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Cannabis and sleep architecture: A systematic review and meta-analysis

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ABSTRACT

Cannabis use for sleep is increasingly prevalent, yet its effects on sleep architecture remain unclear. This systematic review and meta-analysis examined polysomnographic evidence on cannabis' impact on sleep parameters. Eighteen studies were identified, with nine suitable for meta-analysis. Findings indicate that cannabis administration does not consistently alter sleep duration, latency, wake time, efficiency, or sleep staging. While early studies suggested reductions in rapid eye movement sleep, these were primarily based on small-scale trials with high tetrahydrocannabinol doses and significant methodological limitations. More recent studies using larger samples and lower therapeutic doses of tetrahydrocannabinol have reported mixed (and often no) evidence of rapid eye movement (REM) suppression, and the evidence base remains very limited. However, withdrawal from active cannabis use was consistently associated with sleep disturbances, including reduced total sleeping times and prolonged sleep onset latency, as well as REM rebounds. Variability in study outcomes highlights the influence of factors such as dosage, cannabinoid composition, prior cannabis use, and health conditions. Further research using standardised protocols and larger samples is needed to clarify the relationship between cannabis and sleep architecture and to address the discrepancies between subjective sleep improvements and objective sleep metrics.

Glossary of terms

Anxiolytic	A property of a substance that helps reduce anxiety
Antipsychotic	A property of a substance that helps manage symptoms of psychosis
Cannabidiol	A non-psychoactive compound in cannabis often associated with therapeutic effects, known as CBD
Cannabis-naïve	Individuals who have little to no prior experience using cannabis
Chronic cannabis use	Daily cannabis use continued over the course of at least one year
Dependence	A state where continued use of a substance is needed to avoid withdrawal symptoms
Dronabinol	A synthetic form of tetrahydrocannabinol, available in capsule form
Objective measures of sleep	Sleep assessments based on measurable physiological data

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Anxiolytic	A property of a substance that helps reduce anxiety
Nabilone	A synthetic cannabinoid that mimics tetrahydrocannabinol with a slightly different chemical structure, available in capsule form
Oromucosal	Administration of a drug through the mucous membranes in the mouth, absorbed under the tongue or inside the cheek
Polysomnography	A diagnostic tool that records brain waves, oxygen levels, heart rate, and other physiological parameters during sleep
Rapid eye movement sleep	A sleep stage characterised by rapid eye movements, playing a significant role in memory consolidation and emotional regulation
Sedative properties	Characteristics of a substance that promote relaxation and help induce sleep
Sleep efficiency	The ratio of total time spent asleep to the total time spent in bed, expressed as a percentage

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Abbreviations

CBD	Cannabidiol
CI	Confidence interval
EEG	Electroencephalogram
INS	Insomnia
OSA	Obstructive sleep apnea
PD	Parkinson's disease
PSG	Polysomnography
PTSD	Post-traumatic-stress-disorder
RBD	REM behaviour disorder
RCT	Randomised controlled trial
REM	Rapid eye movement
RLS	Restless legs syndrome
RoB	Risk of bias
ROBINS-I	Risk of bias in non-randomised studies - interventions
SMD	Standardised mean difference
THC	Tetrahydrocannabinol
US	United States

(*continued*)

Anxiolytic	A property of a substance that helps reduce anxiety
Sleep onset latency	The time it takes for a person to transition from being awake to falling asleep
Slow wave sleep	A deep sleep stage in non-REM sleep, important for physical recovery and immune function
Tetrahydrocannabinol	The primary psychoactive compound in cannabis, known as THC, responsible for the "high" sensation
Tolerance	A reduced response to a substance after repeated use, requiring higher doses to achieve the same effect
Transdermal	A method of drug administration where cannabinoids are absorbed through the skin
Wake after sleep onset	The amount of time spent awake after initially falling asleep, often used as a measure of sleep disturbance
Withdrawal symptoms	Negative physical or psychological effects experienced when a substance is reduced or discontinued after chronic use

1. Introduction

As cannabis has become increasingly available by legal means across Canada and in most jurisdictions in the US [1], a growing number of North Americans are using cannabis to manage poor sleep, particularly symptoms relating to insomnia [2]. Surveys suggest that sleep is the most commonly reported symptom that cannabis users target, with up to 85 percent of medical cannabis users reporting sleep improvements, in addition to a large number of recreational users [3,4]. Patients thus increasingly seek guidance from healthcare professionals regarding the use of cannabis to manage sleep [5]. A relatively large number of adolescents appear to use cannabis to improve their sleep as well, with one study finding that eight percent of students at a Northeastern US public high school had used cannabis as a sleep aid [6].

Despite its perceived benefits, cannabis use carries risks. While acute effects may provide temporary relief from symptoms of depression, anxiety, and stress [7], long-term chronic use—typically defined as daily or near-daily use continued over the course of several years [8]—has been associated with worsening of these symptoms [9]. Adolescent cannabis use in particular has been linked to an increased risk of developing depression and suicidal behaviour later in life [9], and a higher likelihood of psychotic illness [10,11], although the directionality of these relationships is debated [12,13]. Chronic cannabis use has also been associated with cognitive impairments such as reduced memory function and attention [14], especially at high doses of tetrahydrocannabinol (THC) [15]—cannabis' main psychoactive compound that produces the "high" sensation. Other reported associations include reduced respiratory function, cardiovascular disorders, and impaired educational attainment [8]. In relation to sleep, chronic cannabis users tend to display increased daytime sleepiness [16].

Studying cannabis' effects on sleep is complicated by several factors. Chronic users may develop tolerance to certain effects and exhibit withdrawal symptoms upon cessation due to dependence [8], thereby making cannabis use necessary for sleep [17]. In contrast, cannabis-naïve individuals may experience adverse effects from first-time cannabis use that can harm their sleep [18], such as headaches or increased anxiety [8]. Sleep outcomes are also influenced by cannabinoid type, dose, and intake method. The primary compounds of the cannabis plant, cannabidiol (CBD) and THC [19], have opposing psychoactive effects [20], where THC is recognised for its sedative properties, while CBD has been associated with anxiolytic and antipsychotic properties [21]. Some research suggests that THC may exert more beneficial effects on sleep than CBD [21,22], but the specific effects of dosing remain elusive [22]. Additionally, the cannabis delivery system—commonly smoked, vaporised, or orally ingested, but also consumed through oromucosal sprays and transdermal applications [23]—may differentially affect sleep outcomes [24]. Further, comorbidities play an important role, where it has been suggested that sleep improvements may be partly or even fully mediated by cannabis' effects on pain or other medical symptoms [25].

Another challenge in interpreting cannabis' effects on sleep is the inconsistency between subjective perceptions of sleep and objective metrics; an issue that is well-documented [26,27]. For example, one study found that while individuals reported falling asleep faster after cannabis use, their polysomnography (PSG) assessments indicated no corresponding decrease in sleep onset latency [28]. Moreover, certain perceived improvements may have detrimental long-term health consequences. A notable example is reductions in rapid eye movement (REM) sleep, which may contribute to the short-term subjective feeling of improved sleep among users, as REM sleep disturbances are frequently reported by individuals with sleep disorders [29]. Over time, however, the reduction in REM sleep has implications for cognitive functions and mood, given the role of REM sleep in memory consolidation and emotional regulation [30]. Similarly, while acute cannabis use may improve sleep, developing chronic use patterns may cause harm to one's health and sleep over time.

The current evidence regarding cannabis and sleep architecture is thus difficult to interpret. Cannabis is generally believed to reduce sleep onset latency [25], which is likely related to THC's soporific effects and CBD's anxiolysis potentially contributing indirectly [18]. Studies using subjective measures, such as sleep satisfaction scales, often report improvements, particularly in patients with pain symptoms [22]. However, diagnostic evidence gathered through polysomnography (PSG)—the gold standard for sleep studies to record brain waves, oxygen levels, heart rate, and eye and leg movements during sleep [31]—suggests that cannabis use is associated with reduced REM sleep [32–34]. Existing reviews on cannabis and sleep have specifically focused on cannabis-naïve subjects—excluding many potential studies involving participants that have some prior use [22], or mixed objective and subjective measures without meta-analysis [35], while the only meta-analytic review to date has focused on subjective sleep outcomes [36]. No systematic review has quantitatively synthesized the objective evidence on cannabis's effects on sleep architecture across key polysomnographic variables. This review addresses this gap by conducting a series of meta-analyses to provide a comprehensive quantitative synthesis of cannabis's impact on sleep architecture.

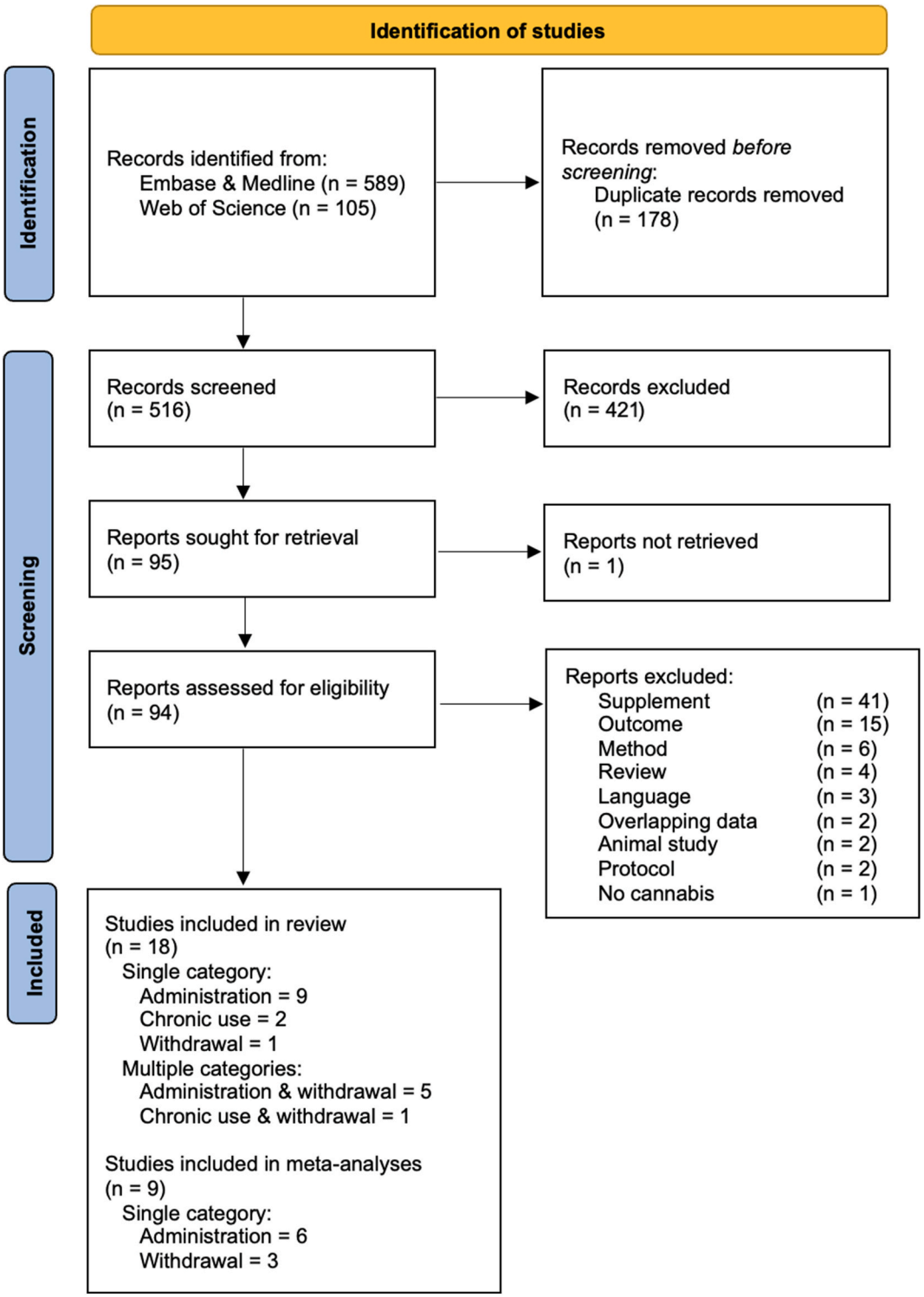

Fig. 1. PRISMA flow diagram of included studies.

