Neuroscience and Biobehavioral Reviews 172 (2025) 106092

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Identifying EEG markers related to acute cannabis consumption: A systematic review

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ARTICLE INFO

Keywords:
Cannabis
THC
EEG
Neurocognition

ABSTRACT

Background/rationale: Electroencephalography (EEG) has potential to provide a sensitive measure of the acute neurophysiological response to cannabis administration. As delta-9-tetrahydrocannabinol (THC; the psychoactive constituent of cannabis) can induce transient neurocognitive impairments that differ as a function of tolerance and dose, understanding the neural profile related to intoxication would be of great benefit in the wake of increasing recreational and medicinal use. Accordingly, the present systematic review examined the current research literature related to acute cannabis administration and EEG measures.
Methods: Peer-reviewed articles published from 2000 were assessed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Studies that administered non-synthetic cannabis, containing THC (orally or inhaled) and reported differences in EEG outcomes within the acute time frame (<6 hours post-administration) as compared to baseline or placebo, were eligible for inclusion.
Results/discussion: A total of 16 studies were eligible for inclusion, of which 11 reported differences in the amplitude/latency of event-related potentials (ERPs) and 9 reported changes in frequency band power. Of the ERPs, the P3 was identified as a potential indicator of recent cannabis consumption, as demonstrated by decreased P3 amplitude across various doses (generally exhibiting small-to-moderate magnitude effects where effect sizes were reported). Oscillatory activity in the theta frequency band power range (typically 4–7 Hz) was impacted following cannabis administration, with some support for a dose-dependent change in power. The present results highlight the potential utility of some EEG measures as markers of recent cannabis consumption, although great heterogeneity in participant characteristics and reported data limits conclusions from these results. It is also evident that EEG changes in highly tolerant user groups (such as those who use cannabis medicinally), require further exploration.

The increasing global acceptance of cannabis use (both medicinal and recreational) has led to a need to further understand how this substance might affect daily functions, especially in relation to safety-sensitive tasks. Delta-9-tetrahydrocannabinol (THC), the key psychoactive component of cannabis (Lafaye et al., 2017), interacts as an agonist within the human endocannabinoid system, disrupting typical functioning in key cortical networks (Pertwee, 2008; Ramaekers et al., 2021). There is significant variability in the behavioural effects of THC across individuals and cognitive domains, adding uncertainty in the assessment of these effects (Arkell et al., 2022; Bossong et al., 2012; Dellazizzo et al., 2022; Desrosiers et al., 2015). Electroencephalography (EEG) based assessment has the potential to avoid variability in behavioural responses by directly assessing psychophysiological responses at the neural level to further clarify the complex effects of cannabis. EEG assessments enable evaluation of brain activity during completion of structured cognitive tasks designed to target specific skills and processes while responding to visual or auditory stimuli. With medicinal cannabis prescription rates also steadily growing in Australia (MacPhail et al., 2022), there is a critical need to identify biomarkers of recent cannabis consumption, which may shed light on the neural functions most impacted by this psychoactive substance.

Cannabis (containing THC) is known to influence neurocognitive function, potentially impacting functions and skills necessary for daily activities (Dellazizzo et al., 2022; McCartney et al., 2021). However, attempts to accurately identify and assess the neural effects of cannabis have proven challenging. For example, blood/oral fluid samples only

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https://doi.org/10.1016/j.neubiorev.2025.106092
Received 11 September 2024; Received in revised form 26 February 2025; Accepted 5 March 2025
Available online 6 March 2025

identify the presence of THC in the circulatory system, not the recency of use, THC dose or quantity of cannabis consumed (Arkell et al., 2021; McCartney et al., 2022). In addition, findings on behavioural and cognitive outcomes arising from cannabis consumption are variable (Dellazizzo et al., 2022; Hart et al., 2001; Morgan et al., 2018; Ramaekers et al., 2009; Schwope et al., 2012). This variability may reflect the impact of factors including tolerance and dose, which are known to moderate the individual response to THC (Ramaekers et al., 2020, 2021). For example, those who frequently use cannabis (e.g., medically) or consume low doses may demonstrate little or no impairment to functions typically affected by THC (Olla et al., 2021; Pelletti et al., 2021; Ramaekers et al., 2020; Schwope et al., 2012; Wallace et al., 2015). However, behavioural cognitive tasks used to examine these effects may be limited in their sensitivity to detect changes of a more subtle nature, suggesting that techniques such as EEG may be more appropriate for assessing these effects.

A key strength of EEG is the high temporal resolution of measurement, allowing for precision when it comes to the timing and tracking of activity across brain regions (Luck, 2014). EEG is a non-invasive technique, with demonstrable utility for the understanding of the neurophysiology of drug dynamics, as seen with common prescription (and non-prescription) substances (Bewernitz and Derendorf, 2012; Lansbergen et al., 2011). While brain activity can be assessed at rest (e.g., relaxing with eyes closed), specific stimuli can be used to assess various aspects and stages of cognitive processing and responding. For example, studies have investigated how cannabis might influence common EEG components such as event-related potentials (ERPs). These can provide time-locked information related to cognitive task performance (e.g., reaction time, accuracy, errors), with some findings revealing an influence of cannabis on neural activity, even in the absence of observable behavioural changes (Hart et al., 2010; Richard et al., 2021; Roser et al., 2008). This suggests that cannabis may exert subtle influences on early cognitive processes that cannot be captured by common cognitive assessments. However, the present research literature is highly variable in controlling for potential confounds including participant characteristics, cannabis administration routes and doses administered. This variability in research design and methodology creates significant barriers to determining a clear neurocognitive profile of cannabis-related impairment. To date, this literature has yet to be systematically reviewed, making it difficult to delineate between these factors.

The current review synthesises research examining electrophysiological outcomes following acute oral or inhaled cannabis administration, to summarise the present understanding of the neural profile related to recent cannabis consumption. By examining changes in EEG waveform following THC administration, there is opportunity to further clarify current understanding of the specific brain response to cannabis.

1. Method

The Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) guidelines were followed in this review. See Supplementary file (Appendix A) for full details of the search protocol. Citations were screened by CS and a research assistant at each stage, with the final selection completed by CS. KS and MS provided advice when required. Ethics approval was not required for this review as no participants were recruited.

1.1. Search strategy & selection criteria

A research assistant independently completed searches of PubMed, ProQuest, APA PsycINFO and Scopus in July 2022. Searches were restricted by title, abstract, English language, peer-review status and scholarly journals, where relevant (see S1 for further information). The presented search strategy also resulted in a separate review of the effects of acute cannabis administration on standardised neuropsychological test measures (see Stefanidis et al., 2024). Both reviews followed the same protocol, with differences emerging only at the full-text screening stage. As such, while the primary search terms presented here encapsulated all articles related to cannabis, cognitive function and EEG, this review focussed only on those that involved an EEG assessment.

The search keywords are as follows:

1. *THC, tetrahydrocannabinol, cannabi* ,* or *mari*uana*

2. *cognit*, neuropsycholog* , electroencephalogra*, EEG, event-related potential,* or *ERP*

Upon completion of the database searches, citations were screened in Endnote to identify and remove duplicates. After duplicate removal, a total of 7861 citations were imported into Rayyan for title and abstract review (Ouzzani et al., 2016). All articles were screened by both CS and a research assistant, first by title and then by abstract to determine their relevance. Articles published before the year 2000 were deemed ineligible for inclusion in this review. Systematic and meta-analytic reviews pertaining to cannabis (containing THC) and cognition/EEG were flagged to allow for later reference list screening to ensure comprehensiveness of the search strategy. This search resulted in a total of 84 relevant full-text articles (including those articles related to cognitive assessment). Full-text screening of EEG-related articles was completed by CS.

Synthetic THC (e.g., dronabinol, nabilone) is delivered as an isolate, lacking the whole phytocannabinoid profile that is found in cannabis (Greydanus et al., 2015; Roque-Bravo et al., 2023; Russo, 2011; Sholler et al., 2020). While not the focus of this review, such compounds found in the cannabis plant (e.g., cannabidiol) may affect psychoactive outcomes (Freeman et al., 2019). As such, considering the sensitivity of EEG, the present review excluded synthetic THC to reduce heterogeneity introduced by potentially differing pharmacological effects, in order to focus on the effects of cannabis. Studies were included on the basis that they examined EEG outcomes after oral or inhaled administration of cannabis (cannabis flower or derived from cannabis). Studies using intravenous methods of administration were excluded.

