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Cannabis use and associated sleep-disordered breathing among US adults

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ABSTRACT

Study objectives: As sleep-disordered breathing has become more prevalent, so has cannabis use due to legalization across many regions of the United States of America (USA). This study sought to determine if there is an association between cannabis use and signs of sleep-disordered breathing.
Methods: This was a cross-sectional study of a sample of 14,485 US adults from the 2017 to 2018 Behavioral Risk Factor Surveillance System (BRFSS). Data from state-administered telephone surveys were used. We utilized survey-weighted multivariable logistic regression to investigate the association between self-reported frequency of cannabis use with witnessed apnea during sleep and reported loud snoring.
Results: Cannabis use and signs of sleep-disordered breathing were present in 5.5% and 44.7%, respectively, of the cohort. Compared to no cannabis use, non-regular use (1-15 days of use in last 30) was associated with witnessed apnea during sleep (OR 1.41; 95%CI 1.05, 1.86), loud snoring (1.26; 1.01, 1.57), and either sign (1.33; 1.07, 1.65). Regular use of cannabis (16-30 days of use) was associated with witnessed apnea during sleep (1.73; 1.27, 2.33), loud snoring (1.68; 1.32, 2.14), and either sign (1.82; 1.43, 2.33). Additionally, each additional day per month of cannabis use was associated with increased odds of reported witnessed apnea during sleep, reported loud snoring, and either sleep-disordered breathing sign.
Conclusions: Cannabis use was associated with a greater odds of signs of sleep-disordered breathing. More research is required to examine the causality of this relationship and to determine if the potential effect is mediated by the method of cannabis use.

Introduction

Sleep-disordered breathing (SDB) constitutes a range of pathologies, with the obstructive type characterized by the presence of increased respiratory effort due to increased upper airway narrowing.[1] SDB is highly prevalent in the general population, with a population-scale sleep study finding mild of greater SDB in 83.8% of males and 60.8% of females, though estimates vary based on definition of SDB or OSA.[2-7] While continuous positive airway pressure (CPAP), surgical options, and mandibular repositioning devices can treat OSA, each of these approaches has important limitations.[8-11]

Meanwhile, recent increases in the use and non-medical use of cannabis—the most frequently used substance in the US and worldwide after alcohol and tobacco—have been linked to both its sleep-promoting effects and its popularity as an alternative to medical treatment.[12-16] Insomnia and sleeping disorders consistently represent the most cited reasons for cannabis self-medication.[14,15,17] While survey studies indicate that patients with sleep disorders consistently report positive effects from cannabis,[18-20] double-blind, placebo-controlled studies yield mixed results that vary according to study population, primary disorder, cannabinoid studied and route of administration utilized.[21-24] Of note, while THC,

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Impact and relevance

What was known

Prior studies regarding cannabis use and its influence on sleep have suggested that cannabis may influence sleep physiology and autonomic function in a variety of ways. While cannabis has been explored as a potential treatment for sleep disorders, evidence regarding its relationship with sleep-disordered breathing (SDB) remains limited and conflicting. Importantly, no prior population-based studies had examined the association between cannabis use and common signs of SDB in a nationally representative sample.

What this study adds

This study demonstrates an association between cannabis use and sleep-disordered breathing—including witnessed apneas and loud snoring—through use of a large, nationally representative sample of adults. This study identified an exposure-dependent relationship, whereby increasing frequency of cannabis use was associated with incrementally greater odds of SDB signs. These findings raise concern about cannabis use in individuals with or at risk for SDB and support current recommendations against its use as a treatment for obstructive sleep apnea.

the primary psychoactive and intoxicating component of the cannabis plant, may improve some sleep outcomes (sleep onset latency and hours slept),[25,26] it has a paradoxical effect on sleep quality including reducing REM.[27] For patients that regularly use cannabis with THC, interruption in use (i.e., skipping a day or two) can elicit withdrawal symptoms that are marked by sleep disruptions and perhaps worsening of SDB.[28,29]

Preclinical studies suggest THC's therapeutic potential in treating OSA.[30,31] However, clinical trials have demonstrated some positive but overall heterogeneous results.[25,26] Furthermore, these studies assessed oral THC administration while inhalation remains the most common route of administration in medical and non-medical cannabis users.[32] In light of this heterogeneity, the American Academy of Sleep Medicine formally recommends against the use of cannabis to treat OSA.[33] Given that many utilize cannabis for insomnia and other sleep-related disorders, it is crucial to understand how cannabis use may relate to OSA and sleep disordered breathing generally. No study to date has used a population-based approach to investigate the prevalence of cannabis use among people with OSA or associations between OSA signs and cannabis use, despite potential mechanisms linking cannabis to OSA and SDB, including airway inflammation, increased upper airway muscle relaxation leading to greater airway collapsibility.[34,35]

Thus, the objective of this study was to use the Behavioral Risk Factor Surveillance System (BRFSS) database to assess the association between SDB signs and cannabis use among a national sample of adults. Unlike other nationally representative surveys, the BRFSS provides rich data related to frequency of current cannabis (marijuana) use, primary mode of administration, and SDB signs, information that is critical in understanding cannabis use prevalence in people with SDB and associations between SDB signs and frequency of cannabis use. We hypothesize that cannabis use would be associated with increased signs of SDB in a frequency of use-dependent manner.