2. Methods

2.1. Protocol registration

The review was conducted in accordance with PRISMA guidelines for performing systematic reviews. The protocol was registered in the International Prospective Register of Systematic Reviews (PROSPERO, registration number CRD42023462484).

2.2. Search strategy

Following recommendations made by Bramer et al. [37], we searched Embase, Medline, and Web of Science for peer-reviewed articles in scientific journals. We based our search terms around three key concepts: cannabis, sleep architecture measurement, and sleep disorders:

(mari*uana or cannab* or tetrahydrocannabinol or nabilone or dronabinol) AND (polysomnogra* or polygra* or electroencephalogra*

Table 1
Summary of included studies categorised by administration, chronic use, and withdrawal.

	Design					Sample					Cannabis			Sleep								Bias	MA
Author(s). Year.	R	B	C	T	# days	Use	Sex (M)	Age	Condition	Size	Method	Type	Dose (mg)	TST	SOL	WASO	SE	N1	N2	SWS	REM	REML		
Administration																								
Pivik et al. [50]	NR	DB	PC	CO	8	MU*	100 %	YA	NA	4	Oral	THC	13–17	↑	=	↓	NA	↓	=	↑	↓	NA	H	X
Hosko et al., 1973 [51].	NR	SB	PC	CO	8	MU*	100 %	24–28	NA	7	Oral	THC	0.2–0.4/ kg	NA	NA	=	NA	NA	NA	=	=	NA	H	X
Freemon. 1974.* [46]	NR	DB	PC	PL	14	MU*	?	21–29	NA	T5 | C1	Oral	THC	20	=	=	=	NA	=	=	=	↓	NA	H	X
Feinberg et al., 1975.* [42]	NR	DB	PC	CO	30	CU	100 %	?	NA	7	Oral	THC	70–210	=	=	=	NA	NA	=	=	↓	=	H	X
Feinberg et al., 1976.* [43]	NR	SB	PC	PP	32	CU	100 %	~26	NA	11	Oral	THC/GC	70–210	=	=	=	NA	NA	=	=	↓	↓	H	X
Tassinari et al., 1976 [52].	NR	SB	UC	PP	26	M	?	21–25	NA	11	Oral	THC	0.7–1.4/ kg	NA	NA	NA	NA	↓	↑	↓	↓	NA	H	X
Freemon & Al-Marashi. 1977.* [44]	NR	DB	PC	CO	30	SU	100 %	YA	NA	2	Oral	THC	20	=	↑	=	NA	NA	NA	=	=	NA	H	X
Freemon. 1982.* [45]	NR	DB	PC	CO	35	MU*	100 %	24	NA	2	Oral	THC	30	=	=	=	NA	NA	↑	↓	=	NA	H	X
Nicholson et al., 2004 [18].	R	DB	PC	CO	4	MU*	50 %	25.3	NA	8	Oral	THC:CBD	15:0; 5:5; 15:15	=	=	=	=	=	=	=	=	=	L	✓
Farabi et al., 2014 [53].	NR	SB	UC	PP	21	U	40 %	51.7	OSA	15	Oral	Dronabinol	T. 2.5–10	=	=	=	=	=	↓	=	=	=	L	✓
Carley et al., 2018 [38].	R	DB	PC	PL	42	MU	71 %	54.8	OSA	T48 | C25	Oral	Dronabinol	T. 2.5–10	=	=	=	=	NA	NA	=	↑	=	L	✓
Linares et al., 2018 [39].	R	DB	PC	CO	14	MU	46 %	29.3	NA	26	Oral	CBD	300	=	=	=	=	=	=	=	=	=	L	✓
Walsh et al., 2021 [40].	R	DB	PC	CO	28	MU*	17 %	53	INS	23	Oral	THC:CBN: CBD	10:1:0.5	=	=	=	=	=	=	=	=	↑	L	✓
De Almeida et al., 2021 [41].	R	DB	PC	PL	84	U	64 %	57.6	RBD + PD	T17 | C16	Oral	CBD	T. 75-300	=	=	=	=	=	=	=	↓	=	L	✓
Chronic use																								
Pranikoff et al., 1973.* [49]	NR	UB	CG	PL	2	CU	100 %	YA	NA	T10 | C10	Inhaled	GC	NA	=	=	NA	=	=	=	=	=	=	H	X
Karacan et al., 1976 [57].	NR	UB	CG	PL	8	CU	100 %	30.3	NA	T32 | C32	Inhaled	GC	NA	=	=	NA	NA	=	=	=	↑	=	H	X
Bolla et al., 2008 [56].	NR	UB	CG	PL	2	CU	65 %	21.2	NA	T17 | C14	Inhaled	GC	NA	↓	=	=	=	NA	NA	↓	=	=	H	X
Withdrawal																								
Pranikoff et al., 1973.* [49]	NR	UB	CG	PL	2	NA	100 %	20–25	NA	T20 | C20	Inhaled	GC	NA	=	=	=	=	=	↓	↑	=	=	H	X
Freemon. 1974.* [46]	NR	DB	UC	PP	14	NA	?	21–29	NA	T5 | C1	Oral	THC	20	=	=	=	NA	=	=	=	=	NA	H	X
Feinberg et al., 1975.* [42]	NR	SB	PC	CO	30	NA	100 %	?	NA	7	Oral	THC	70–210	↓	↑	↑	↓	NA	=	=	↑	↓	H	✓
Feinberg et al., 1976.* [43]	NR	SB	PC	PP	32	NA	100 %	~26	NA	11	Oral	THC/GC	70–210	↓	↑	=	NA	NA	=	=	↑	↓	H	✓
Freemon & Al-Marashi. 1977.* [44]	NR	DB	PC	CO	30	NA	100 %	YA	NA	2	Oral	THC	20	=	↑	↑	NA	NA	NA	↓	=	NA	H	X
Freemon. 1982.* [45]	NR	DB	PC	CO	35	NA	100 %	24	NA	2	Oral	THC	30	=	↑	↑	NA	NA	↑	↓	=	NA	H	X
Vandrey et al., 2011 [48].	NR	DB	UC	PP	20	NA	85 %	29	NA	20	Inhaled	GC	NA	↓	↑	=	↓	NA	NA	=	↑	↓	L	✓

Table 2
ROBINS-I assessment for non-randomised trials.

	D1	D2	D3	D4	D5	D6	D7	Overall
Pivik et al. 1972.	X	X	X	-	-	-	-	X
Hosko et al. 1973.	X	+	+	-	+	-	-	X
Pranikoff et al. 1973.	X	-	+	+	+	+	X	X
Freemon. 1974.	X	X	-	-	+	+	-	X
Feinberg et al. 1975.	X	X	+	-	-	X	+	X
Feinberg et al. 1976.	X	X	-	+	+	X	-	X
Karacan et al. 1976.	X	-	+	+	-	+	-	X
Tassinari et al. 1976.	X	-	-	X	X	X	+	X
Freemon & Al-Marashi. 1977.	X	-	+	+	+	+	+	X
Freemon. 1982.	-	X	+	+	+	+	-	X
Bolla et al. 2008.	-	-	+	+	+	X	-	X
Vandrey et al. 2011.	+	+	-	+	+	+	+	+
Farabi et al. 2014.	-	+	+	+	+	-	+	+

Domains: D1 = Bias due to confounding; D2 = Bias due to selection of participants; D3 = Bias in classification of interventions; D4 = Bias due to deviations from intended interventions; D5 = Bias due to missing data; D6 = Bias in measurement of outcomes; D7 = Bias in selection of the reported result.

Table 3
RoB 2.0 assessment for randomised trials.

	D1	D2	D3	D4	D5	Overall
Nicholson et al. 2004.	+	+	+	+	+	+
Carley et al. 2018.	+	+	+	+	+	+
Linares et al. 2018.	+	+	+	+	+	+
Walsh et al. 2021.	+	+	+	+	+	+
De Almeida et al. 2021.	+	+	+	+	+	+

Domains: D1 = Bias arising from the randomization process; D2 = Bias due to deviations from intended intervention; D3 = Bias due to missing outcome data; D4 = Bias in measurement of the outcome; D5 = Bias in selection of the reported result.

or sleep architecture or actimet* or actigra*) AND (sleep* or insomnia or rapid eye movement or narcolepsy or hypersomnolence or nightmare disorder* or restless leg syndrome or parasomnia* or circadian rhythm* or drows* or wake*)

The use of wildcards and Boolean operators was tailored to each database. No restrictions were used and the search was performed on September 2, 2023. All extracted studies were imported into the systematic review software Covidence.

2.3. Eligibility

Eligibility criteria included 1) a sample using any form of cannabis, including but not limited to THC, CBD, nabilone, dronabinol, and whole plant cannabis; 2) use of diagnostic measurements for sleep outcomes through the use of electroencephalogram, polysomnography, polygraphy, or actigraphy; 3) at least one of the following outcomes: total sleeping time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), sleep efficiency (SE), stage 1 sleep (N1), stage 2 sleep (N2), slow wave sleep (SWS), REM sleep, REM latency (REML); and 4) study design inclusive of a comparative group; either pre-post or a control group. Studies were excluded if cannabis use was only employed as a moderator rather than a main variable, if cannabis was studied in combination with other substances without isolating cannabis-specific effects, or if the publication language was not in English. Titles and abstracts of the studies were screened using double-review by two reviewers (SW and RB), where RV and AM resolved conflicts.