As such, primary eligibility criteria for this review were defined as follows: (1) examined change or difference in EEG measures following acute non-synthetic cannabis administration via inhaled or orally administered routes (<4 hours post-administration); (2) participants aged over 18 years (both healthy and clinical samples); (3) Cannabis was administered in isolation or with placebo (participants usual medication and tobacco were accepted); and, (4) a clearly defined task paradigm or resting state measure (i.e., eyes closed, eyes opened) was utilised. Studies were excluded where the type of task/paradigm used, or the timing of EEG acquisition and task performance could not be determined from available (or requested) information. For each neurophysiological measure, data from at least two independent studies was required for inclusion in this review. As such, components that had only one data point available, were excluded.

An updated search was conducted in February 2024 by CS, aiming to identify all relevant articles published after 2022 (as part of a wider review concerning cannabis and neurocognition). As such, PubMed, ProQuest, APA PsycINFO and Scopus were searched using the predefined strategy outlined above. This updated search resulted in the identification of 2412 citations. After removing duplicates in Endnote, 1695 citations remained for title and abstract screening according to relevance to cannabis and EEG. As a result, eight full-text articles were screened, with CS identifying two additional studies for inclusion.

1.2. Risk of bias assessment

To assess methodological quality and address potential sources of bias, key criteria were addressed for each included study (adapted from Higgins et al., 2011), as outlined in the protocol (Appendix A). Author CS screened articles for the following: (1) EEG analysis; (2) cannabis

administration; (3) performance indicators; (4) reporting; and (5) other unspecified bias. The criteria were each given a rating of 'low risk', 'moderate risk', or 'high risk' (see Section 7 of protocol, Appendix A), with KS reviewing each rating. Any conflicts were resolved through discussion.

1.3. Data extraction

All information relating to sample characteristics, cannabis use history criteria, cannabis intervention (and timing), task paradigm, task performance and EEG data was extracted (where available) from each included article. Where sufficient information was available for the calculation of effect sizes (Cohen's *d*), G*Power was used (version 3.1.9.7, Dusseldorf University). The magnitude of effect was interpreted as small, medium or large, relating to cut-offs of.2,.5 and.8, respectively (Cohen, 1988). Outcomes relating to significance were provided where effect sizes could not be calculated (e.g., data not provided in article or upon request).

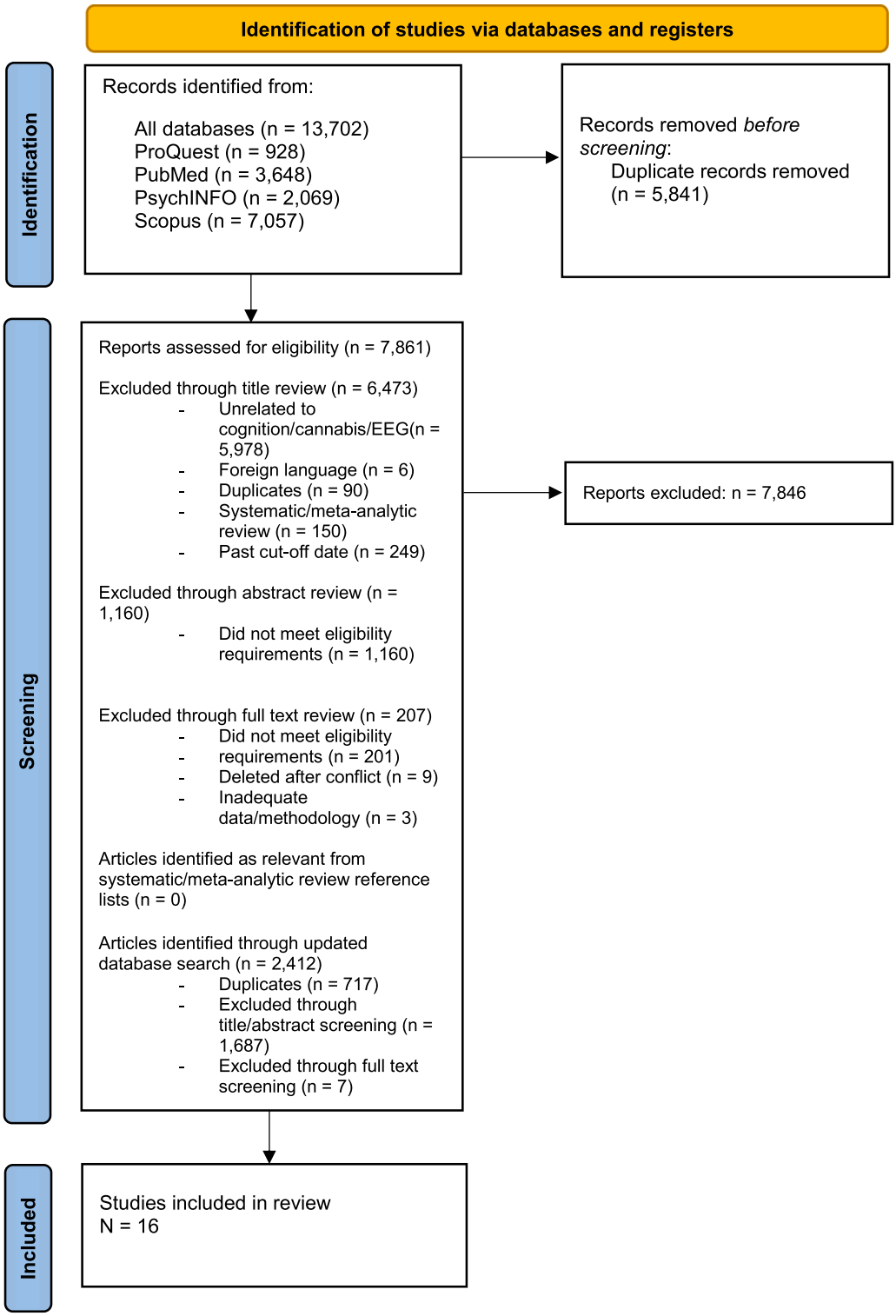

Fig. 1. Search strategy.

1.4. Data analysis

After extraction, it became apparent that most of the studies identified reported insufficient data to enable effect size calculation (authors with valid correspondence details were contacted to collect additional data for effect size calculation). Due to the limited data, meta-analysis was not feasible throughout this review, leading to a qualitative summary of findings for each measure. While three studies provided data that may have been eligible for meta-analysis (for the P3 component only), it was deemed inappropriate to summarise this data quantitatively. This was due to heterogeneity in task paradigms, timing of task completion and electrode sites analysed across these studies. Where effect sizes were available, they have been discussed in the context of the results and the statistical significance of the findings.

Across the literature identified, studies primarily focussed on event-related potentials and spectral power analyses when assessing changes following cannabis consumption. As such, this review focused on ERPs and frequency band data. ERPs were examined in terms of changes to latency and/or amplitude after cannabis administration. Modulations to each frequency band following cannabis consumption were noted in terms of increases/decreases in power. While connectivity analyses (e.g., coherence, mutual information) were also identified, there was insufficient data (i.e., data from more than two independent studies) for inclusion.

2. Results

After full-text and reference list screening, a final sample of 16 studies examined neural changes in response to cannabis consumption via EEG remained (see Fig. 1). Of these studies, 11 examined ERPs, with 8 components identified having sufficient data for inclusion. Another 9 studies provided information pertaining to frequency band alterations. Three studies that examined the acute effects of cannabis administration on EEG outcomes were excluded on the basis that they reported isolated components/analysis methods not reported elsewhere, resulting in insufficient data for synthesis. Extracted participant and study characteristic data of the final sample are presented in Table 1. Table 2 provides a summary and description of the included EEG components.

3. Event-related potentials

3.1. ERN

ERN amplitude appears to be negatively impacted by higher doses of cannabis. Although there was limited data to examine this component (Table 3), Kowal et al. (2015) found that the high dose group demonstrated a large magnitude reduction in amplitude. All three studies recorded ERN amplitude while participants completed a task requiring the recruitment of attention and inhibitory control skills. A significant interaction effect reported by Spronk et al. (2011) highlights the diminished ERN response from placebo to post-administration of cannabis. Spronk et al. (2016b) also administered a 'booster' dose, in which participants received an additional dose of 0.15 mg/kg THC (e.g., 11.25 mg THC for a weight of 75 kg), which may result in a total dose of THC in line with Kowal et al.'s (2015) high dose condition.