Methods

This study was deemed exempt from institutional review board approval, as it is a secondary analysis of existing data, does not involve intervention or interaction with human subjects, and is fully deidentified in accordance with the deidentification standard defined in Section *§164.514(a)* of the Health Insurance Portability and Accountability Act (HIPAA) Privacy Rule. STROBE reporting guidelines for an observational cross-sectional study were followed and are available as Supplementary material.

Study population

This was a nationally representative, population-based, retrospective cross-sectional study comprised of adults, aged ≥18 years, who completed the 2017 to 2018 Behavioral Risk Factor Surveillance System (BRFSS) survey, including modules on cannabis/marijuana use and sleep disorders.[36] The BRFSS, conducted by the Centers for Disease Control and Prevention (CDC), is a health-related telephone survey that collects state data (from all 50 states) about US residents regarding their health-related behaviors, chronic health conditions, and use of preventative services. Those who chose not to answer or did not know their cannabis use in the last 30 days (n = 139; 0.6%) were excluded from the initial sample. Both modules were offered nationally to be selected by states for inclusion in their administration of the BRFSS survey. Two states (Minnesota and Tennessee) chose to include both optional modules in their surveys during the time period analyzed, so respondents from these states who completed the BRFSS were included. Due to statistical considerations (described in statistical analysis), a stratified random sample of 60% of all eligible individuals was selected to be utilized in the study sample (n = 14,485).

Study measures

Current cannabis use was measured by self-report via the question "During the past 30 days, on how many days did you use marijuana or cannabis?" The BRFSS did not ask about use of cannabis derivatives, such as tetrahydrocannabinol (THC) or cannabidiol (CBD). Cannabis use was incorporated into regression models as both a continuous and categorical variable. Individuals were assigned to the no current use (0 days of use in the previous 30 days), non-regular use (1–15 days of use in the previous 30 days,) or regular use (16–30 days of use in the last 30 days) subgroups (categorical), as in previous research.[37]

Presence of SDB was conceptualized in 3 ways: (1) answering yes to the defined "apnea" question: "Has anyone ever observed that you stop breathing during your sleep?" (2) answering yes to the defined "loud snoring" question: "Have you ever been told that you snore loudly?" or (3) answering yes to either or both questions.

Covariates included in multivariable regression models were selected *a priori* based on prior literature and biologic plausibility demonstrating associations with cannabis use, sleep-disordered breathing, or both. These included demographic characteristics, socioeconomic indicators, cardiometabolic risk factors, and substance use behaviors that are established or potential confounders of the cannabis–SDB relationship.

Other variables utilized in multivariable regression include sex, age, race/ethnicity, insurance status, education, income, body mass index (BMI), cigarette use, electronic cigarette use, alcohol use, and diabetes. Sex was reported as male or female. Age was grouped in 6 categories (18–24, 24–34, 35–44, 45–64, and 65 and older), reflecting meaningful life stages as selected by BRFSS. BRFSS's 8-category combined race/ethnicity variable was utilized. Insurance status was

binary (yes/no). Education and income were analyzed categorically as asked in the BRFSS. BMI was utilized as a continuous variable. Cigarette and electronic cigarette use was categorically defined as use "Not at all (Never smoker)," "Not at all (Former smoker)," "Some days (current smoker)," and "Every day (current smoker)." Alcohol use was analyzed as a continuous variable as number of days per 30 days of drinking alcohol; if asked in days per week, data was multiplied to the implied number of days per 30. Diabetes status was binary (yes/no). Although categorical modeling of several covariates increased the number of parameters, this approach was chosen to preserve clinical interpretability and alignment with established BRFSS coding conventions. Given the large analytic sample size, model convergence and stability were maintained, as described in statistical analysis. Respondents who either did not respond, were unsure, or declined to respond to the cannabis use question or did not respond to either aforementioned sleep-associated question were not included in the analyses. Individuals were also asked in multiple-choice fashion, "What was the reason you used marijuana?" and "During the past 30 days, how did you primarily use marijuana?" Since data suggests that users of cannabis often use cannabis via multiple modalities, individuals with all primary methods of use were equally included in analysis.[38,39]

Statistical analysis

Baseline characteristics of all individuals with data on cannabis use and SDB, this sample and the entire 2017 to 2018 BRFSS sample were described without weighting to provide a description of the study sample (rather than provide population prevalence estimates). Subsequently, this sample with cannabis and SDB data and the entire 2017 to 2018 BRFSS sample were compared with Pearson's $\chi 2$ tests for categorical variables and independent-sample t-tests for continuous variables.

The association between cannabis use and SDB signs was analyzed with logistic regression, with BRFSS survey weighting applied.[40] Since the data available for analysis was a very small subset of the overall sample (2 of 50 states), survey weighting was initially rendered inappropriate.[41] Of the 887,452 individuals who completed the BRFSS survey from 2017 to 2018, 23,719 participants completed both the cannabis use and sleep disorder modules and were initially eligible for analysis. Among these, 18,696 participants had complete data for all covariates required for stratified sampling. Given the small subsample available for analysis, we utilized a stratified random sample of 60% of the available data, with stratification based on age group, sex, race, education, and income, to ensure BRFSS survey weighting would be appropriately representative of the US population, as previously suggested and performed.[42,43] After performing stratified random sampling to match the BRFSS population demographics, the final analytic cohort included 14,485 participants. Overall, approximately 98% of the original survey population were excluded due to module non-completion or missing data.

