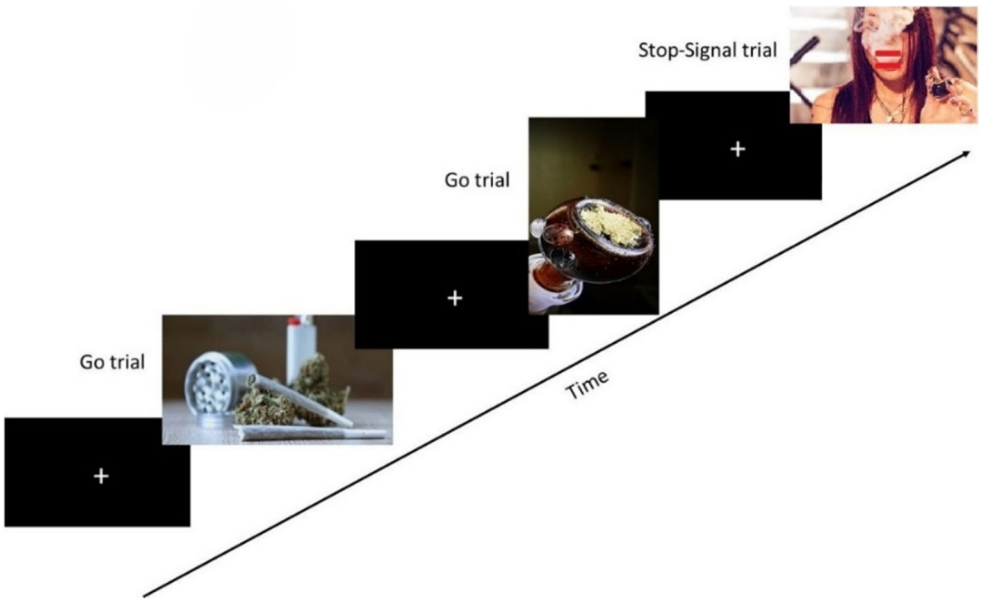

Fig. 1 Schematic diagram of the stop-signal task. *Note.* The figure illustrates a typical sequence of the Stop-Signal Task, with time progressing from left to right. Labels ('Go trial', 'Stop-Signal trial') apply directly to the image positioned to the right of each label

CUDIT-R: cannabis use disorders identification test-revised (Adamson et al. 2010)

A revised version of the CUDIT containing 8 items, two each from the domains of consumption, cannabis problems (abuse), dependency, and psychological factors. Scores of 8 points or more indicate hazardous cannabis use. Scores of 12 or more indicate possible CUD. The CUDIT-R demonstrated good internal consistency in the present sample (ω_t = 0.84).

Behavioural task

Cannabis cue - stop-signal task (Verbruggen and Logan 2009)

The task used in the experiment was based on the protocol developed by Verbruggen and Logan (2009), with a cannabis cue stimulus set developed by Macatee et al. (2021).

The task was split into three main sections: Firstly, a short practice block to familiarise participants with the task. Secondly, a priming block to establish a dominant response tendency. Thirdly, an experimental block of 200 trials, (75% 'Go' trials, 25% 'Stop-signal' trials presented in randomised order). The 200 trials were divided into 10 blocks of 20 trials, with 10-second rest intervals between blocks. Full task parameters are provided in Appendix B.

The primary outcome measure was SSRT, estimated using the integration method, in accordance with best practice guidelines (Verbruggen et al. 2019). Data from the initial practice and priming blocks were not recorded. See Fig. 1 for a schematic of the task. A split-half correlation between the first and second halves of the task indicated good internal consistency (r = .83; r_{SB} = 0.91).