3.2. P1

There is insufficient data to determine a clear influence of cannabis on the P1 (Table 4). While Spronk et al.'s (2016b) found no effect of cannabis on P1 amplitude, Theunissen et al. (2012) found a significant decrease in P1 amplitude in their occasional user group (with a slightly longer analysis window) associated with moderate to large effect sizes. While the change was not significant, the heavy user group demonstrated a large magnitude increase in P1 amplitude to target stimuli, suggesting this component might be impacted in more experienced cannabis users, specifically for salient stimuli (Theunissen et al., 2012). There was a trend towards amplitude increases in experienced cannabis users while a decrease in amplitude was observed in those who did not use cannabis often.

3.3. N1

Overall, cannabis did not reliably affect N1 amplitude or latency. While Ilan et al. reported a decrease in N1 amplitude, this was the only study to report such a difference (see Table 5). These discrepancies could be explained by the tasks themselves, with Ilan et al. (2004) examining memory, and the remaining studies focusing on inhibitory control. Adding to this Ilan et al.'s (2004) timing bracket for this component began later than all others reported in Table 4. Ilan et al.'s (2004) results also suggest that cannabis did not impact the speed of response, as seen through the non-significant change to latency, but there was no other data identified in the other studies to corroborate this.

3.4. P2

There is limited and inconsistent evidence of an influence of cannabis on the P2 component. Only two studies examined the P2 component, with one (Ilan et al., 2004) reporting a significant decrease in P2 amplitude post-cannabis flower smoking (Table 6). In contrast, Richard et al. (2021) did not identify a significant change in P2 amplitude following a comparatively high dose of vaporised THC. However, the task used involved sustained attention rather than the memory-based task used by Ilan et al. (2004). Ilan et al. (2004) also found that the latency of the P2 was not affected by cannabis smoking. Both studies involved small samples (<15) of varied cannabis use history, including frequent and infrequent users.

3.5. N2

Seven studies analysed the N2 component (see Table 7), with mixed findings. Of these, only one reported data for the calculation of effect sizes, with Theunissen et al. (2012) reporting non-significant small to moderate size effects, suggesting cannabis' effects on the N2 may not be of practical relevance, or that the study may have been under-powered (N = 24). Two other studies reported a significant effect of cannabis administration on N2 amplitude, with one showing a decrease and the other an increase in N2 amplitude. This might be attributed to differences in the cognitive paradigm utilised, with Ilan et al. (2005) using an episodic memory task, whereas Spronk et al. (2016b) used an inhibitory control task. Cannabis may differentially affect the recruitment of these skills and consequently the ERP response. These samples also differed markedly in cannabis use history, although when analysing the influence of previous cannabis exposure, Theunissen et al. (2012) found no consistent difference in effect on the N2. Across the remaining studies, there was no effect of cannabis consumption on N2 latency or amplitude.

3.6. P3

Collectively, cannabis administration appeared to reduce P3 amplitude when compared against either baseline or to a placebo condition. Most studies ($n = 8$, 80 %) demonstrated a reduction in P3 amplitude after cannabis administration, with the remaining two studies reporting no significant change. Cannabis consistently resulted in a typically medium magnitude decline in P3 amplitude, with Theunissen et al. (2012) reporting the only large effect size in their occasional user group. It appears that the P3 amplitude is negatively impacted across different doses, independent of cannabis use history. Of those studies reporting latency data, results were mixed, with non-significant small and moderate effect sizes. Spronk et al. (2016a), (2016b) observed a significant increase in P3 latency after cannabis administration (with a moderate

Table 1
Study characteristics.

Study	Design	Total sample	Characteristics	Cannabis use history criteria	Cannabis intervention	Stimulus/task paradigm	EEG
Bocker et al. (2010a)	Within-subjects Randomised, double-blind, placebo-controlled crossover	N = 23 (Split sample, n = 11 lower half, n = 12 upper half)	M (SD) age total sample = 24 (4)	2–18 cannabis cigarettes per month	Cannabis flower cigarette (with tobacco): 29.3 mg THC, 49.2 mg THC & 69.4 mg THC Smoked in 22 mins	Visual Selective Attention Task; 2hrs post consumption	N2b, P3
Bocker et al. (2010b)	Within-subjects Randomised, double-blind, placebo-controlled crossover	N = 16 males	N.R	2–9 cannabis cigarettes per month	Cannabis flower cigarette (with tobacco): 29.3 mg THC, 49.1 mg THC & 69.4 mg THC Smoked in 22 mins	RSEC, General Attention Task; 150 mins post onset of cannabis intake.	Delta, Theta, Alpha, Beta
Brown et al. (2019)	Within-subjects Randomised, placebo-controlled crossover	N = 11	N.R	> 1 exposure in 3-month period but < 4 x per week	Cannabis flower (6.7 % THC) 500 mg by vaporisation (approximately 33.5 mg THC). Inhaled over 10 mins	RSEO, RSEC, Three Choice Vigilance, Standard Image Recognition, Verbal Memory Scan, modified TMT, 45 min driving simulator task; 30 mins post consumption.	LPP, Theta
Brown et al. (2020)	Within-subjects Randomised, placebo-controlled crossover	N = 10	N.R	N.R	Cannabis flower (6.7 % THC) 500 mg by vaporisation (approximately 33.5 mg THC). Inhaled over 10 mins	RSEO, RSEC, Attention & Memory Tasks, 45 min simulated drive; 30 mins post consumption	Delta, Theta, Alpha, Beta, Gamma
Hart et al. (2010)	Within-subjects Balanced, double-blind, placebo-controlled crossover	N = 24 (11 female) *only data from 23 used for EEG analyses	M (SD) age = 25.8 (4.1)	No criteria apparent. Participants reported almost daily use, averaging 4 cannabis cigarettes per day	Cannabis flower cigarette (1.8 % or 3.9 % THC) 1 g = Approximately 18 or 39 mg total THC	Word Presentation, Working Memory (Spatial N-back), Word Recognition; 15–200 mins post consumption	P3, N4, Theta, Alpha, Beta
Ilan et al. (2004)	Within-subjects Placebo-controlled crossover	N = 10 (5 male)	M age = 26.7	Casual smokers: 1x per week to 1x per month over past year	Cannabis flower cigarette (3.45 % THC) 6 puffs	RSEO, RSEC, Word presentation, Working Memory (Spatial N-Back), Word recognition; 20–200 mins post consumption.	N1, P2, P3, N4, Theta, Alpha, Beta
Ilan et al. (2005)	Within/between Randomised, double-blind, placebo-controlled mixed (between subject allocation to low or high dose THC)	N = 23 *only data from 22 used for EEG analyses	M (SD) age low dose = 25.7 (3.1) M (SD) age high dose = 26.4 (4.8)	≥ 10 lifetime exposures	Cannabis flower cigarette (1.8 % or 3.6 % THC) + varied levels of CBC & CBD 5 puffs over 10 mins	RSEO, RSEC, Word presentation, Working Memory (Spatial N-Back), Word recognition; 20–140 mins post consumption	N2, P3, N4, Theta, Alpha, Beta
Kowal et al. (2015)	Between-subjects Randomised, double-blind, placebo-controlled	N = 55 (49 male) n = 18 (high dose) n = 18 (low dose) n = 19 (placebo)	M (SD) age placebo = 21.3 (2.3) M (SD) age 5.5 mg = 21.1 (2.1) M (SD) age 22 mg = 22.3 (2.3)	Frequent users: ≥ 4x per week for at least 2 years	Cannabis flower (19 % THC) by vaporisation. 5.5 mg or 22 mg THC	Flanker Task (adapted version); between 6 and 35 mins post consumption	ERN, N1, N2, P3
Lansbergen et al. (2011)	Within-subjects Randomised, double-blind, placebo-controlled crossover	N = 16 (12 male)	M (SEM) age = 21 (0.5) Regular ecstasy users	Regular users: at least 2x per week for past year	Cannabis flower by vaporisation. 4, 6 & 6 mg THC administered at 90 min intervals + Placebo MDMA condition	RSEC; 2 hrs post first dose, 30 mins post second dose	Theta, Alpha
Richard et al. (2021)	Within-subjects Randomised, double-blind, placebo-controlled crossover	N = 14 (8 male)	M (SD) age = 24.1 (1.4)	No criteria apparent. Varied use history (ranged from 5 to 6x per year, to 4x per week)	Cannabis flower (6.7 % THC) 500 mg by vaporisation (approximately 33.5 mg THC). Inhaled over 10 mins	RSEO, RSEC, Sternberg Verbal Memory Scan, Three-Choice Vigilance Task; 10 mins post consumption	P2, LPP, Delta, Theta, Alpha, Beta, Gamma
Roser et al. (2008)	Within-subjects Randomised, double-blind, placebo-controlled crossover	N = 20 (10 male)	M (SD) age = 28.2 (3.1)	Previous occasional exposure but no use in last month.	4 capsules containing cannabis extract totalling either: 10 mg THC or 10 mg THC + 5.4 mg CBD	Choice Reaction Task; 3hrs post consumption	P3
Spronk et al. (2011)	Within-subjects Randomised, double-blind, placebo-controlled crossover	N = 10 (8 male)	M age = 20.6 Regular ecstasy users	Regular users: at least 2x per week for past year	Cannabis flower via vaporisation. Subsequent doses of 4, 6 & 6 mg THC at 90 min intervals	Modified Flanker Task*	ERN, N1, N2, P3