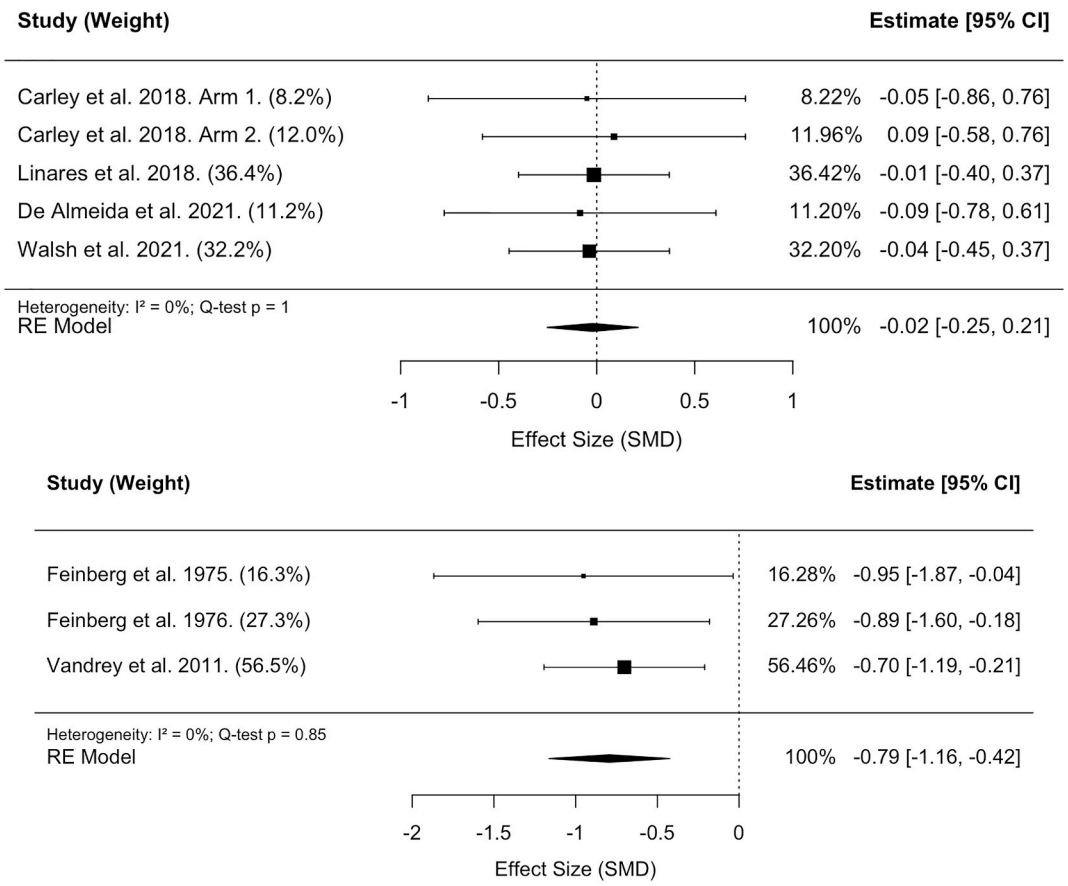

Fig. 2. a. Administration - Total sleeping time. b. Withdrawal - Total sleeping time.

2.4. Data acquisition

Sample characteristics (sex, age, health condition, ethnicity, prior cannabis use patterns), study design (administration, comparison between user and non-user, withdrawal), sleep assessment methods (EEG, PSG, actigraphy), methodological factors (randomisation, blinding, controls, designs, duration, day(s) of sleep measurement), cannabis details (type, dose, route of delivery, timing), and sleep outcomes (means, standard errors/deviations and/or p-values for the specified outcomes) were collected and charted. Each charted study underwent double-review.

2.5. Bias assessment

Risk of Bias assessment was performed for RCTs using Cochrane's Risk of Bias 2.0 tool and ROBINS-I tool for non-randomised trials. It was recognised that while these tools are useful in assessing bias for the outcomes of this study, they do not necessarily reflect study quality.

2.6. Data analysis

Data from the studies were charted by study design, sample, and cannabis characteristics. Sleep data from the included studies were analysed to evaluate the effects of cannabis on sleep architecture across TST, SOL, WASO, SE, N1, N2, SWS, REM, and REML. Statistical significance was assessed based on reported p-values, using a threshold of $\alpha = 0.05$.

Meta-analyses were planned using a random-effects model to account for variability across studies. Standardised mean differences (SMDs) with 95 % confidence intervals (CIs) were calculated for continuous outcomes. Heterogeneity across studies was assessed using the I^2 statistic, with values of 25 %, 50 %, and 75 % interpreted as low, moderate, and high heterogeneity, respectively. Funnel plots and Egger's tests were planned to assess publication bias. Missing standard deviations were imputed from p-values or standard errors when not explicitly reported. For crossover designs, pooled means and standard deviations were calculated to avoid overrepresentation of participants.

Sensitivity analyses were performed to evaluate the robustness of findings, including the exclusion of studies with high risk of bias, studies that did not confirm participants' prior cannabis use status, and studies focusing specifically on THC or CBD. Meta-analyses were performed separately for studies examining cannabis administration and cannabis withdrawal. Meta-analyses were conducted using *R* 4.4.2 with the "meta" and "metafor" packages. Graphs were created to visualise effect sizes and heterogeneity across studies.

3. Results

Eighteen studies met the selection criteria (Fig. 1). These were tabulated in Table 1.

3.1. Data assessment

3.1.1. Study periods

The identified studies came from two separate periods: 1972–1982 and 2004-present, with no studies identified between 1983 and 2003. There are several significant differences between these two periods in terms of study design. The early cannabis administration studies' sample sizes ranged from 2 to 11, with an average of 5.6 participants, while those of modern studies ranged between 8 and 73, averaging 29.7 participants. THC doses ranged from 0.2 mg/kg to 210 mg in early studies, whereas modern studies used 2.5–15 mg. All eight early studies were

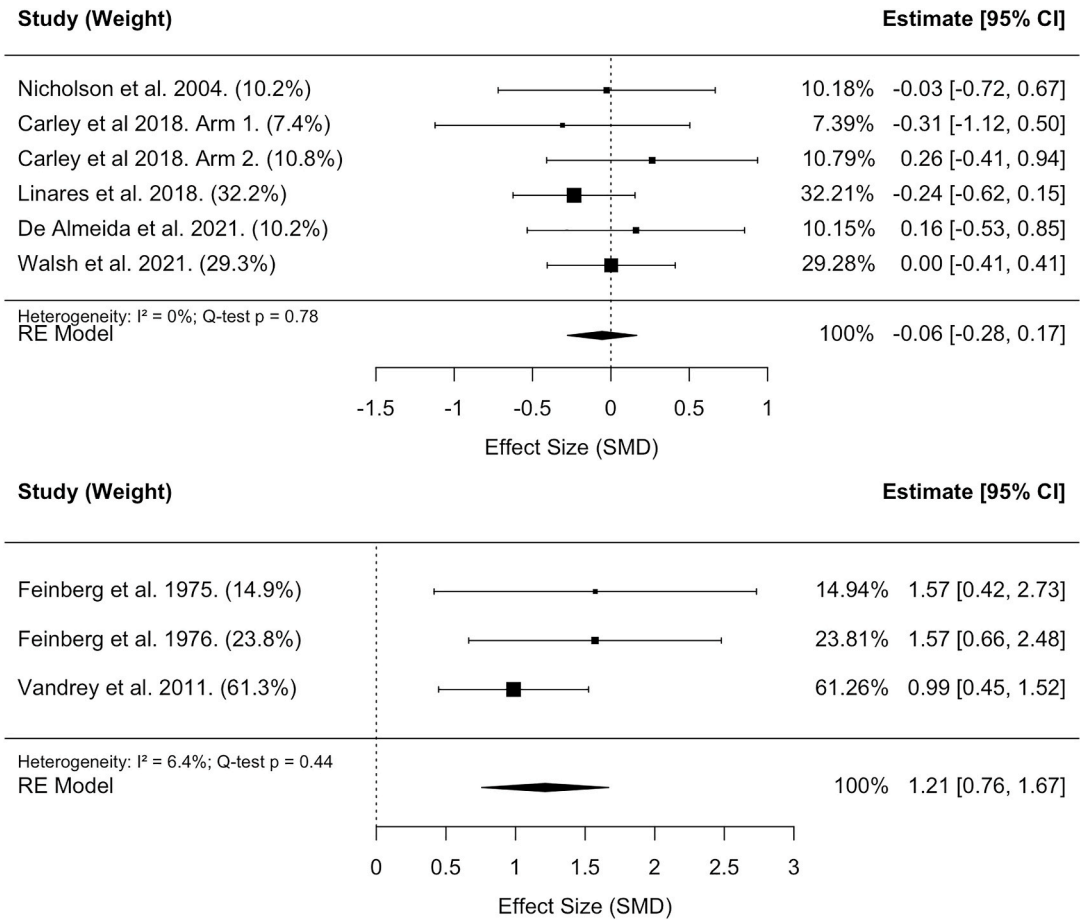

Fig. 3. a. Administration - Sleep onset latency. b. Withdrawal - Sleep onset latency.

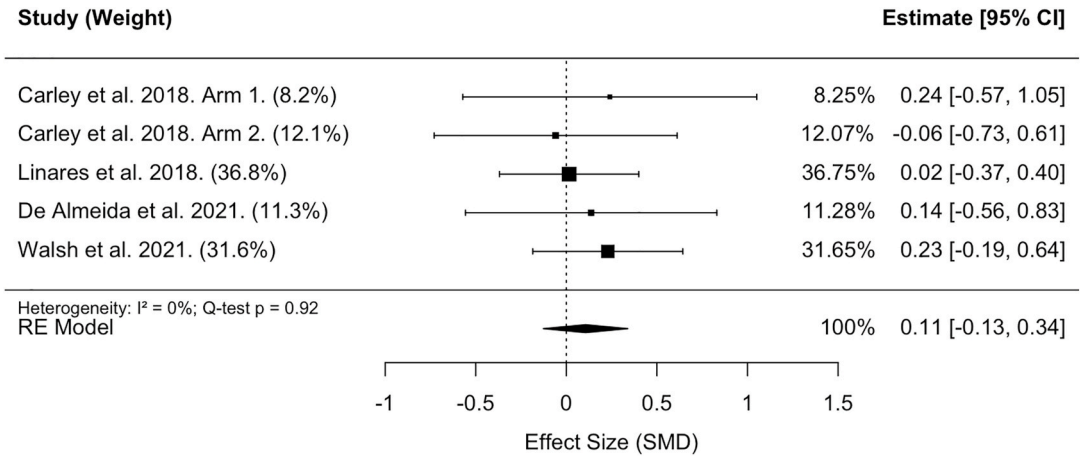

Fig. 4. Administration - Wake after sleep onset.

conducted with "healthy" adults to study sleep patterns, while four out of six modern studies addressed specific sleep conditions. Early studies all recruited "young adult" men, with participants ranging from 21 to 29 years of age; modern studies had samples that averaged 49.8 years of age and the percentage of males averaged 55.5 %.

3.1.2. Study designs

Nine studies focused on administration, one on chronic use, and one on withdrawal exclusively. Five studies examined both administration and withdrawal, and one study both chronic use and withdrawal. Five out of a total of 14 administration studies were considered randomised double-blind placebo-controlled trials [18,38–41]; three used crossover and two used parallel designs. Other administration studies used a mix of single-, double-, and unblinded as well as pre-post, crossover, and parallel designs. Every study included reported measurements collected through PSG or EEG; no eligible actigraphy-only studies were identified. One study used both PSG and actigraphy [40], where PSG data was used for meta-analysis. Comparisons between chronic cannabis users and non-users in a total of three studies were non-randomised by nature.