Multivariable models were sequentially adjusted for demographic characteristics (specified above) and other exposures known to be associated with SDB, including body mass index (BMI), cigarette use, electronic cigarette use, alcohol use, and diabetes. Sequential adjustment was used to assess for potential confounding; minimal change in effect estimates across sequential adjustment steps confirmed absence of gross confounding. Furthermore, a corrective factor created by the BRFSS for the interaction between cigarette use and electronic cigarette use was included in multivariable analysis. Since survey weighting with a small subsample is controversial due to modeling instability created by small samples, a supplementary logistic regression model using all available data from individuals with responses to both cannabis use and sleep disorder modules but without survey weighting was also created. While survey-weighted models incorporate sampling weights to support inference to the US non-institutionalized adult population, unweighted models assess the robustness of these associations within the analytic sample itself. Statistical significance was set at $P < .05$. All analyses were conducted using STATA Version 18.0 Standard Edition (StataCorp LLC).

Results

Data from 14,485 individuals were utilized in this study. Supplementary Table 1 presents the sample characteristics compared to other 2017 to 2018 BRFSS participants. These samples had statistically significant (but clinically small) differences in age, sex, race/ethnicity, education, annual income, diabetes status, insurance status, and cigarette and electronic cigarette smoking status. The BRFSS subset utilized for further analysis was more likely to be aged 65 years or older (n = 4245; 29.8%), female (n = 7760; 54.4%), non-Hispanic white (n = 10,573; 74.1%), college-educated (n = 5171; 36.3%), and insured (n = 12,875; 90.3%) with mean BMI of 28.4 (SD: 6.3).

For the subgroup included in this study, Table 1 describes characteristics by cannabis use frequency, including among 13,461 non-users, 438 non-regular users, and 364 regular users of cannabis. The most common route of consumption of cannabis was smoking in the non-regular use group (n = 381; 87.0%), followed by vaporizing (n = 22; 5.0%), and eating (n = 20; 4.6%). Smoking was also the most common route of consumption in the regular use group (n = 319; 87.6%), followed by vaporizing (n = 16; 4.4%), and eating (7; 1.9%). Most common reason for cannabis use was non-medical/recreational in the non-regular use group (n = 270; 61.6%) and medical only in the regular use group (n = 137; 37.6%). Other variables used in

Table 1
Unweighted sample characteristics of sample described by cannabis use group characteristics

Characteristic (n, %)	Cannabis use group		
	No current use (n = 13,461)	Non-regular use (n = 438)	Regular use (n = 364)
Cannabis use (continuous, days/month; mean, standard deviation)	0 (0)	4.7 (4.2)	27.7 (4.0)
Primary method of cannabis use			
Smoking	-	381 (87.0)	319 (87.6)
Eating	-	20 (4.6)	7 (1.9)
Drink	-	0 (0.0)	6 (1.7)
Vaporize	-	22 (5.0)	16 (4.4)
Dabbing	-	6 (1.4)	5 (1.4)
Some other way	-	4 (0.9)	7 (1.9)
Refused or Unknown	-	5 (1.1)	4 (1.1)
Reason for cannabis use			
Medical only	-	113 (25.8)	137 (37.6)
Non-medical/Recreational	-	270 (61.6)	136 (37.4)
Both medical and non-medical	-	45 (10.3)	90 (24.7)
Refused or Unknown	-	10 (2.3)	1 (0.3)
Witnessed apnea during sleep			
Yes	2057 (15.3)	75 (17.1)	72 (19.8)
No	11,299 (83.9)	359 (82.0)	292 (80.2)
Refused or Unknown	105 (0.8)	4 (0.9)	0 (0.0)
Witnessed snoring loudly during sleep			
Yes	5553 (41.3)	195 (44.5)	182 (50.0)
No	7778 (57.8)	239 (54.6)	181 (49.7)
Refused or Unknown	130 (1.0)	4 (0.9)	1 (0.3)
Either witnessed apnea or loud snoring during sleep			
Yes	5963 (44.3)	212 (48.4)	201 (55.2)
No	7484 (55.6)	226 (51.6)	163 (44.8)
Refused or Unknown	14 (0.1)	0 (0.0)	0 (0.0)

Table 2
Unweighted sample characteristics of sample described by cannabis use group characteristics