(*continued on next page*)

Table 1 (*continued*)

Study	Design	Total sample	Characteristics	Cannabis use history criteria	Cannabis intervention	Stimulus/task paradigm	EEG
Spronk et al. (2016a)	Within-subjects Randomised, double-blind, placebo-controlled crossover	N = 38 (29 male) n = 31 completed task in cannabis condition	M (SD) age = 22.1 (4.6) Non-addicted polydrug users	≥ 2x per week	Cannabis flower via vaporisation. 0.3 mg/kg THC, Booster dose of 0.15 mg/kg THC given 1 hr after 1st dose + Placebo cocaine condition	Go/No-go Task; 45 mins post consumption of booster dose, approximately 1 hr 45 mins post 1st dose	N2, P3
Spronk et al. (2016b)	Within-subjects Randomised, double-blind, placebo-controlled crossover	N = 61 Only n = 52 for placebo condition	M (SD) age = 22.6 (4.3) Non-addicted polydrug users	≥ 2 joints per week	Cannabis flower via vaporisation. 0.3 mg/kg THC, Booster dose of 0.15 mg/kg THC given 1 hr after 1st dose + Placebo cocaine condition	Modified Flanker Task; approximately 15 mins post consumption of booster dose, approximately 1 hr 15 mins post 1st dose	ERN, N1, P1, N2, P3
Struve et al. (2003)	Within-subjects Double-blind, placebo-controlled crossover	N = 8 (4 male)	M (SD) age = 24.6 (5.2)	History of infrequent/casual THC use, no chronic daily use.	Cannabis flower cigarette. Low dose: 1.77 % THC High dose: 3.54 % THC 8 mins smoking, 8 inhalations	RSEC; During, immediately post, 26 mins, 1 hr & 4hrs post consumption	Delta, Theta, Alpha, Beta
Theunissen et al. (2012)	Within-subjects Double-blind, placebo-controlled crossover	N = 24 (17 male)	M (SD) age occasional = 22.8 (2.3) M (SD) age heavy = 23.2 (3.3)	Heavy users: > 4x per week Occasional users: < 2x per week	Cannabis flower cigarette (13 % THC, with tobacco) 0.5 mg/kg THC Smoked over the course of 10–15 mins	Divided Attention Task, Stop Signal Task; 20 mins post consumption.	P1, N2, P3

*Note that although the task was performed after THC administration. exact timing was unclear for this study only.
N.R = not reported; RSEC = Resting state eyes closed; RSEO = Resting state eyes open; ERN = Error-related negativity; LPP = Late positive potential

Table 2
ERP components and frequency band descriptions.

Neurophysiological measure	Description
ERPs	
Negative deflections	
Error-Related Negativity (ERN)	Elicited by an incorrect motor response to a stimulus.
N100 (N1)	Elicited by an unexpected stimulus, influenced by selective attention and perceptual processing.
N200 (N2)	Elicited by repetitive non-target stimulus, reflects processes of attention and conflict monitoring.
N400 (N4)	Elicited by violations of semantic expectancies such as pairing words that do not belong.
Positive deflections	
P100 (P1)	Associated with early visual and attentional selection.
P200 (P2)	Associated with perceptual processing of visual stimuli, often larger for stimuli with target features.
P300 (P3)	Associated with information processing, attention, memory and stimulus categorisation, typically elicited by an oddball paradigm.
Late Positive Potential (LPP)	Typically elicited by repetition paradigms, reflects processes of memory, encoding and retrieval and is associated with emotional processing.
Frequency bands	
Delta (0.5–4 Hz)	Apparent when asleep.
Theta (4–8 Hz)	Apparent in a relaxed but awake state, associated with drowsiness.
Alpha (8–13 Hz)	Apparent when awake but 'zoned out' or reflecting (eyes closed).
Beta (13–30 Hz)	Related to a mentally active and focussed or alert state.
Gamma (30–80 Hz)	Associated with perception and associative learning, as well as short-term memory and sensory processing.

Kumar and Bhuvaneswari (2012); Luck (2014); Polich (2007); Schomer and Lopes (2010); Sur and Sinha (2009); Woodman (2010).

effect size), demonstrating a prolonged stimulus evaluation timeframe. Nonetheless, the findings on latency outcomes were inconsistent. Findings from each study are reported in Table 8.

3.7. N4

Two of the three studies that examined the N4 observed a decrease in amplitude after cannabis smoking, with Ilan et al. (2005) suggesting this decrease was dose-dependent. Ilan et al. (2004) reported no effect of cannabis on N4 amplitude, even while the dose and task paradigms were similar to that used in the high dose condition reported by Ilan et al. (2005). However, this outcome was based on a small sample (N = 10; Ilan et al., 2004), so there may have been insufficient power to detect a change for this component. No data were reported concerning N4 latency, as reported in Table 9.

3.8. LPP

Cannabis appeared to have a significant effect on LPP amplitude, albeit only two studies reported this component (Table 10). Both studies utilised vaporised cannabis administration and observed a change in LPP amplitude using word recognition paradigms, while LPP latency was not reported. Brown et al. (2019) also found that the power attenuation of the LPP was related to decrements in driving performance during a simulated driving task, as measured through an increase to the standard deviation of the lateral position (lane weaving). While Richard et al. (2021) reported a large magnitude increase in LPP amplitude at the frontal electrode site, this was accompanied primarily by a pattern of amplitude decreases at occipital sites. As such, amplitude attenuation as a result of cannabis consumption appeared to be localised to parietal-occipital regions when compared to a placebo condition.

Table 3
Cannabis and the ERN.

Study	Comparison	ERN timing	Paradigm	Assessment timing	Latency	Amplitude
Kowal et al. (2015)	Placebo to vaped cannabis (5.5 mg or 22 mg THC) (between-subjects)	50–100 ms at Fz, FCz & Cz	Flanker Task (modified version - letters)	6–35 mins post	N.R	**X** Low dose Cohen's *d*: −0.18 ▼ High dose Cohen's *d*: −0.85
Spronk et al. (2011)	Placebo to vaped cannabis (16 mg THC)	Difference between negative peak 0–200 ms and positive peak 80 ms before & after response onset at FCz & Cz	Flanker Task (modified version - letters)	N.R	N.R	ERN difference was significant in placebo but not THC condition (correct vs incorrect trial responses)
Spronk et al. (2016b)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg THC, 0.15 mg/kg THC booster)	Difference between negative peak 0–200 ms and positive peak 80 ms before & after response onset at FCz & Cz	Flanker Task (modified version - letters)	15 mins post booster dose (1 hr 15 mins post first dose)	N.R	▼

Notes: N.R = not reported; **X** *= non-significant,* ▼ = significantly reduced

Table 4
Cannabis and the P1.

Study	Comparison	P1 timing	Paradigm	Assessment timing	Latency	Amplitude
Spronk et al. (2016b)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg THC, 0.15 mg/kg THC booster)	70–130 ms at Oz	Flanker Task (modified version - letters)	15 mins post booster dose (1 hr 15 mins post first dose)	N.R	**X**
Theunissen et al. (2012)	Placebo to smoked cannabis (0.5 mg/kg THC)	80–200 ms at Oz	Divided Attention Task	20 mins post	**X** Occasional users Cohen's *d*: −0.27 (distractors) −0.36 (targets) **X** Heavy users Cohen's *d*: 0.31 (distractors) 0.07 (targets)	▼ Occasional users Cohen's *d*: −0.43 (distractors) −0.75 (targets) **X** Heavy users Cohen's *d*: 0.18 (distractors) 0.83 (targets)

Notes: N.R = not reported; **X** *= non-significant,* ▼ = significantly reduced

Table 5
Cannabis and the N1.