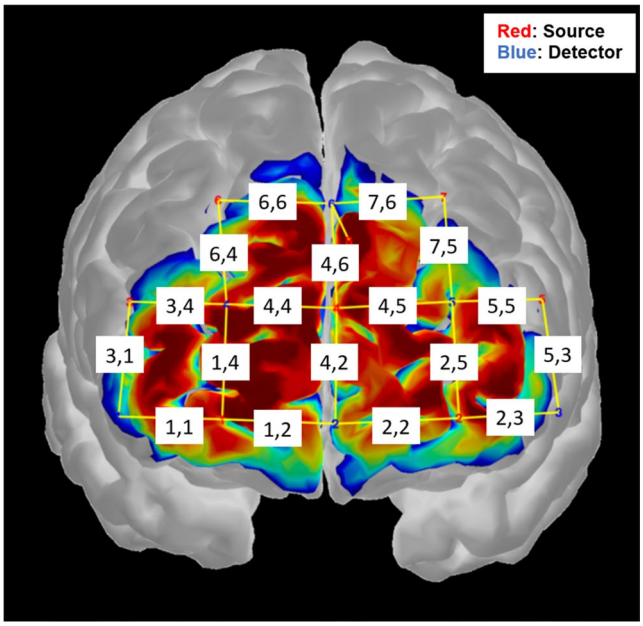

Fig. 2 fNIRS array map with channel labels

fNIRS data collection

Participants wore a flexible, anatomically registered EASYCAP head-cap (EASYCAP GmbH, Germany), with 8 source and 6 detector fibres, creating 18 functional channels. The optode array targeted multiple regions of the prefrontal cortex. Data were acquired using the NTS2 fNIRS system (Gowerlabs Ltd, London, UK). See Fig. 2 for the

optode layout and channel labelling. Full acquisition and preprocessing parameters are provided in Appendix C.

fNIRS data analysis: pre-processing

Preprocessing was conducted using Homer3 (Huppert et al. 2009) within MATLAB (The Mathworks Inc. 2022) and included channel pruning, motion correction, filtering, and conversion to relative oxyHb/deoxyHb values using the Modified Beer–Lambert Law. Full preprocessing parameters and quality control procedures are detailed in Appendix C.

Block averaging and HRF extraction

A -5 to 0 s baseline relative to block onset was used and a standardised universal HRF mean was calculated using the 5 to 20-second window post-block onset. Full robustness checks using alternative windows and first-half analyses are reported in Appendix D.

Procedure

Prior to attending the lab, potential participants completed a brief pre-screening questionnaire on XM Qualtrics to determine eligibility and group allocation. This included a cannabis-use screening item and, where applicable, the CUDIT-R (Adamson et al. 2010). Full wording of the pre-screening item is provided in Appendix A. A unique ID was created for each participant, to match their responses to their pre-screen data while retaining anonymity. Ineligible participants were screened out and their data were not recorded.

Participants attended the Brain and Behaviour Laboratory in the University of Liverpool's Department of Psychology for a single session lasting approximately 2 h. After providing informed consent, a head measurement was taken to ensure the correct sized fNIRS cap could be fitted. Participants completed the Background Drug Use Questionnaire, AUDIT-C, Mood Adjective Checklist, and sets D and E of Raven's Progressive Matrices. Participants were instructed to abstain from cannabis use on the day of testing. Self-report measures completed immediately prior to testing confirmed that no participants had consumed alcohol or other illicit substances on the day of the session.

After having the fNIRS cap fitted, participants completed the SST, whilst brain activity was recorded using fNIRS. Following task completion, participants had the cap removed, and completed the NASA-TLX, before being fully debriefed. Participants were compensated with a £20 shopping voucher or psychology undergraduate degree experimental participation points if applicable.

Table 1 Demographic, cognitive, mood, and alcohol-use characteristics by group

Measure	Non-user controls (N=30)	Low-CUD-risk (N=21)	High-CUD-risk (N=30)
Sex (F/M)	21 / 9	13 / 8	15 / 15
Age (years)	21.42 (±4.82)	24.25 (±7.92)	21.62 (±5.29)
RPM	20.03 (±2.21)	18.57 (±3.17)	19.20 (±2.60)
MAC Anxiety	12.31 (±2.29)	11.20 (±2.14)	12.00 (±2.50)
MAC Arousal	21.31 (±2.63)	21.80 (±3.03)	22.00 (±2.80)
MAC Hedonic Tone	24.03 (±2.65)	24.60 (±2.92)	23.80 (±2.40)
AUDIT-C	5.23 (±2.92)	6.33 (±2.64)	6.93 (±2.64)

Values are presented as mean (±SD) unless otherwise indicated, RPM=Raven's Progressive Matrices, MAC=Mood Adjective Checklist, AUDIT-C=Alcohol Use Disorders Identification Test - Consumption

Statistical analysis

Group differences in task performance were assessed using a one-way analysis of variance (ANOVAs), with group (high-CUD-risk individuals who use cannabis, low-CUD-risk individuals who use cannabis, and non-user controls) as the between-subjects factor and SSRT as the dependent variable.