Characteristic (*n*, %)	Cannabis use group		
	No current use (*n* = 13,461)	Non-regular use (*n* = 438)	Regular use (*n* = 364)
Age group (years)			
18-24	718 (5.33)	76 (17.4)	73 (20.1)
25-34	1542 (11.5)	110 (25.1)	100 (27.5)
35-44	1773 (13.2)	85 (19.4)	61 (16.8)
45-54	2244 (16.7)	64 (14.6)	54 (14.8)
55-64	2993 (22.2)	76 (17.4)	49 (13.5)
65 or older	4191 (31.1)	27 (6.2)	27 (7.4)
Sex			
Male	5971 (44.4)	297 (67.8)	231 (63.5)
Female	7486 (55.6)	141 (31.3)	133 (36.5)
Refused or Unknown	4 (< 0.1)	0 (0.0)	0 (0.0)
Race			
White only, non-Hispanic	10,080 (74.9)	274 (62.6)	219 (60.2)
Black only, non-Hispanic	1109 (8.2)	39 (8.9)	60 (16.5)
American Indian or Alaskan Native, non-Hispanic	279 (2.1)	13 (3.0)	17 (4.7)
Asian only, non-Hispanic	257 (1.9)	9 (2.1)	8 (2.2)
Native Hawaiian or other Pacific Islander only, non-Hispanic	25 (0.2)	8 (1.8)	2 (0.6)
Other race, non-Hispanic	74 (0.6)	2 (0.5)	3 (0.8)
Multiracial, non-Hispanic	281 (2.1)	23 (5.3)	27 (7.4)
Hispanic	1132 (8.4)	66 (15.1)	24 (6.6)
Refused or Unknown	224 (1.7)	4 (0.9)	4 (1.1)
Education			
Never attended school or only kindergarten	35 (0.3)	0 (0.0)	0 (0.0)
Grades 1 through 8	354 (2.6)	6 (1.4)	6 (1.7)
Grades 9 through 11	663 (4.9)	27 (6.2)	27 (7.4)
Grade 12 or GED	3551 (26.4)	135 (30.8)	128 (35.2)
College 1 year to 3 years	3864 (28.7)	131 (29.9)	121 (33.2)
College 4 years or more	4954 (36.8)	136 (31.1)	81 (22.3)
Refused or Unknown	40 (0.3)	3 (0.7)	1 (0.3)
Annual income			
< $10,000	528 (3.9)	26 (5.9)	24 (6.6)
$10,000 to $14,999	571 (4.2)	34 (7.8)	24 (6.6)
$15,000 to $19,999	840 (6.3)	26 (5.9)	46 (12.6)
$20,000 to $24,999	1073 (8.0)	38 (8.7)	44 (12.1)
$25,000 to $34,999	1279 (9.5)	27 (6.2)	39 (10.7)
$35,000 to $49,999	1627 (12.1)	49 (11.2)	56 (15.4)
$50,000 to $74,999	1780 (13.2)	74 (16.9)	39 (10.7)
≥$75,000	3707 (27.5)	113 (25.8)	52 (14.3)
Refused or Unknown	2056 (15.3)	51 (11.6)	40 (11.0)
Insurance status			
Yes	12,221 (90.8)	365 (83.3)	289 (79.4)
No	1172 (8.7)	71 (16.2)	71 (19.5)
Refused or Unknown	68 (0.5)	2 (0.5)	5 (1.1)
Diabetes status			
Yes	1865 (13.9)	36 (8.2)	20 (5.5)
No	11,583 (86.1)	401 (91.6)	343 (94.2)
Refused or Unknown	13 (0.1)	1 (0.2)	1 (0.3)
BMI (mean, standard deviation)	28.5 (6.3)	27.3 (5.8)	27.1 (6.0)
Alcohol use (days/month; mean, standard deviation)	4.5 (7.8)	8.5 (9.3)	8.5 (10.0)
Cigarette smoking status			
Every day	1426 (10.6)	110 (25.1)	153 (42.0)
Some days	617 (4.6)	57 (13.0)	44 (12.1)
Not at all (Former Smoker)	3671 (27.3)	107 (24.4)	90 (24.7)
Not at all (Never Smoker)	7655 (56.9)	164 (37.4)	73 (20.1)
Refused or Unknown	92 (0.7)	0 (0.0)	4 (1.1)
Electronic cigarette smoking status			
Every day	241 (1.8)	41 (9.4)	53 (14.6)
Some days	140 (1.0)	17 (3.9)	16 (4.4)
Not at all (Former Smoker)	1716 (12.8)	184 (42.0)	163 (44.8)
Not at all (Never Smoker)	11,342 (84.3)	196 (44.8)	132 (36.3)
Refused or Unknown	22 (0.2)	0 (0.0)	0 (0.0)

multivariable regression are summarized by cannabis use group in Table 2.

Table 3 describes the results of a multivariable logistic regression testing the association between cannabis use category and SDB signs, with the same adjustment variables. Compared to non-users, non-regular cannabis users had greater odds of reported witnessed apnea (OR 1.41; 95%CI 1.05, 1.86), reported loud snoring (1.26; 1.01, 1.57), and either SDB sign (1.33; 1.07, 1.65). The magnitude of the association between regular cannabis use and reported witnessed apnea (1.73; 1.27, 2.33), reported loud snoring (1.68; 1.32, 2.14), and either SDB sign (1.82; 1.43, 2.33) was greater for regular users compared to non-regular users. When analyzing results without survey weighting, non-regular and regular cannabis use were associated with witnessed apnea during sleep. Regular cannabis use was also associated with reported loud snoring and either SDB sign, but non-regular use was not significantly associated with these signs.

Supplementary Table 2 describes the results of multivariable logistic regression of cannabis use as a continuous variable with SDB signs while controlling for age, sex, race/ethnicity, education, income, insurance status, presence/absence of diabetes, BMI, cigarette smoking status, and electronic cigarette smoking status. With survey-weighting, cannabis use was positively associated with reported witnessed apnea (OR 1.02; 95%CI 1.01, 1.03), reported loud snoring (1.02; 1.01, 1.03), and either SDB sign (1.02; 1.01, 1.03). Without survey weighting, each additional day of cannabis use was associated with increased odds of witnessed apneas during sleep and either reported loud snoring or apnea during sleep, but not loud snoring alone.