Study	Comparison	N1 timing	Paradigm	Assessment timing	Latency	Amplitude
Ilan et al. (2004)	Placebo to 6 puffs smoked cannabis (3.45 % THC)	WM: 110–250 ms at Pz WR: 90–220 ms at Oz	Spatial N-back WM task & Word Recognition task	20 mins post	X	▼
Kowal et al. (2015)	Placebo to vaped cannabis (5.5 mg or 22 mg THC) (between-subjects)	65–115 ms at FCz, Cz and Pz	Flanker Task (modified version - letters)	6–35 mins post	N.R	X
Spronk et al. (2016b)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg THC, 0.15 mg/kg THC booster)	50–150 ms at FCz, Cz and Pz	Flanker Task (modified version - letters)	15 mins post booster dose (1 hr 15 mins post first dose)	N.R	X
Spronk et al. (2011)	Placebo to vaped cannabis (16 mg THC)	50–150 ms at FCz, Cz and Pz	Flanker Task (modified version - letters)	N.R	N.R	X

Notes: N.R = not reported, WM = Working memory; **X** *= non-significant,* ▼ = significantly reduced

4. Frequency bands

4.1. Delta

There is insufficient data available to determine a clear influence of cannabis on delta power, as presented in Table 11. Bocker et al. (2010b) reported that cannabis at varying doses had no significant impact on delta power in a sample of occasional cannabis users (two to nine cannabis cigarettes a month). While three additional studies examined this frequency range, outcomes relating to power were not reported or were unclear.

4.2. Theta

A decrease in power following cannabis administration was consistent across all studies reporting theta band activity (Table 12). Of these, only Lansbergen et al. (2011) reported sufficient data to calculate effect size, with the power decrease in the moderate-to-large effect size range,

Table 6
Cannabis and the P2.

Study	Comparison	P2 timing	Paradigm	Assessment timing	Latency	Amplitude
Ilan et al. (2004)	Placebo to 6 puffs smoked cannabis (3.45 %THC)	200–300 ms at FCx	Spatial N-back WM task	20 mins post	**X**	▼
Richard et al. (2021)	Placebo to vaped cannabis (33.5 mg THC)	150–250 ms	Verbal Memory Scan and Three-Choice Vigilance Task	10 mins post	N.R	Assumed non-significant based on information provided.

Notes: N.R = not reported, WM = Working memory; **X** *= non-significant,* ▼ = significantly reduced

Table 7
Cannabis and the N2.

Study	Comparison	N2 timing	Paradigm	Assessment timing	Latency	Amplitude
Bocker et al. (2010a)	Placebo to smoked cannabis (29.3 mg, 49.2 mg or 69.4 mg THC)	N2b: 275–325 ms at Fz	Visual Selective Attention Task	2hrs post	N.R	**X**
Ilan et al. (2005)	Baseline to 5 puffs smoked cannabis (1.8 % or 3.6 % THC)	150–250 ms at P0z	Word Recognition task	20 mins post	N.R	▼
Kowal et al. (2015)	Placebo to vaped cannabis (5.5 mg or 22 mg THC) (between-subjects)	280–330 ms at FCz	Flanker Task (modified version - letters)	6–35 mins post	N.R	**X**
Spronk et al. (2016a)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg THC, 0.15 mg/kg THC booster)	Subtracted most negative peak at 200–350 ms from proceeding positive peak at FCz	Go/NoGo Task	45 mins post booster dose (1 hr 45 mins post first dose)	**X**	**X**
Spronk et al. (2016b)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg THC, 0.15 mg/kg THC booster)	Subtracted most negative peak at 200–350 ms from proceeding positive peak at FCz	Flanker Task (modified version - letters)	15 mins post booster dose (1 hr 15 mins post first dose)	N.R	▲
Spronk et al. (2011)	Placebo to vaped cannabis (16 mg THC)	200–350 ms at FCz	Flanker Task (modified version - letters)	N.R	N.R	**X**
Theunissen et al. (2012)	Placebo to smoked cannabis (0.5 mg/kg THC)	150–300 ms at Fz	Stop Signal Task	20 mins post	**X** Heavy users Cohen's *d*: −0.08 (Go) −0.06 (NoGo50) −0.52 (NoGo150) −0.75 (NoGo250) −0.07 (NoGo350) **X** Occasional users Cohen's *d*: 0.02 (Go) 0.52 (NoGo50) 0.26 (NoGo150) −0.19 (NoGo250) 0.12 (NoGo350)	**X** Heavy users Cohen's *d*: 0.29 (Go) 0.28 (NoGo50) −0.21 (NoGo150) 0.24 (NoGo250) −0.17 (NoGo350) **X** Occasional users Cohen's *d*: −0.11 (Go) 0.48 (NoGo50) −0.23 (NoGo150) 0.75 (NoGo250) 0.31 (NoGo350)

Notes: N.R = not reported, WM = Working memory; WR = Word recognition; **X** *= non-significant,* ▼ = significantly reduced; ▲ = significantly increased

highlighting the practical significance of this measure. Of interest, Lansbergen et al. (2011) also reported an increase in theta power after placebo administration (medium effect size). These influences on theta power appeared to be dose-dependent, with studies reporting greater reductions in theta power after a larger dose of THC was delivered (Bocker et al., 2010b; Hart et al., 2010). The consistent influence on theta power persisted across doses, paradigms (e.g., resting state and active conditions) and irrespective of the cannabis use history of the sample.

4.3. Alpha

Cannabis administration most often decreased alpha band power, although findings were sometimes inconsistent. As reported in Table 13, decreases were evident across four studies while participants were resting or completing tasks. These decreases were most consistently apparent during the completion of memory-related cognitive tasks. Lansbergen et al. (2011) found that this decrease produced a significant medium effect in the Lower-1 range (6.4–8.4 Hz), although the defined frequency analysis windows may have influenced the overall alpha results for this study. While Struuve et al. (2003) found an increase in absolute power, this was the only study to find this effect. However, this study had the longest assessment timing after cannabis in addition to participants having their eyes closed (Struve et al., 2003), likely influencing alpha activity.

Table 8
Cannabis and the P3.

Study	Comparison	P3 timing	Paradigm	Assessment timing	Latency	Amplitude
Bocker et al. (2010a)	Placebo to smoked cannabis (29.3 mg, 49.2 mg or 69.4 mg THC)	400–500 ms at Pz	Visual Selective Attention Task	2hrs post	**X**	▼
Hart et al. (2010)	Baseline to smoked cannabis (18 mg or 39 mg THC)	290–570 ms at Cz, slow wave 400–700 ms at POz	Spatial N-Back WM Task	15–200 mins post	**X**	▼ Low-load phase (after high dose only - 60 mins post). ▼ High-load phase (after low dose only - 60 mins post).
Ilan et al. (2004)	Placebo to 6 puffs smoked cannabis (3.45 %THC)	270–450 ms at Pz	Spatial N-back WM task	20 mins post	**X**	▼ greater in low load condition
Ilan et al. (2005)	Baseline to 5 puffs smoked cannabis (1.8 % or 3.6 % THC)	300–450 ms at POz	Spatial N-back WM task	20 mins post	N.R	▼
Kowal et al. (2015)	Placebo to vaped cannabis (5.5 mg or 22 mg THC) (between-subjects)	350–400 ms at FCz, Cz & Pz	Flanker Task (modified)	6–35 mins post	N.R	**X**
Roser et al. (2008)	Placebo to oral cannabis extract (10 mg THC)	250–600 ms at Fz, Cz & Pz	Choice Reaction Task	3hrs post	**X** Pure THC Cohen's *d*: −0.03 (Fz) 0.11 (Cz) 0.09 (Pz) **X** Cannabis extract Cohen's *d*: −0.41 (Fz) −0.37 (Cz) −0.41 (Pz)	▼ Pure THC Cohen's *d*: −0.38 (Fz) −0.41 (Cz) −0.47 (Pz) ▼ Cannabis extract Cohen's *d*: −0.40 (Fz) −0.62 (Cz) −0.49 (Pz)
Spronk et al. (2016a)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg THC, 0.15 mg/kg THC booster)	300–600 ms at Pz & FCz	Go/No-go Task	45 mins post booster dose (1 hr 45 mins post first dose)	▲ Cohen's *d*: 0.57	▼ Average NoGo P3 Cohen's *d*: −0.53 P3 amplitude from Pz Cohen's *d*: −0.55
Spronk et al. (2016b)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg THC, 0.15 mg/kg THC booster)	300–500 ms at FCz, Cz & Pz	Flanker Task (modified)	15 mins post booster dose (1 hr 15 mins post first dose)	▲	▼
Spronk et al. (2011)	Placebo to vaped cannabis (16 mg THC)	300–500 ms at FCz, Cz & Pz	Flanker Task (modified)	N.R	N.R	**X**
Theunissen et al. (2012)	Placebo to smoked cannabis (0.5 mg/kg THC)	275–650 at Pz	Divided Attention Task	20 mins post	**X** Heavy users Cohen's *d*: −0.70 (distractors) −0.02 (targets) **X** Occasional users Cohen's *d*: 0.02 (distractors) −0.37 (targets)	▼ Those who reported greater intoxication showed the largest decreases. No significant effect of cannabis use history. Heavy users Cohen's *d*: −0.10 (distractors) −0.51 (targets) Occasional users Cohen's *d*: −0.32 (distractors) −0.98 (targets)

Notes: N.R = not reported, **X** *= non-significant,* ▼ = significantly reduced; ▲ = significantly increased

Table 9
Cannabis and the N4.