All seven withdrawal studies were non-randomised and employed a

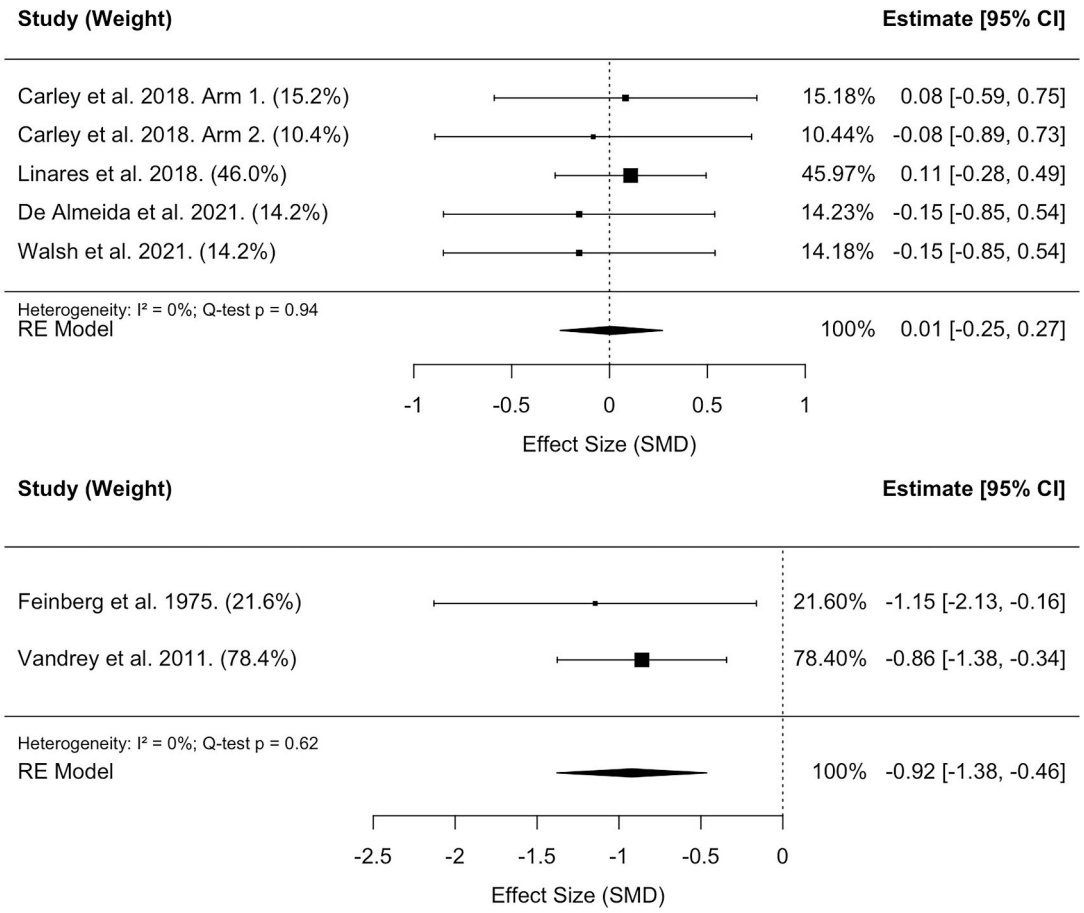

Fig. 5. aAdministration - Sleep efficiency. b. Withdrawal - Sleep efficiency.

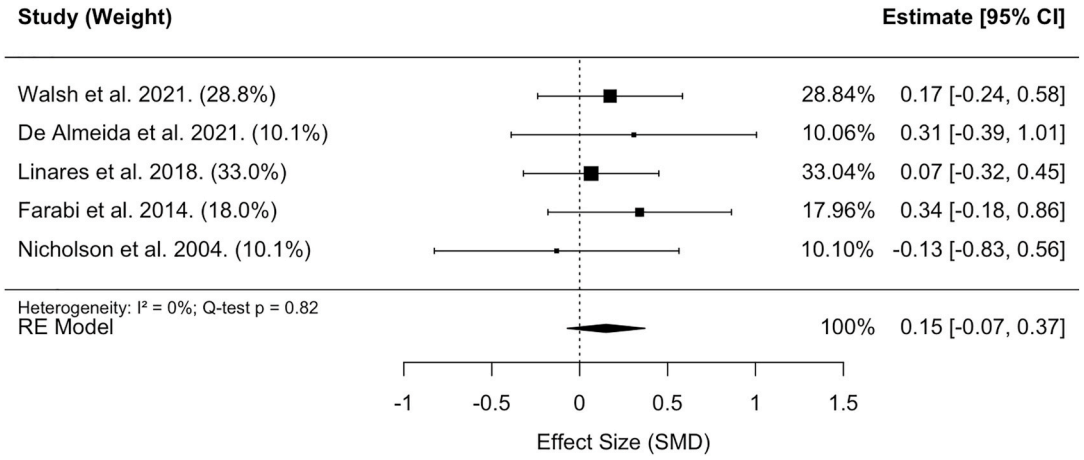

Fig. 6. Administration - N1.

mix of different designs: four used placebo controls [42–45]—replacing a cannabis capsule with a placebo capsule, inducing withdrawal; three used pre-post measures [46–48]—comparing baseline sleep before abrupt discontinuation with sleep during withdrawal, where participants were aware of cessation; and one used a control group [49]—comparing cannabis users going through withdrawal to non-users. In five studies, withdrawal effects were measured after conducting a cannabis administration phase [42–46]. Two studies specifically focused on withdrawal effects among active users [48,49].

3.1.3. *Prior use patterns*

Prior use patterns were recorded differently across cannabis administration studies. Prior use of cannabis was considered minimal in eight studies. Washout periods were employed in six of these studies [18,40,45,46,50,51], with durations ranging between two weeks [40,45,51] and two months [50], while two studies relied on self-reporting of abstinence prior to enrolment in the study [38,39]. Three studies used urinalysis to confirm abstinence from cannabis use [38,40,51]. One study included one two-year cannabis user who smoked one or two joints per week among ten cannabis-naïve subjects [52], without

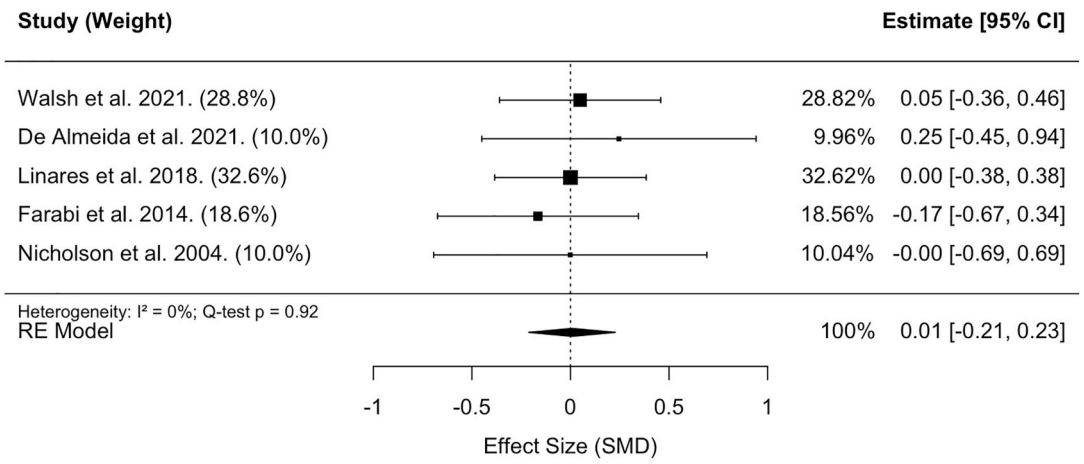

Fig. 7. Administration - N2.

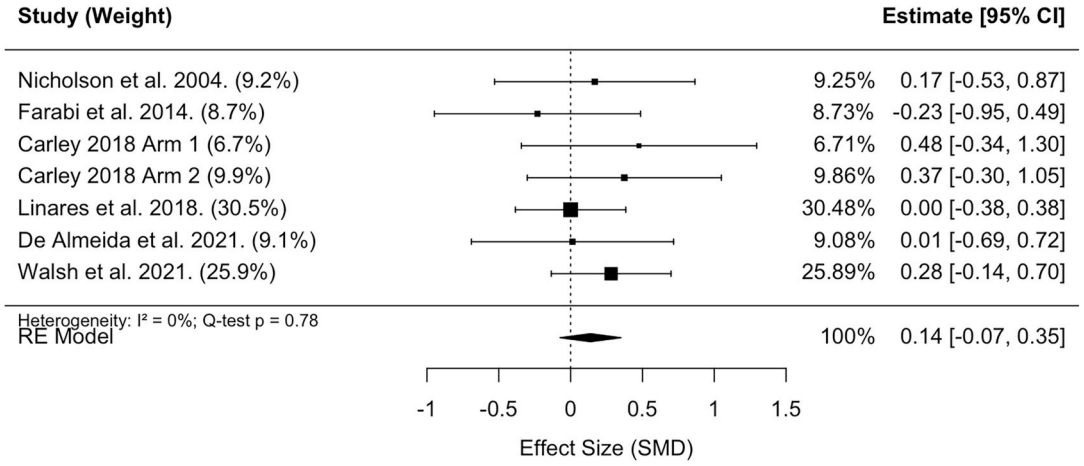

Fig. 8. Administration - Slow wave sleep.

explanation for this mixed sample. Another, which did not utilise a washout period, included two brothers who used cannabis about once monthly [44]. Two studies used "experienced marijuana users" who had a history of daily cannabis use [42,43]. Last, two studies did not specify their participants' prior use, nor confirmed they had used a washout period [41,53].

3.1.4. Bias assessment

All studies published between 1972 and 1982 showed high risk of bias, particularly due to confounding, selection of participants, and bias in measurement of outcomes. Studies comparing users and non-users were inherently at risk of bias, given the many potential confounding variables that predispose people to cannabis use. The bias assessment of non-randomised studies using ROBINS-I can be found in Table 2 and the assessment of randomised studies using RoB 2.0 in Table 3.

While the randomised trials had low risk of bias in terms of internal validity as assessed by RoB 2.0, confounding still posed a risk to the studies' results. Three of the five studies included samples of participants with sleep disorders; obstructive sleep apnea and REM behaviour disorder in Parkinson's Disease, potentially affecting results. Due to the limited number of studies included in the meta-analyses, an Egger's test for publication bias was not conducted.

3.1.5. Data for meta-analyses

Several data alterations were made to perform meta-analyses. In two studies, standard deviations were derived from p-values [42,43], and in one study from its standard error [48]. For one study, given that it used the same crossover sample to test three combinations of THC:CBD ratios (15:0; 5:5; 15:15 mg), the means and standard deviations were recalculated and pooled to not overrepresent the participants in the study [18].

For withdrawal, data from the first three days was used in two studies [42,43] and days four to six in one [48]. Withdrawal effects are theorised to primarily occur within the first few days of cannabis withdrawal [54,55], so the timing of measurement was standardised to the extent possible. For administration data, measurement timing ranged from first night [18,39] to 12 weeks [41].

All early era cannabis administration studies were ineligible for meta-analysis due to the lack of standard deviation notation and/or imprecise p-values (i.e. p = NS or p = < 0.1). Two early era studies were included in withdrawal meta-analyses, as reporting for these specific analyses was precise [42,43].

3.2. Meta-analyses and results interpretation

3.2.1. Total sleeping time

Meta-analysis of four studies [38–41] found no significant effect of cannabis administration on TST (SMD: 0.021; 95 % CI: [−0.253, 0.211], $p = 0.86$, $I^2 = 0.00$ %), indicating no heterogeneity (see Fig. 2a). Of the 12 administration studies that assessed TST, one reported a significant increase, at 13–17 mg THC (n = 4) [50], while the remaining 11 found no significant change.