Discussion

To our knowledge, this is the first population-based study examining the association between cannabis use and SDB. In this nationally representative sample, regular and non-regular cannabis use were independently associated with a greater odds of SDB signs, as compared to no current cannabis use. The magnitude of the association with reported witnessed apneas, loud snoring, and loud snoring and/or witnessed apneas was greater for those with regular use compared to those with non-regular cannabis use, who also had greater odds than non-users.

Smoking cannabis was the predominant method of consumption reported in this sample, and this method of consumption specifically may contribute to SDB. While cannabis use may be associated with tobacco smoking and electronic cigarette use, our study controlled for these factors, showing an independent association between cannabis use and SDB.

Previous work has found strong associations between tobacco smoking and the presence and severity of OSA due to the inflammatory effect of inhalational smoke exposure on the airway.[44–47] Cannabis inhalation may produce a similar effect. Cannabis smoking is often unfiltered and consumed through deeper breaths compared to tobacco smoking.[48] Furthermore, cannabis burns at a higher temperature relative to tobacco, and as such its inhalation can result in increased thermal injury to the airway.[34] Through these means, it is possible that cannabis use may contribute to upper airway obstruction that is often implicated in OSA and SDB.

There was an exposure-dependent relationship between cannabis use and SDB signs. Each additional day of cannabis use per month was associated with nearly 2% greater odds of witnessed apneas, loud snoring, or both signs. This estimate should not be interpreted as a strict per-day effect of cannabis use on sleep-disordered breathing, but rather as an overall summary of the association across increasing frequency of use. This phenomenon may be due to the biphasic impact cannabis consumption (regardless of method of consumption) has on the autonomic nervous system; although lower doses of cannabis result in sympathetic activation,

Table 3
Multivariable logistic regression of cannabis use (as a categorical variable) with sleep disordered breathing[a]

Weighting	Dependent variable[b]	Adjusted odds ratio	95% CI	*P*-value
Weighted	Witnessed apnea during sleep			
	Non-regular cannabis use	1.41	**1.05-1.86**	**.02**
	Regular cannabis use	1.73	**1.27-2.33**	**<.001**
	Reported loud snoring			
	Non-regular cannabis use	1.26	**1.01-1.57**	**.04**
	Regular cannabis use	1.68	**1.32-2.14**	**<.001**
	Reported loud snoring and/or apnea			
	Non-regular cannabis use	1.33	**1.07-1.65**	**.01**
	Regular cannabis use	1.82	**1.43-2.33**	**<.001**
Unweighted	Witnessed apnea during sleep			
	Non-regular cannabis use	1.27	**1.00-1.62**	**.05**
	Regular cannabis use	1.58	**1.22-2.06**	**.001**
	Reported loud snoring			
	Non-regular cannabis use	1.06	0.88-1.27	.53
	Regular cannabis use	1.24	**1.01-1.53**	**.043**
	Reported loud snoring and/or apnea			
	Non-regular cannabis use	1.14	0.95-1.37	.17
	Regular cannabis use	1.36	**1.10-1.68**	**.004**

Bold indicates statistical significance at alpha=0.05.

[b] Individuals were categorized as non-regular cannabis users if they reported 1 to 15 days of cannabis use in the last 30 days. Regular cannabis use was defined as use 16 to 30 days in the last 30 days.

[a] No cannabis use utilized as reference group. Variables utilized in multivariable analysis included: age, sex, race/ethnicity, education, income, insurance status, presence/absence of diabetes, BMI, alcohol use, cigarette smoking status, electronic cigarette smoking status, and a corrective factor for the interaction between cigarette/e-cigarette smoking status.

high-dose consumption subsequently leads to parasympathetic activation.[49] As such, increased consumption may further exacerbate the natural relaxation of upper airway dilator muscles that occurs during sleep.[35] As tolerance to cannabis can develop within days, frequent users of cannabis may require higher doses to experience the same effects.[49,50] In doing so, the resultant parasympathetic activation and relaxation of upper airway muscles may ultimately increase the risk of airway collapse and SDB.

Finally, the use of cannabis for medical reasons was the primary motivation among regular users in our sample and was also commonly cited among non-regular users. Though we do not have data regarding reason for use among those using for medicinal reasons, this phenomenon may, in part, stem from the frequent use of consumer cannabis as a sleep aid and the increasing prevalence of favorable medical cannabis legislation nationwide.[15,51]

Our findings, considered alongside the existing literature, underscore the need for caution in using cannabis as a treatment for obstructive sleep apnea or sleep-disordered breathing. While the estimated effect associated with each additional day of cannabis use was statistically significant but clinically small (approximately 2% increased odds), the cumulative effect observed among individuals with regular or heavy use compared with non-use appears more clinically meaningful (up to 82% increased odds for either SDB sign). The American Academy of Sleep Medicine currently recommends against cannabis for the treatment of SDB and OSA, and our results align with that position as cannabis use is frequently associated with SDB. However, given the cross-sectional nature of our study, we cannot determine the direction of this association—it is possible that individuals with SDB are more likely to use cannabis in an effort to manage their sleep difficulties. In light of these findings, physicians should counsel patients on the potential risks of cannabis use, particularly in those with or at risk for OSA. Further research is needed to clarify the relationship between cannabis, sleep, and sleep disorders.