Study	Comparison	N4 timing	Paradigm	Assessment timing	Latency	Amplitude
Hart et al. (2010)	Baseline to smoked cannabis (18 mg or 39 mg THC)	325–445 ms at Cz	Word Recognition task	15–200 mins post	N.R	▼ Low and high dose condition.
Ilan et al. (2004)	Placebo to 6 puffs smoked cannabis (3.45 %THC)	WP: 350–450 ms at Fz WR: 300–450 ms at Cz	Word Presentation task & Word Recognition task	20 mins post	N.R	**X**
Ilan et al. (2005)	Baseline to 5 puffs smoked cannabis (1.8 % or 3.6 % THC)	WR: 300–400 ms at Cz	Word Recognition task	20 mins post	N.R	▼ Greater in high dose condition.

Notes: N.R = not reported, **X** *= non-significant,* ▼ = significantly reduced

Table 10
Cannabis and the LPP.

Study	Comparison	LPP timing	Paradigm	Assessment timing	Latency	Amplitude
Brown et al. (2019)	Placebo to vaped cannabis (33.5 mg THC)	400–800 ms	Verbal Memory Scan Task and 45 min simulator drive	30 mins post	N.R	▼ Correlated with increase in SDLP.
Richard et al. (2021)	Placebo to vaped cannabis (33.5 mg THC)	500–800 ms	Verbal Memory Scan and Three-Choice Vigilance Task	10 mins post	N.R	▼ Target amplitude Cohen's *d*: 1.66 (Fp) −0.60 (O1) −0.55 (O2) Non-Target amplitude Cohen's *d*: −0.70 (Fp1) −0.56 (POz) *Increased amplitude to stimuli during placebo visit, but no difference after THC during Verbal Memory Scan.

Notes: N.R = not reported; ▼ = significantly reduced*; SDLP = Standard deviation of lateral position*

Table 11
Cannabis and Delta band activity.

Study	Comparison	Frequency range	Paradigm	Assessment Timing	Power
Bocker et al. (2010b)	Placebo to smoked cannabis (29.3 mg, 49.2 mg or 69.4 mg THC)	0.05–4 Hz	General Attention Task	150 mins post	**X**
Brown et al. (2020)	Placebo to vaped cannabis (33.5 mg THC)	1–3 Hz	RSEO, RSEC, Attention & memory tasks (cog battery 60 mins), simulated drive (45 mins)	30 mins post	N.R
Richard et al. (2021)	Placebo to vaped cannabis (33.5 mg THC)	1–3 Hz	RSEO, RSEC, Sternberg's Verbal Memory Scan Task & Three-Choice Vigilance Task	10 mins post	N.R
Struve et al. (2003)	Baseline to 8 inhalations smoked cannabis (1.77 % or 3.54 % THC)	1.5–3.5 Hz	RSEC	0–4hrs post	N.R

Notes: N.R = not reported, **X** *= non-significant,* ▼ = significantly reduced; RSEC = Resting state eyes closed; RSEO = Resting state eyes open

4.4. Beta

The most common finding was for a decrease in beta power following cannabis administration, as seen across four studies in Table 14. A total of seven identified studies assessed beta activity, of which only four provided relevant data concerning changes to power in the wake of cannabis administration. Hart et al. (2010) reported that the observed beta power attenuation reversed as the effects of cannabis dissipated, with beta power then increasing over the course of the session. The dose of THC delivered also appears to affect beta activity, with Bocker et al. (2010b) reporting differential dose effects. Beta power increased from placebo as compared to the low and medium dose conditions, before then decreasing in the high dose condition, highlighting a potential inverse U-shaped dose function of THC on beta power.

4.5. Gamma

Limited data concerning the effects of cannabis on gamma activity were available, as seen in Table 15. Of the two studies identified, only one reported an outcome for this measure, finding cannabis increased power while participants were in the resting state eyes closed condition.

4.6. Task paradigms

Of the studies identified, N = 12 reported outcomes where a cognitive task/paradigm was completed while EEG was recorded. These results are denoted as changes to reaction time, accuracy or number of errors in Table 16 below. Cannabis most consistently impacted reaction time, with many showing slower responses after administration. While accuracy was often impacted, there were many cases where although participants were slower to react, accuracy remained unchanged from baseline/placebo performance. This did not seem to be consistently related to dose or cannabis use history, although the mixed cannabis experience in some of the samples may have complicated these findings.

5. Discussion

This systematic review synthesised available research literature examining the acute effects of cannabis administration across multiple EEG components. Across the studies identified, results indicate that changes to specific ERP/frequency band measures are associated with recent cannabis consumption, as demonstrated through reductions in amplitude or power. However, contradictory findings, heterogeneity and limited data hinder conclusions for some components. Where cognitive tasks were completed, performance indicators such as reaction time and accuracy were often impacted in the wake of cannabis administration. Taken together, these outcomes suggest that some EEG measures may help to identify recent cannabis consumption.

The present findings highlight the potential for ERPs to assist in understanding the specific psychophysiological responses to cannabis consumption, further elucidating the complex outcomes of this substance. Studies examining ERPs primarily focussed on the P3, revealing this component may be sensitive to recent cannabis consumption, finding reductions in amplitude as compared to baseline or placebo (Böcker et al., 2010a; Hart et al., 2010; Ilan et al., 2005, 2004; Roser et al., 2008; Spronk et al., 2016a, 2016b; Theunissen et al., 2012). The P3 component of the ERP is associated with attentional processing, with changes to P3 amplitude indicating that attentional decrements occur following cannabis consumption. These reductions were typically of a small-to-medium magnitude effect, with most data points demonstrating this effect was consistent across samples and paradigms, further supporting the notion of a meaningful influence of cannabis on the P3 component (Roser et al., 2008; Spronk et al., 2016a; Theunissen et al., 2012). P3 latency effects were less clear, with both increases and

Table 12
Cannabis and Theta band activity.

Study	Comparison	Frequency range	Paradigm	Assessment Timing	Power
Bocker et al. (2010b)	Placebo to smoked cannabis (29.3 mg, 49.2 mg or 69.4 mg THC)	4–8 Hz	General Attention Task	150 mins post	▼ Greater after high dose. *THC-related theta effects were correlated with memory performance.
Brown et al. (2019)	Placebo to vaped cannabis (33.5 mg THC)	3–5 Hz (slow band)	RSEO, RSEC, Three Choice Vigilance, Standard Image Recognition, Verbal Memory Scan, modified TMT, Driving simulator tasks	30–100 mins post	▼ Correlated with increased SDLP.
Brown et al. (2020)	Placebo to vaped cannabis (33.5 mg THC)	4–7 Hz at Fz	RSEO, RSEC, Attention & memory tasks (cognitive battery 60 mins), simulated drive (45 mins)	30–100 mins post	▼ Correlated with increased SDLP.
Hart et al. (2010)	Baseline to smoked cannabis (18 mg or 39 mg THC)	4–7 Hz at Fz	Word Presentation, Working Memory (Spatial N-back) & Word Recognition	15–200 mins post	▼ All task/resting conditions. Greater from high dose. Effects were greatest 15 mins post, but remained until 60 mins (1.8 %THC) and 110 mins (3.9 % THC) post.
Ilan et al. (2004)	Placebo to 6 puffs smoked cannabis (3.45 %THC)	4–6 Hz at AFz	RSEO, RSEC, Word Presentation task, Spatial N-back WM task & Word Recognition task	20–200 mins post	▼ All task/resting conditions.
Ilan et al. (2005)	Baseline to 5 puffs smoked cannabis (1.8 % or 3.6 % THC)	4–7 Hz at Fz	Word Presentation task, Spatial N-back WM task & Word Recognition task	20–140 mins post	▼ All tasks. Greatest 20 mins post, but remained at 140 mins post.
Lansbergen et al. (2011)	Placebo to vaped cannabis (4, 6 & 6 mg THC) at 90 min intervals	4.4–6.4 Hz	RSEC	30 mins post second dose	▼ Cohen's *d*: −0.73 Found that power increased after placebo, Cohen's *d*: 0.49
Richard et al. (2021)	Placebo to vaped cannabis (33.5 mg THC)	Slow: 3–5 Hz Fast: 5–7 Hz	RSEO, RSEC, Sternberg's Verbal Memory Scan Task & Three-Choice Vigilance Task	10 mins post	▼ In both resting state conditions (most pronounced in RSEC)
Struve et al. (2003)	Baseline to 8 inhalations smoked cannabis (1.77 % or 3.54 % THC)	3.5–7.5 Hz	RSEC	0–4hrs post	N.R