For the fNIRS data, changes in oxyHb and deoxyHb concentrations from baseline were analysed separately for each channel using a series of ANOVAs. A partial correction of $p < .01$ was applied for multiple comparisons.

Exploratory analyses assessed whether cannabis use indices (i.e., lifetime dose, age of onset, CUDIT-R score) were associated with SSRT and differences in oxyHb and deoxyHb concentrations. To account for potential task-related slowing effects across task blocks, supplementary analyses were also conducted on the first half of the task.

Group differences in mood and affect measures were examined using one-way ANOVAs. These variables were not entered as covariates in the primary behavioural or neuroimaging analyses.

Results

Baseline demographic, cognitive, mood, and alcohol-use characteristics are summarised in Table 1.

There were no significant group differences in age, $F(2, 44.25)=1.10$, $p = .343$, $\eta^2 = 0.041$, or sex distribution, $\chi^2(2, N=81)=2.53$, $p = .282$. One-way ANOVAs, or Welch's ANOVAs where assumptions of homogeneity were violated, were used to compare groups. There were no significant group differences in RPM scores, $F(2, 78)=0.88$, $p = .417$, $\eta^2 = 0.022$. A significant difference in alcohol consumption was observed across groups on the AUDIT-C, $F(2, 49.78)=6.89$, $p = .002$, $\eta^2 = 0.163$. Bonferroni-corrected

post hoc tests indicated that high-CUD risk individuals who use cannabis reported significantly greater alcohol use than non-user controls ($p < .001$). No other between-group comparisons were significant ($ps \geq 0.05$). All groups reported average AUDIT-C scores above five, indicative of harmful alcohol use (Bush et al. 1998). There were no significant group differences in anxiety, $F(2, 44.13) = 2.54$, $p = .091$, $\eta^2 = 0.077$; hedonic tone, $F(2, 45.72) = 1.21$, $p = .308$, $\eta^2 = 0.024$; or arousal, $F(2, 78) = 1.48$, $p = .234$, $\eta^2 = 0.037$.

Cannabis use and lifetime substance exposure

Table 2 summarises core indices of cannabis use (abstinence, CUDIT-R score, age of onset, and lifetime cannabis dose) and reported lifetime use of other substances in the two cannabis user groups (low and high CUD risk). High-CUD risk individuals who use cannabis reported greater lifetime cannabis exposure and earlier age of onset. Reported lifetime use of other substances (e.g., cocaine, ecstasy, ketamine, LSD) varied across individuals and substances. Participants in the non-user control group did not report any lifetime cannabis or other illicit substance use.

Recent substance use (Past 3 Months)

Table 3 summarises the number of participants in each group who reported using the following substances within the past three months. For reporting clarity, frequency responses ('Less than monthly', 'at least once per month', 'at least once per week', and 'daily') were collapsed into a single category: 'Used in last 3 months'.

Given its known effects on cognitive and neurovascular function, reflecting evidence that nicotine may exert acute cognitive-enhancing effects, tobacco use in the sample was analysed (Valentine and Sofuoglu 2018). Daily use was reported by 15 high-CUD risk individuals who use cannabis, 4 low-CUD risk individuals who use cannabis and 2 non-user controls, suggesting that a subset of participants, especially in the high-CUD risk cannabis user group, may have used tobacco on the day of testing.

A chi-square test indicated a significant association between group and smoking status, $\chi^2(2, N = 81) = 38.68$, $p < .001$. Significantly more high-CUD risk individuals who use cannabis were classified as current smokers (i.e., reported tobacco use in the past three months) compared to controls. A follow-up comparison showed that low-CUD risk individuals who use cannabis were also significantly more likely to report recent tobacco use than controls, $\chi^2(1, N = 51) = 5.81$, $p = .016$. However, the difference between high- and low-CUD-risk individuals who use cannabis was not statistically significant.