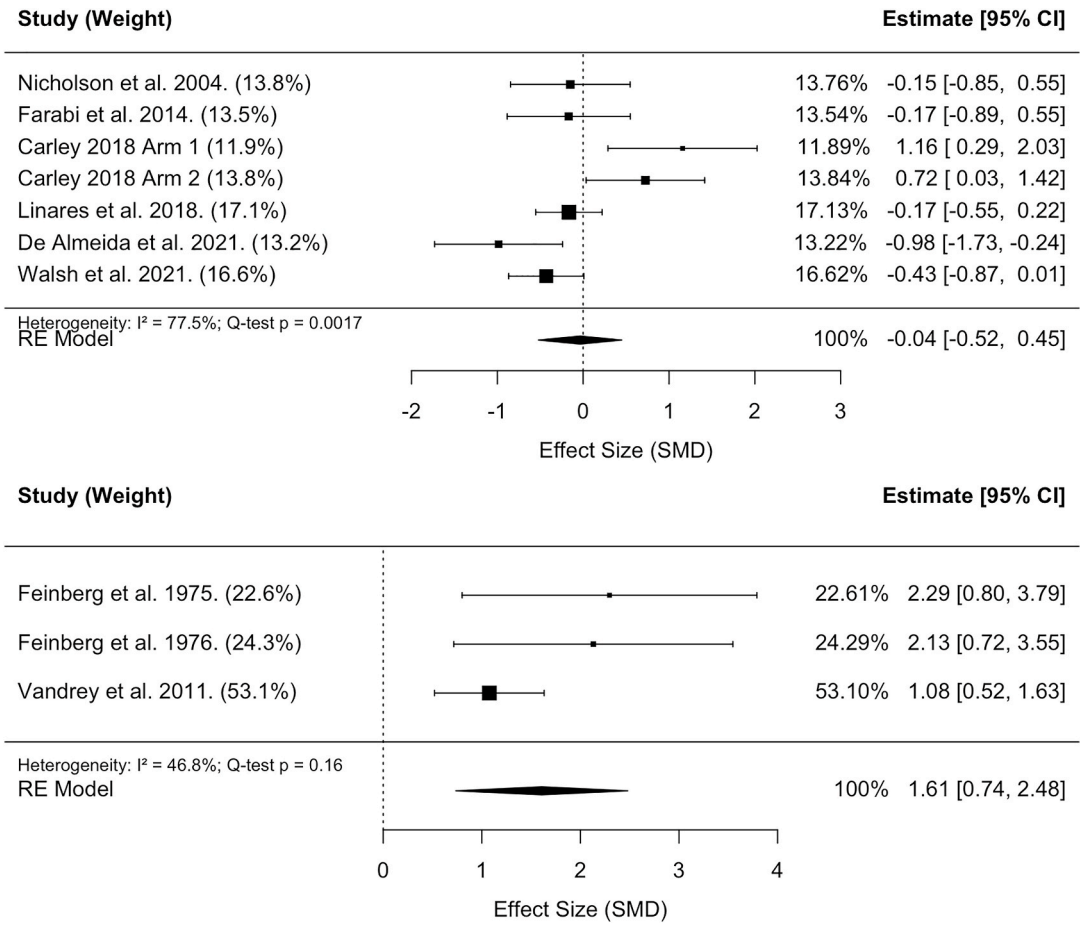

Fig. 9. a. Administration - REM sleep. b. Withdrawal - REM sleep.

In three studies comparing the sleep of cannabis users to non-users, one found shorter TST [56], with the other two finding no significant difference.

For cannabis withdrawal, pooling the results of three eligible studies for meta-analysis [42,43,48] showed a moderate to large reduction in TST (SMD = −0.794, 95 % CI: [−1.163, −0.424], $p < 0.0001$, $I^2 = 0.00$ %) with no heterogeneity (see Fig. 2b). Three out of seven withdrawal studies found a significant reduction in TST [42,43,48], with the remaining four reporting no change.

3.2.2. Sleep onset latency

Meta-analysis of five studies [18,38–41] found no significant effect of cannabis administration on SOL (SMD: 0.056; 95 % CI: [−0.277, 0.165], $p = 0.62$, $I^2 = 0$ %), with no heterogeneity (see Fig. 3a). Among the 12 administration studies that measured SOL, one reported a significant increase, at 20 mg THC (n = 2) [44], while the remaining 11 studies found no change.

Among three studies comparing cannabis users' SOL to that of non-users, none reported significant differences.

For cannabis withdrawal, pooling the results of three eligible studies for meta-analysis [42,43,48] showed a large increase in SOL (SMD = 1.213, 95 % CI: [0.757, 1.669], $p < 0.0001$, $I^2 = 6.43$ %) (see Fig. 3b). Five out of seven withdrawal studies reported increases in SOL [42–45, 48], with the remaining two reporting no change.

3.2.3. Wake after sleep onset

Meta-analysis of four studies [38–41] found no significant effect of cannabis administration on WASO (Overall SMD = 0.106, 95 % CI: [−0.127, 0.339], $p = 0.37$, $I^2 = 0.00$ %), indicating no heterogeneity (see Fig. 4). Among 13 administration studies that measured WASO, one found a statistically significant decrease, at 13–17 mg THC (n = 4) [50], with the remaining 12 studies finding no change.

One study comparing users to non-users reported WASO and found no significant difference [56].

Only one study provided the statistical reporting necessary to calculate effects for WASO, rending meta-analysis impossible. Three out of seven withdrawal studies showed an increase in WASO [42,44,45]. The remaining four studies showed no change.

3.2.4. Sleep efficiency

Meta-analysis of four studies [38–41] found no significant effect of cannabis administration on SE (SMD = −0.009, 95 % CI: [−0.252, 0.271], $p = 0.94$, $I^2 = 0.00$ %), indicating no heterogeneity (see Fig. 5a). None of the six administration studies measuring SE showed a significant effect.

Two studies comparing cannabis users to non-users included measures of sleep efficiency, finding no significant differences.

Pooling effects of two studies eligible for meta-analysis for cannabis withdrawal [42,48] showed a large reduction in SE (SMD = −0.922, 95 % CI: [−1.379, −0.464], $p < 0.0001$, $I^2 = 0.00$ %) with no heterogeneity (see Fig. 5b). Out of three studies reporting on SE in cannabis withdrawal, two reported a decrease [42,48].

3.2.5. N1 sleep

Meta-analysis of five studies [18,39–41,53] found no significant effect of cannabis administration on N1 (SMD = 0.150, 95 % CI: [−0.071, 0.372], $p = 0.18$, $I^2 = 0.00$ %), indicating no heterogeneity (see Fig. 6). Two out of eight administration studies reporting on N1 sleep found decreases, at 13–17 mg THC (n = 4) [50] and 0.7–1.4 mg/kg THC (n = 11) [52], while in the remaining six studies no change was found.

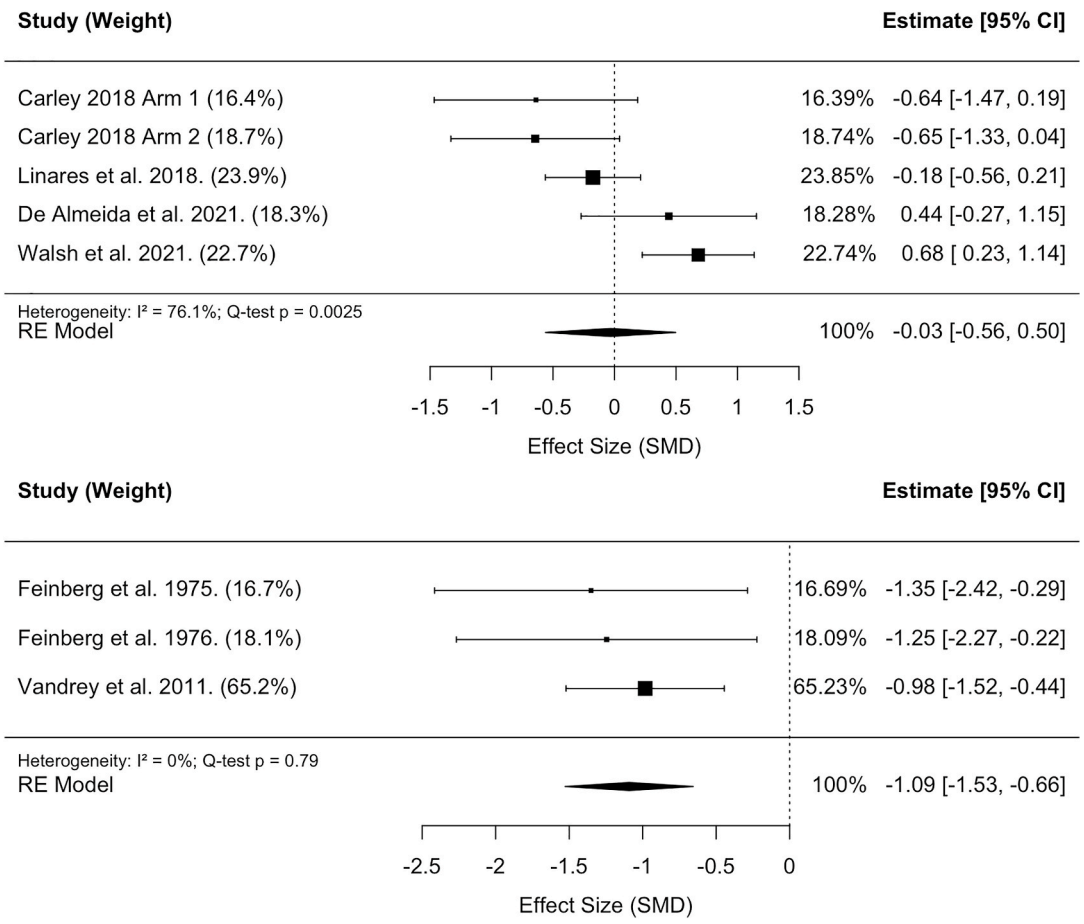

Fig. 10. a. Administration - REM latency. b. Withdrawal - REM latency.

In the three studies comparing cannabis users to non-users no differences were found in N1 sleep.

For withdrawal, none of the studies provided the statistical reporting necessary to calculate effects for N1, rending meta-analysis impossible. A total of two studies reported on N1, finding no change.

3.2.6. *N2 sleep*

Meta-analysis of five studies [18,39–41,53] found no significant effect of cannabis administration on N2 (SMD = 0.008, 95 % CI: [−0.212, 0.227], p = 0.94, I^2 = 0.00 %), indicating no heterogeneity (see Fig. 7). In 11 studies reporting N2, two reported an increase, at 0.7–1.4 mg/kg THC (n = 11) [52] and 30 mg THC (n = 2) [45], and one a decrease [53], at 2.5–10 mg dronabinol (n = 15) [53], with the remaining eight studies reporting no change.

In the three studies comparing cannabis users to non-users no differences were found in N2 sleep.

For withdrawal, none of the studies provided the statistical reporting necessary to calculate effects for N2, rending meta-analysis impossible. Qualitatively, one study found an increase [45] and one a decrease [49] in N2, while three found no change.

3.2.7. *Slow wave sleep*

Pooling results of six eligible studies [18,38–41,53] showed no significant effect of cannabis administration on SWS (SMD = 0.138, 95 % CI: [−0.074, 0.351], p = 0.201, I^2 = 0.00 %), with no heterogeneity (see Fig. 8). Among the 14 administration studies, one study showed an increase, at 13–17 mg THC (n = 4) [50], two studies showed a decrease, at 0.7–1.4 mg/kg THC (n = 11) [52] and 30 mg THC (n = 2) [45], with the remaining 11 studies showing no change.

Comparing cannabis users and non-users, one study showed lower SWS [56], and two no change.