Our study is limited by a variety of factors. Due to our use of a retrospective cross-sectional survey, we were able to assess the association between cannabis use and SDB signs but could not establish causality between the 2 variables, though the dose-response relationship uncovered does provide some support toward a case for causality. Additionally, the assessment of dose-response was limited, as duration and quantity of cannabis use were only measured through days utilized in the past 30 days rather than dosage of cannabinoid exposure. The data in BRFSS only asks regarding last 30 days of exposure, so inferences regarding chronic use cannot be made from this data. Because BRFSS is a telephone-based survey, selection bias is possible, as individuals without reliable phone access or those less likely to participate in surveys may be underrepresented. Because BRFSS does not comprehensively capture many conditions that may motivate medical cannabis use, residual confounding and indication bias related to reason for use remain possible. Individuals using cannabis for medical purposes may differ from non-users with respect to underlying health conditions, concurrent medications, and health care utilization patterns that could influence sleep-disordered breathing symptoms. Future studies with more detailed clinical information are needed to better address these potential sources of bias. Furthermore, SDB was established in our study through patient self-report of witnessed apneas or snoring, which may be less accurate than classification through clinical diagnosis through physician exam or objective measurement with sleep testing. Whereas our multivariable model controlled for multiple demographic variables and risk factors associated with SDB, residual confounding is possible, as we were unable to account for unmeasured factors such as intensity of alcohol use, sedative or hypnotic medication use, and psychiatric comorbidities, which may influence both cannabis use and sleep-related outcomes. Our patient sample had similar but not identical characteristics to the US adult population, though the application of survey weighting helps to increase applicability to a representative US population.

Future directions may include objective measurement of SDB with sleep testing, investigating the impact of consumption method (inhalation, oral, sublingual, and topical), and examining the individual components within cannabis.

Conclusions

This cross-sectional study suggests an independent association between cannabis use and SDB, including reported witnessed apnea during sleep and reported loud snoring, after controlling for demographics and medical risk factors. While per-day increases in cannabis use were associated with statistically significant but clinically small effects, the cumulative association observed with regular or heavy use appears more clinically meaningful. Although certain components of cannabis may provide therapeutic benefits for patients with SDB, the

use of cannabis, particularly when inhaled regularly, may be detrimental to sleep-related health and outcomes. Further work is needed to understand the mechanisms of the observed association, including the impacts of dosing and route of consumption.

Author contributions

Tyler J. Gallagher: Conception and design of work, data acquisition and analysis, interpretation of data, drafting of manuscript, critical revision. **Matthew E. Lin**: Conception and design of work, data acquisition and analysis, interpretation of data, drafting of manuscript, critical revision. **Ian Kim**: Conception and design of work, data analysis, interpretation of data, critical revision. **Ziva D. Cooper**: Conception and design of work, interpretation of data, critical revision. **Sebastian M. Jara**: Conception and design of work, interpretation of data, critical revision. **Eric J. Kezirian**: Conception and design of work, interpretation of data, critical revision. All authors contributed to and approved of this work.

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Declaration of generative AI and AI-assisted technologies in the writing process

No AI or AI-assisted technologies were utilized in the creation of this manuscript.

Data sharing

All data are publicly available through the Centers for Disease Control Behavioral Risk Factor Surveillance System Website.

Declaration of conflicts of interest

All authors declare no conflicts of interest related to this manuscript.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.sleh.2026.07.003.