Table 2 Indices of cannabis use and lifetime dose of other substances within cannabis user groups (values are Mean ± SD)

	Low CUD risk cannabis user	High CUD risk cannabis user
Cannabis		
Abstinence (Days)	2.62 (±1.98)	1.75 (1.60)
CUDIT-R Score	5.10 (±1.99)	17.07(±3.453)
Age of Onset (Years)	17.09 (±2.02)	16.33 (±1.82)
Lifetime Dose (Total SJUs)	769.67 (±1762.50)	3734.98 (±6384.16)
Lifetime Doses		
Cocaine (grams)	76.87 (±119.18), *n*=7	44.92 (±135.58), *n*=18
Ecstasy (miligrams)	8687.50 (±12278.98), *n*=6	20925.00.00 (±66090.20), *n*=14
Ketamine (grams)		37.90 (±47.92), *n*=5
LSD (Tabs)	52.00, *n*=1	5709, *n*=1

ns vary by substance, as not all participants reported lifetime use of other drugs. Control participants reported no lifetime cannabis or illicit substance use

Table 3 Recent drug use (Within Last 3 Months) by group

Substance	Non-user control	Low-CUD-risk	High-CUD-risk
Alcohol	26	21	28
Amphetamine	0	0	1
Cocaine	1	6	15
Ecstasy	0	3	5
Ketamine	1	2	9
LSD	0	0	3
Mushrooms (Psilocybin)	0	4	10
Poppers (Alkyl nitrites)	1	4	5
Tobacco	7	15	25

In addition to the summary data in Tables 2 and 3, Appendix E provides detailed substance use statistics, including abstinence and last-use dose, 30-day quantity estimates, and full frequency distributions, allowing finer-grained inspection of dosage patterns and substance-specific variability across groups.

Hypothesis 1: Inhibitory control deficits in high-CUD-risk users

It was hypothesised that, relative to controls and low-CUD-risk users, participants in the high-CUD-risk group would exhibit significantly slower SSRTs, indicative of impaired inhibitory control.

The assumption of homogeneity of variances was met, $p = .226$. The ANOVA revealed no statistically significant differences in SSRT across groups, $F(2, 78) = 0.665$, $p = .517$, $\eta^2 = 0.017$. Group means and dispersion are presented in Table 4.

Table 4 Mean stop-signal reaction times (SSRT) by group

Group	Mean (ms)
Non-user control	276.27 ± 149.01
Low-CUD-risk	243.19 ± 60.54
High-CUD-risk	265.27 ± 53.47

The high standard deviation in the control group was driven by one participant with a mean SSRT exceeding 900 ms. A sensitivity analysis excluding this participant yielded comparable results, so they were retained in the main analysis

Table 5 Mean oxyHb concentration changes (μmol/L) across groups and channels

Channel	Non-user control	Low-CUD-risk	High-CUD-risk
1,1	0.027 (±0.189)	0.050 (±0.200)	−0.003 (±0.214)
1,2	−0.039 (±0.232)	0.009 (±0.210)	−0.037 (±0.217)
1,4	0.027 (±0.291)	−0.012 (±0.201)	−0.009 (±0.171)
2,2	0.006 (±0.601)	−0.030 (±0.184)	−0.037 (±0.169)
2,3	−0.007 (±0.326)	0.048 (±0.213)	−0.025 (±0.235)
2,5	0.054 (±0.387)	−0.016 (±0.136)	−0.016 (±0.143)
3,1	0.061 (±0.257)	0.007 (±0.231)	0.035 (±0.194)
3,4	0.007 (±0.219)	0.025 (±0.225)	0.022 (±0.188)
4,2	0.018 (±0.283)	0.004 (±0.156)	0.022 (±0.180)
4,4	−0.015 (±0.215)	0.018 (±0.199)	−0.004 (±0.211)
4,5	−0.033 (±0.215)	0.013 (±0.168)	−0.002 (±0.199)
4,6	0.051 (±0.214)	0.035 (±0.208)	0.037 (±0.215)
5,3	0.021 (±0.178)	0.031 (±0.207)	0.001 (±0.184)
5,5	0.023 (±0.280)	0.060 (±0.299)	−0.028 (±0.233)
6,4	0.010 (±0.168)	0.003 (±0.153)	0.023 (±0.158)
6,6	−0.001 (±0.201)	−0.017 (±0.170)	0.043 (±0.169)
7,5	0.011 (±0.213)	0.046 (±0.197)	0.014 (±0.143)
7,6	0.005 (±0.264)	0.002 (±0.180)	0.036 (±0.232)

Exploratory analyses confirmed a universal slowing effect across task blocks, with no group-specific differences. Supplementary analyses restricted to the first half of the task (Blocks 1–5) yielded comparable null results (see Appendix D).