Due to only one study providing the statistical reporting necessary to calculate effects for SWS, no meta-analysis was performed. Among the seven withdrawal studies, one showed an increase [49] and two a decrease [44,45], with the remaining four studies showing no change.

3.2.8. *REM sleep*

Meta-analysis of six studies [18,38–41,53] found no significant effect of cannabis administration on REM sleep (Overall SMD: 0.0356; 95 % CI: [−0.521, 0.449], p = 0.89, I^2 = 77.48 %), with high heterogeneity (see Fig. 9a), which sensitivity analyses did not resolve. Among the 14 administration studies, six studies showed a decrease, at 13–17 mg THC (n = 4) [50], 20 mg THC (n = 5) [46], 70–210 mg THC (n = 7 [42]; n = 11 [43]), 0.7–1 mg/kg THC (n = 7) [52], and 300 mg CBD (n = 17) [41], and one an increase, at 2.5–10 mg dronabinol (n = 48) [38], with the remaining seven studies finding no change.

In studies comparing cannabis users and non-users, one study showed higher REM among cannabis users [57], with the other two showing no significant differences.

For withdrawal, the pooled result of three studies eligible for meta-analysis [42,43,48] showed a large increase in REM (SMD = 1.609, 95 % CI: [0.737, 2.479], p = 0.003, I^2 = 46.8 %), with moderate heterogeneity (see Fig. 9b). Three out of seven withdrawal studies showed an increase [42,43,48], with four studies showing no change.

3.2.9. *REM latency*

Meta-analysis of four eligible studies on the effects of cannabis administration on REM latency [38–41] showed no significant effect (SMD = −0.032, 95 % CI: [−0.561, 0.497], p = 0.906, I^2 = 76.12 %), with no heterogeneity (see Fig. 10a), which sensitivity analyses did not

resolve. One study showed a decrease, at 70–210 mg THC (n = 11) [43], one an increase, at 10:1:0.5 mg THC:CBN:CBD (n = 23) [40], with six studies showing no change.

In studies comparing cannabis users to non-users, none of the three available studies found a significant difference in REM latency.

For withdrawal, meta-analysis of three eligible studies [42,43,48] showed a large decrease in REM latency (SMD = −1.092, 95 % CI: [−1.527, −0.657], $p < 0.0001$, $I^2 = 0$ %), with no heterogeneity (see Fig. 10b). Three out of four studies measuring REM latency showed a decrease [42,43,48], with one study showing no change.

3.2.10. Sensitivity analyses

For cannabis administration meta-analyses, removing two studies that failed to specify prior use [41,53] and/or two CBD-only studies [39,41] did not change results in terms of statistical significance for any measured outcome. Heterogeneity was also not significantly reduced by the removal of these studies for REM sleep or REM latency.

4. Discussion

This review highlights both the complexity and scarcity of the evidence surrounding cannabis use and sleep architecture. Cannabis administration did not produce consistent effects on any key sleep parameter. While some studies reported REM impairments, these were mainly observed in trials with notable methodological limitations, such as small sample sizes, excessive dosing, and study designs that may have influenced sleep outcomes. In contrast, evidence regarding cannabis withdrawal, though sparse, was more consistent, demonstrating reductions in total sleep duration and increases in sleep onset latency and REM.

The two eras in which the studies were conducted, 1972–1982 and 2004 onward, differ significantly in terms of design, dose, and outcome. Early studies largely focused on the mechanistic effect of cannabis on sleep, used mostly high-dose THC, and frequently reported REM suppression, while the latter period consists of low-dose cannabis trials to treat specific sleep disorders, often finding no change in sleep architecture. The 22-year hiatus in research coincides with the height of the US 'War on Drugs,' which Ronald Reagan officially declared in 1982 [58]; the same year the last scientific journal article of the early period was published [45]. The revival of human laboratory studies on cannabis post-2000 coincided with shifting public attitudes and the expanding legalisation of cannabis in the US [59], as well as the emergence of standardised oral and spray-based medical cannabinoid preparations and a move away from smoked administration, which enabled more controlled research designs (despite ongoing regulatory constraints) [60]. That said, few clinical trials utilising polysomnography to study the effects of cannabis have been published in the past two decades, and the evidence base for cannabis' effects on sleep architecture remains thin: only six administration and three withdrawal studies in this review were deemed suitable for meta-analysis.

Several methodological limitations stand out in the studies that found REM impairments. Reporting "relatively slight" REM decrements, Pivik et al. used a very small sample size (n = 4) and used no formal significance testing, instead relying on a binomial test of rankings, limiting its reliability and generalisability [50]. Similarly, Freemon observed initial REM suppression in a very small sample (n = 5) and found that among the three participants who received THC for six consecutive nights, REM sleep exceeded baseline in one, returned to baseline in another, and remained suppressed in the third [46]. In two studies, Feinberg et al. used very high doses of THC, at 70–210 mg among small samples (n = 7) [42] (n = 11) [43], where the authors noted that REM suppression was dose-dependent and exhibited partial tolerance over time. They also acknowledged that their protocol, which involved waking participants up at 4 a.m. for THC administration, may have fragmented sleep and disrupted REM. A "complete suppression" of REM sleep was reported by Tassinari et al. [52], in another small sample using cannabis-naive subjects (n = 7) who received very high single oral doses of 0.7–1 mg/kg THC (~45–75 mg), where the authors acknowledged that "the heavy doses given resulted in such severe intoxication with clinical and neurological manifestations that our study could hardly be compared with others in the literature" [52], limiting the generalisability of their findings.

The serious methodological limitations of these studies complicate interpretations about REM sleep. A daily dose of 210 mg THC, as used by Feinberg et al. [42,43], is more than twenty times what is considered a "single serving of an edible" by many legislatures today [61], so it is questionable whether these findings constitute reliable evidence for the effects of cannabis on REM sleep under real-world dosing conditions. However, it is worth noting that among chronic cannabis users the median daily THC intake is estimated to be approximately 90–150 mg [62–65], with a 2024 survey suggesting the upper quantile of cannabis users in the US consume ≥290 mg daily [65], suggesting that the doses used in these studies may be somewhat representative of habitual heavy use. Whether these study designs realistically model real-world consumption patterns is debatable.

In contrast to early studies, none of the four included modern studies that administered THC reported REM suppression, with one study showing a REM increase [38]. However, this study was conducted with participants with obstructive sleep apnea, where THC may reduce apnea severity by stabilising upper airway muscle tone through its interaction with the endocannabinoid system [35], such that the observed REM increase may be a secondary effect of improved sleep continuity. It is plausible that the absence of REM suppression in modern studies reflects pharmacological tolerance. Most of these studies measured effects after a period of up to 12 weeks, at which point tolerance to THC's REM-suppressing effects may have emerged. Indeed, early high THC studies reporting REM reductions documented partial or complete tolerance over time in certain subjects [42,43,46].

THC's effects on REM may also be dose-dependent, such that a single therapeutic low dose of 2.5–15 mg THC at night may cause minimal or no REM impairment, as observed in modern studies, whereas high-dose THC may lead to more pronounced disruptions, as observed in early studies. While no formal quantitative dose-response assessment can be established in this review due to limited statistical reporting in early trials, the lowest THC dose associated with a statistically significant reduction in REM sleep was 20 mg [46], with all other such findings at ≥30 mg. This pattern aligns with literature suggesting that the therapeutic window for THC typically falls below 20 mg [66], and that doses exceeding 20 mg THC tend to produce more noticeable adverse effects [22]. That said, a 2025 study has reported a reduction in REM after a single THC:CBD 10:200 mg capsule in insomnia patients on the first night [67], and in our review, Walsh et al. reported directional evidence of REM suppression following 14 nights of insomnia treatment with a THC:CBN:CBD 10:1:0.5 mg formulation, with the effect falling just short of statistical significance ($p = 0.055$) [40].

It should also be noted that stage-based metrics alone do not fully capture the effects of cannabis on sleep architecture, as the neural quality of each stage may also be altered. For example, high-density EEG analysis has shown that cannabis use reduces delta power during N3 sleep and increases alpha and beta activity during REM sleep [67]; changes associated with lighter, less restorative sleep. Similarly, in OSA patients, dronabinol shifted EEG power toward theta frequencies and decreased sigma power, while strengthening ultradian oscillations, suggesting altered sleep depth and structure not detectable through stage scoring alone [53].

Another line of evidence to support claims about suppressed REM sleep is the observation of REM rebounds upon withdrawal. Indeed, two early studies and one modern study reported these [42,43,48], though it has been suggested that the rebound observed in these studies may be accentuated by their use of very high oral THC doses [47]. Further, increased nightmares and vivid dreaming upon withdrawal are commonly reported by users [17,68,69], which aligns with the REM

Practice Points

1. Cannabis administration shows no consistent effect on sleep parameters, including total sleeping time, sleep onset latency, wake after sleep onset, light sleep, deep sleep, and sleep efficiency.
2. High-dose 1970s trials consistently showed THC-related REM suppression; in modern therapeutic-dose trials REM suppression has been reported, but findings are mixed and the overall evidence base remains thin.
3. Withdrawal from chronic cannabis use significantly disrupts sleep, reducing total sleeping time and increasing sleep onset latency, though polysomnographic evidence is scarce.
4. Variability in outcomes across studies is influenced by differences in dosages, study designs, and participant characteristics—particularly prior cannabis use and health conditions.

rebound hypothesis. Similarly, administration of dronabinol has been found to reduce nightmare frequency and intensity in several studies on patients with post-traumatic stress disorder (PTSD) [70], suggesting possible REM suppression, though it is not clear that cannabis suppresses nightmares for individuals who do not have PTSD [71].

Although numerous studies have assessed the sleep of cannabis users with subjective measures [72–75], and the perception that cannabis can improve sleep onset appears common [76,77], few have compared the sleep architecture of users to that of non-users using actigraphy or polysomnography. Among the three studies meeting inclusion criteria in this review, none reported significant differences in sleep parameters. One possible explanation for this discrepancy is that cannabis may exert indirect effects on sleep by alleviating symptoms such as pain and anxiety [25]. Our review suggests that no polysomnographic studies have specifically examined individuals with these conditions, despite prior research suggesting that a majority of daily cannabis users consume the substance specifically to manage these symptoms [3].

Reflecting a previous review article [36], our meta-analyses aggregated results from studies examining both THC and CBD. However, sensitivity analyses suggested that separating the studies had minimal impact—likely due to the small number of CBD-studies and the large number of null findings. Evidence for CBD remains too thin for firm guidance: only two eligible CBD-only trials were identified, with one showing a modest reduction in REM sleep [41]. Further, it should be noted that the studies included do not reflect the most common method of use—inhalation [78]. Indeed, the clinical literature almost exclusively studies oral consumption [22,36].

For specific sleep disorders, it is too early to comment on how cannabis may affect outcomes. To our knowledge, there are no studies employing cannabis to treat periodic limb movement disorder, narcolepsy [79], or parasomnias. There have been reports of cannabis improving restless legs syndrome [80], but this is yet to be researched in clinical trials [81]. While short-term benefits for obstructive sleep apnea have been suggested [33], the *American Academy of Sleep Medicine* has made a position statement that cannabis should not be used for the treatment of sleep apnea due to insufficient evidence of effectiveness, tolerability, and safety [82], as others have noted that sleep apnea being added as a qualifying condition for medical cannabis in certain US states was premature and potentially harmful [83].