References

1. Panossian L, Daley J. Sleep-disordered breathing. *Contin Lifelong Learn Neurol.* 2013;19(1):86–103. https://doi.org/10.1212/01.CON.0000427211.98305.c6
2. Harris CC, Hollstein M. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med.* 1993;329(18):1318–1327.
3. Ancoli-Israel S, Kripke DF, Klauber MR, Mason WJ, Fell RKO. Sleep-disordered breathing in community-dwelling elderly. *Sleep.* 1991;14(6):486–495. https://doi.org/10.1093/sleep/14.6.486
4. Senaratna CV, Perret JL, Lodge CJ, et al. Prevalence of obstructive sleep apnea in the general population: a systematic review. *Sleep Med Rev.* 2017;34:70–81. https://doi.org/10.1016/j.smrv.2016.07.002
5. Heinzer R, Vat S, Marques-Vidal P, et al. Prevalence of sleep-disordered breathing in the general population: the HypnoLaus study. *Lancet Respir Med.* 2015;3(4):310–318. https://doi.org/10.1016/S2213-2600(15)00043-0
6. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Original contribution increased prevalence of sleep-disordered breathing in adults. *Am J Epidemiol.* 2013;177(9):1006–1014. https://doi.org/10.1093/aje/kws342
7. Benjafield AV, Ayas NT, Eastwood PR, et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. *Lancet Respir Med.* 2026;7:9–12. https://doi.org/10.1016/S2213-2600(19)30198-5 (August 2019).
8. McEvoy RD, Antic NA, Heeley E, et al. CPAP for prevention of cardiovascular events in obstructive sleep apnea. *N Engl J Med.* 2016;375(10):919–931. https://doi.org/10.1056/nejmoa1606599
9. Strollo PJ, Soose RJ, Maurer JT, et al. Upper-airway stimulation for obstructive sleep apnea. *N Engl J Med.* 2014;370(2):139–149. https://doi.org/10.1056/nejmoa1308659
10. Martínez-García MA, Campos-Rodríguez F, Catalán-Serra P, et al. Cardiovascular mortality in obstructive sleep apnea in the elderly: role of long-term continuous positive airway pressure treatment: a prospective observational study. *Am J Respir Crit Care Med.* 2012;186(9):909–916. https://doi.org/10.1164/rccm.201203-0448OC
11. Santilli M, Manciocchi E, D'addazio G, et al. Prevalence of obstructive sleep apnea syndrome: a single-center retrospective study. *Int J Environ Res Public Health.* 2021;18(19):10277. https://doi.org/10.3390/ijerph181910277
12. Yu B, Chen X, Chen X, Yan H. Marijuana legalization and historical trends in marijuana use among US residents aged 12-25: results from the 1979-2016 National Survey on drug use and health. *BMC Public Health.* 2020;20(1):1–10. https://doi.org/10.1186/s12889-020-8253-4
13. WDR-2010. United Nations: Office on Drugs and Crime.
14. Hazekamp A, Ware MA, Muller-Vahl KR, Abrams D, Grotenhermen F. The medicinal use of cannabis and cannabinoids-an international cross-sectional survey on administration forms. *J Psychoact Drugs.* 2013;45(3):199–210. https://doi.org/10.1080/02791072.2013.805976
15. Bachhuber M, Arnsten JH, Wurm G. Use of cannabis to relieve pain and promote sleep by customers at an adult use dispensary. *J Psychoact Drugs.* 2019;51(5):400–404. https://doi.org/10.1080/02791072.2019.1626953
16. Hasin DS, Shmulewitz D, Sarvet AL. Time trends in US cannabis use and cannabis use disorders overall and by sociodemographic subgroups: a narrative review and new findings. *Am J Drug Alcohol Abuse.* 2019;45(6):623–643. https://doi.org/10.1080/00952990.2019.1569668
17. Feingold D, Weinstein A. Cannabis and depression. *Adv Exp Med Biol.* 2021;1264:67–80. https://doi.org/10.1007/978-3-030-57369-0_5
18. Suraev A, Mills L, Abelev SV, Arkell TR, McGregor IS, Lintzeris N. Medical cannabis use patterns for sleep disorders in Australia: results of the cross-sectional CAMS-20 Survey. *Nat Sci Sleep.* 2023;15(April):245–255. https://doi.org/10.2147/NSS.S390583
19. Cahill SP, Lunn SE, Diaz P, Page JE. Evaluation of patient reported safety and efficacy of cannabis from a survey of medical cannabis patients in Canada. *Front Public Health.* 2021;9(May):1–15. https://doi.org/10.3389/fpubh.2021.626853
20. Lee RM, Donnan J, Harris N, Garland SN. A cross-sectional survey of the prevalence and patterns of using cannabis as a sleep aid in Canadian cancer survivors. *J Cancer Surviv.* 2023;19(1):386–396. https://doi.org/10.1007/s11764-023-01474-2
21. National Academies of Sciences, Engineering and M. *The Health Effects of Cannabis and Cannabinoids: The Current State of Evidence and Recommendations for Research.* National Academies Press; 2017https://doi.org/10.17226/24625
22. Walsh JH, Maddison KJ, Rankin T, et al. Treating insomnia symptoms with medicinal cannabis: a randomized, crossover trial of the efficacy of a cannabinoid medicine compared with placebo. *Sleep.* 2021;44(11):1–8. https://doi.org/10.1093/sleep/zsab149
23. Bonn-Miller MO, Feldner MT, Bynion TM, et al. A double-blind, randomized, placebo-controlled study of the safety and effects of CBN with and without CBD on sleep quality. *Exp Clin Psychopharmacol.* 2023;32(3):277–284. https://doi.org/10.1037/pha0000682
24. Ried K, Tamanna T, Matthews S, Sali A. Medicinal cannabis improves sleep in adults with insomnia: a randomised double-blind placebo-controlled crossover study. *J Sleep Res.* 2023;32(3):1–12. https://doi.org/10.1111/jsr.13793
25. Prasad B, Radulovacki MG, Carley DW. Proof of concept trial of dronabinol in obstructive sleep apnea. *Front Psychiatry.* 2013;4(JAN):1–5. https://doi.org/10.3389/fpsyt.2013.00001
26. Carley DW, Prasad B, Reid KJ, et al. Pharmacotherapy of apnea by cannabimimetic enhancement, the PACE clinical trial: effects of dronabinol in obstructive sleep apnea. *Sleep.* 2018;41(1):1–13. https://doi.org/10.1093/sleep/zsx184
27. Veitch MR, Jairam S, Gurges P, et al. Cannabinoid use and obstructive sleep apnea: a retrospective cohort study. *Can J Neurol Sci.* 2024;51(6):828–835. https://doi.org/10.1017/cjn.2024.25