NASA-TLX

One-way ANOVAs revealed no significant group differences across any of the NASA-TLX subscales (all *ps* > 0.05) indicating that subjective task demands were similar between groups. Mean ratings for each subscale are reported in Appendix F.

Hypothesis 2: Differential prefrontal and orbitofrontal activity in high-CUD-risk users

It was hypothesised that, relative to controls and low-CUD-risk individuals who use cannabis, participants in the high-CUD-risk group would exhibit differences in blood oxygenation in PFC regions associated with cognitive control, as well as increased activation in OFC regions implicated in reward processing.

Table 6 Mean deoxyHb concentration changes (μmol/L) across groups and channels

Channel	Control	Low-CUD-risk	High-CUD-risk
1,1	.017 (±.087)	.010 (±.079)	.013 (±.077)
1,2	.025 (±.058)	.020 (±.061)	.019 (±.049)
1,4	.018 (±.060)	.011 (±.051)	.004 (±.042)
2,2	.019 (±.150)	.025 (±.051)	.019 (±.052)
2,3	.012 (±.118)	.006 (±.055)	.019 (±.081)
2,5	.011 (±.117)	.017 (±.047)	.019 (±.043)
3,1	.005 (±.102)	.009 (±.062)	-.024 (±.113)
3,4	.011 (±.065)	.008 (±.036)	-.005 (±.091)
4,2	.020 (±.063)	.024 (±.053)	.016 (±.073)
4,4	.017 (±.071)	-.001 (±.067)	.019 (±.062)
4,5	.038 (±.082)	.009 (±.045)	.006 (±.053)
4,6	-.005 (±.083)	.005 (±.051)	.013 (±.075)
5,3	.008 (±.056)	-.020 (±.071)	-.005 (±.123)
5,5	.010 (±.066)	.010 (±.077)	.027 (±.139)
6,4	.021 (±.046)	.015 (±.036)	.019 (±.051)
6,6	.020 (±.064)	.026 (±.052)	-.011 (±.063)
7,5	.025 (±.052)	.008 (±.034)	.010 (±.043)
7,6	.004 (±.112)	.034 (±.057)	-.007 (±.079)

No statistically significant group differences were observed on any channel (all *ps* > 0.01). Assumptions of homogeneity of variance were assessed using Levene's test; although one channel showed violations, results from Welch's test were non-significant, consistent with the standard ANOVA. Effect sizes were uniformly small (all $\eta^2 < 0.03$), indicating minimal group-level variation. Descriptive statistics of mean concentration changes in oxyHb and deoxyHb for each group by channel are reported in Tables 5 and 6. For full ANOVA summaries, see Appendix D.

When collapsing across groups, the Stop-Signal Task elicited significant task-related haemodynamic responses in several prefrontal channels, both across the full task and when analyses were restricted to the first five blocks (see Appendix D).

To assess the robustness of the between-group results across different HRF extraction windows, additional ANOVAs were conducted using 0–20 s and 0–25 s windows. No significant group differences were observed (all ps > 0.01). Exploratory regression models including cannabis use indices (i.e., lifetime dose, age of onset, and CUDIT-R score) as predictors of SSRT and oxyHb/deoxyHb concentration changes likewise yielded no significant effects (see Appendix D).

Discussion

The current study comprehensively assessed behavioural performance and OFC/PFC activation patterns using fNIRS across individuals who use cannabis with high- and low-CUD-risk scores, and individuals not reporting cannabis

use. No significant differences were found in task performance or oxyHb/deoxyHb responses across any assessed channels. Supplementary analyses across a range of data extraction parameters confirmed these null results.