We acknowledge that many of the studies included in this review are at high risk of bias, use diverse cannabis types, dosages, and (likely) purity levels (which are not commonly reported), as well as include participants with different conditions. However, we believe it is of value to quantitatively synthesise the existing evidence for each sleep parameter to gain a better understanding of where findings align and where and why they conflict. We also believe it is important to differentiate between the evidence base stemming from the 1970s and the modern era, which Gates et al. alluded to in a previous review [84]. Indeed, the meta-analyses in this review are biased towards modern studies, as all early era administration studies lacked the statistical reporting necessary to meet meta-analysis inclusion criteria.

In sum, our results suggest that clinicians should not view cannabis as a reliable sleep aid. Administration trials suggest that a single dose does not reliably improve a patient's sleep, and user-versus-non-user comparisons do not show significantly improved sleep onset or other such changes, with both lines of evidence converging on negligible changes in sleep architecture. By contrast, withdrawal effects appear to be more consistent, with moderate-to-large decrements in total sleeping time and sleep onset in the first week of cessation. However, these quantitative estimates derive from tiny cohorts and should be interpreted cautiously. Clinicians should therefore prepare regular users for transient insomnia when quitting, pair tapering schedules with behavioural sleep interventions (such as CBT-I), and avoid presenting cannabis as a benign alternative to licensed hypnotics.

Next-step trials should be adequately powered and preregistered, clearly specify formulation, route, and dose (including real-world inhaled and self-titrated bedtime use), stratify by prior use and clinical indication (focusing especially on those with pain and anxiety disorders), and incorporate spectral EEG to resolve mixed findings. As cannabis is increasingly used as a sleep aid by high school and college students across North America [6,85] and its use continues to expand globally [86,87], addressing these methodological and evidence gaps is essential for guiding clinical recommendations and shaping public health policy.

Research Agenda

1. Future research should employ standardised protocols, including washout periods, pre-specified dosing regimens, explicit cannabinoid profiles, route of administration (including inhaled), and participant selection criteria, to improve comparability across studies.
2. Future research should more closely investigate the effects of THC dosing on REM sleep.
3. Future research should address the gap between subjective improvements in sleep quality and the lack of improvements in objective measurements. For example, studies using polysomnography might be performed with populations that note subjective improvements in sleep when using cannabis, such as those with pain or anxiety disorders.

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References

[1] Goodman S, Wadsworth E, Leos-Toro C, Hammond D. Prevalence and forms of cannabis use in legal vs. illegal recreational cannabis markets. Int J Drug Pol 2020 Feb 1;76:102658.
[2] Leung J, Chan G, Stjepanović D, Chung JYC, Hall W, Hammond D. Prevalence and self-reported reasons of cannabis use for medical purposes in USA and Canada. Psychopharmacology (Berl) 2022 May 1;239(5):1509–19.
[3] Walsh Z, Callaway R, Belle-Isle L, Capler R, Kay R, Lucas P, et al. Cannabis for therapeutic purposes: patient characteristics, access, and reasons for use. Int J Drug Pol 2013;24(6):511–6.
[4] Turna J, Balodis I, Munn C, Van Ameringen M, Busse J, MacKillop J. Overlapping patterns of recreational and medical cannabis use in a large community sample of cannabis users. Compr Psychiatry 2020 Oct;102:152188.
[5] Lintzeris N, Driels J, Elias N, Arnold JC, McGregor IS, Allsop DJ. Medicinal cannabis in Australia, 2016: the cannabis as medicine survey (CAMS-16). Med J Aust 2018;209(5):211–6.
[6] Goodhines PA, Wedel AV, Dobani F, Zaso MJ, Gellis LA, Park A. Cannabis use for sleep aid among high school students: Concurrent and prospective associations with substance use and sleep problems. Addict Behav 2022 Nov 1;134:107427.
[7] Cuttler C, Spradlin A, McLaughlin RJ. A naturalistic examination of the perceived effects of cannabis on negative affect. J Affect Disord 2018 Aug 1;235:198–205.
[8] Hall W, Degenhardt L. The adverse health effects of chronic cannabis use. Drug Test Anal 2014;6(1–2):39–45.
[9] Gobbi G, Atkin T, Zytynski T, Wang S, Askari S, Boruff J, et al. Association of cannabis Use in adolescence and risk of depression, anxiety, and Suicidality in young adulthood: a systematic review and meta-analysis. JAMA Psychiatry 2019 Apr 1;76(4):426–34.
[10] Hall W, Degenhardt L. Cannabis use and the risk of developing a psychotic disorder. World Psychiatry 2008 Jun;7(2):68–71.
[11] Hasan A, von Keller R, Friemel CM, Hall W, Schneider M, Koethe D, et al. Cannabis use and psychosis: a review of reviews. Eur Arch Psychiatr Clin Neurosci 2020 Jun 1;270(4):403–12.
[12] Beletsky A, Liu C, Lochte B, Samuel N, Grant I. Cannabis and anxiety: a critical review. Med Cannabis Cannabinoids 2024 Feb 23;7(1):19–30.
[13] Walsh Z, Gonzalez R, Crosby K, Thiessen M S, Carroll C, Bonn-Miller MO. Medical cannabis and mental health: a guided systematic review. Clin Psychol Rev 2017 Feb;51:15–29.
[14] Figueiredo PR, Tolomeo S, Steele JD, Baldacchino A. Neurocognitive consequences of chronic cannabis use: a systematic review and meta-analysis. Neurosci Biobehav Rev 2020 Jan 1;108:358–69.
[15] Wieghorst A, Roessler KK, Hendricks O, Andersen TE. The effect of medical cannabis on cognitive functions: a systematic review. Syst Rev 2022 Oct 3;11(1):210.
[16] Ogeil RP, Phillips JG, Rajaratnam SMW, Broadbear JH. Risky drug use and effects on sleep quality and daytime sleepiness. Hum Psychopharmacol Clin Exp 2015;30(5):356–63.
[17] Gates P, Albertella L, Copeland J. Cannabis withdrawal and sleep: a systematic review of human studies. Subst Abuse 2016 Jan 1;37(1):255–69.
[18] Nicholson AN, Turner C, Stone BM, Robson PJ. Effect of Δ-9-tetrahydrocannabinol and cannabidiol on Nocturnal sleep and early-morning behavior in young adults. J Clin Psychopharmacol 2004 Jun;24(3):305.
[19] Atakan Z. Cannabis, a complex plant: different compounds and different effects on individuals. Ther Adv Psychopharmacol 2012 Dec 1;2(6):241–54.
[20] Brunt TM, van Genugten M, Höner-Snoeken K, van de Velde MJ, Niesink RJM. Therapeutic satisfaction and subjective effects of different strains of pharmaceutical-grade cannabis. J Clin Psychopharmacol 2014 Jun;34(3):344.
[21] Lavender I, Garden G, Grunstein RR, Yee BJ, Hoyos CM. Using cannabis and CBD to sleep: an updated review. Curr Psychiatry Rep 2024 Dec 1;26(12):712–27.
[22] Velzeboer R, Malas A, Boerkoel P, Cullen K, Hawkins M, Roesler J, et al. Cannabis dosing and administration for sleep: a systematic review. Sleep 2022 Nov 9;45(11):zsac218.
[23] Bruni N, Della Pepa C, Oliaro-Bosso S, Pessione E, Gastaldi D, Dosio F. Cannabinoid delivery systems for pain and inflammation treatment. Molecules 2018 Oct;23(10):2478.
[24] Ware MA, Wang T, Shapiro S, Robinson A, Ducruet T, Huynh T, et al. Smoked cannabis for chronic neuropathic pain: a randomized controlled trial. Can Med Assoc J 2010 Oct 5;182(14):E694–701.
[25] Sznitman SR, Shochat T, Greene T. Is time elapsed between cannabis use and sleep start time associated with sleep continuity? An experience sampling method. Drug Alcohol Depend 2020 Mar 1;208:107846.
[26] Cudney LE, Frey BN, McCabe RE, Green SM. Investigating the relationship between objective measures of sleep and self-report sleep quality in healthy adults: a review. J Clin Sleep Med 2022;18(3):927–36.
[27] Zinkhan M, Berger K, Hense S, Nagel M, Obst A, Koch B, et al. Agreement of different methods for assessing sleep characteristics: a comparison of two actigraphs, wrist and hip placement, and self-report with polysomnography. Sleep Med 2014 Sep 1;15(9):1107–14.
[28] Haupts M, Vila C, Jonas A, Witte K, Álvarez-Ossorio L. Influence of previous failed antispasticity therapy on the efficacy and tolerability of THC:CBD oromucosal spray for multiple sclerosis spasticity. Eur Neurol 2016 May 10;75(5–6):236–43.
[29] van der Helm E, Yao J, Dutt S, Rao V, Saletin JM, Walker MP. REM sleep depotentiates amygdala activity to previous emotional experiences. Curr Biol 2011 Dec;21(23):2029–32.
[30] Gujar N, McDonald SA, Nishida M, Walker MP. A role for REM sleep in recalibrating the sensitivity of the human brain to specific emotions. Cerebr Cortex 2011 Jan 1;21(1):115–23.
[31] Rundo JV, Downey R. Chapter 25 - polysomnography. In: Levin KH, Chauvel P, editors. Handbook of clinical neurology [internet], vol. 160. Elsevier; 2019. p. 381–92 (Clinical Neurophysiology: Basis and Technical Aspects; [cited 2024 Feb 23], https://www.sciencedirect.com/science/article/pii/B9780444640321000254.
[32] Edwards D, Filbey FM. Are sweet dreams made of these? Understanding the relationship between sleep and cannabis use. Cannabis Cannabinoid Res 2021 Dec 9;6(6):462–73.
[33] Babson KA, Sottile J, Morabito D. Cannabis, cannabinoids, and sleep: a review of the literature. Curr Psychiatry Rep 2017 Apr;19(4):23.
[34] Garcia AN, Salloum IM. Polysomnographic sleep disturbances in nicotine, caffeine, alcohol, cocaine, opioid, and cannabis use: a focused review. Am J Addict 2015;24(7):590–8.
[35] Suraev AS, Marshall NS, Vandrey R, McCartney D, Benson MJ, McGregor IS, et al. Cannabinoid therapies in the management of sleep disorders: a systematic review of preclinical and clinical studies. Sleep Med Rev 2020 Oct;53:101339.
[36] AminiLari M, Wang L, Neumark S, Adli T, Couban RJ, Giangregorio A, et al. Medical cannabis and cannabinoids for impaired sleep: a systematic review and meta-analysis of randomized clinical trials. Sleep 2022 Feb 1;45(2):zsab234.
[37] Bramer WM, Rethlefsen ML, Kleijnen J, Franco OH. Optimal database combinations for literature searches in systematic reviews: a prospective exploratory study. Syst Rev 2017 Dec 6;6(1):245.
[38] Carley DW, Prasad B, Reid KJ, Malkani R, Attarian H, Abbott SM, et al. Pharmacotherapy of apnea by cannabimimetic enhancement, the PACE clinical trial: effects of dronabinol in obstructive sleep apnea. Sleep 2018 Jan 1;41(1):zsx184.
[39] Linares IMP, Guimaraes FS, Eckeli A, Zuardi AW, Souza JDS, Hallak JE, et al. No acute effects of cannabidiol on the sleep-wake cycle of healthy subjects: a randomized, double-blind, placebo-controlled, crossover study. Front Pharmacol 2018 Apr 5;9:315.
[40] Walsh JH, Maddison KJ, Rankin T, Murray K, McArdle N, Ree MJ, et al. Treating insomnia symptoms with medicinal cannabis: a randomized, crossover trial of the efficacy of a cannabinoid medicine compared with placebo. Sleep 2021 Nov 1;44(11):zsab149.
[41] de Almeida CMO, Brito MMC, Bosaipo NB, Pimentel AV, Tumas V, Zuardi AW, et al. Cannabidiol for rapid eye movement sleep behavior disorder. Mov Disord 2021;36(7):1711–5.
[42] Feinberg I, Jones R, Walker JM, Cavness C, March J. Effects of high dosage delta-9-tetrahydrocannabinol on sleep patterns in man. Clin Pharmacol Ther 1975;17(4):458–66.
[43] Feinberg I, Jones R, Walker J, Cavness C, Floyd T. Effeets of marijuana extract and tetrahydrocannabinol on electroencephalographic sleep patterns. Clin Pharmacol Ther 1976;19(6):782–94.
[44] Freemon FR, Al-Marashi MS. Long-term changes in the sleep of normal volunteers administered multiple doses of delta-9-tetrahydrocannabinol. Drug Alcohol Depend 1977;2(1):39–43.
[45] Freemon FR. The effect of chronically administered delta-9-tetrahydrocannabinol upon the polygraphically monitored sleep of normal volunteers. Drug Alcohol Depend 1982;10(4):345–53.
[46] Freemon FR. The effect of δ9-tetrahydrocannabinol on sleep. Psychopharmacologia 1974 Mar 1;35(1):39–44.
[47] Bolla KI, Lesage SR, Gamaldo CE, Neubauer DN, Wang NY, Funderburk FR, et al. Polysomnogram changes in marijuana users who report sleep disturbances during prior abstinence. Sleep Med 2010 Oct 1;11(9):882–9.
[48] Vandrey R, Smith MT, McCann UD, Budney AJ, Curran EM. Sleep disturbance and the effects of extended-release zolpidem during cannabis withdrawal. Drug Alcohol Depend 2011 Aug 1;117(1):38–44.
[49] Pranikoff K, Karacan I, Larson EA, Williams RL, Thornby JI, Hursch CJ. Effects of marijuana smoking on the sleep EEG. Preliminary studies. JFMA J Fla Med Assoc Fla Med Assoc 1973 Mar;60(3):28–31.
[50] Pivik RT, Zarcone V, Dement WC, Hollister LE. Delta-9-tetrahydrocannabinol and synhexl: effects on human sleep patterns. Clin Pharmacol Ther 1972;13(3):426–35.
[51] Hosko MJ, Kochar MS, Wang RI. Effects of orally administered delta-9-tetrahydrocannabinol in man. Clin Pharmacol Ther 1973;14(3):344–52.
[52] Tassinari CA, Ambrosetto G, Peraita-Adrado MR, Gastaut H. The neuropsychiatric syndrome of δ9-tetrahydrocannabinol and cannabis intoxication in naive subjects. In: Nahas GG, Sutin KM, Harvey D, Agurell S, Pace N, Cancro R, editors. Marihuana and medicine [internet]. Totowa, NJ: Humana Press; 1976. p. 649–64. https://doi.org/10.1007/978-1-59259-710-9_64 [cited 2025 Jan 6].
[53] Farabi SS, Prasad B, Quinn L, Carley DW. Impact of dronabinol on quantitative electroencephalogram (qEEG) measures of sleep in obstructive sleep apnea syndrome. J Clin Sleep Med 2014;10(1):49–56.
[54] Bonnet U, Preuss UW. The cannabis withdrawal syndrome: current insights. Subst Abuse Rehabil 2017 Apr 27;8:9–37.
[55] Connor JP, Stjepanović D, Budney AJ, Le Foll B, Hall WD. Clinical management of cannabis withdrawal. Addiction 2022;117(7):2075–95.