28. Haney M, Cooper ZD, Bedi G, Vosburg SK, Comer SD, Foltin RW. Nabilone decreases marijuana withdrawal and a laboratory measure of marijuana relapse. *Neuropsychopharmacology*. 2013;38(8):1557–1565. https://doi.org/10.1038/npp.2013.54
29. Herrmann ES, Cooper ZD, Bedi G, et al. Effects of zolpidem alone and in combination with nabilone on cannabis withdrawal and a laboratory model of relapse in cannabis users. *Psychopharmacology*. 2016;233(13):2469–2478. https://doi.org/10.1007/s00213-016-4298-6
30. Calik MW, Radulovacki M, Carley DW. Intranodose ganglion injections of dronabinol attenuate serotonin-induced apnea in Sprague-Dawley rat. *Respir Physiol Neurobiol*. 2014;190(1):20–24. https://doi.org/10.1016/j.resp.2013.10.001
31. Petrunich-Rutherford ML, Calik MW. Effects of cannabinoid agonists and antagonists on sleep in laboratory animals. *Adv Exp Med Biol*. 2021;1297:97–109. https://doi.org/10.1007/978-3-030-61663-2_7
32. National Academies of Sciences, Engineering and M. *Cannabis Policy Impacts Public Health and Health Equity*. The National Academies Press; 2024https://doi.org/10.17226/27766
33. Ramar K, Rosen IM, Kirsch DB, et al. Medical cannabis and the treatment of obstructive sleep apnea: an American Academy of Sleep Medicine position statement. *J Clin Sleep Med*. 2018;14(4):679–681. https://doi.org/10.5664/jcsm.7070
34. Rawal S, Tatakis D, Tipton D. Periodontal and oral manifestations of marijuana use. *J Tenn Dent Assoc*. 2012;92(2):26–31.
35. Remmers JE, DeGroot WJ, Sauerland EK, Anch AM. Pathogenesis of upper airway occlusion during sleep. *J Appl Physiol Respir Environ Exerc Physiol*. 1978;44(6):931–938. https://doi.org/10.1152/jappl.1978.44.6.931
36. Centers for Disease Control and Prevention The BRFSS Data User Guide. Published online 2013:1-10.
37. Orozco FR, Lin M, Hur K. Cannabis use and sinonasal symptoms in US adults. *JAMA Otolaryngol Head Neck Surg*. 2022;148(9):854–861. https://doi.org/10.1001/jamaoto.2022.2001
38. Kim N, Flora S, Macander CE. Multi-modal cannabis use among U.S. young adults: findings from the 2022 and 2023 BRFSS in 23 States. *Int J Environ Res Public Health*. 2025;22(4):1–15. https://doi.org/10.3390/ijerph22040495
39. Steigerwald S, Wong PO, Cohen BE, et al. Smoking, vaping, and use of edibles and other forms of marijuana among U.S. adults. *Ann Intern Med*. 2018;169(12):890–892. https://doi.org/10.7326/M18-1681
40. Weighting the BRFSS Data
41. Carle AC. Fitting multilevel models in complex survey data with design weights: recommendations. *BMC Med Res Methodol*. 2009;9(1):1–13. https://doi.org/10.1186/1471-2288-9-49
42. Lohr SL. *Sampling: Design and Analysis. First*. Duxbury Press; 1999.
43. Woodruff TJ, Zota AR, Schwartz JM. Environmental chemicals in pregnant women in the United States: NHANES 2003–2004. *Environ Health Perspect*. 2011;119(6):878–885. https://doi.org/10.1289/ehp.1002727
44. Kashyap R, Hock LM, Bowman TJ. Higher prevalence of smoking in patients diagnosed as having obstructive sleep apnea. *Sleep Breath*. 2001;5(4):167–172. https://doi.org/10.1007/s11325-001-0167-5
45. Wetter DW, Young TB, Bidwell TR, Badr MS, Palta M. Smoking as a risk factor for sleep-disordered breathing. *Arch Intern Med*. 1994;154(19):2219–2224.
46. Kim KS, Kim JH, Park SY, et al. Smoking induces oropharyngeal narrowing and increases the severity of obstructive sleep apnea syndrome. *J Clin Sleep Med*. 2012;8(4):367–374. https://doi.org/10.5664/jcsm.2024
47. Krishnan V, Dixon-Williams S, Thornton JD. Where there is smoke…there is sleep apnea. *Chest*. 2014;146(6):1673–1680. https://doi.org/10.1378/chest.14-0772
48. Moore BA, Augustson EM, Moser RP, Budney AJ. Respiratory effects of marijuana and tobacco use in a U.S. sample. *J Gen Intern Med*. 2005;20(1):33–37. https://doi.org/10.1111/j.1525-1497.2004.40081.x
49. Latif Z, Garg N. The impact of marijuana on the cardiovascular system: a review of the most common cardiovascular events associated with marijuana use. *J Clin Med*. 2020;9(6):1–16. https://doi.org/10.3390/jcm9061925
50. Sidney S. Cardiovascular consequences of marijuana use. *J Clin Pharmacol*. 2002;42(11 suppl):64–70. https://doi.org/10.1002/j.1552-4604.2002.tb06005.x
51. NCSL. State Medical Cannabis Laws. Published 2024. Accessed October 15, 2024. https://www.ncsl.org/health/state-medical-cannabis-laws.