These findings are contrary to our hypotheses, which were based on prior studies suggesting that more problematic cannabis use (e.g., presence vs. absence of CUD, higher vs. lower dependence severity indicated by higher CUDIT-R scores) may result in distinct neural differences compared to lower-risk users and controls (e.g. Filbey and Yezhuvath 2013; Morie and Potenza 2021; Tapert et al. 2007; Wrege et al. 2014), or increased OFC activation due to the use of cannabis cues (Sehl et al. 2021; Zhou et al. 2019). Notably, Liu et al. (2019) reported delayed SSRT in relation to lifetime cannabis exposure in a mega-analysis, but cannabis use was operationalised as a binary exposure indicator, limiting inference about problematic use or CUD-risk. In contrast, the present study tested group differences using a priori CUDIT-R score stratification. To the authors' knowledge, this is the first study to examine this paradigm using this stratification.

Despite stratifying participants by CUDIT-R score, the present study did not detect statistically significant group differences under the current methodological conditions. These findings should be interpreted cautiously and do not preclude the possibility of cortical differences in other samples or under other task conditions. As such, it remains unclear whether the present study's selected cutoff (CUDIT-R score $\geq$ 12) sufficiently distinguishes between subsets of individuals who use cannabis at the neurobiological level. It is possible that such differences emerge only at higher severity thresholds of CUD (i.e., dependency) or may only manifest in subcortical structures beyond the spatial resolution of fNIRS.

Emerging evidence suggests that subcortical brain regions, rather than cortical structures, may be more sensitive to cannabis-related changes in problematic use. These include the hippocampus (Lorenzetti et al. 2024; Solowij et al. 2013; Wang et al. 2021) and other subcortical reward-related regions (e.g., caudate, nucleus accumbens, amygdala) among dependent individuals who use cannabis (Chye et al. 2017; Filbey et al. 2009, 2014; Lorenzetti et al. 2020; Zhou et al. 2018). These findings emphasise subcortical neural differences which are not readily detected by cortical imaging techniques such as fNIRS.

However, not all brain imaging studies relating to dependence have reported consistent findings. For example, Cousijn et al. (2022) found no association between cannabis dependence severity and white matter microstructure, suggesting that some neurobiological changes may not be universal across all dependent users. This suggests that dependence-related neural differences may vary in magnitude, regional specificity, or only emerge in clinical populations. Our findings may therefore reflect either subtle or absent effects at the cortical level in this subclinical sample, or a threshold effect not captured by CUDIT-R screening alone. Future work might benefit from combining fNIRS with complementary neuroimaging techniques, such as fMRI, offering a more comprehensive understanding of both cortical and subcortical mechanisms underlying cannabis-related cognitive function.

Another explanation is that laboratory-based inhibitory tasks do not adequately reflect the motivational and emotional pressures under which self-regulation tends to fail in everyday life. Research on hot and cold executive functions (Metcalfe and Mischel 1999; Zelazo and Carlson 2012) suggests that inhibitory control may operate differently in affectively neutral ('cold') versus emotionally charged or motivationally salient ('hot') contexts. Inhibitory control deficits in individuals who use cannabis may therefore be context-dependent, emerging primarily in situations involving reward or affective cues (e.g., Field and Cox 2008; Franken 2003; Griffith-Lendering et al. 2012). The laboratory setting used here was clinical and low-risk and may not have elicited meaningful conflict between inhibitory and reward-related processes.

Previous work has highlighted a general disconnect between laboratory task performance and real-world behaviour (Eisenberg et al. 2019), particularly in substance use, where decision-making impairments often manifest in emotionally or socially charged situations (Ekhtiari et al. 2017). Supporting this, Zech et al. (2023) found that smartphone-based inhibitory control tasks administered in participants' natural environments were more sensitive to alcohol use patterns than traditional lab-based measures, underscoring the importance of ecological validity in substance use research (see also; Jones et al. 2018). Future studies should therefore incorporate ecologically valid, real-world assessment methods to capture context-specific self-regulation processes that may not be evident in laboratory settings.