[56] Bolla KI, Lesage SR, Gamaldo CE, Neubauer DN, Funderburk FR, Cadet JL, et al. Sleep disturbance in heavy marijuana users. Sleep 2008 Jun;31(6):901–8.
[57] Karacan I, Fernández-Salas A, Coggins WJ, Carter WE, Williams RL, Thornby JI, et al. Sleep electroencephalographic-electrooculographic characteristics of chronic marijuana users: Part I. Ann N Y Acad Sci 1976;282(1):348–74.
[58] Schoenfeld H. The war on Drugs, the politics of crime, and mass incarceration in the United States. J Gend Race Justice 2012;15:315.
[59] Lashley K, Pollock TG. Waiting to inhale: reducing stigma in the medical cannabis industry. Adm Sci Q 2020;65(2):434–82.
[60] Pisanti S, Bifulco M. Modern history of medical cannabis: from widespread use to prohibitionism and back. Trends Pharmacol Sci 2017 Mar 1;38(3):195–8.
[61] Barrus DG, Capogrossi KL, Cates SC, Gourdet CK, Peiper NC, Novak SP, et al. Tasty THC: promises and challenges of cannabis edibles. Methods rep, RTI. Press; 2016 Nov;2016. https://doi.org/10.3768/rtipress.2016.op.0035.1611.
[62] Mehndiratta SS, Wig NN. Psychosocial effects of longterm cannabis use in India A study of fifty heavy users and controls. Drug Alcohol Depend 1975 Sep 1;1(1):71–81.
[63] Budney AJ, Borodovsky JT, Struble CA, Habib MI, Shmulewitz D, Livne O, et al. Estimating THC consumption from smoked and vaped cannabis products in an online survey of adults who use cannabis. Cannabis Cannabinoid Res 2024 Apr;9(2):688–98.
[64] Larsen SF, Johnson AJ, Larimer ME, Dager SR, Kleinhans NM. Self-report methodology for quantifying standardized cannabis consumption in milligrams delta-9-tetrahydrocannabinol. Am J Drug Alcohol Abuse 2023 Nov 2;49(6):723–32.
[65] Borodovsky JT, Hasin DS, Wall M, Struble CA, Habib MI, Livne O, et al. Quantity of delta-9-tetrahydrocannabinol consumption and cannabis use disorder among daily cannabis consumers. Addiction 2025 Apr;120(4):676–85.
[66] Arnold JC, Nation T, McGregor IS. Prescribing medicinal cannabis. Aust Prescr 2020 Oct;43(5):152–9.
[67] Suraev A, McGregor IS, McCartney D, Marshall NS, Kao CH, Wassing R, et al. Acute effects of oral cannabinoids on sleep and high-density EEG in insomnia: a pilot randomised controlled trial. J Sleep Res 2025 Jul 9:e70124.
[68] Vorspan F, Guillem E, Bloch V, Bellais L, Sicot R, Noble F, et al. Self-reported sleep disturbances during cannabis withdrawal in cannabis-dependent outpatients with and without opioid-dependence. Sleep Med 2010 May;11(5):499–500.
[69] Allsop DJ, Norberg MM, Copeland J, Fu S, Budney AJ. The Cannabis Withdrawal Scale development: patterns and predictors of cannabis withdrawal and distress. Drug Alcohol Depend 2011 Dec 1;119(1):123–9.
[70] Kuhathasan N, Dufort A, MacKillop J, Gottschalk R, Minuzzi L, Frey BN. The use of cannabinoids for sleep: a critical review on clinical trials. Exp Clin Psychopharmacol 2019 Aug;27(4):383–401.
[71] Amaral C, Carvalho C, Scaranelo A, Chapman K, Chatkin J, Ferreira I. Cannabis and sleep disorders: not ready for prime time? A qualitative scoping review. J Clin Sleep Med 2023;19(5):975–90.
[72] Winiger EA, Hitchcock LN, Bryan AD, Cinnamon Bidwell L. Cannabis use and sleep: expectations, outcomes, and the role of age. Addict Behav 2021 Jan 1;112:106642.
[73] Cebrian J, Gonzalez-Cuevas G. Cannabis linked to improved sleep quality: a preliminary study. Eur Psychiatry 2021 Apr;64(S1):S564–5.
[74] Hser YI, Mooney LJ, Huang D, Zhu Y, Tomko RL, McClure E, et al. Reductions in cannabis use are associated with improvements in anxiety, depression, and sleep quality, but not quality of life. J Subst Abuse Treat 2017 Oct 1;81:53–8.
[75] Coelho J, Montagni I, Micoulaud-Franchi JA, Plancoulaine S, Tzourio C. Study of the association between cannabis use and sleep disturbances in a large sample of University students. Psychiatry Res 2023 Apr 1;322:115096.
[76] Altman BR, Mian MN, Slavin M, Earleywine M. Cannabis expectancies for sleep. J Psychoact Drugs 2019 Oct 20;51(5):405–12.
[77] Celmer L. Marijuana use leads to lost sleep for half of Americans [Internet]. Ame Acad of Sleep Med – Association for Sleep Clinicians Res 2023 [cited 2025 Jul 29]. Available from: https://aasm.org/marijuana-use-leads-to-lost-sleep-for-half-of-americans/.
[78] Schauer GL, Njai R, Grant-Lenzy AM. Modes of marijuana use – smoking, vaping, eating, and dabbing: results from the 2016 BRFSS in 12 States. Drug Alcohol Depend 2020 Apr 1;209:107900.
[79] Thorpy MJ, Dauvilliers Y. Clinical and practical considerations in the pharmacologic management of narcolepsy. Sleep Med 2015 Jan 1;16(1):9–18.
[80] Ghorayeb I. More evidence of cannabis efficacy in restless legs syndrome. Sleep Breath 2020 Mar 1;24(1):277–9.
[81] Ghorayeb I. Cannabis for restless legs syndrome. In: Monti JM, Pandi-Perumal SR, Murillo-Rodríguez E, editors. Cannabinoids and sleep: molecular, functional and clinical aspects [internet]. Cham: Springer International Publishing; 2021. p. 173–81. https://doi.org/10.1007/978-3-030-61663-2_13 [cited 2024 Mar 7]. (Advances in Experimental Medicine and Biology).
[82] Ramar K, Rosen IM, Kirsch DB, Chervin RD, Carden KA, Aurora RN, et al. Medical cannabis and the treatment of obstructive sleep apnea: an American Academy of sleep medicine position statement. J Clin Sleep Med. 14(4):679–681.
[83] Kolla BP, Mansukhani MP, Olson EJ, Louis EKS, Silber MH, Morgenthaler TI. Medical cannabis for obstructive sleep apnea: premature and potentially harmful. Mayo Clin Proc 2018 Jun 1;93(6):689–92.
[84] Gates PJ, Albertella L, Copeland J. The effects of cannabinoid administration on sleep: a systematic review of human studies. Sleep Med Rev 2014 Dec 1;18(6):477–87.
[85] Graupensperger S, Hultgren Brittney A, Fairlie Anne M, Lee Christine M, Larimer ME. Using alcohol and cannabis as sleep aids: associations with descriptive norms among college students. Behav Sleep Med 2023 Jan 2;21(1):84–96.
[86] Rafei P, Englund A, Lorenzetti V, Elkholy H, Potenza MN, Baldacchino AM. Transcultural aspects of cannabis use: a descriptive overview of cannabis use across cultures. Curr Addict Rep 2023 Sep 1;10(3):458–71.
[87] Wanke M, Sandberg S, Macit R, Gülerce H. Culture matters! Changes in the global landscape of cannabis. Drugs Educ Prev Pol 2022 Jul 4;29(4):317–22.