Notes: N.R = not reported, WM = Working memory; **X** *= non-significant;* ▼ = significantly reduced; RSEC = Resting state eyes closed; RSEO = Resting state eyes open

decreases evident, depending on dose, electrode and cannabis use history, with these outcomes primarily failing to meet statistical significance. These results suggests that while overall efficiency of neural processing might remain unchanged, the strength of attentional allocation is modulated by cannabis consumption.

While studies examining the ERN, N4 and LPP also report amplitude attenuation following cannabis intake, there were insufficient data to support this conclusion across the body of research reviewed. Of these, only Richard et al. (2021) and Kowal et al. (2015) provided sufficient data for the calculation of effect sizes, with both medium and large effects observed for the ERN and LPP, suggesting that ERN and LPP may be a potential indicator of recent cannabis intake. Some studies also reported a dose-dependent effect of cannabis, with attenuation of amplitude increasing following higher THC doses (e.g., Ilan et al., 2005; Kowal et al., 2015), although further research is required to explore these effects. Taken together, it appears that cannabis has a greater influence on EEG components that reflect more complex underlying cognitive processes involved in attentional control, memory processing and the emotional processing of stimuli, highlighting their potential utility as markers when it comes to understanding the effects of cannabis on such skills.

This review also identified an effect of cannabis on oscillatory activity within the theta frequency range (typically 4–7 Hertz), with consistent reports of power decreases that were sometimes dose-dependent in nature (Böcker et al., 2010b; Brown et al., 2019, 2020; Hart et al., 2010; Ilan et al., 2005, 2004; Lansbergen et al., 2011; Richard et al., 2021). Only one study reviewed provided sufficient data for the calculation of effect size (Lansbergen et al., 2011). While this study reported a larger effect size, the outcomes from the other reported studies were based on statistical significance testing. As such, further research is required to confirm whether this finding is meaningful. Irrespective of this, the cannabis-related effects on theta power may be a potential marker of recent consumption, as assessed with task paradigms primarily involving the recruitment of skills such as attention and memory. These changes were significantly correlated with observable task changes, suggesting a potential link between these EEG-measured components and behavioural effects (Böcker et al., 2010b; Brown et al., 2019). Frontal theta activity is related to complex underlying processes pertaining to learning, encoding and memory (Herweg et al., 2020; Jensen and Tesche, 2002)). Collectively, these results suggest that acute cannabis consumption may interrupt activity within this range, potentially leading to difficulty recruiting these skills in everyday life. As such, theta oscillatory activity may provide useful insight into the specific mechanisms of cannabis intoxication and warrants further investigation as a potential biomarker related to recent consumption.

While alpha and beta power also appeared to be affected by cannabis administration, the evidence was limited, with some contradictory findings (e.g., Böcker et al., 2010b; Struve et al., 2003), suggesting further research is needed to elucidate the effects of cannabis within these frequency ranges. In relation to other reported frequency bands (i.e., delta, gamma) current evidence appears mixed or net yet sufficient to draw strong conclusions.

6. Limitations & future directions

There are several limitations that should be acknowledged. First, the range in participant characteristics (such as health and cannabis use history) may have influenced present outcomes. For example, while

Table 13
Cannabis and Alpha band activity.

Study	Comparison	Frequency range	Paradigm	Assessment Timing	Power
Bocker et al. (2010b)	Placebo to smoked cannabis (29.3 mg, 49.2 mg or 69.4 mg THC)	8–12 Hz	General Attention Task	150 mins post	**X**
Brown et al. (2020)	Placebo to vaped cannabis (33.5 mg THC)	8–12 Hz	RSEO, RSEC, Attention & memory tasks (cog battery 60 mins), simulated drive (45 mins)	30 mins post	N.R
Hart et al. (2010)	Baseline to smoked cannabis (18 mg or 39 mg THC)	7–11 Hz at POz	Word Presentation, Working Memory (Spatial N-back) & Word Recognition	15–200 mins post	▼ WM task at 15, 160 and 200 mins.
Ilan et al. (2004)	Placebo to 6 puffs smoked cannabis (3.45 %THC)	8–12 Hz at Pz	RSEO, RSEC, Word Presentation task, Spatial N-back WM task & Word Recognition task	20–200 mins post	▼ WM task
Ilan et al. (2005)	Baseline to 5 puffs smoked cannabis (1.8 % or 3.6 % THC)	8–11 Hz at Cz	Word Presentation task, Spatial N-back WM task & Word Recognition task	20–140 mins post	▼ WP task 20 mins post but recovered. ▼ WM task
Lansbergen et al. (2011)	Placebo to vaped cannabis (4, 6 & 6 mg THC) at 90 min intervals	Lower−1: 6.4–8.4 Hz Lower−2: 8.4–10.4 Upper: 10.4–12.4	RSEC	30 mins post second dose	▼ Lower−1 Cohen's *d*: −0.51 **X** Lower−2 Cohen's *d*: 0.14 **X** Upper Cohen's *d*: 0.17
Richard et al. (2021)	Placebo to vaped cannabis (33.5 mg THC)	Slow alpha: 8–10 Hz Fast alpha: 10–12 Hz	RSEO, RSEC, Sternberg's Verbal Memory Scan Task & Three-Choice Vigilance Task	10 mins post	▼ RSEO at Cz, PO & T3.
Struve et al. (2003)	Baseline to 8 inhalations smoked cannabis (1.77 % or 3.54 % THC)	7.5–12.5 Hz	RSEC	0–4hrs post	▲ Greater increases in high dose condition.

Notes: N.R = not reported, WM = Working memory; **X** *= non-significant;* ▼ = significantly reduced; ▲ = significantly increased; RSEC = Resting state eyes closed; RSEO = Resting state eyes open

Table 14
Cannabis and Beta band activity.

Study	Comparison	Frequency range	Paradigm	Assessment Timing	Power
Bocker et al. (2010b)	Placebo to smoked cannabis (29.3 mg, 49.2 mg or 69.4 mg THC)	12–30 Hz	RSEC, General Attention Task	150 mins post	▲ 29.3 mg ▲ 49.2 mg ▼ 69.4 mg
Brown et al. (2020)	Placebo to vaped cannabis (33.5 mg THC)	13–30 Hz	RSEO, RSEC, Attention & memory tasks (cog battery 60 mins), simulated drive (45 mins)	30 mins post	N.R
Hart et al. (2010)	Baseline to smoked cannabis (18 mg or 39 mg THC)	13–18 Hz at Fz	Word Presentation, Working Memory (Spatial N-back) & Word Recognition	15–200 mins post	▼ Increased as the effects of cannabis dissipated.
Ilan et al. (2004)	Placebo to 6 puffs smoked cannabis (3.45 %THC)	13–18 Hz at Cz	RSEO, RSEC, Word Presentation task, Spatial N-back WM task & Word Recognition task	20–200 mins post	▼ Resting state and task conditions (greater in the low load task).
Ilan et al. (2005)	Baseline to 5 puffs smoked cannabis (1.8 % or 3.6 % THC)	13–18 Hz at Cz	Word Presentation task, Spatial N-back WM task & Word Recognition task	20–140 mins post	▼ WM task.
Richard et al. (2021)	Placebo to vaped cannabis (33.5 mg THC)	Slow: 13–20 Hz Fast: 21–30 Hz	RSEO, RSEC, Sternberg's Verbal Memory Scan Task & Three-Choice Vigilance Task	10 mins post	N.R
Struve et al. (2003)	Baseline to 8 inhalations smoked cannabis (1.77 % or 3.54 % THC)	12.5–20 Hz	RSEC	0–4hrs post	N.R