Participants were drawn from a non-clinical sample; therefore, CUDIT-R scores function as a proxy for CUD-risk, rather than diagnostic confirmation of CUD. Moreover, while the CUDIT-R is a well-validated screening tool, it does not confer a clinical diagnosis. As such, heterogeneity in symptom severity and cannabis-related impairment may persist within both cannabis user groups (e.g., some individuals scoring $<$ 8 may meet diagnostic criteria for CUD, while others scoring $\geq$ 12 may not). Further stratification of individuals who use cannabis based on usage characteristics, (e.g., clinical diagnosis, SCID outcomes, symptom severity), may help to reveal subgroup-specific differences in cue-specific inhibitory control that were not evident in the present sample.

Theoretically, the results do not allow strong inferences about the association with cannabis use and impaired reward and inhibitory control processes in the same way, or to the same extent, as substances such as alcohol or cocaine (see Smith et al. 2014). While classic addiction models emphasise neural differences in reward and executive control networks in problematic substance users (e.g., Everitt and Robbins 2016; Goldstein and Volkow 2011; Noël et al. 2006, 2013), the current findings support a more nuanced interpretation in individuals who use cannabis, in which neurocognitive changes may be subtler, more variable, and context dependent.

There are several methodological limitations that should be considered. First, the sample reflected CUDIT-R-stratified patterns of cannabis involvement within a non-clinical cohort in which alcohol, tobacco, and other substance use were present. Substance-specific attribution of the behavioural or haemodynamic findings therefore cannot be made. However, co-use of multiple substances is common among individuals who use cannabis in non-clinical populations and may therefore reflect ecologically valid patterns of use rather than an atypical sample composition (Rosen et al. 2018).

A the use of a universal HRF extraction window may have introduced inconsistencies in timing, leading to data loss or misalignment. Although supplementary analyses using alternative windows were consistent with the main results, a custom-coded approach that models each participant's block durations individually may enhance sensitivity. Thirdly, recent substance use was not objectively verified using biological specimens (e.g., urine toxicology). However, agreement between self-report and biological metrics is typically high, confirming reliability of self-report metrics (Bharat et al. 2023; Large et al. 2012).

Large-scale evidence suggests that behavioural differences in SSRT associated with cannabis use are small in magnitude (Liu et al. 2019). Detecting effects of this size would require substantially larger samples than are typically feasible for task-based neuroimaging studies. Therefore, the current sample may have been underpowered to detect behavioural differences. As such, future work aiming to detect subtle behavioural differences may benefit from large-scale or multi-site designs, or from alternative inhibitory control paradigms.

While larger samples may be required to detect subtle behavioural differences, the present sample size was comparable to, or larger than, previous fNIRS studies which have successfully identified neurophysiological differences in substance users (e.g., Roberts and Montgomery 2015a, b; Montgomery et al. 2017). Lastly, although tobacco use differed between groups and some participants may have used nicotine on the day of testing, nicotine exposure was not experimentally controlled and its effects on haemodynamic responses cannot be ruled out. Future work may therefore benefit from explicitly accounting for nicotine use in analyses.

In summary, this study found no statistically significant differences in behavioural performance or cortical haemodynamic responses between individuals scoring in the higher CUD-risk range (CUDIT-R score $\geq$ 12), those scoring in the low CUD-risk range (CUDIT-R score $<$ 8), and individuals not reporting cannabis use. These findings should be interpreted cautiously given sample characteristics, co-use patterns, and reliance on self-report measures. Future research may benefit from examining clinical or treatment-seeking populations, or from employing more sensitive behavioural and neuroimaging paradigms.

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Author contributions C.P., C.R., and P.C. conceptualised and designed the study. M.G. assisted with task programming. C.P. collected and analysed the data, prepared all tables and figures, and drafted the manuscript. P.C., V.L., and A.J. contributed to the interpretation of data and provided critical feedback and editorial input. C.R. (primary supervisor) and P.C. (secondary supervisor) supervised the project. All authors reviewed and approved the final version of the manuscript.

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Data availability The anonymised datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate This study was approved by the University of Liverpool Central University Research Ethics Committee D (Ref: 14067). All procedures were conducted in accordance with the Declaration of Helsinki and institutional guidelines. All participants were aged 18 years or over and provided written informed consent prior to participation.

Consent to participate All participants provided written informed consent to take part in this study.

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