Notes: N.R = not reported, WM = Working memory; **X** *= non-significant;* ▼ = significantly reduced; ▲ = significantly increased; RSEC = Resting state eyes closed; RSEO = Resting state eyes open

some studies employed 'regular' or 'frequent' user groups, others provided no cannabis use criteria or involved varied usage patterns within the sample (Table 1). This heterogeneity hinders further inspection of the relationship between acute cannabis consumption, tolerance and EEG outcomes. Some studies also included current poly-drug users in their sample (Lansbergen et al., 2011; Spronk et al., 2016a; Spronk et al., 2016b) which may also influence neurophysiological responses (Ceballos et al., 2009; Liu et al., 2022). Second, a range of doses were administered across the studies, with some studies involving an imprecise dose of THC delivery (e.g., '6 puffs of a cannabis cigarette'). Further, as these studies were synthesised qualitatively, specific dose-related effects cannot be examined directly. In addition, the exclusion of studies that administered synthetic THC limits insights concerning the psychoactive constituent (THC) beyond that which was delivered via cannabis. Third, as task paradigms, stimulus characteristics and analyses differed across studies, these differences may lead to differences in study

Table 15
Cannabis and Gamma band activity.

Study	Comparison	Frequency range	Paradigm	Assessment Timing	Power
Brown et al. (2020)	Placebo to vaped cannabis (33.5 mg THC)	25–40 Hz	RSEO, RSEC, Attention & memory tasks (cog battery 60 mins), simulated drive (45 mins)	30 mins post	N.R
Richard et al. (2021)	Placebo to vaped cannabis (33.5 mg THC)	30–40 Hz	RSEO, RSEC, Sternberg's Verbal Memory Scan Task & Three-Choice Vigilance Task	10 mins post	▲ RSEC.

Notes: N.R = not reported; **X** *= non-significant;* ▲ = significantly increased; RSEC = Resting state eyes closed; RSEO = Resting state eyes open

outcomes. However, the high number of randomised-controlled trials included in the present review may mitigate the impact of additional confounding variables. Finally, while many studies reported differences in EEG outcomes in the wake of cannabis administration, there was a noted lack of data available for the estimation of effect size (lending to a reliance on statistical significance throughout this review). Due to this, the magnitude of these outcomes cannot be determined for many of the ERP components/frequency bands reported in this review. Further research is required to provide a more comprehensive estimate of the overall effect of cannabis on EEG.

It is also important to note that no study attempted to examine effects in those who use cannabis medicinally. As such, while the aforementioned measures may be sensitive to cannabis consumption in recreational or occasional user groups, it remains unclear as to whether these findings will emerge in medicinal populations. A key point to highlight is the cannabis use history reported by the studies described (Table 1), where even the 'frequent' user groups would not approach the repetitive daily, long-term use patterns that would be related to a prescribed schedule (e.g., multiple administrations per day). Prescribed regular intake of cannabis is likely to be related to higher tolerance, in addition to the interaction of symptom management where the symptoms may exert independent effects that may interrupt typical functioning (Ramaekers et al., 2021). This review has identified a pressing need for further research examining the acute neural response to cannabis consumption in medicinal cannabis patients.

7. Conclusion

This systematic review attempted to identify EEG markers that are most sensitive to acute cannabis (containing THC) administration. A consistent effect of cannabis on amplitude of the P3 ERP (e.g., attention, memory) suggests that this component may be a sensitive neural indicator of recent cannabis consumption, warranting further investigation. While other ERP components also emerged as potential measures that may have utility when examining cannabis-related effects on error processing and emotional stimuli (e.g., ERN, N4, LPP), mixed findings and limited data hinder the ability to draw any conclusions. Some EEG frequency bands also appeared to display a notable effect following cannabis consumption, with the most robust evidence emerging for theta activity interruptions, likely related to changes in cognitive recruitment. These outcomes highlight the potential utility of EEG

Table 16
Cannabis and task performance.

Study	Comparison	Task paradigm	Results
Bocker et al. (2010a)	Placebo to smoked cannabis (29.3 mg, 49.2 mg or 69.4 mg THC)	Visual Selective Attention Task; 2hrs post	Reaction time▲ Accuracy▼ Both with a linear dose-related effect.
Bocker et al. (2010b)	Placebo to smoked cannabis (29.3 mg, 49.2 mg or 69.4 mg THC)	General Attention Task; 150 mins post	Reaction time▲ Errors ▲
Brown et al. (2019)	Placebo to vaped cannabis (33.5 mg THC)	Three Choice Vigilance, Standard Image Recognition, Verbal Memory Scan, TMT (modified), 45 min driving simulator task; 30 mins post	Cognitive tasks: N.R Simulator task: SDLP▲
Brown et al. (2020)	Placebo to vaped cannabis (33.5 mg THC)	Attention, Memory Tasks, 45 min simulated drive; 30 mins post	Cognitive tasks: N.R Simulator task: SDLP▲
Hart et al. (2010)	Baseline to smoked cannabis (18 mg or 39 mg THC)	Word Presentation, Working Memory (Spatial N-back), Word Recognition; 15–200 mins post	WM task: Reaction time▲ Accuracy **X** *WR* task: Reaction time▲ Accuracy **X**
Ilan et al. (2004)	Placebo to 6 puffs smoked cannabis (3.45 %THC)	Word Presentation, Working Memory (Spatial N-Back), Word recognition; 20–200 mins post	WP task: Reaction time **X** Accuracy **X** WM task: Reaction time▲ Accuracy▼ (high load condition only) *WR* task: Reaction time▲ (new words only) Accuracy▼
Ilan et al. (2005)	Baseline to 5 puffs smoked cannabis (1.8 % or 3.6 % THC)	Word Presentation, Working Memory (Spatial N-Back), Word recognition; 20–140 mins post	WM task: Reaction time▲ Accuracy▼ (both dose conditions) *WR* task: Reaction time **X** Accuracy▼
Kowal et al. (2015)	Placebo to vaped cannabis (5.5 mg or 22 mg THC) (between-subjects)	Flanker Task (modified version); between 6 and 35 mins post	Accuracy▼ (greater in high dose condition) Reaction time **X**
Richard et al. (2021)	Placebo to vaped cannabis (33.5 mg THC)	Sternberg Verbal Memory Scan, Three-Choice Vigilance Task; 10 mins post	Reaction time **X** Accuracy **X**
Roser et al. (2008)	Placebo to oral cannabis extract (10 mg THC)	Choice Reaction Task; 3hrs post	Reaction time **X**
Spronk et al. (2011)	Placebo to vaped cannabis (16 mg THC)	Flanker Task (modified version); N.R	Reaction time **X** Accuracy **X**
Spronk et al. (2016a)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg THC, 0.15 mg/kg THC booster)	Go/No-go Task; 45 mins post booster dose (1 hr 45 mins post first dose)	Reaction time▲ Accuracy▼
Spronk et al. (2016b)	Placebo (cocaine & cannabis) to vaped cannabis (0.3 mg/kg	Flanker Task (modified version); 15 mins post booster dose (1 hr 15 mins post first dose)	Reaction time▲ Accuracy **X**

(*continued on next page*)

Table 16 (*continued*)

Study	Comparison	Task paradigm	Results
	THC, 0.15 mg/kg THC booster)		
Theunissen et al. (2012)	Placebo to smoked cannabis (0.5 mg/kg THC)	Divided Attention Task, Stop Signal Task; 20 mins post	DAT: Reaction time▲ Errors▲ Accuracy▼ Effects greater in occasional users. SST: Reaction time▲ Accuracy **X** No effect of cannabis use history.

Notes: N.R = not reported; **X** *= non-significant;* ▲ = significantly increased; ▼ = significantly decreased; *SDLP = Standard deviation of lateral position; WM = working memory; WR = word recognition; WP = word presentation; DAT = divided attention task; SST = stop signal task*

biomarkers in understanding and tracking the profile of cannabis-related effects on brain activity, especially considering the more subtle changes that may be apparent in tolerant user groups. As both recreational and medicinal cannabis use increases globally, such knowledge will assist in further attempts to understand and accurately identify the point at which safety-sensitive tasks might be compromised due to cannabis consumption.

Funding statement

This research was funded by the MAIC/UniSC Road Safety Research Collaboration Grant.

Declaration of competing interest

The authors report there are no conflicts of interest to declare.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2025.106092.

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